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Cerebellar stroke without motor deficit: Clinical evidence for motor and non-motor domains within the human cerebellum

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Abstract

Objective—To determine whether there are non-motor regions of cerebellum in which sizeable infarcts have little or no impact on motor control.

Experimental procedures—We evaluated motor deficits in patients following cerebellar stroke using a modified version of the International Cooperative Ataxia Rating Scale (MICARS). Lesion location was determined using MRI and CT. Patients were grouped by stroke location – Group 1, stroke within the anterior lobe (lobules I–V); Group 2, anterior lobe and lobule VI; Group 3, posterior lobe (lobules VI – IX; including flocculonodular lobe, lobule X); Group 4, posterior lobe but excluding lobule VI (i.e., lobules VII–X); Group 5, stroke within anterior lobe plus posterior lobe.

Results—Thirty-nine patients were examined 8.0 ± 6.0 days following stroke. There were no Group 1 patients. As mean MICARS scores for Groups 2 through 5 differed significantly (one-way ANOVA, $F(3,35) = 10.9$, $p = 0.00003$), post-hoc Tukey's Least Significant Difference tests were used to compare individual groups. Group 2 MICARS scores ($n = 6$; mean ± SD, 20.2 ± 6.9) differed from Group 3 ($n = 6$; 7.2 ± 3.8 ; $p = 0.01$) and Group 4 ($n = 13$; 2.5 ± 2.0 ; $p = 0.00002$); Group 5 ($n = 14$; 18.6 ± 12.8) also differed from Group 3 ($p = 0.009$) and Group 4 ($p = 0.00002$). There were no differences between Groups 2 and 5 ($p = 0.71$), or between Group 3 and Group 4 ($p = 0.273$). However, Group 3 differed from Group 4 when analyzed with a two-sample t-test unadjusted for multiple comparisons ($p = 0.03$). Thus, the cerebellar motor syndrome resulted from stroke in the anterior lobe, but not from stroke in lobules VII – X (Groups 2 plus 5, $n = 20$, MICARS 19.1 ± 11.2 , vs. Group 4; $p = 0.000002$). Strokes involving lobule VI produced minimal motor impairment.

Conclusion—These findings demonstrate that cerebellar stroke does not always result in motor impairment, and they provide clinical evidence for topographic organization of motor versus nonmotor functions in the human cerebellum.

Keywords

Cerebellum; ataxia; motor control; functional topography

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The notion that the cerebellum is devoted purely to the coordination of gait, extremity and oculomotor movement, and articulation has been deeply entrenched in medical and neurological texts. Evidence pointing to non-motor functions of the cerebellum (see Schmahmann, 1991) is beginning to alter this conventional wisdom. Recent findings include the description of the cerebellar cognitive affective syndrome in adults (CCAS; Schmahmann and Sherman, 1998) and children (Levisohn et al., 2000), the demonstration of reciprocal connections between cerebellum and cerebral association and paralimbic cortices (Schmahmann and Pandya, 1997; Kelly and Strick, 2003), and functional imaging studies (see Desmond and Fiez, 1998; Stoodley and Schmahmann, 2009) showing cerebellar activation in cognitive and emotional paradigms. These observations notwithstanding, some clinical neurologists and neuroscientists remain skeptical of a cerebellar contribution to functions beyond motor control.

We have proposed that there is topographic organization of function in the cerebellum such that sensorimotor function is represented predominantly in the anterior lobe (lobules I-V) with a second representation in lobule VIII; cognitive processing is subserved by the posterior lobe (lobules VI and VII in particular); and the cerebellar vermis and fastigial nuclei constitute the limbic cerebellum (Schmahmann, 1991, 1996, and 2004).

There is a time-honored tradition in clinical neurology of lesion-deficit correlation in order to derive new insights into the functions of cerebral cortical and white matter structures (e.g., Wernicke, 1874; Broca, 1878; Dejerine, 1892; Geschwind, 1965a,b) as well as of cerebellum in humans and animals (e.g., Luciani, 1891; Ferrier and Turner, 1893; Russell, 1894; Bolk, 1906; Holmes, 1930). We draw on this method here, using the tools of the neurological examination in patients with focal strokes to test our anatomical-functional hypothesis. If the traditional view that the role of the cerebellum is confined to motor control is correct, then acute stroke anywhere in cerebellum should, by definition, impair motor function. In contrast, if the topography hypothesis is correct, then there should be non-motor regions of cerebellum in which a sizeable infarct would have no impact on motor control. In this study we examined patients with cerebellar stroke, documented their motor impairments using an ataxia rating scale, and analyzed the relationships between motor scores and locations of the infarcts.

Experimental Procedures

Patient recruitment

We prospectively reviewed the clinical records of adults admitted to inpatient neurology in Partners Healthcare hospitals over a 4-year period, with the clinical and/or radiographic diagnosis of stroke involving cerebellum. Computer generated lists of admissions to the Massachusetts General Hospital neurology wards were monitored daily, and those with cerebellar stroke as part of the admitting diagnosis were screened for entry in the study. Patients with cerebellar stroke were also referred by residents and faculty of the Brigham and Women's Hospital and Newton Wellesley Hospital, and screened for possible inclusion in the study. Neuroimaging was performed as part of routine clinical service. MRI scans included diffusion weighted imaging (DWI), T1-weighted and T2-weighted sequences, and fluid attenuated inversion recovery sequencing (FLAIR). CT scans only were performed in some patients unable to undergo MRI. Screening of records and imaging was performed by JM, and appropriateness for the study was confirmed by JDS. Detailed evaluation of the neuroimaging, including identification of lobules and assignment to Groups, was performed by JDS after completion of the clinical study and without regard to the clinical findings.

Inclusion criteria

Patients ≥ 18 years of age were evaluated for inclusion into the study if there was neuroimaging evidence of recent stroke in the cerebellum, regardless of their clinical presentation.

Exclusion criteria

Patients were excluded if neuroimaging revealed 1) evidence of previous cerebral, brainstem or cerebellar infarcts, and 2) acute stroke outside the cerebellum involving the brainstem and/or cerebral hemispheres.

Assessment of cerebellar motor impairment

Patients eligible for the study were examined (JDS) on the ward at the time of their hospitalization. A number of patients had minimal motor impairments and were discharged from the hospital by the clinical house staff prior to being evaluated for this study; these subjects returned for examination in the outpatient clinic. This accounted for the time difference from stroke onset to examination between Groups. We set a cut-off time from stroke onset to examination at one month post-stroke.

Patients were evaluated using the Modified Cooperative Ataxia Rating Scale (Schmahmann et al., 2009; Table 1). This validated semi-quantitative 120-point rating scale for the assessment of ataxia is based upon the International Cooperative Ataxia Rating Scale (Trouillas et al., 1997; Storey et al., 2004). MICARS measures posture and gait, kinetic function of the arms and legs (appendicular dysmetria), speech disorders, and oculomotor impairment. Normal subjects score ≤ 4 (Schmahmann et al., 2009), and therefore patients with a MICARS score ≤ 4 were regarded as motorically normal.

This study was approved by the Partners Human Research Committee at the Massachusetts General Hospital, and all participants provided written, informed consent.

Lesion localization

After the clinical examinations were performed and the collection of data completed, the neuroimaging studies (MRI in 32 cases; CT scan in 7) were reviewed in detail and the location of the infarct was determined (JDS) with reference to the MRI Atlas of the Human Cerebellum (Schmahmann et al., 2000). The anatomical localization was performed at the conclusion of this 4-year study without reference to the MICARS score obtained in each subject. In most cases, 9 to 10 images of the cerebellum in the axial plane from superior to inferior were available for comparison with equivalent sections in the Atlas. For each case, we used the Atlas to identify the primary fissure that demarcates the anterior lobe from lobule VI, and the superior posterior fissure that separates lobule VI from lobule VII (Figure 1). Lobule VII comprises lobule VIIA, including the vermis regions VIIAf and VIIAt, and hemispheric regions crus I and II; and lobule VIIB at the vermis and hemispheres (Schmahmann et al., 2000). Lesioned areas were recorded on a standard template of axial sections of cerebellum derived from the Atlas (Figure 2). Blood vessel territory (superior cerebellar artery [SCA], anterior inferior cerebellar artery [AICA] and posterior inferior cerebellar artery [PICA]) was not used for lesion localization, as the territories are not lobule-specific, relative sizes and territories irrigated are not constant, and anastomoses may occur between terminal branches (Tatu et al., 1996).

The locations of the strokes in all 39 cases were also analyzed with respect to the degree of involvement of the deep cerebellar nuclei. This analysis was performed blinded to MICARS score or Group membership. Cerebellar nuclei are poorly visible on MRI, and not identifiable on CT. As determined with reference to the cryosection data in the MRI Atlas, however, the axial sections in this series that contain the cerebellar nuclei are sections 5 and 6 (from superior to inferior), equivalent to $z = -29$ and $z = -37$ in the Atlas. The fastigial and dentate nuclei are

identified in these horizontal sections in the Atlas with confidence, but the globose and emboliform nuclei interposed between the fastigial and the dentate, cannot be isolated from each other. We therefore regarded the globose and emboliform together as the interpositus nucleus. The involvement of the different nuclei in each case (fastigial, interpositus, dentate) was then examined by comparison of levels 5 and 6 on the MRI with the horizontal sections $z = -29$ and $z = -37$ in the MRI Atlas to determine whether the infarct included the expected location of the nuclei as defined in the Atlas. A measure of extent of involvement of the nuclei was determined: grade 1, minimal encroachment on any nucleus in either of the two levels; 2, clear involvement of any nucleus in one of the two levels; 3, clear involvement of any nucleus in both levels; 4, apparent complete involvement of the nuclei in both levels.

We also tested whether medial versus lateral location of the cerebellar stroke influenced the MICARS score. We used the coordinate system in the Atlas to identify the midline for each of the 9 horizontal sections, and then measured 10mm laterally in each direction from the midline. The cerebellum was thus divided into a medial versus a lateral zone for each hemisphere. Location of the stroke in the medial sector, the lateral sector, or both was then determined.

Data analysis

Our *a priori* approach was to divide the patients into 5 Groups by location of the lesion as identified on neuroimaging within different combinations of 3 clusters of cerebellar lobules. These clusters were (i) the cerebellar anterior lobe (lobules I – V); (ii) lobule VI; and (iii) the cerebellar posterior lobe and flocculonodular lobe without lobule VI (i.e., lobules VII – X).

This determination of the Groups was driven by the hypothesis of the study. Specifically, we wished to determine whether lesions in lobules I – V (Group 1) would result in characteristic cerebellar motor impairments as opposed to lesions in lobules VII – X (Group 4) that we predicted would not. We were unable to distinguish lobule X (flocculonodular lobe) from adjacent lobules in this study, and thus included lobule X with our evaluation of the posterior lobe. The literature is mixed regarding lobule VI. Connectional and physiological studies indicate that lobule VI is part of the motor system (Schmahmann and Pandya, 1997; Kelly and Strick, 2003), whereas fMRI (see Stoodley and Schmahmann, 2009) and fcMRI studies (Krienen and Buckner, 2009) suggest that it plays a role in cognition. We therefore defined Group 2 as having a lesion in the anterior lobe (lobules I – V) plus lobule VI; and Group 3 with lesion involving the remainder of the posterior lobe (lobules VII – X) plus lobule VI, to determine whether the addition of lobule VI adds to the motor deficit in either Group. Finally, to determine whether a lesion in lobules VI – X would compound the motor deficit from lesions of the anterior lobe, we included a group, Group 5, in which stroke was present in some part of all 3 clusters – namely, anterior lobe, lobule VI, and lobules VII – X.

Statistical methods

We used one-way analysis of variance (ANOVA) to determine whether the mean MICARS scores for the different groups were statistically significant or not. We then performed pairwise *post-hoc* comparisons of all groups using Tukey's Least Significance Difference test to determine which groups were different from each other. The utility of Group, age of patient, and time from stroke onset to examination as predictors of MICARS scores was assessed using multiple linear regression. Pairwise comparisons of mean MICARS scores between Groups were then made using two-tailed t-tests. Comparisons in 2x2 tables were made using Fisher's exact test. The null hypothesis of the same proportion of males as females in the population of eligible cases was evaluated using an exact binomial test. An ANOVA model was used with MICARS score as response and Group membership, nuclear involvement, and their interaction as factors, to test whether nuclear involvement is related to MICARS score. ANOVA was also

used to test whether there was a relationship between degree of involvement of the nuclei and MICARS score.

Results

Participants

The clinical records and neuroimaging findings of 110 patients with stroke that involved the cerebellum were evaluated. Of these, 39 patients (ages 50.8 ± 16.6 , range 20 to 83) met the inclusion criteria of stroke isolated to cerebellum with no prior neurological events clinically or on imaging, no ischemic deficit outside of cerebellum, and examination within one month of stroke onset (Figure 2).

Patients were examined (mean $\pm$ S.D.) 8.0 days $\pm$ 6.0 days following the stroke (range 1 – 30 days; mode 5 days). Craniotomy for prevention of herniation was necessary in Case 5, who was examined 11 days following surgery, and in Case 25, examined 12 days post-operatively.

There were more males than females in the cohort of 39 patients (M = 27 [69%], $p=0.01$). All 6 patients in Group 3 were male. Gender division was equal, however, in the patients in Groups 2 and 5 (65% male) and those in Group 4 (62% male).

Data analysis by lesion location

There were no patients in this cohort in whom the stroke was present exclusively rostral to the primary fissure, i.e., restricted to any part of lobules I – V of the anterior lobe. The number of patients in Group 1 was therefore 0. Table 2 shows the number of participants and MICARS scores for Groups 2–5.

The MICARS score means for the four Groups (2 through 5) were significantly different (one-way ANOVA, $F(3,35) = 10.9$, $p=3.4 \times 10^{-5}$). Pairwise post-hoc comparison of all Groups using Tukey's Least Significant Difference test was then performed. Four of the mean MICARS score pairings were significantly different: Group 2 differed from Group 3 ($p=0.01$) and from Group 4 ($p=0.00002$); and Group 5 differed from Group 3 ($p=0.009$) and from Group 4 ($p=0.00002$). The other two Group comparisons were not significant: Group 2 was not different from Group 5 ($p=0.71$); and Group 3 was not different from Group 4 ($p=0.273$). Note however, that a two-sample t-test for Groups 3 and 4 alone, unadjusted for multiple comparisons, did reach statistical significance ($T=2.8$, $p=0.03$). The discrepancy between this test and the corresponding *post-hoc* result is due to the fact that the S.D.s for the two groups (3.8 for Group 3 and 2.0 for Group 4) were substantially less than the residual ANOVA S.D. of 8.4 used in the Tukey LSD test.

The MICARS scores for patients with anterior lobe involvement (Groups 2 plus 5, $n=20$; 19.1 ± 11.2) differed from the mean MICARS scores in Group 3 ($n=6$; $p=0.0005$) and Group 4 ($n=13$, $p<0.0001$), in which the strokes spared the anterior lobe.

With respect to the impact on MICARS score of involvement of the deep cerebellar nuclei in addition to the location of the cortical stroke, 7 cases had no stroke in sections 5 and 6 ($z = -29$ and $z = -37$); and another 7 had stroke in these two levels but the infarct did not encroach on the nuclei but was confined to the cortex. The remaining 25 stroke cases involved the nuclei in addition to the cerebellar cortex. The degree of nuclear involvement was grade 1 in 6 cases; grade 2 in 3; grade 3 in 15; and grade 4 in 1. Based on an ANOVA model with MICARS score as response and Group membership, nuclear involvement, and their interaction as factors, the null hypothesis that nuclear involvement is unrelated to MICARS cannot be rejected ($F(4,35) = 0.17$, $P=0.95$). For strokes that involved the nuclei, there was no statistically significant effect

on MICARS score attributable to severity of the nuclear involvement (one-way ANOVA, $F(3,21) = 0.75$, $P = 0.53$).

In terms of the medial-lateral location of the stroke and the impact on the motor outcome, we observed that in 100% of the 39 cases the cerebellar stroke included regions both medial to, and lateral to, the 10mm dividing line that separates the vermal from the paravermal regions. Medial versus lateral location of the stroke therefore cannot be considered a significant factor in contributing to the different MICARS scores.

Contribution of patient age and time to examination

We examined whether patient age or interval between stroke and examination contributed to the MICARS results in this cohort by using a multiple regression model with Group, age and time to examination as covariates. After adjusting for age and time to examination, Group remained a highly significant predictor of the MICARS score ($p=0.005$), whereas age ($p=0.95$) and time to examination ($p=0.95$) did not. The adjusted R^2 indicated that 39% of the variability in the MICARS score was explained by the regression model. Removal of both age and time to examination from the model did not have a significant effect on the adequacy of the model ($F(2,32)=0.0043$, $p=0.996$). The simple regression model with Group as the only covariate had an adjusted R^2 of 42%, an improvement over the 39% for the more complex model. We therefore conclude that in the population of patients studied over this time course in this analysis there is no evidence to support the usefulness of age and/or time to examination as a predictor of MICARS score. There was also no difference in time to examination between patients in Groups 2 plus 5 (6.2 ± 6.5 days; $p=0.45$) and those in Group 3 (8.2 ± 5.1 days); but there was a difference in time to examination between patients in Groups 2 plus 5 and those in Group 4 (10.7 ± 4.9 days; $p=0.03$). Any attempt to adjust the p-values in this study for multiple comparisons, however, would render this result not statistically significant.

Clinical presentations by Group

Of the 39 patients, vertigo with nausea and emesis was present at onset in 30 (76.9%), nausea and emesis without vertigo in 3 (7.7%), and vertigo without nausea or emesis in 2 (5.1%). These symptoms did not differ according to Group (one-way ANOVA, $p=0.39$). Only 4 patients (10.3%) presented without any of these features – 2 had headache only (both in Group 4); 1 reported non-vertiginous dizziness with fatigue and arm clumsiness (Group 5); and 1 noted headache, tinnitus, dysarthria and ataxia (Group 2).

Thirteen of the 39 patients (33%) were motorically normal ($\text{MICARS} \leq 4$) at the time of examination. Twelve (92 %) of these patients were in Group 4 (stroke confined to some part of lobules VII – X). The remaining case was in a patient in Group 3 (lobule along with lobules VII – X). Five of the 13 motorically normal cases scored 1 out of possible 8 points on the oculomotor component of the MICARS; 2 had hypermetric saccades, 2 others had “very subtle” hypermetric saccade, and 1 had transient gaze evoked nystagmus. None of the 13 cerebellar stroke subjects with a normal MICARS score had infarction that involved the anterior lobe.

Discussion

In this study, 13 of 39 patients with cerebellar stroke (33.3%) examined 8.0 $\pm$ 6.0 days after onset of stroke were motorically normal, demonstrating none of the signs of the cerebellar motor syndrome characterized by gait ataxia, appendicular dysmetria, or dysarthria. The apparent dichotomy between those patients who were motorically impaired versus those who were motorically normal was accounted for by the location of the cerebellar lesion. In patients with motor findings the lesions involved the anterior lobe (lobules I – V). In patients with minor or absent motor findings, the lesions spared the anterior lobe and were confined instead to

lobules VII – X of the posterior lobe. Patients with infarction involving lobule VI along with lobules VII – X, but sparing the anterior lobe, had a minor degree of motor impairment. Additional involvement of the deep cerebellar nuclei did not impact the MICARS scores. All cases had some involvement of both medial and lateral cerebellar regions, and therefore medial vs. lateral location of stroke did not contribute differentially to total MICARS scores.

Patients in Group 4 (with no motor deficit) were examined a little later than patients in the other Groups. This occurred because these patients were healthy enough to be discharged prior to our reaching them in the hospital, and we had to call them back at their convenience. This fact notwithstanding, the time to examination did not prove to be significant in the regression analysis and thus in this cohort, there is not sufficient evidence to point to time as a significant factor contributing to Group differences in MICARS score.

Motor representation has been demonstrated repeatedly in the anterior lobe of the human cerebellum, particularly in lobules III through V, and to a lesser extent also in lobule VI of the posterior lobe, in clinical (Ackermann et al., 1992; Kase et al., 1993; Terao et al., 1996; Urban et al., 2003) and morphometric analyses of stroke patients (Schoch et al., 2006) and in functional imaging studies (Fox et al., 1985; Nitschke et al., 1996; Rijntjes et al., 1999; Urban et al., 2003; Grodd et al., 2005; Stoodley and Schmahmann, 2009). In contrast, the absence of motor impairment following strokes, particularly in lobule VII of the posterior lobe, has not received similar attention. Schoch et al. (2006) previously reported that motor deficits result from lesions of the “superior cerebellum”, and our findings are consistent with their observation. Schoch et al. noted, however, that lesions of the posterior cerebellum were not followed by significant motor impairment because (i) posterior lobe may be “merely involved” (i.e., not necessary) for motor control, and (ii) because “lesions including somatotopic representations in the posterior cerebellum may be followed by motor dysfunction only in the very early stage of the (stroke) disease” (Schoch et al., 2006). Our Group 4 patients with stroke in lobules VII – X who had normal MICARS scores were examined 10.7 $\pm$ 4.9 days after stroke, 2 weeks before the Schoch et al. acute stroke patients (n=20) who were examined on average 24 days post-stroke. We view the silence of the posterior lobe with respect to motor deficit following stroke from a different perspective.

Anatomical tract tracing studies help explain the present observations. The cerebellar anterior lobe and parts of lobule VI receive spinal afferents through the spinocerebellar tracts (Oscarsson, 1965), and are reciprocally interconnected with motor cortices via motor corticopontine projections (Brodal, 1978; Hartmann-von Monakow et al., 1981; Schmahmann et al., 2004) and through feedback to motor regions from cerebellar nuclei via thalamus (Thach, 1987; Kelly and Strick, 2003). The anterior lobe is also reciprocally linked with the medial and dorsal accessory nuclei of the inferior olivary complex, which in turn receive afferents from spinal cord (Brodal, 1981; Voogd, 2004). In contrast, cerebellar lobule VII is essentially devoid of connections with the motor cortex or spinal cord (Brodal, 1981; Voogd, 2004). It is linked instead in a reciprocal feedforward and feedback manner with cerebral cortical association areas – prefrontal cortex, posterior parietal cortex, superior temporal polymodal regions, cingulate gyrus, and posterior parahippocampal area (Schmahmann, 1991, 1996; Schmahmann and Pandya, 1997; Kelly and Strick, 2003). Further, lobule VII is reciprocally linked with the principal olivary nucleus that has minimal spinal cord input (Sugihara and Shinoda, 2004).

These anatomical findings in monkey receive support from functional connectivity magnetic resonance imaging (fcMRI) studies in humans that show a dichotomy between anterior lobe connections with motor related cortices but posterior lobe links with association areas in the prefrontal, posterior parietal and superior temporal regions (Krienen and Buckner, 2009), and from functional magnetic resonance imaging (fMRI) studies showing that sensorimotor tasks activate the anterior lobe and lobule VI to some degree, whereas cognitive paradigms activate

lobules VI and VII of the cerebellar posterior lobe (e.g., Blackwood et al., 2004; Frings et al., 2006; see Stoodley and Schmahmann, 2009). The clinical role of lobule VIII remains to be established. It has been identified as a second motor area in physiological (Snider and Stowell, 1944) and fMRI studies (Nitschke et al., 1996; Grodd et al., 2005), but it was not clearly implicated as a cause of motor incapacity in this clinical study or in that of Schoch et al. (2006), and it is recruited in some cognitive paradigms in fMRI studies.

The notion that the cerebellum is purely a motor control device is not supported by review of the stroke literature. Most previous studies have been described in terms of the affected blood vessel, but this approach is anatomically imprecise. Vascular territories are not invariant between different brains, and they do not respect lobular boundaries (Tatu et al., 1996). This is exemplified by the SCA territory that does not abruptly end at the primary fissure (e.g., Amarenco and Hauw, 1990). Indeed, none of our cases of stroke within the SCA territory were confined exclusively to the anterior lobe, but all involved lobule VI to some degree. AICA infarcts may damage pons, spinocerebellar tracts and vestibular nuclei (Amarenco et al., 1993; Roquer et al., 1998). PICA strokes may involve the inferior cerebellar peduncle as part of the lateral medullary syndrome (Amarenco et al., 1989; Barth et al., 1994; Chaves et al., 1994), and the present findings, as well as other (unpublished) cases we have examined, imply that it may be the infarct in the lateral medulla rather than involvement of the cerebellar posterior lobe that accounts for persistent motor incoordination in these patients. Reports of PICA stroke patients with severe ataxia and depressed level of arousal generally refer to those with edema and brainstem compression (Sybert and Alvord, 1975; Kase et al., 1993), thus providing no useful data on specific functions of the posterior lobe. Vertigo, nausea and emesis at onset of PICA stroke (involving lobules IX and X) are clinically indistinguishable from those of acute peripheral vestibulopathy, a point emphasized in prior reports (Duncan et al., 1975; Guiang and Ellington, 1977; Lee et al., 2006). Vertigo, even from peripheral vestibulopathy, impairs ambulation (Baloh and Honrubia, 1979), and therefore gait unsteadiness in this setting does not make the case that the cerebellar posterior lobe is involved in coordination of gait. Silent infarcts, or only subtle cerebellar findings from PICA strokes, have been described (Amarenco et al., 1989; Kumral et al., 2005) but this has not prompted the fundamental reconsideration of cerebellar function that now appears warranted.

Our study has clinical implications. The observation that a patient with a sizeable cerebellar stroke in the posterior lobe has no or minimal cerebellar motor signs is consistent with prior reports of a purely vestibular presentation of PICA stroke (Duncan et al., 1975; Lee et al., 2006). The unfortunate phenomenon of “fatal gastroenteritis” is now recognized; the case of the patient with abrupt onset of nausea and vomiting but no overt neurological findings mistakenly sent out of the emergency room with a diagnosis of gastrointestinal distress, only to return some hours later with cerebellar swelling and brainstem compression from the infarcted PICA territory.

Our results also provide support for theoretical notions regarding the wider role of the cerebellum (Leiner et al., 1986; Schmahmann and Pandya, 1989; Schmahmann, 1991, 1996, 2000; Middleton and Strick, 1994). The CCAS, characterized by executive, linguistic, visual-spatial and affective impairments, arises from lesions of the cerebellar posterior lobe but not the anterior lobe (Schmahmann and Sherman, 1998; Exner et al., 2004). In contrast, we show here that the cerebellar motor syndrome arises when lesions involve principally the anterior lobe, and to a lesser degree lobule VI, but not when the lesion involves lobule VII in the posterior lobe. It thus appears that there is a double dissociation between the motor cerebellum in the anterior lobe (lobules I – V), and the cognitive cerebellum situated within the posterior lobe (predominantly lobule VII). Lobule VI appears to occupy an intermediate role in this motor – nonmotor dichotomy, perhaps representing the cerebellar equivalent of a premotor or supplementary motor region.

We recognize certain limitations of our study. (i) Anatomical definition of cerebellar lobules is suboptimal on CT scan. For the purposes of our study we were able to use the MRI Atlas to identify the primary and superior posterior fissures on CT scan with confidence. (ii) The numbers of patients in Group 2 (infarction in lobules I–V plus lobule VI) and Group 3 (infarction in lobules VI – X) were small, limited to 6 in each Group. These numbers were nevertheless sufficient to demonstrate that the difference in MICARS scores between the Groups was significant. (iii) The same investigator (JDS) performed both the MICARS evaluations and the delineation of the anatomical lesions according to the MRI Atlas. In order to prevent inadvertent bias, the anatomical definitions were performed without reference to the MICARS score. Further, in many cases the detailed anatomical determination of lesion location was completed years after the patients were examined in this 4-year prospective study. This is nevertheless a potential limitation of the study that can be addressed in future investigations designed to challenge or replicate the present observations.

These limitations notwithstanding, the novel observation in this lesion-deficit correlation study that sizeable strokes in the cerebellum produced no appreciable motor deficit when the lesion avoided the anterior lobe (lobules I – V) provides clinical evidence in support of functional topography in the human cerebellum. We consider the significance of this finding both for clinical neurology, and for theoretical formulations of the wider role of the cerebellum.

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Figure 1.

A. Selected horizontal MRI images through the cerebellum with fissures and lobules identified. Inferior (posterior lobe) at top left, to superior (anterior lobe) at bottom right. Fissures are color coded: pink, precentral fissure (f.), between lobules I/II and lobule III; light green, preculminate f., between lobules III and IV; purple, intraculminate f., between lobules IV and V; red, primary f., between lobules V and VI; light blue, superior posterior f., between lobule VI and crus I; yellow, horizontal f., between crus I and crus II; dark blue, ansoparamedian f., between crus II and lobule VIIIB; dark green, prebiventer f., between lobules VIIIB and VIIIA; brown, intrabiventer f., between lobules VIIIA and VIIIB; mauve, secondary f., between lobules VIIIB

and IX. The posterolateral f. between lobules IX and X is seen at the vermis. (From Schmahmann et al., 2000).

B. Diagram of horizontal sections through cerebellum taken from the MRI images above, demarcating the anterior lobe (shaded black) rostral to the primary fissure (PF); lobule VI (shaded gray) between the primary and superior posterior fissures (SPF); and posterior lobe plus lobule X (not shaded) caudal to the superior posterior fissure.

Figure 2.

Imaging data acquired in the 39 patients with cerebellar stroke in this study, with MICARS score for each case, and anatomical Group as outlined in the text. Cerebellar outlines as in Figure 1B. Infarcted tissue is shaded black.

TABLE 1

The Modified International Cooperative Ataxia Rating Scale (MICARS). Tests and measures of severity derived from ICARS are shown in regular font; the 7 additional tests, which when added form the MICARS, are shown in bold font.

I. POSTURE AND GAIT DISTURBANCES

1. Walking Capacities

0: Normal

1: Almost normal naturally, but unable to walk with feet in tandem position

2: Walking without support, but clearly abnormal and irregular

3: Walking without support but with considerable staggering; difficulties in half turn

4: Walking with autonomous support no longer possible; the patient uses the episodic support of the wall for 10-meter test.

5: Walking only possible with one stick

6: Walking only possible with two special sticks or with a Stroller.

7: Walking only with accompanying person.

8: Walking impossible, even with accompanying person (wheelchair).

2. Gait Speed

0: Normal

1: Slightly reduced

2: Markedly reduced

3: Extremely Slow

4: Walking with autonomous support no longer possible

3. Standing Capacities, Eyes open

0: Normal: able to stand on one foot more than 10 seconds.

1: Able to stand with feet together, but no longer able to stand on one foot more than 10 seconds.

2: Able to stand with feet together, but no longer able to stand with feet in tandem position.

3: No longer able to stand with feet together, but able to stand in natural position without support, with no or moderate sway.

4: Standing in natural position without support, with considerable sway and considerable corrections.

5: Unable to stand in natural position without strong support of one arm.

6: Unable to stand at all, even with strong support of two arms.

4. Spread of feet in natural position without support, Eyes open

0: Normal (< 10 cm)

1: Slightly enlarged (> 10 cm)

2: Clearly enlarged (25 cm < spread < 35 cm)

3: Severely enlarged (> 35 cm)

4: Standing in natural position impossible

5. Body sway with feet together, eyes open

0: Normal (< 10 cm)

1: Slight oscillations

2: Moderate oscillations (< 10 cm at the level of head)

3: Severe oscillations (> 10 cm at the level of head), threatening the upright position

4: Immediate falling

6. Body sway with feet together, eyes closed

0: Normal (< 10 cm)

1: Slight oscillations

2: Moderate oscillations (< 10 cm at the level of head)

3: Severe oscillations (> 10 cm at the level of head), threatening the upright position

4: Immediate falling

7. Quality of sitting position

0: Normal

1: With slight oscillations of the trunk

2: With moderate oscillations of the trunk and legs

3: With severe dysequilibrium

4: Impossible

II. KINETIC FUNCTIONS

8. Knee-tibia test (decomposition of movement and intention tremor)

(Left and Right scored)

0: Normal

1: Lowering of heel in continuous axis, but the movement is decomposed in several phases, without real jerks, or abnormally slow

2: Lowering jerkily in the axis

3: Lowering jerkily with lateral movements

4: Lowering jerkily with extremely long lateral movements or test impossible

9. Action tremor in the heel-to-knee test (Left and Right scored)

0: Normal

1: Tremor stopping immediately when the heel reaches the knee

2: Tremor stopping < 10 seconds after reaching the knee

3: Tremor continuing > 10 seconds after reaching knee

4: Uninterrupted tremor or test impossible

10. Decomposition of leg movement (Left and Right scored)

0: Normal

1: Corners or edges on the circle

2: Markedly decomposed attempts at circle

11. Decomposition of leg tapping (Left and Right scored)

0: Normal

1: Slightly slow and irregular

2: Clearly slow and irregular

12. Finger-to-nose test: decomposition and dysmetria

(Left and Right scored)

0: Normal

1: Oscillating movement without decomposition of the movement

2: Segmented movement in 2 phases and/or moderate dysmetria in reaching nose

3: Segmented movement in more than 2 phases and/or considerable dysmetria in reaching nose

4: Dysmetria preventing the patient from reaching nose.

13. Finger-to-nose test: intention tremor of the finger

(Left and Right scored)

0: Normal

1: Simple swerve of the movement

2: Moderate tremor with estimated amplitude < 10 cm

3: Tremor with estimated amplitude between 10 cm and 40 cm.

4: Severe tremor with estimated amplitude > 40 cm

14. Finger-finger test (action, tremor and/or instability)

(Left and Right scored)

- 0: Normal
- 1: Mild instability
- 2: Moderate oscillations of finger with estimated amplitude < 10 cm
- 3: Considerable oscillations of finger with estimated amplitude between 10 and 40 cm
- 4: Jerky movements > 40 cm of amplitude

15. Pronation-supination alternating movements(Left and Right scored)

- 0: Normal
- 1: Slightly irregular and slowed
- 2: Clearly irregular, and slowed movement, but without elbow sway
- 3: Extremely irregular, and slowed, but with sway of the elbow
- 4: Movement completely disorganized or impossible

16. Rebound of the arms (Left and Right scored)

- 0: None
- 1: Less than 10 cm
- 2: Greater than 10 cm

17. Overshoot of the arms (Left and Right scored)

- 0: None
- 1: Less than 10 cm
- 2: Greater than 10 cm

18. Drawing of Archimedes' spiral on a predrawn pattern

- 0: Normal
- 1: Impairment and decomposition, the line quitting the pattern slightly, but without hypermetric swerve
- 2: Line completely out of the pattern with recrossings and/or hypermetric swerves
- 3: Major disturbances due to hypermetria and decomposition
- 4: Drawing completely disorganized or impossible

III. SPEECH DISORDERS

19. Dysarthria: fluency of speech

0: Normal

1: Mild modification of fluency

2: Moderate modification of fluency

3: Considerable slow and dysarthric speech

4: No speech

20. Dysarthria: Clarity of speech 0: Normal

1: Suggestion of slurring

2: Definite slurring, most words understandable

3: Severe slurring, speech not understandable

4: No speech

21. Dysarthria: Alternating syllables

0: Normal

1: Slightly irregular

2: Clearly irregular, dysrhythmic and slurred

IV. OCULOMOTOR DISORDERS

22. Abnormal eye movements at rest

0: Absent

1: Present

23. Gaze-evoked nystagmus

0: Normal

1: Transient

2: Persistent but moderate

3: Persistent and severe

24. Abnormalities of the ocular pursuit

0: Normal

1: Slightly saccadic

2: Clearly saccadic

25. Dysmetria of the saccade

0: Absent

1: Bilateral clear overshoot or undershoot of the saccade

26. Saccadic intrusions into vestibulo-ocular reflex cancellation

0: Absent

1: Present

Table 2
Data grouped by anatomical location of lesion, and MICARS scores for patients in these Groups.

	Group 1	Group 2	Group 3	Group 4	Group 5
Lobules involved	I-V only	I-V plus lobule VI	VII-X plus lobule VI	VII-X only	I-X
Number of cases	0	6	6	13	14
MICARS-TOTAL (120)	0	20.2 +/- 6.9	7.2 +/- 3.8	2.5 +/- 2.0	18.7 +/- 12.8
GAIT (34)	0	6.8 +/- 5.0	3.8 +/- 3.9	1.2 +/- 1.1	8.4 +/- 7.4
ARM (44)	0	7.7 +/- 2.3	2.2 +/- 1.5	0.8 +/- 1.2	4.4 +/- 4.0
LEG (24)	0	3.0 +/- 2.4	0.7 +/- 1.0	0.2 +/- 0.4	2.3 +/- 1.9
SPEECH (10)	0	1.3 +/- 1.6	0	0	1.1 +/- 1.4
OCCULOMOTOR (8)	0	1.3 +/- 1.4	0.5 +/- 0.5	0.4 +/- 0.5	2.6 +/- 1.4