Right in Comparison to Left Cerebral Hemisphere Damage by Stroke Induces Poorer Muscular Responses to Stance Perturbation Regardless of Visual Information

Daniel Boari Coelho, PhD,*'† Corina Aparecida Fernandes, Master,* Alessandra Rezende Martinelli, PhD,* and Luis Augusto Teixeira, PhD*

Objective: Fast and scaled muscular activation is required to recover body balance following an external perturbation. An issue open to investigation is the extent to which the cerebral hemisphere lesioned by stroke leads to asymmetric deficits in postural reactive responses. In this experiment, we aimed to compare muscular responses to unanticipated stance perturbations between individuals who suffered unilateral stroke either to the right or to the left cerebral hemisphere. *Methods:* Stance perturbations were produced by releasing a load attached to the participant's trunk, inducing fast forward body oscillation. Electromyography was recorded from the gastrocnemius medialis and biceps femoris muscles. Muscular activation from age-matched healthy individuals was taken as reference. *Results:* Analysis indicated that damage to the right hemisphere induced delayed activation onset, and lower rate and magnitude of activation of the proximal and distal muscles of the paretic leg. Those deficits were associated with stronger activation of the nonparetic leg. Comparisons between left hemisphere damage and controls showed deficits limited to activation of the biceps femoris of the paretic leg. Manipulation of visual information led to no significant effects on muscular responses. *Conclusions:* These results suggest that right cerebral hemisphere damage by stroke leads to more severe deficits in the generation of reactive muscular responses to stance perturbation than damage to the left cerebral hemisphere regardless of visual information.
Key Words: Muscular responses—electromyography—cerebral lesion—hemiparesis

Introduction

Damage to different cortical and subcortical areas provoked by cerebral stroke leads to deficits in quiet and dynamic upright postural control[1]. For reactive postural responses to unanticipated perturbations, in particular, cases of unilateral stroke have been shown to impair control of both legs[2], but with more critical deficits in the paretic limb. That asymmetric effect of unilateral stroke has been evidenced through delayed[2−5] and weaker[4,6] muscular activation of the paretic leg to stance perturbation in comparison with healthy controls. Particularly detrimental to stance control seems to be damage to the right cerebral hemisphere. Of particular interest for the present investigation, the preeminence of the right cerebral hemisphere for stance control has been observed also in postural reactive responses. This effect has been observed in reduced velocity of center of pressure displacement across the feet soles and lower muscular activation of the paretic

From the *Human Motor Systems Laboratory, School of Physical Education and Sport, University of São Paulo, SP, Brazil; and †Biomedical Engineering, Federal University of ABC, São Bernardo do Campo, SP, Brazil.

Received October 4, 2018; revision received November 22, 2018; accepted December 14, 2018.

Funding: This study was funded through grants provided by the Foundation for Research Support of the State of São Paulo, Brazil (FAPESP, CAF #2011/16667-9, ARM #2011/18173-3, and LAT #2012/05769-8), and by the Brazilian Council of Science and Technology (CNPq, LAT #302628/2013-4).

Address correspondence to: Daniel Boari Coelho, Centre for Engineering, Modelling and Applied Social Sciences (CECS), Federal University of ABC (UFABC), Alameda da Universidade, s/no, Bairro Anchieta, São Bernardo do Campo, SP 09606-045, Brazil. E-mail: daniel.boari@ufabc.edu.br.

1052-3057/$ - see front matter

https://doi.org/10.1016/j.jstrokecerebrovasdis.2018.12.021

leg in the recovery of body balance following a sudden perturbation in individuals with right in comparison with left hemisphere damage by stroke.[7] Hence, previous results in stroke patients have converged to show increased importance of the right cerebral hemisphere for generation of postural responses.

An explanation for the preeminence of right hemisphere for stance control is associated with processing of proprioceptive information from the lower limbs. Goble[8] found that neural activation resulting from stimulation of ankle muscle spindles was predominantly localized to right-sided areas of the brain. Given that the neural activation was correlated with balance stability, increased deficits of stance regulation in individuals with right hemisphere damage might be associated with proprioception processing. In this case, one could expect that impaired capacity to processes proprioception from the lower limbs might be compensated for by increased reliance on another relevant sensory source for stance regulation like vision. On this point, research has suggested greater visual dependence for the control of balance stability in poststroke than healthy controls[9−11]. In the comparison between hemispheres, Manor et al[12] showed that damage to the right in comparison to the left cerebral hemisphere led to reduced balance stability particularly in the condition of visual occlusion. From these findings on quiet balance control, it could be thought that visual information is critical for balance recovery following a sudden perturbation in stroke lesions to the right cerebral hemisphere. From an alternative perspective, it has been proposed that the right cerebral hemisphere is specialized for regulating postural stability. That hemispheric specialization has been proposed to take place through exploration of stiffness- and viscous-like properties of muscular responses,[13] in addition to proprioceptive reflex gains and thresholds.[14−16] Comparisons between individuals who suffered stroke to the right versus left cerebral hemisphere in the performance of ipsilateral target-oriented manual movements have lent support for this theorization by showing that right but not left cerebral hemisphere damage induces deficits in the final hand positioning over a spatial target.[17−21] When initial directional errors were augmented by means of visuomotor rotations, right hemisphere damage was found to impair particularly online corrections to movements toward the target.[20,22] Although the conceptualization of right hemisphere specialization for postural stability has been formulated based on research of manual movements, it has important implications for stance control. For situations of perturbed stance, in particular, generation of postural responses to recover a stable upright posture following a perturbation could be thought to be regulated through similar mechanisms. From this rationale, one would expect that damage to the right cerebral hemisphere leads to poorer muscular responses to unanticipated perturbations in comparison with damage to the left hemisphere.

In the present experiment, we aimed to compare muscular responses to unanticipated perturbations between individuals who suffered unilateral stroke either to the right or to the left cerebral hemisphere.[1] In preliminary research on this topic,[7] it was shown poorer balance recovery following perturbation in individuals with right as compared to left cerebral hemisphere damage. In the present investigation, we performed a detailed analysis of activation of upper and lower posterior leg muscles, including activation onset latency, gain rate, and magnitude of activation in different phases of the response both in the paretic and nonparetic legs in individuals with unilateral damage either to the right or left cerebral hemisphere. The proposition of right cerebral hemisphere preeminence for the control of postural stabilization led us to predict poorer muscular responses in individuals who suffered right cerebral hemisphere damage in comparison with groups of left hemisphere damage and healthy controls. To evaluate the extent to which sensory processes affect the generation of reactive postural responses, we conducted the evaluation with manipulation of visual information based on the following rationale: if right hemisphere damage leads to a deficit of visual processing in the generation of reactive postural responses, we expected to find differences between groups in the condition of full vision only, due to the supposed intact processing of visual information in left hemisphere damage and controls, while under visual occlusion all individuals would be in equivalent conditions for generation of muscular responses. For the proposition that the right hemisphere plays a prominent role for processing of proprioceptive signals from the ankles,[8] conversely, only the condition of visual occlusion would be expected to lead to poor performance of individuals with right hemisphere damage due to inability to compensate for the deficit in the processing of somesthetic information by weighting up vision in sensorimotor integration.

Methods

Participants

Selection of poststroke participants with damage to the right (RHD, $n = 11$, age range 55-83 years), or left (LHD, $n = 11$, age range 54-83 years) cerebral hemisphere, and healthy controls (CO, $n = 24$, age range 46-76 years) was made from the same hospital database. Inclusion criteria for poststroke participants were the following: single unilateral stroke, time range following stroke of 6 months to 3 years, mild to moderate functional disability (Rankin scale, Wilson[23]), behavioral manifestation of hemiparesis, absence of neurologic or orthopedic diseases other than those induced exclusively by the stroke, absence of damage to the cerebellum or brain stem, and scores 1-2 in the modified Ashworth's spasticity scale.[24] Exclusions were

[1]Joints' kinematics and center of pressure data are reported elsewhere (Fernandes et al., 2018, Experiment 2).

due to incapacity to perform the experimental task under visual occlusion (left hemisphere stroke, $n = 1$), and detection of stroke symptoms, based on the stroke symptoms questionnaire[25] (control group, $n = 1$). Data from 5 participants of each group were discarded because activation of the muscle biceps femoris was too low for identification of its activation onset in 1 or both of the legs. Low activation of that muscle in the paretic leg in participants of both stroke groups was due to almost exclusive responses of the nonparetic leg, while almost exclusive responses of the lower legs led also to exclusions (all groups). Full analysis was performed on 6 participants of RHD (3 men, mean age = 68.17 years, $SD = 6.49$), 6 participants of LHD (3 men, mean age = 59.67 years, $SD = 11.59$), and 18 controls (7 men, mean age = 74.11 years, $SD = 11.73$). The lesioned cortical areas (as indicated by magnetic resonance imaging), in addition to Fugl-Meyer scale scores for sensitivity and mobility, of the poststroke participants are presented in Table 1. The modified minimental state examination[26] was used to detect possible cognitive problems, with acceptable minimum scores varying as a function of level of education: 13 points for the uneducated, 18 for the low and middle school education, and 26 for high school education. All participants achieved the respective required score. Participants provided informed consent, and the local university ethics committee approved the study protocol according to the Declaration of Helsinki.

Tasks and Apparatus

The task consisted of recovering stable upright stance following unanticipated perturbations keeping the feet in place. Stance perturbation was induced by using a custom-built load release system, as described in the following.

Table 1. *Neural areas damaged and Fugl-Meyer scale score*

	Right	Left
Stroke site (n)		
Parietal	1	1
Parietal and temporal	1	1
Parietal and frontal		1
Parietal, temporal, frontal and Basal ganglia	1	
Occipital	1	
Occipital, temporal and thalamus		1
Basal ganglia		1
Frontal		1
Frontal and temporal	1	
Frontal and Basal ganglia	1	
Fugl-Meyer (score)		
Sensitivity	10	10
Mobility	22.5	25.5

Stroke sites. Cortical areas: frontal, temporal, parietal, and occipital cortices, temporo-parietal junction. Subcortical: thalamus, basal ganglia, and insula. Maximum score for the Fugl-Meyer scale (Fugl-Meyer, Jääskö, Leyman, Olsson, & Steglind, 1975) is 12 for sensitivity and 34 for mobility.

Participants stood barefoot upright on a rigid surface, feet oriented approximately in parallel hip-width apart, resisting against a load pulling backward the participant's trunk. To maintain stance while being pulled by the load, participants leant the body slightly forward to assume a stable equilibrium position, keeping legs and trunk aligned, with the feet in full contact with the support base. The arms were maintained relaxed beside the body in parallel with the trunk. They wore a 20-cm wide harness positioned at the lumbar-sacral region with an electromagnetic system fixed on its backside. A steel cable tied to a magnet was linked to the harness by means of an electromagnet. The cable crossed a pulley attached to a height-adjustable support, with a load hung at the end of the cable (Fig. 1, A). A remote switch was used to release the load at a time unbeknownst to the participant. Load release induced a fast forward participant's body sway, requiring fast postural responses mainly through activation of the posterior muscles of the legs (see Martinelli,[27] for a detailed description). Activation of the muscles gastrocnemius medialis (GM) and biceps femoris (BF) of both legs was measured by means of wireless surface electrodes (Delsys Trigno Wireless System, Boston, MA). It should be observed regarding muscles selection that body balance recovery in our task required a shift in activation between anterior (preperturbation) and posterior (postperturbation) leg muscles to prevent forward body displacement leading to balance loss. Then, our task imposed a particular reactive response based on activation of the posterior muscles of the legs rather than coactivating anterior and posterior muscles following load release.

Experimental Design and Procedures

Participants were tested under full vision and visual occlusion, with sequence of visual conditions alternated across participants within group. For the condition of full vision, participants gazed at a 1-cm diameter black spot at the top of a vertical shaft, positioned in front of them at the eyes height 1.5 m away. For the condition of visual occlusion, participants were blindfolded and oriented their head as if gazing at the visual target. For familiarization, the perturbation task was performed preliminarily with progressive loads. The first trial was performed with the load of 1 kg, the second with 2 kg, and the third with 5% of the personal body mass (3−4.5 kg across participants). Following those familiarization trials (discarded from analysis), they were evaluated by using the 5% load, performing 3 trials in block under each visual condition.

Electrodes were attached to the GM and BF muscles of both legs, following the SENIAM recommendations for electromyography (EMG) measurements (http://www.seniam.org/). In cases that the participant moved their feet in response to stance perturbation that trial was discarded and immediately repeated. Feet positions were outlined with adhesive tapes on the ground to ensure correct repositioning in those cases. Intertrial intervals within visual conditions were 30-s long, time during which participants rested in unloaded upright stance. Between visual conditions, participants rested sitting during an interval of 2 minutes. EMG data acquisition was sampled at the frequency of 2000 Hz.

Analysis

EMG signals were band-pass filtered online between 20 and 400 Hz. They were rectified and Butterworth low-pass filtered offline at 30 Hz. Analysis of muscular activation was conducted in the period immediately following load release. We evaluated the following variables: (1) latency of muscular activation onset, having as criterion the value of 2 standard deviations above the values in the period of 200 milliseconds preceding perturbation; (2) gain rate of muscular activation, given by the slope of the line connecting the values at muscular activation onset and at the ensuing 100 milliseconds in the integrated EMG signal; and magnitude of muscular activation in the (3) primary 0−150 milliseconds and (4) secondary 150−300 milliseconds epochs following muscular activation onset, given by the normalized integrated EMG signal. A representative signal from the GM muscle showing the 2 epochs analyzed is presented in Figure 1, B. Normalization was made by the individual maximum muscular activation across experimental conditions

Half the participants of the CO group had their right leg labeled as paretic and the left leg as nonparetic, while for the other half of the participants in this group we used the opposite labeling. Legs labeling across CO participants was randomized. Statistical analysis was made by means of 3-way 3 (group: RHD, LHD, and CO) × 2 (vision: full, occlusion) × 2 (leg: paretic × nonparetic) ANOVAs with repeated measures on the last 2 factors. Post hoc comparisons were performed through Newman-Keuls procedures. We report significant differences ($P < .05$) accompanied by the respective effect sizes given by the partial eta squared (η_p^2). We employed the classification of η_p^2 values as small (<.06), moderate (≥.06-.14) and large (>.14) (based on Cohen[28]).

Results

A few trials were repeated due to a compensatory stepping in response to the perturbation: RHD = 2, LHD = 3, and CO = 2. Representative EMG signals from individual trials are presented in Figure 2. In that figure, we show the profile of activation of the GM muscle, for the nonparetic (left panels) and paretic (right panels) legs, of participants who suffered right cerebral damage in comparison with left hemisphere damage and controls. Analysis of activation onset latency for the GM muscle indicated a significant group × leg interaction, $F(2, 27) = 6.96$, P less than .01, $\eta_p^2 = .34$. Post hoc comparisons revealed that the interaction was due to longer activation delays of the RHD's paretic leg in comparison with their nonparetic leg, and with the corresponding legs of the LHD and CO (Fig 3, A). Analysis of activation onset latency of the BF muscle indicated a significant group × leg interaction, $F(2, 27) = 3.39$, $P = .05$, $\eta_p^2 = .20$. Post hoc comparisons revealed significantly longer activation delays for RHD's paretic leg in comparison with CO, and for LHD's paretic leg in comparison with their nonparetic leg and the respective leg of CO (Fig 3, B).

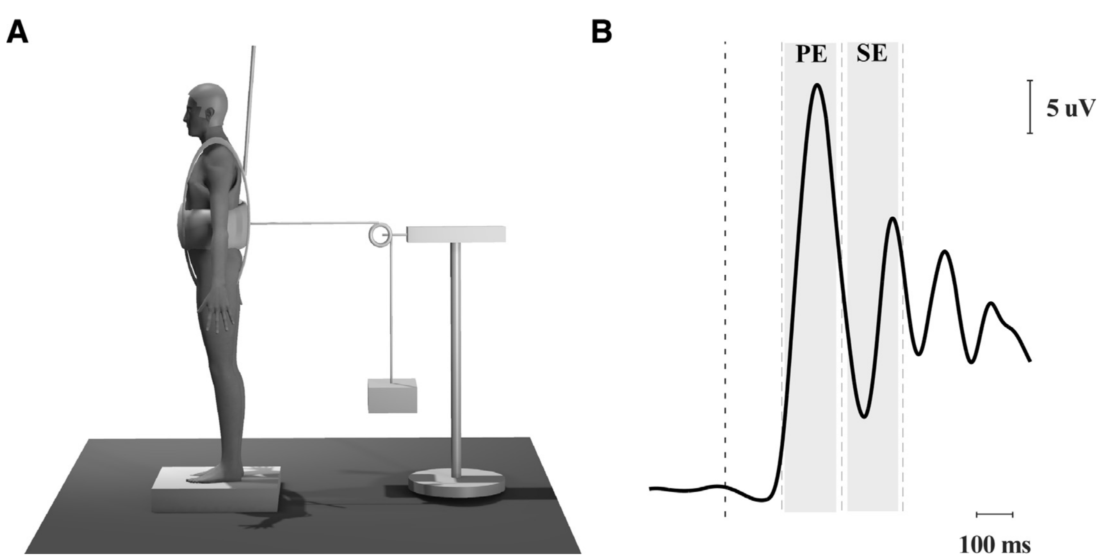

Figure 1. *(A) Representation of the perturbed balance task, with the load pulling the participants' trunk backward in the period preceding load release. (B) Representative signal of the activation of the gastrocnemius medialis muscle, with indication of the primary (PE: 0-150 milliseconds) and secondary (SE: 150-300 milliseconds) epochs employed in the analysis.*

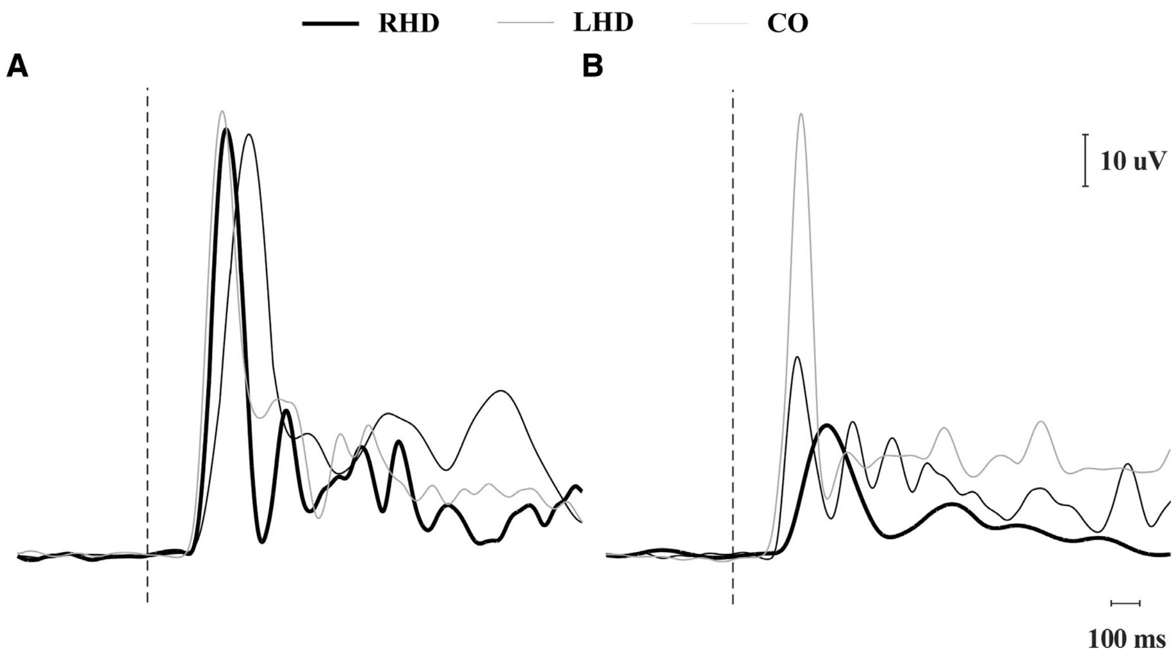

Figure 2. *Representative single-trial electromyography signals in response to perturbation (time indicated by vertical dashed lines), showing the main characteristics of muscular activation of the (A) nonparetic and (B) paretic legs of each group. RHD, right hemisphere damage; LHD, left hemisphere damage; CO, controls.*

Analysis of rate of muscular activation (slope) for the GM muscle indicated a significant group × leg interaction, $F(2, 27) = 3.48$, $P = .05$, $\eta_p^2 = .20$. Post hoc comparisons revealed lower values for RHD's paretic leg in comparison with their nonparetic leg and with the corresponding leg of CO (Fig 3, C). Analysis of rate of muscular activation for the BF muscle (Fig 3, D) indicated a significant main effect of leg, $F(1, 27) = 4.65$, $P = .04$, $\eta_p^2 = .15$. That effect was due to significant lower values for the paretic ($M = .44$, SE = .06) than for the nonparetic leg ($M = .58$, $SE = .08$).

Analysis of magnitude of activation in the primary epoch of the GM muscle activation indicated a significant group × leg interaction, $F(2, 27) = 3.69$, $P = .04$, $\eta_p^2 = .21$. Post hoc comparisons revealed weaker muscular activation of the RHD's paretic leg in comparison with their nonparetic leg and with the corresponding leg of CO (Fig 4, A). Analysis of that epoch for the BF muscle activation indicated a significant group × leg interaction, $F(2, 27) = 3.29$, $P = .05$, $\eta_p^2 = .20$. Post hoc comparisons revealed a similar effect for both poststroke groups, with lower activation of the paretic in comparison with the nonparetic leg (Fig 4, B).

For the secondary epoch of muscular activation, analysis of the GM muscle indicated a significant group × leg interaction, $F(2, 27) = 11.17$, P less than .01, $\eta_p^2 = .45$. Post hoc comparisons revealed significantly stronger activation of the RHD's nonparetic leg in comparison with their paretic leg, and with the corresponding leg in LHD and CO. Figure 4, C suggests weaker activation of the RHD's paretic leg in the comparison with controls, but the difference fell short of significance ($P = .07$). Analysis of the secondary epoch of the BF muscle activation failed to show any significant effects (Fig 4, D).

Manipulation of visual information led to no significant main effects or associated interactions on muscular responses, with analysis indicating F values less than 2.60, P values greater than .10, and η_p^2 values less than .09, across variables.

Discussion

Results from previous research showing that right as compared to left cerebral hemisphere damage by stroke leads to impoverished control in quiet[12,29–32] and dynamic[33–35] body balance might be interpreted as being due to deficits in visual or proprioceptive processing. Lack of effect of vision manipulation is contradictory to those assumptions. The finding that visual occlusion did not impair muscular responses, however, suggests that vision plays a minor role in the evaluated task in comparison with quiet stance control cf.[36] Sensory information from somatosensory receptors located at the feet soles,[37–40] and at muscles spindles in the ankles[40] and in the upper legs or trunk[41,42] could be thought to be the main sensory sources used for triggering and scaling reactive postural responses. Further on this point, it has been shown that weighting of proprioceptive information can be set independently for each leg based on lateralized reliability of sensory information for stance control.[43] From these observations, it is possible that proprioceptive information from the RHD's paretic leg is downregulated, leading to generation of delayed and weaker muscular responses of that leg in comparison with controls. This line of reasoning, then, contributes to a putative

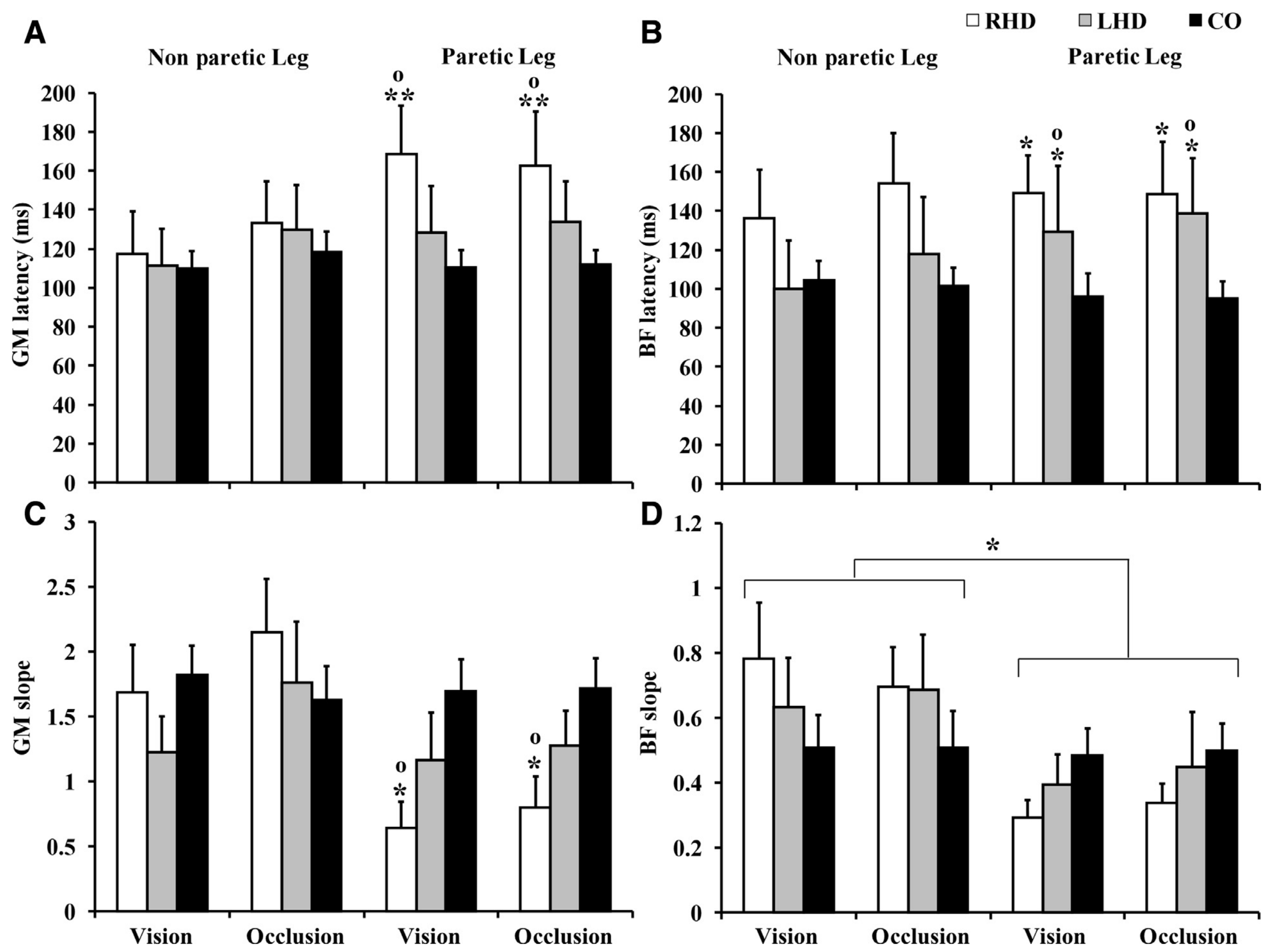

Figure 3. *Average values (standard errors in vertical bars) for (A) latency of activation onset of the muscles gastrocnemius medialis (GM) and (B) biceps femoris (BF), and slope (gain rate) of muscular activation for (C) GM and (D) BF. Comparisons are organized as a function of leg (nonparetic × paretic) and vision (vision × occlusion). Significant interactions are marked as different from CO (*), different from CO and LHD (**), and different from the opposite leg (º).*

lateralized sensorimotor integration deficit rather than to a disruption of processing of proprioceptive information in both legs as one might predict from the proposition of preeminence of the right cerebral hemisphere for the processing of ankles' proprioception.[8]

Previous studies have reported that brain damage by stroke leads to asymmetric impairment of muscular responses to unanticipated stance perturbations, with delayed[3–5,44] and weaker[4] muscular activation of the paretic leg. Our results qualify that conclusion by showing that deficits of muscular activation of the paretic leg were more extensive in RHD than LHD. In particular, activation of the paretic leg's GM muscle was significantly affected in RHD only, with delayed, slower increment and lower magnitude of activation in comparison with controls. On the other hand, LHD showed a pattern of the GM muscle activation in the paretic leg more similar to controls, and absence of significant between-leg asymmetries. Interestingly, deficits in the activation of the proximal BF muscle were equivalent between the RHD and LHD groups. We suggest that these results may reflect crossed innervation for the distal lower leg muscles, while the proximal upper leg muscles could be thought to receive part of the descending efferent signals from the ipsilateral cerebral hemisphere.[45] From this perspective, our results lead to the interpretation that the right cerebral hemisphere plays a prominent role in the generation of reactive muscular responses of the lower legs in the contralesional side, and possibly affecting muscular responses of the upper leg in both body sides. Based on our findings of reduced gain rate and lower magnitude of muscular activation in the affected leg muscles, it is apparent that the observed deficits result from reduced motor unit firing rates[46] or insufficient motor unit recruitment[47] to generate reactive responses to stance perturbation.

As a further point of interest, our results revealed that deficits of the RHD's paretic leg were compensated for by stronger activation of the nonparetic leg, an effect reaching statistical significance in the secondary epoch of activation of the GM muscle (but see similar trends in slope and magnitude of activation in the primary epoch). That increased muscular response of the nonparetic leg seems to be compensatory to delayed, slower and weaker activation of the paretic leg in the RHD group. Analogous compensatory behavior between legs has been reported previously in self-

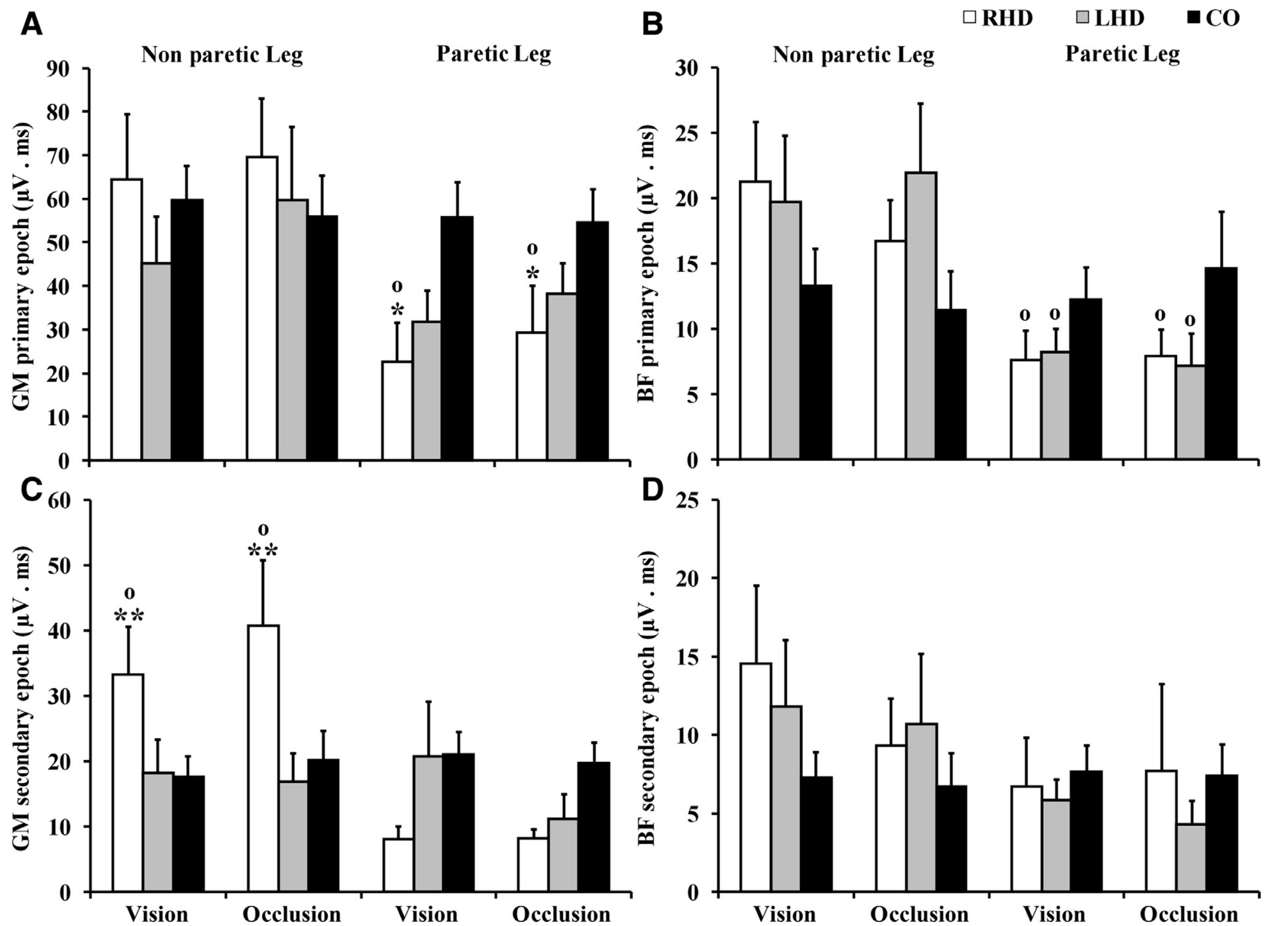

Figure 4. *Average values (standard errors in vertical bars) for magnitude of muscular activation in the (A, B) primary and (C, D) secondary epochs for the muscles gastrocnemius medialis (GM, left) and biceps femoris (BF, right). Comparisons are organized as a function of leg (nonparetic × paretic) and vision (vision × occlusion). Significant interactions are marked as different from CO (*), different from CO and LHD (**), and different from the opposite leg (°).*

induced postural perturbations by means of fast arm raising.[48] We interpret these results as suggesting that the postural control system scales up muscular activation of the nonparetic leg to compensate for the impaired responses of the paretic leg. As this compensatory muscular activation was observed only in the secondary epoch of the GM muscle activation, it is plausible that feedforward and feedback processes took place to induce increased activation of the nonparetic leg. That adaptive behavior of muscular activation between the legs indicates the relevance of higher order neural structures for the generation of automatic postural responses. The fact that the lesioned sites of the right cerebral hemisphere leading to poor muscular responses were distinct across participants within group suggests that corrective responses are organized in a highly distributed neural network, with damage to part of it impairing the generation of appropriate reactive muscular responses of the paretic leg.

Conclusions

Our results indicate that damage to the right in comparison to the left cerebral hemisphere led to more dramatic deficits in the activation of the paretic leg muscles relevant to reactive responses to unanticipated stance perturbations regardless of visual information. Those deficits were featured by longer muscular activation onset delays, decreased gain rate and reduced magnitude of activation of posterior muscles of the lower and upper legs of the paretic hemibody. Our results suggest that increased attention should be given to rehabilitation of upright balance control in individuals suffering from right hemisphere damage due to stroke, with special emphasis to reactive responses. As a major limitation of our investigation, the cerebral damage groups were reduced in size. This may have prevented observation of significant effects associated with visual manipulation. Additionally, we measured bilaterally activation of 2 posterior leg muscles only. From these observations, we recommend that our conclusions be considered preliminary until further investigations can be undertaken with larger numbers of participants and additional postural muscles examined.

Acknowledgments

The authors would like to thank Paulo Lotufo, Isabela Benseñor e Alessandra Goulart for their technical support, and to Leon Barbosa for his assistance in data collection.

Conflict of Interest

The authors declare that they have no conflict of interest.

References

1. Teixeira LA. Interlateral asymmetries of body balance control resulting from cerebral stroke. Locomotion and Posture in Older Adults 2017: 291-305.
2. Marigold DS, Eng JJ. Altered timing of postural reflexes contributes to falling in persons with chronic stroke. Exp Brain Res 2006;171:459-468.
3. Di Fabio RP, Badke MB, Duncan PW. Adapting human postural reflexes following localized cerebrovascular lesion: analysis of bilateral long latency responses. Brain Res 1986;363:257-264.
4. Ikai T, Kamikubo T, Takehara I, et al. Dynamic postural control in patients with hemiparesis. Am J Phys Med Rehabil 2003;82:463-469.
5. Marigold DS, Eng JJ, Inglis JT. Modulation of ankle muscle postural reflexes in stroke: Influence of weight-bearing load. Clin Neurophysiol 2004;115:2789-2797.
6. Garland SJ, Ivanova TD, Mochizuki G. Recovery of standing balance and health-related quality of life after mild or moderately severe stroke. Arch Phys Med Rehabil 2007;88:218-227.
7. Fernandes CA, Coelho DB, Martinelli AR, et al. Right cerebral hemisphere specialization for quiet and perturbed body balance control: Evidence from unilateral stroke. Hum Mov Sci 2018;57:374-387.
8. Goble DJ, Coxon JP, Van Impe A, et al. Brain activity during ankle proprioceptive stimulation predicts balance performance in young and older adults. J Neurosci 2011;31:16344-16352.
9. Bensoussan L, Viton JM, Schieppati M, et al. Changes in postural control in hemiplegic patients after stroke performing a dual task. Arch Phys Med Rehabil 2007;88:1009-1015.
10. Bonan IV, Colle FM, Guichard JP, et al. Reliance on visual information after stroke. Part I: Balance on dynamic posturography. Arch Phys Med Rehabil 2004;85:268-273.
11. Oliveira CB, Medeiros IR, Greters MG, et al. Abnormal sensory integration affects balance control in hemiparetic patients within the first year after stroke. Clinics (Sao Paulo) 2011;66:2043-2048.
12. Manor B, Hu K, Zhao P, et al. Altered control of postural sway following cerebral infarction: a cross-sectional analysis. Neurology 2010;74:458-464.
13. Shadmehr R, Arbib MA. A mathematical analysis of the force-stiffness characteristics of muscles in control of a single joint system. Biol Cybern 1992;66:463-477.
14. Mutha PK, Boulinguez P, Sainburg RL. Visual modulation of proprioceptive reflexes during movement. Brain Res 2008;1246:54-69.
15. Crevecoeur F, Kurtzer I, Bourke T, et al. Feedback responses rapidly scale with the urgency to correct for external perturbations. J Neurophysiol 2013;110:1323-1332.
16. Kurtzer IL, Crevecoeur F, Scott SH. Fast feedback control involves two independent processes utilizing knowledge of limb dynamics. J Neurophysiol 2014;111:1631-1645.
17. Haaland KY, Prestopnik JL, Knight RT, et al. Hemispheric asymmetries for kinematic and positional aspects of reaching. Brain 2004;127:1145-1158.
18. Schaefer SY, Haaland KY, Sainburg RL. Ipsilesional motor deficits following stroke reflect hemispheric specializations for movement control. Brain 2007;130:2146-2158.
19. Schaefer SY, Haaland KY, Sainburg RL. Hemispheric specialization and functional impact of ipsilesional deficits in movement coordination and accuracy. Neuropsychologia 2009;47:2953-2966.
20. Schaefer SY, Haaland KY, Sainburg RL. Dissociation of initial trajectory and final position errors during visuomotor adaptation following unilateral stroke. Brain Res 2009;1298:78-91.
21. Schaefer SY, Mutha PK, Haaland KY, et al. Hemispheric specialization for movement control produces dissociable differences in online corrections after stroke. Cereb Cortex 2012;22:1407-1419.
22. Mani S, Mutha PK, Przybyla A, et al. Contralesional motor deficits after unilateral stroke reflect hemisphere-specific control mechanisms. Brain 2013;136:1288-1303.
23. Wilson JT, Hareendran A, Grant M, et al. Improving the assessment of outcomes in stroke: Use of a structured interview to assign grades on the modified Rankin Scale. Stroke 2002;33:2243-2246.
24. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. Phys Ther 1987;67:206-207.
25. Abe IM, Goulart AC, Santos Junior WR, et al. Validation of a stroke symptom questionnaire for epidemiological surveys. Sao Paulo Med J 2010;128:225-231.
26. Bertolucci PH, Brucki SM, Campacci SR, et al. The Mini-Mental State Examination in a general population: impact of educational status. Arq Neuropsiquiatr 1994;52:1-7.
27. Martinelli AR, Coelho DB, Magalhaes FH, et al. Light touch modulates balance recovery following perturbation: from fast response to stance restabilization. Exp Brain Res 2015;233:1399-1408.
28. Cohen J. Statistical Power Analysis for the Behavioral Sciences. New York, NY: Routledge Academic; 1988.
29. Peurala SH, Kononen P, Pitkanen K, et al. Postural instability in patients with chronic stroke. Restor Neurol Neurosci 2007;25:101-108.
30. Rode G, Tiliket C, Boisson D. Predominance of postural imbalance in left hemiparetic patients. Scand J Rehabil Med 1997;29:11-16.
31. Perennou DA, Mazibrada G, Chauvineau V, et al. Lateropulsion, pushing and verticality perception in hemisphere stroke: a causal relationship? Brain 2008;131:2401-2413.
32. Laufer Y, Sivan D, Schwarzmann R, et al. Standing balance and functional recovery of patients with right and left hemiparesis in the early stages of rehabilitation. Neurorehabil Neural Repair 2003;17:207-213.
33. Ioffe ME, Chernikova LA, Umarova RM, et al. Learning postural tasks in hemiparetic patients with lesions of left versus right hemisphere. Exp Brain Res 2010;201:753-761.
34. Ustinova KI, Chernikova LA, Ioffe ME, et al. Impairment of learning the voluntary control of posture in patients with cortical lesions of different locations: The cortical mechanisms of pose regulation. Neurosci Behav Physiol 2001;31:259-267.
35. Ishii F, Matsukawa N, Horiba M, et al. Impaired ability to shift weight onto the non-paretic leg in right-cortical brain-damaged patients. Clin Neurol Neurosurg 2010;112:406-412.
36. Vieira OJ, Coelho DB, Teixeira LA. Asymmetric balance control between legs for quiet but not for perturbed stance. Exp Brain Res 2014;232:3269-3276.
37. Meyer PF, Oddsson LI, De Luca CJ. Reduced plantar sensitivity alters postural responses to lateral perturbations of balance. Exp Brain Res 2004;157:526-536.
38. Meyer PF, Oddsson LI, De Luca CJ. The role of plantar cutaneous sensation in unperturbed stance. Exp Brain Res 2004;156:505-512.

39. Perry SD, McIlroy WE, Maki BE. The role of plantar cutaneous mechanoreceptors in the control of compensatory stepping reactions evoked by unpredictable, multi-directional perturbation. Brain Res 2000;877:401-406.
40. Thompson C, Belanger M, Fung J. Effects of plantar cutaneo-muscular and tendon vibration on posture and balance during quiet and perturbed stance. Hum Mov Sci 2011;30:153-171.
41. Bloem BR, Allum JH, Carpenter MG, et al. Is lower leg proprioception essential for triggering human automatic postural responses? Exp Brain Res 2000;130:375-391.
42. Bloem BR, Allum JH, Carpenter MG, et al. Triggering of balance corrections and compensatory strategies in a patient with total leg proprioceptive loss. Exp Brain Res 2002;142:91-107.
43. Pasma JH, Boonstra TA, Campfens SF, et al. Sensory reweighting of proprioceptive information of the left and right leg during human balance control. J Neurophysiol 2012;108:1138-1148.
44. Di Fabio RP. Lower extremity antagonist muscle response following standing perturbation in subjects with cerebrovascular disease. Brain Res 1987;406:43-51.
45. Brinkman J, Kuypers HG. Splitbrain monkeys: cerebral control of ipsilateral and contralateral arm, hand, and finger movements. Science 1972;176:536-539.
46. Pollock CL, Ivanova TD, Hunt MA, et al. Behavior of medial gastrocnemius motor units during postural reactions to external perturbations after stroke. Clin Neurophysiol 2015;126:1951-1958.
47. Garland SJ, Gray VL, Knorr S. Muscle activation patterns and postural control following stroke. Motor Control 2009;13:387-411.
48. Garland SJ, Willems DA, Ivanova TD, et al. Recovery of standing balance and functional mobility after stroke. Arch Phys Med Rehabil 2003;84:1753-1759.