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ORIGINAL ARTICLE

Postural asymmetry correlated with lateralization of cerebellar perfusion in persons with chronic stroke: A role of crossed cerebellar diaschisis in left side

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ABSTRACT

Objective: Hemiplegia after stroke leads to impairment of the affected limbs and induces more weight on the non-paretic lower limb to form postural asymmetry. Studies of asymmetric cerebral functions have found similarly asymmetric functions in the cerebellum. Crossed cerebellar diaschisis (CCD) is defined as reduced blood flow and hypometabolism in the cerebellar hemisphere contralateral to supratentorial cerebral pathology. No study explored the relationship between posture (standing balance) and CCD in those persons yet. It was hypothesized that CCD would impair postural control and tend toward lateralization of cerebellar perfusion.

Methods: To determine the relationship between postural asymmetry and CCD among patients with chronic stroke while testing in the upright position. Based on images from Tc-99m-ECD brain perfusion, 42 patients were retrospectively allocated into three groups: left CCD, right CCD and no CCD. The ability to maintain an upright stance as assessed by postural parameters was evaluated using a force platform.

Results: The sway intensity differed significantly between the groups with left CCD and no CCD (p = 0.0052), as did the sway velocities (p = 0.0010). The association between the duration of stroke and sway intensity was highly significant (p < 0.0001). The interval from the stroke onset to the postural analysis was significantly associated with sway intensity and velocity.

Conclusions: This study indicates that the impairment of posture sway control was more severe in left CCD than the other CCD types. The results support a relationship between the postural asymmetry and lateralization of CCD in patients with chronic stroke.

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Introduction

Patients with stroke often display exaggerated postural sway, abnormal gait and severe balance dysfunction with disturbed equilibrium reactions [1]. Hemiplegia after stroke not only leads to impairment of the affected limbs, but also produces abnormal posture, inducing weight-bearing asymmetry [2]. Postural asymmetry means more weight on the non-paretic lower limb and many factors have been demonstrated to contribute to the failure of compensation for it by loading the paretic lower limb [3–6]. After the patient is able to stand for a while without any demand for support, objective postural sway tests can assess stationary standing stability in the early stages of rehabilitation. Therefore, the biomechanical analysis of postural disturbances, among patients with hemiplegia may identify patients with postural asymmetry and, thus, aid locomotor outcome after stroke.

Based on established neuroimaging studies, asymmetry of brain function has been demonstrated in the motor cortex and cerebellum within the tasks of the upper extremity [7] and of lower extremity [8] function. Lateralization is one of the characteristics of human cultures with a right-sided tendency, which may be socially as well as biologically enforced. Furthermore, it emerges in the brain as a unitary property of large-scale circuits that connect the cerebral cortex and cerebellum. Neuroimaging studies of asymmetric cerebral functions have often found similarly asymmetric (but flipped) functional responses in the cerebellum [9]. For example, in most people, language processing activates the left, inferior frontal gyrus and the superior temporal lobe as well as the right cerebellum, including crus I/II and lobule VI. In contrast, spatial processing often involves the right angular gyrus, supramarginal gyrus, insula and the left cerebellar lobule VI. A reasonable hypothesis based on the anatomic and neuroimaging observation is that functional asymmetries within the cerebellum will show distinct postural sway control.

Reduction of blood flow and hypometabolism in the cerebellar hemisphere contralateral to supratentorial cerebral pathology is defined as crossed cerebellar diaschisis (CCD) [10]. Studies of single-photon emission computed tomography (SPECT) and positron emission tomography (PET) demonstrated that CCD can be caused by lesions in a variety of cortical areas; such lesions include cerebral haematomas, head injury and epilepsy [10–13]. CCD can

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also co-exist in patients who have complex regional pain syndrome [14] and parakinesia brachialis oscitans [15]. CCD can only be diagnosed by PET or SPECT and there are no screening tests for CCD. However, a work from Germany showed that dynamic susceptibility, contrast perfusion weighted magnetic resonance (MR) imaging might also detect CCD [16].

In this research, no study was found exploring the relationship between posture (standing balance) and CCD in subjects with stroke [3–6,17–22]. It was hypothesized that CCD would impair postural control and that a tendency toward lateralization of cerebellar perfusion existed. Therefore, the authors sought to determine the relationship between upright postural control and cerebellar hypoperfusion among patients with chronic hemiplegic stroke during eye open and eye closed conditions. The study was conducted to learn whether postural function would be affected in patients with chronic stroke and CCD during testing with eyes open and closed when resting in the upright position. This study also evaluated the possibility of postural asymmetry.

Materials and methods

Patients

In this retrospective study encompassing January 2008–December 2011, patients, either inpatient or outpatient, who were diagnosed with ischaemic stroke were screened from the medical records database for the study, as shown in the flow chart in Figure 1.

Exclusion criteria were (1) Concomitant cerebellar abnormalities or bilateral cerebral pathology, (2) Small lesion in the cerebral hemisphere (such as lacunar infarction), (3) Carotid artery disease or any intracranial space-occupying lesion or any other cerebral pathology such as haemorrhage, tumour or infection, (4) Spinocerebellar atrophy, intracranial surgery, visual neglect or of cerebellar signs, such as pathological nystagmus, dysarthria and ataxia, (5) Lack of information on the modified Rankin Scale and Mini-Mental State Exam, (6) Insufficient or incomplete data for quantitative measurement of the postural sway, (7) Unreliable postural sway testing data due to having orthopaedic and other postural control influencing diseases, such as arthrosis or total hip joint replacement, (8) The interval between the SPECT scan and the investigation of postural sway larger than 3 weeks, (9) Blurred images of brain perfusion SPECT due to a patient's intolerance or uncooperative behaviour during an examination (ineligible for an brain perfusion SPECT), (10) Cerebellar diaschisis ipsilateral to the supratentorial hemispheric ischaemia, and (11) Significantly decreased tracer uptake within the bilateral cerebellum.

Inclusion criteria were (1) MR imaging including diffusion-weighted imaging (DWI) to confirm hemispheric supratentorial ischaemic stroke. MR scan was performed for all patients; (2) brain perfusion SPECT was performed in all eligible cases in the Department of Nuclear Medicine; (3) all of the patients had minimal or no cognitive impairment completed postural sway testing, Brunnstrom stage IV–VI in lower limbs, at least.

Examination of SPECT images

Brain SPECT images were obtained after intravenous injection of ethyl cysteinate dimer (Tc-99m-ECD), which was prepared from a commercial kit (Neurolite Du Pont Merck Pharmaceutical Company, Billerica, MA) by adding 25 mCi of freshly eluted Tc-99m pertechnetate to 5 mL of saline

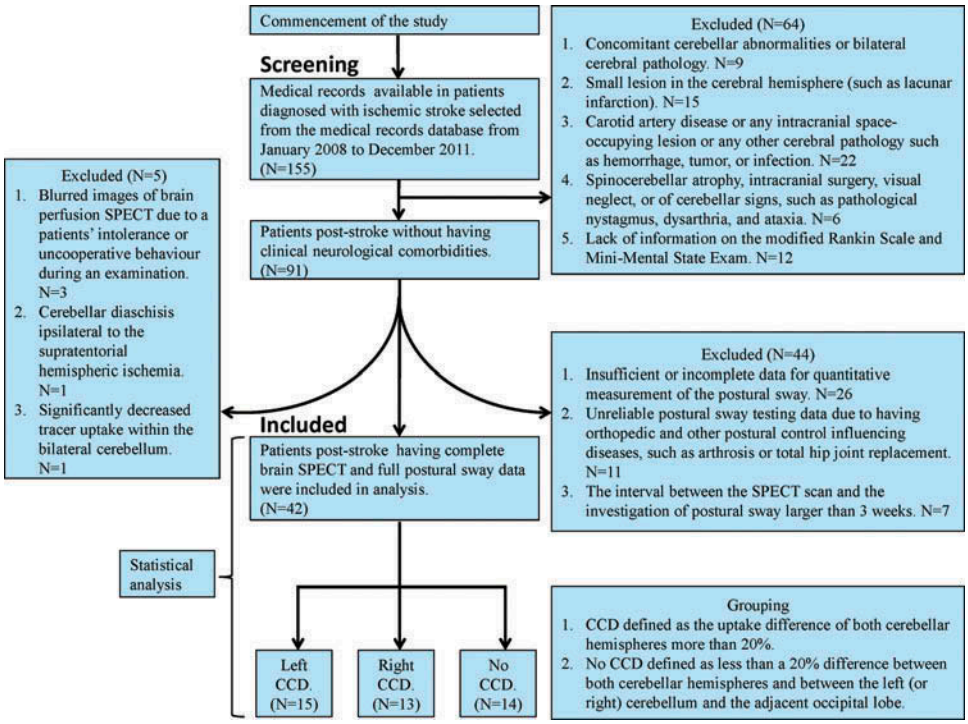

Figure 1. A flowchart of the retrospective study design.

solution. After injection of the radiotracer, imaging commenced 30 minutes later using a dual-headed camera equipped with ultra-high resolution fan-beam collimators. Data were acquired in a 128 × 128 matrix with a 1.4-times zoom through 360° (180° for each head) with rotation at 3° intervals, for 30 seconds per angle step. Images were reconstructed using a back-projection method [23] with a Metz filter. SPECT images were analysed from three axial planes (axial, sagittal and coronal slices, with a 0.3-cm slice thickness) and yielded a complete set of axial tomographic slices from the posterior cranial fossa to the vertex. CCD was identified when the uptake difference of both cerebellar hemispheres was more than 20% [24,25]. No CCD was defined as less than a 20% difference between both cerebellar hemispheres and between the left (or right) cerebellum and the adjacent occipital lobe on the reconstructed images. Thus, three groups were identified: left CCD, right CCD and no CCD (Figure 2).

Image analysis

Grey-scale median (GSM) has been investigated on computer analysis for hypoechoic and echogenic superficial femoral artery occlusive plaque as the study by Marks et al. [26]. Densitometric analysis was carried out using the Adobe Photoshop software (version CS3, Adobe Systems Inc, San Jose, CA) and grey-scale histogram facility. With use of this software, GSM value analysis quantifies uptake of bilateral cerebellar hemispheres in a series of axial cuts of the images seen on brain SPECT after converting the images to grey-scale. Patients who had post-stroke images available in the clinical practice and that were amenable to GSM analysis were identified as a sub-set for further investigation. The GSM of brain SPECT images can be calculated, categorizing the cerebellum into less or normal perfusion hemispheres by placing appropriate regions of interest (ROIs) on positions standardized under MRI guidance. With this software, a GSM was obtained by using the lasso tool from the tool palette in a grey-scale document when ROIs were placed in the affected (A) and mirrored unaffected (U) cerebellar hemispheres. A linear grey-scale of 0 (absolute dark) to 255 (absolute white) obtained from the software facilities was applied as a reference. Brain SPECT images amenable to GSM analysis were defined as those in which the lesion could reasonably be identified by grey-scale data. The outline of the cerebellar hemisphere was determined and drawn manually in five cerebellar slices with higher activity.

Figure 3 shows an example of computer analysis for GSM evaluation on the CCD lesions. From the value obtained from the representatives of A and U cerebellar hemisphere (Figure 3), the interhemispheric relative uptake difference was calculated by assessing the asymmetry index (AI) between the U and the A in the 42 patient as the follow equation, which was used by Kim and Lee [25]. For instance, Mr Shaw FW, one of the cases, whose GSM values of both hemispheres of the cerebellum were measured as 139 (U) and 73 (A), separately. With both U and A values known, AI could be calculated.

$$\mathrm{AI} = [(\mathrm{U} - \mathrm{A})/\mathrm{U}] \times 100\% = (139 - 73)/139 = 47\%$$

Testing of postural sway

Testing of postural sway was performed using a CATSYS platform system (Danish Product Development Ltd, Copenhagen, Denmark). Postural sway was measured by asking the patient to stand quietly, upright and still, on a force platform at ground level for 60 seconds. The postural sway test was performed with eyes open and again with eyes closed to determine the effectiveness of visual feedback on the postural parameters. The values of sway intensity and velocity were recorded via the force platform and the mean of those obtained from three trials were compared between groups.

The sway parameter definitions were previously defined [27,28]. In brief, sway intensity was defined as the root mean square of acceleration. Sway velocity was defined as the average travel speed of the centre of pressure, calculating by dividing the total length of the trajectory of the centre of

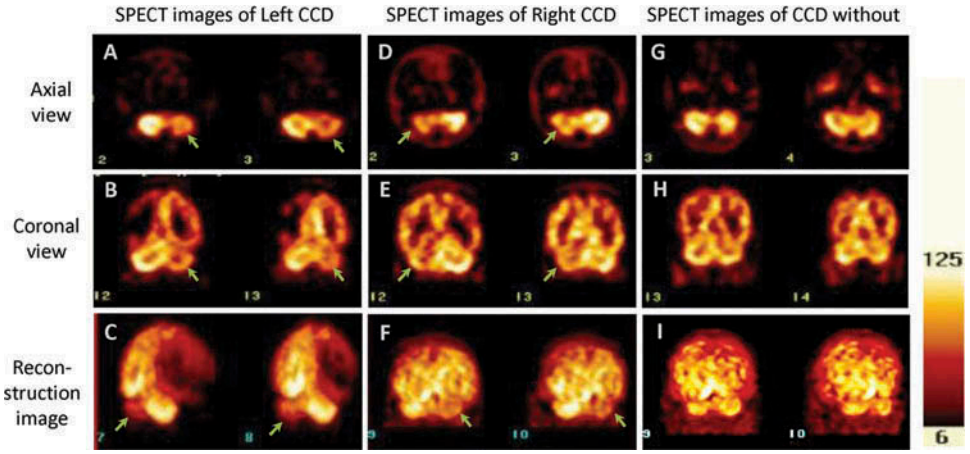

Figure 2. Representative single-photon emission computed tomographic images of a single subject in each group obtained from two consecutive axial and coronal planes and 3D reconstructed images. The representative patient with left CCD shows reduced perfusion in the left cerebellar hemisphere (indicated by arrows in (*a–c*)). The representative patient with right CCD shows reduced perfusion in the right cerebellar hemisphere (indicated by arrows in (*d–f*)). No abnormality is visible on either side of the cerebellum in the representative images of the patient without CCD (*g–i*).

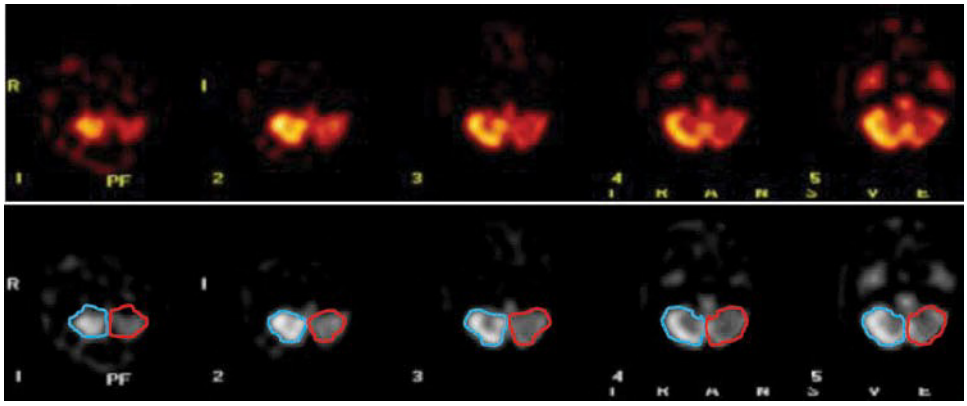

Figure 3. SPECT images showing representative slices of the cerebellum in one patient with left crossed cerebellar diaschisis (CCD). SPECT images of the five slices of cerebellum before grey-scaling (upper panel). After converting the colour images to grey-scale, the regions of interest (ROIs) of grey-scale median values were obtained on sections of axial scan (lower panel). The ROIs were symmetrically drawn in both cerebellar hemispheres on the selected slices.

pressure by the recording period length (mm s^{-1}). Sway intensity and velocity are the most sensitive and reliable measures of standing steadiness. They have been used successfully to observe changes in postural control between patients with their eyes open or closed [27,29,30].

Ethics statement

The human ethics committee of the medical centre approved this study (TSGHIRB No.: 2-102-05-135), which conformed to the principles of the Declaration of Helsinki. With the approval of the Institutional Review Board, informed consent was waived due to the retrospective nature of the study.

Statistics

The differences in patient characteristics were compared across the three groups in those 42 patients with the use of analysis of variance or the Chi-squared test. The generalized estimating equations (GEEs) were used to determine the associations of various effects (group, age and eyes open/closed) in the three groups and to adjust the correlations between repeated measurements in the same subjects obtained under different conditions (eyes open vs eyes closed). In addition, this study adjusted for potential confounders (age, gender and interval duration). A p-value of 0.05 or less indicated a statistically significant difference. Contrast estimates were calculated after establishing the final GEE models for sway intensities and velocities. All data analyses were performed using the PROC GENMOD function of SAS version 9.13 (Carry, NC).

Results

This study retrospectively reviewed the data of 155 consecutive patients. It excluded 113 patients who had clinical neurological comorbidities (n = 64) and inappropriate data regarding SPECT including blurred images of brain perfusion SPECT due to a patient's intolerance or uncooperative behaviour during an examination (n = 3); cerebellar diaschisis ipsilateral to the supratentorial hemispheric ischaemia (n = 1); and significantly decreased tracer uptake within the bilateral cerebellum (n = 1). In addition, 44 patients were excluded due to insufficient or incomplete data for quantitative measurement of the postural sway testing due to the inability to independently stand, to understand and follow simple verbal instructions and presence of serious hearing disorder (n = 26); unreliable postural sway testing data due to having orthopaedic and other postural control influencing diseases, such as arthrosis or total hip joint replacement (n = 11); the interval between the SPECT scan and the investigation of postural sway larger than 3 weeks (n = 7) (Figure 1). Finally, 42 patients included with a diagnosis of ischaemic stroke completed clinical charts that fit the inclusion criteria. The interval between stroke onset and the time of the postural investigation ranged from 0.5–1.5 years, which was measured from the included 42 patients. The interval between the SPECT scan and the investigation of postural sway ranged from 10 days to 3 weeks. The selected sample consisted of 35 men and seven women, aged 42–79 years (mean = 56 ± 13.61 years) (Table I). Forty patients were right-handers and two were left-handers. The infarct locations were comparable between right and left hemispheres of the brain.

After the adjustment for gender, it was found that CCD (left vs none) was positively significantly associated with sway intensity (β = 2.70, p = 0.0052) (Table II) and sway velocity (β = 8.18, p = 0.0010) (Table III), respectively. Eye condition (open vs closed) and stroke onset-test interval in years were negatively and significantly associated with sway intensity (β = −1.62, p = 0.0004; β = −0.03, p = 0.0001) (Table II) and sway velocity (β = −2.78, p < 0.0001; β = −0.03, p < 0.0001) (Table III), respectively. In addition, patient age was positively and significantly associated with sway intensity (Table II, β = 0.09, p = 0.0365) but not sway velocity (Table III, β = 0.17, p = 0.0577).

An increase of 1 year in age increased sway intensity by 0.09 units (Table II). Left CCD sway intensity was 170% (Table UU) greater than that of no CCD and left CCD sway velocity was 718% (Table III) greater than that of no CCD. However, the difference was not significant between right CCD and no CCD for sway intensity (Table II) and sway velocity (Table III). Moreover, sway intensity and sway

Table I. Demographic characteristics of the study groups.

	Left CCD	Right CCD	No CCD	*p*-value
Male sex, *n* (%)	13 (86.7)	11 (84.6)	11 (78.6)	0.20
Mean (± SD) age at stroke onset, years	59.1 ± 13.5	61.3 ± 13.6	60.3 ± 12.7	0.09
Side of hemiplegia/hemiparesis, *n* (%)				0.44
Left	15 (100)	0	6 (42.9)	
Right	0	13 (100)	8 (57.1)	
Handedness, *n* (%)	1:14	0:13	1:13	0.36
Left	1 (6.7)	0	1 (7.1)	
Right	14 (93.3)	13 (100)	13 (92.9)	
Stroke severity (based on modified Rankin scale), *n* (%)				0.22
1	0	0	1 (7.1)	
2	13 (86.7)	10 (76.9)	11 (78.6)	
3	2 (13.3)	3 (23.1)	2 (14.3)	
Co-existing conditions, *n* (%)				
Cardiovascular disease	4 (26.7)	3 (23.1)	4 (28.6)	0.75
Hypertension	13 (86.7)	10 (76.9)	12 (85.7)	0.57
Type 2 diabetes mellitus	5 (33.3)	5 (38.5)	5 (35.7)	0.92
Mean (± SD) cognitive function (Mini-Mental State Exam score)	26.0 ± 4.5	26.3 ± 4.2	26.2 ± 4.0	0.88
Brunnstrom stage	4.8 ± 0.6	5.3 ± 0.8	5.5 ± 0.3	0.62

SD, standard deviation.

p-values were derived from analysis of variance or the Chi-square test.

For the modified Rankin scale, a score of 0 indicates no symptoms, 1 no significant disability, 2 slight disability, 3 moderate disability, 4 moderately severe disability and 5 severe disability.

For the Mini-Mental State Exam, higher scores indicate higher cognitive function, with a score of 0–9 indicating severe impairment and a score of 25 or more indicating normal function.

Table II. Comparison of sway intensity (units) in persons with chronic stroke who had CCD or without.

Parameter	Estimate (β)	SE	95% CL		*Z*	*p*-value
Intercept	3.59	2.86	−2.03	9.19	1.25	0.2114
Patient age (years)	0.09	0.04	0.01	0.17	2.09	0.0365
CCD (left vs none)	2.70	0.97	0.81	4.60	2.79	0.0052
CCD (right vs none)	0.66	1.58	−2.43	3.76	0.42	0.6749
Eyes (open vs closed)	−1.62	0.45	−2.51	−0.73	−3.56	0.0004
Stroke onset-test interval (years)	−0.03	0.01	−0.04	−0.01	−3.86	0.0001

SE, standard error; CL, confidence limit.

The statistical model was assessed using the generalized estimating equations (GEEs) and was adjusted for age and group interaction effects.

velocity were 1.62- (Table II) and 2.78-times (Table III) lower with eyes open than eyes closed, respectively. Finally, with an increase of 1 year in the stroke onset-test interval, sway intensity and sway velocity both decreased by 0.03 units (Table II) and 0.03 mm s^{-1} (Table III), respectively.

Discussion

Postural sway is defined as the small movements made to maintain standing still on both feet. To the authors' knowledge, this is the first report to explore the relationships between these biomechanical parameters and CCD phenomena in a sample of patients with post-stroke hemiplegia. The results show that sway intensity and velocity were significantly increased in left CCD, to a declining extent over the years since the stroke. The results confirmed an association between lateralization of cerebellar perfusion and postural asymmetry in those patients. The results support the hypothesis that CCD impairs control of postural sway in resting upright postures. Both sway intensity and velocity influenced the function of postural sway when CCD appeared after ischaemic stroke. To the authors' surprise, the results showed no significant differences for sway intensity and velocity between right CCD and no CCD. Instead, sway intensity and velocity differed significantly between left CCD and no CCD. It was confirmed that left CCD impaired postural control and that a strong relationship existed between lateralization of cerebellar perfusion and postural asymmetry.

The clinical significance of the CCD phenomenon has been demonstrated in assessing functional deficits and stroke recovery [31,32]; however, some investigators believe that it has yet to be proven to be of clinical importance. Kim et al.

Table III. Comparison of sway velocity (mm s^{-1}) in persons with chronic stroke who had CCD or without.

Parameter	Estimate (β)	SE	95% CL		*Z*	*p*-value
Intercept	9.69	5.98	−2.04	21.42	1.62	0.1054
Patient age (years)	0.17	0.09	−0.01	0.35	1.90	0.0577
CCD (left vs none)	8.18	2.48	3.33	13.03	3.30	0.0010
CCD (right vs none)	0.14	2.78	−5.31	5.59	0.05	0.9607
Eyes (open vs closed)	−2.78	0.47	−3.69	−1.86	−5.95	< 0.0001
Interval (years)	−0.03	0.01	−0.05	−0.02	−4.45	< 0.0001

SE, standard error; CL, confidence limit.

The statistical model was assessed using the generalized estimating equations (GEEs) and was adjusted for age and group interaction effects.

[33] found that none of their patients with CCD showed clear clinical signs of cerebellar dysfunction, although they found that the frequency of CCD was significantly higher in patients with infarction in the frontoparietal lobes or the deep middle cerebral artery territory than in patients with infarction in other regions. In contrast, hemiataxia due to CCD after unilateral supratentorial lesion [24] or thalamic haemorrhage [34] has been reported. Mono-limb ataxia due to CCD was reported after a motor cortex infarction [33]. Except for ataxia, Watanabe et al. [35] studied 20 patients with hemiplegia and found that the degree of CCD differed significantly between Brunnstrom stages II and IV, III and IV and III and V. CCD in Brunnstrom groups II and III was considerably less severe than that of groups IV, V and VI. They concluded that the severity of hemiplegia and the degree of CCD were closely associated. This study found that left CCD increased postural sway, where the left cerebellum projected signal through thalamic relay nuclei to the right motor cortex; it is possible that this system is dominant for some motor functions, as seen with upright postural asymmetry control. Aphasia in the absence of gross hemiparesis can be related to CCD in metabolic disease [36].

Some data on lateralization of cerebellar perfusion may be obtained from studies of neuronal activity. Colombel et al. [37] studied lateralization in the cerebellar hemispheres of rats by observing behaviour in performing motor co-ordination tasks using a rotarod after unilateral removal of the cerebellar hemispheres. They found that worse performances occurred in rats with left hemisphere lesions; the motor deficits on the rotarod were more pronounced after left, as opposed to right, cerebellar damage. They concluded that motor functions lateralized in the left cerebellar hemisphere. Kapreli et al. [38] used functional MR imaging to investigate the existence of an analogous pattern during lower limb joints movements by performing repetitive knee, ankle and toe flexion/extension movements on the right and left sides. They demonstrated that the lateralization index was strongly affected by the anterior lobe of the cerebellum and others and the index increased from proximal joints to distal joints. In these studies, the left CCD group did significantly worse on all sway measures, thus indicating greater postural instability compared to the right CCD and no CCD groups. These findings suggest that patients with chronic stroke who had left CCD are at greater risk of falling than those with right CCD and no CCD, not only because of a reduced ability to maintain muscle strength on the paretic side, but also because of impaired balance function resulting from lateralization of cerebellar perfusion. Impairment of multi-segment or biased egocentric co-ordination of non-paretic and paretic limbs might be one of thhe contributing factors as well [6,39].

The results disclosed postural asymmetry, i.e. a significant difference in sway velocity and intensity in the left CCD group. The findings indicate that right cerebral infarction inducing left CCD impairs the control of posture sway, which could be linked to the symptomology of pusher syndrome. Pusher syndrome is a clinical disorder following cerebral lesions, in which patients actively push away from the non-hemiparetic side, leading to postural imbalance and its underlying mechanism is altered perception of body posture in relation to gravity [40]. The syndrome is more common in patients with strokes in the right hemisphere, so patients push themselves toward the left, an example of postural asymmetry. Interestingly, a brain lesion closely correlated with pusher syndrome occurs in the thalamus [41], which is also a crucial area for the pathomechanism of the CCD due to retrograde degeneration after destruction of the dentato-rubro-thalamo-cortical pathway. Pusher syndrome and left CCD with postural asymmetry share the same neuroanatomic structure, the thalamus, so one had to consider a strong correlation between two. Any link between CCD and pusher syndrome should be further explored.

The phenomenon of CCD is thought to be related to postural control because of the well-known role in balance that the cerebellum plays. CCD is a reflection of reduced neural activation/input into the contralateral cerebellum as a consequence of supratentorial injury. Theoretically, there is no difference between the two sides. These results, however, divulge a relationship that functional asymmetries within the cerebellum will show distinct postural sway control. Multiple lines of evidence suggest that cerebellar asymmetry correlates with cerebral asymmetry. For example, individuals with congenital focal lesions in the left cerebral hemisphere show a re-organized language network involving the left cerebellum [41]. In a study of functional asymmetry, Yan et al. [7] explained the three aspects of intra-task, inter-task and high-order asymmetries to reflect the functional organization of the human brain. They found that, in the finger-to-thumb task, functional asymmetries exist in the insula, temporal lobe, cingulate gyrus, basal ganglion and motor cortex, as well as the cerebellum. The observation that right hemisphere lesions with left cerebellar CCD produce bigger impairment may be a consequence of the higher likelihood of functional cerebellar asymmetry. The involvement of visual neglect in linkage between CD and postural asymmetry could be ignored based on exclusion criteria.

The relationship between handedness and left or right CCD has not yet been elucidated. Cerebellar volume asymmetry not only follows the same laterality pattern in the cerebral cortex, but is associated with handedness [42–44]. Rocca and Filippi [45] investigated brain function lateralization during action execution with the dominant and non-dominant feet in eight left-handers, compared with 13 right-handers who performed the same tasks with their right foot. They found that, in left-handers, the performance of simple motor acts with the dominant lower leg might be achieved through a complex adaptation and neuron– environmental interaction. Nevertheless, the number of left-handers in this study was only two, which was few to discuss their relationship in the study.

The main limitation of this study was its retrospective, cross-sectional design, without intervention and follow-up studies. Therefore, this study could not assess any long-term effects of an eligible intervention. The clinical details of the insult and precise information regarding the onset and evolution of CCD were sometimes not available. This was because such information was usually given by the patients or their relatives based on their memory. The sample size was small (n = 42), which might be the reason why this result of association in patient age and sway velocity failed to reach statistical

significance ($p = 0.0577$). The third was a lack of some clinical assessment tools related to balance measurements in combination with the platform system. Posture control is complicated, but only two parameters (sway intensity and velocity) were measured. Although not the primary objective, presentation of the severity of CCD and precise postural sway testing would have strengthened the study.

Conclusion

Measuring postural parameters in the upright standing position is a reproducible way of measuring postural sway. This study affirmed the relationship between postural asymmetry and lateralization of CCD perfusion. The patients with left CCD had significantly increased postural sway. These findings reinforce the expectation that lateralization of cerebellar perfusion exists in a large-scale neural network from the viewpoint of the functional asymmetries of postural sway.

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Declaration of interest

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