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Crossed Cerebellar Diaschisis in Cerebral Infarction: Correlation of SPECT and Clinical Features†

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= Abstract = **Patients with supratentorial cerebral infarction frequently show depressed metabolic activity in the contralateral cerebellar hemisphere which is known as crossed cerebellar diaschisis(CCD). In order to investigate the relationship between this phenomenon and the characteristics of the supratentorial lesion, we retrospectively evaluated the findings of ^{99m}Tc-HMPAO single photon emission computed tomography (SPECT) in 26 patients with a single supratentorial infarction lesion. A cerebellar asymmetry index (AIcbll), percent difference between both cerebellar hemispheres(Δ%cbll), SPECT volume deficit (SVD), and magnetic resonance volume deficit (MVD) were quantitated. A CCD, defined as AIcbll 〉12%, was observed in 12 of the 26 patients (46. 2%). No correlation was found between the Δ%cbll and duration of disease, SVD, or MVD. SVD and MVD values showed no significant difference between CCD positive and negative groups (71±47ml vs. 70±68ml and 90±84ml vs. 67±77ml, respectively). Patients with frontoparietal lobe or deep middle cerebral artery territory infarctions showed a significantly higher incidence of CCD and lower Δ%cbll values. Patients with severe hemiparesis had a higher incidence of CCD and lower Δ%cbll values than those with milder or no hemiparesis (incidence, 5/5 vs. 6/18, p=0.008; Δ %cbll,-21. 4±3.8% vs -8. 3±11. 1%, p=0. 014). None of the 12 patients with CCD showed clinical signs of cerebellar dysfunction. In conclusion, the location rather than the extent of the lesion appears to be the major determinant for the occurrence and magnitude of CCD in stroke patients.**

Key Words: *Crossed cerebellar diaschisis, Cerebral infarction, SPECT, ^{99m}Tc-HMPAO*

INTRODUCTION

Depressed metabolic activity in the cerebellar hemisphere contralateral to a supratentorial infarction was first described by Baron *et al.* (1980 a,b, 1981 a) using positron emission tomography (PET) and the steadystate oxygen-15 technique. This phenomenon, termed "crossed cerebellar diaschisis" (CCD), was interpreted as being the metabolic counterpart of depressed

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cerebellar function caused by the interruption of the corticopontocerebellar pathway at the supratentorial level (Baron *et al.* 1980 a,b). Subsequent PET studies showed that CCD affects both oxygen consumption and glucose utilization (Lenzi *et al.* 1982; Heiss *et al.* 1983; Martin and Raichle. 1983; Beron *et al.* 1984; Kushner *et al.* 1984; Patronas *et al.* 1984; Patano *et al.* 1986). It was also observed in studies of cerebral perfusion using single photon emission computed tomography (SPECT) (Biersack *et al.* 1984; Meneghetti *et al.* 1984; Cesaro *et al.* 1985; Brott *et al.* 1986; Pantano *et al.* 1987; Vorstrup 1988; De Roo *et al.* 1989; Holman *et al.* 1989).

All these reports are in general agreement that CCD occurs frequently after cerebral hemispheric lesions. However, the relationship between CCD and the size and degree of the hypoperfused supratentorial lesion or its anatomical location remains unclear. Moreover, the time-course of CCD and its clinical correlates are not yet defined There have been no studies where the relationship between radiologically measured infarct size or the degree of cerebral hypoperfusion and the presence of CCD has been evaluated.

We assessed CCD in a series of patients having unilateral supratentorial infarction by quantitating both the degree of their cerebral and cerebellar hypoperfusion using SPECT and the size of their cerebral infarction using magnetic resonance (MR) imaging.

MATERIALS AND METHODS

Patients

The population in this study consisted of 26 patients (range between 34 and 77 years, with a mean age of 55 years; 18 males and 8 females) presenting consecutively with a single supratentorial infarction in the internal carotid artery territory. Patients with clinical or radiological evidence suggesting ischemic or other cerebellar abnormalities were excluded from the study. None of the patients included had either a second symptomatic neurologic event since the initial event or alterations of consciousness.

Patients were evaluated retrospectively by MR, SPECT and a neurologic examination. All the patients underwent these examinations during a 3-day period. The time interval between the onset of the symptom(s) and the SPECT study ranged between 5 days and 6. 2 years. Each of the following factors was determined for each patient to examine its relationship with CCD; (1) the time period between the onset of clinical symptoms and the SPECT study, (2) the severity of cerebral hypoperfusion evaluated by SPECT imaging, (3) the size and anatomical location of the cerebral infarction evaluated by MR imaging, (4) the severity of motor impairment, and (5) the clinical signs of cerebellar dysfunction (Table 1). Motor impairment was graded according to the Medical Research Council Scale of Muscular Strength (Medical Research Council 1983), with a 0 indicating no contraction visible, 1 indicating only a flicker or trace of contraction, 2 indicating active movement at a joint possible with gravity eliminated, 3 indicating active movement possible against gravity, 4 indicating active movement possible against both gravity and resistance, and 5 indicating normal strength. The SPECT study was also conducted in an age-matched control group of 13 volunteers (age range 31-72 years, mean age 52 years) having no history of neurologic disease, psychiatric illness, or vascular risk factors.

Imaging Procedures

SPECT imaging was performed 10-60 minutes after intravenous administration of 740MBq (20mCi) of 99mTc-hexamethylpropylene amine oxime (HMPAO). Using a rotating gamma camera (ROTA ZLC 75. Siemens) interfaced to a dedicated computer system (MicroDELTA, Siemens) and a low-energy high resolution collimator during a 360 rotation, 60 frames of images with a 64x64 matrix were acquired within 30 minutes. 3.6 to 4 million total counts were collected for each study. The transaxial slices were reconstructed by filtered back projection using a Butterworth filter. Each reconstructed slice was corrected for tissue absorption

employing Chang's method (Chang 1978). All slices were 0.625 cm (1 pixel) thick. The SPECT pixel size was 0.625 cm in all three dimensions.

The MR images were obtained on a 0.5-T superconducting unit (Supertec-5000, Goldstar Seoul) in 10 patients and on a 2.0-T superconducting unit (Spectro-20000, Goldstar, Seoul) in 16 patients. The slice thickness/gap was 7mm/2mm for the 0.5-T unit and 5mm/2mm for the 2.0-T unit. The acquisition matrix was 256×256, with a spatial resolution of 1mm×1mm.

Data Analysis

Three consecutive SPECT image slices representing the cerebellum were added to construct a 1.875 cm thick slice. The region of interest (ROI) was created on each cerebellar hemisphere. From the total counts obtained from each cerebellar hemisphere, the following two indices were calculated (Pantano *et al.* 1986); (1) the cerebellar asymmetry index between the right(R) and left(L) cerebellar hemispheres (AIcbll) = R-L / (R+L) x 200, and (2) the percent difference between the contralateral (CCH) and ipsilateral (ICH) cerebellar hemispheres (Δ%cbll)=(CCH-ICH) / ICH x 100. The AIcbll assesses the degree of cerebellar asymmetry irrespective of which side is abnormal, and was used to obtain control values and to determine the significance of the cerebellar asymmetry. A CCD was considered to be present when the AIcbll was greater than the upper limit of 95% confidence interval defined in the age-matched control group, that is more than 12%. The Δ%cbll, on the other hand, expresses the direction of the asymmetry (e. g., it will be negative if CCD is present), and was employed to explore the relationship between CCD and the patient factors as stated above.

To quantitate the severity of the cerebral hypoperfusion, we measured the flow deficit volume from the SPECT image using the method described by Mountz (1989) and Mountz *et al.* (1989). ROIs were drawn around the entire lesion in the involved hemisphere, and around any involved, immediately subjacent brain tissue. The margin of the lesion was defined by the locations where tracer uptake values (count/pixel) around the lesion increased to within 10% of those in the contralateral region of the uninvolved hemisphere. The ROI of each lesion was then drawn as the exact mirror image so that the left side would become the right side and vice versa. This mirror ROI was applied to the cerebral area of the corresponding zone in the uninvolved contralateral side and the counts within both ROIs were measured. In this fashion, a SPECT volume deficit (SVD) in milliliters was determined using the equation: VT = VP x [(Mi-Si) /Mi] x Pi, where VT is the total volume (ml) deficit of the lesion, VP is the volume (ml) of the individual pixel, Si represents the single photon emission counts within the mirrored region of stroke, Mi represents the single photon emission counts within the circumscribed region in the uninvolved hemisphere, and Pi is the number of pixels in the region of interest; the sum of i is taken from all of the scan planes showing a diminution of tracer uptake in the hemisphere containing the stroke. ROIs were also assigned to the entire hemisphere of the involved side to determine the hemispheric volume. The %SVD was defined by the equation: SVD x100 /involved side's hemispheric volume.

The magnetic resonance volume deficit (MVD) was defined as the total volume (ml) of infarcted tissue visible on T2 weighted (2000-3000 /80-100, TR/ TE) MR images as determined by circumscribing all visible areas of high signal intensity in each involved plane and then summing the single-plane volume deficits. The %MVD was defined using MR images. A zone of high signal intensity was clearly visible and sharply demarcated from the normal-appearing brain. The lesion's perimeter was drawn by visually inspecting each MR slice. The product of the area of the lesion calculated by a computer and the thickness of the tomographic slices were used as the volume of the defect in each slice, then was added for all slices demonstrating the lesion as the total MRI volume of the deficit(MVD).

Statistical analysis

Data was expressed as a mean±standard deviation. The Mann-Whitney U test was used to assess differences between two groups regarding Alcbll or Δ%cbll. The significance of the difference in the frequency of CCD between the groups was determined using the chi-square test. Correlations between Δ%cbll and SVD, %SVD, MVD and %MVD were made by regression analysis. A probability value of less than 0.05 was considered significant.

RESULTS

All of the patients' experimental data are summarized in Table 1. The Alcbll increased significantly in the stroke group, as compared to the controls'(14.4±10.6% and 8.2±5.8%, respect-

Table 1. Clinical, SPECT and MRI findings of individual patients

	Sex/age (years)	Days after infarction	SVD(ml) /%SVD	MVD(ml) /%MVD	Infarct location	Muscular strength grade#	Δ%cbll	Cerebellar signs
1	M/65	11	15/1.9	7.6/0.78	P	5	4.7	–
2	M/34	32	40/4.4	NA	PT	5	13.0	–
3	M/55	2250	152/16	194/20	FP	〈5+	−6.4	–
4	F/47	563	18/1.9	1.3/0.10	IC	5	−7.3	–
5	F/40	1278	45/5.1	34/3.6	PO	4	−1.6	–
6	M/64	34	23/2.4	4.1/0.10	BG	5	−8.4	–
7	M/40	55	246/24	158/22	PO	5	−8.8	–
8	F/55	518	93/12	36/4.2	O	5	−5.1	–
9	F/63	790	83/10	52/8.1	T.BG	5	2.2	–
10	M/64	67	NA	232.3	PO	4	−6.3	–
11	M/52	60	22/2.6	1.6/0.23	BG	〈5"	−9.3	–
12	M/36	1182	49/5.1	82/7.9	T.BG.IC	4	0.9	–
13	M/45	660	54/6.3	NA	BG	5	15.7	–
14	M/63	8	NA	201/23	FPT	5	−3.3	–
15	M/60	5	85/10	35/3.8	FP	5	−16.9	–
16	M/62	12	3.2/0.46	NA	BG	5	−14.3	–
17	M/44	1825	127/14	42/6.8	FP.BG	5	−14.6	–
18	M/77	111	103/11	127/14	FPT	5	−30.5	–
19	M/52	25	49/5.4	11/1.2	FP	3	−21.3	–
20	M/57	12	123/12	234/21	FT.BG	2	−26.4	–
21	F/62	498	111/13	186/22	FP	4	−19.2	–
22	M/71	9	66/8.2	167/19	PT	5	−28.8	–
23	M/55	6	2.8/0.29	2.6/0.31	IC	4	−18.7	–
24	F/59	20	47/6.4	90/11	BG.IC	1	−23.5	–
25	M/53	300	116/11.9	NA	FPT	〈5"	−25.3	–
26	F/63	17	17/2.0	11/1.2	IC	3	−16.7	–

Graded according to the Medical Research Council Scale; for details, see "Patients and Methods".

" Hemiparesis present, but grade unknown.

– indicates the presence of crossed cerebellar diaschisis based on SPECT cerebellar asymmetry index.

SVD, SPECT volume deficit; MVD, magnetic resonance deficit; Δ%cbll, percent difference between the contralateral and ipsilateral cerebellar hemispheres on SPECT image; F, frontal; P, parietal; T, temporal; O, occipital lobe; BG, basal ganglia; IC, internal capsule; NA, data not available.

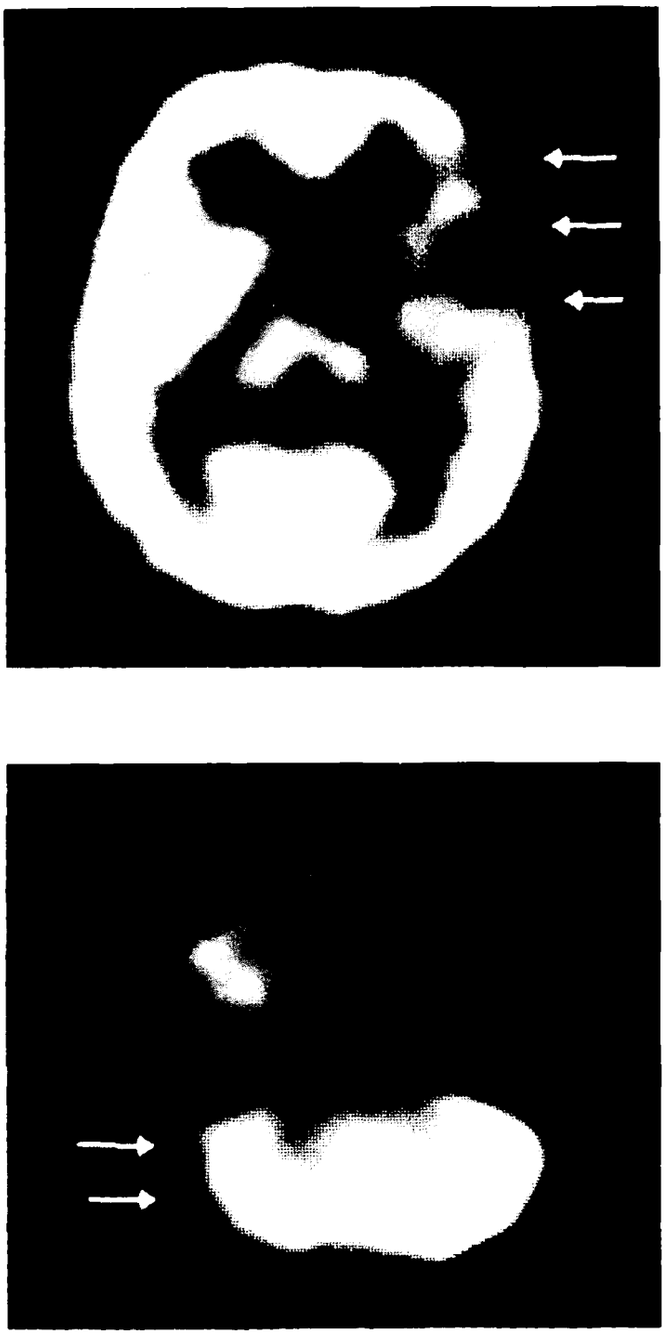

Fig. 1. Single photon emission tomographic images of a patient with cerebral infarction and crossed cerebellar diaschisis. (a) Hypoperfused supratentorial lesion in left frontotemporal area (arrows), and (b) hypoperfusion in contralateral cerebellum (arrows).

ively, p<0.001). Figure 1 demonstrates SPECT images of a typical patient with supratentorial infarction and crossed cerebellar diaschisis. CCD was observed in 12 of the 26 patients (46.2%) with cerebral infarction (Fig. 2). CCD

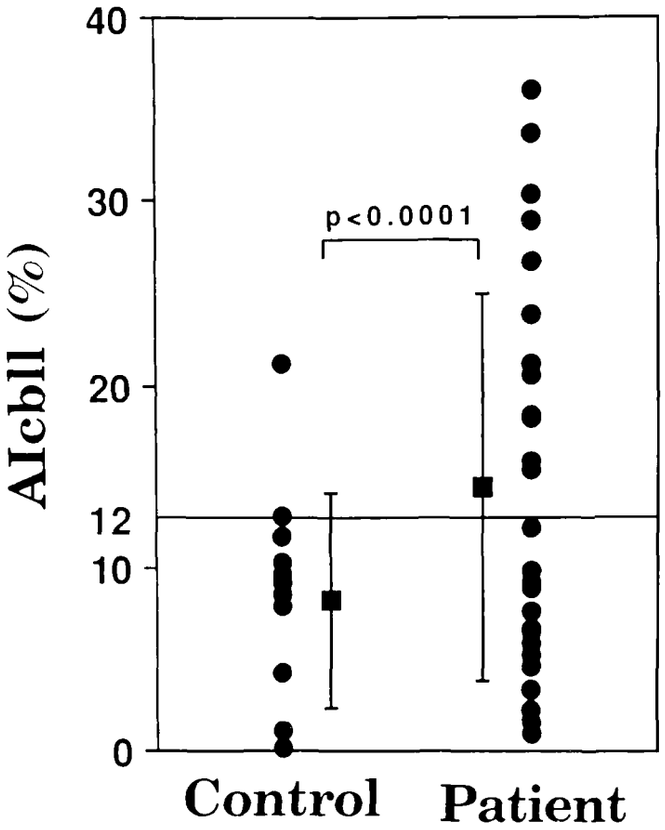

Fig. 2. Alcbll in 13 controls and 26 patients with cerebral infarction. Individual and mean (±SD) values are shown. A crossed cerebellar diaschisis (CCD) was considered to be present when the Alcbll was greater than the upper limit of 95% confidence interval defined in the controls (solid line).

was still present in 3 of the 10 patients more than 10 months after the clinical onset of the infarction.

No significant correlation was found between the Δ%cbll and the time lapse between the clinical onset and the SPECT study, not only for all 26 patients with cerebral infarction ($r = 0.26$) but also for the 12 infarction patients with CCD ($r = 0.35$) (Fig. 3). Furthermore, there was no significant difference in the Δ%cbll between the early (< 30 days) and late (> 30 days) studies of the stroke patients (-16.5 ± 10.3% and -8.3 ± 10.5%, respectively). However, the incidence of CCD was greater during the initial 30 days (8/10) than for the cases studied one month or more after the clinical event (4/16,

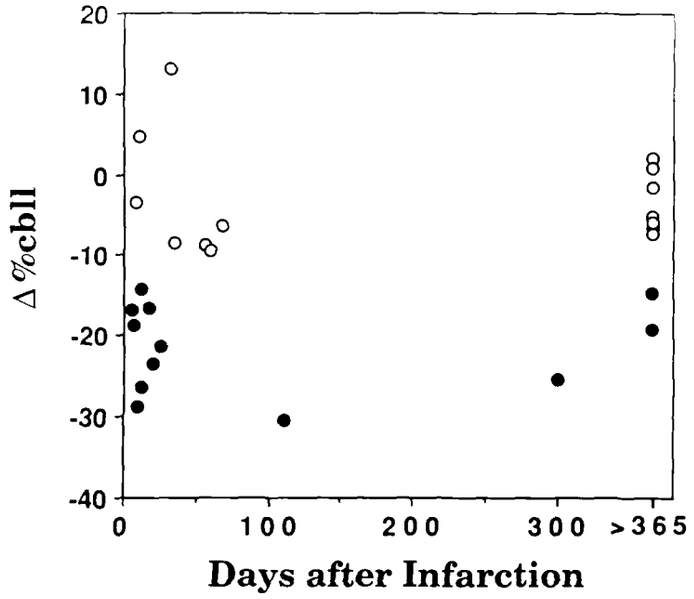

Fig. 3. Δ%cbll versus the duration after stroke in each of 26 patients with cerebral infarction. Values for the patients with CCD are shown as closed circles. There was no significant correlation between the Δ %cbll and the duration after infarction, not only for the 26 patients with cerebral infarction (r=0. 26) but also in the 12 infarction patients with CCD (r=0.35).

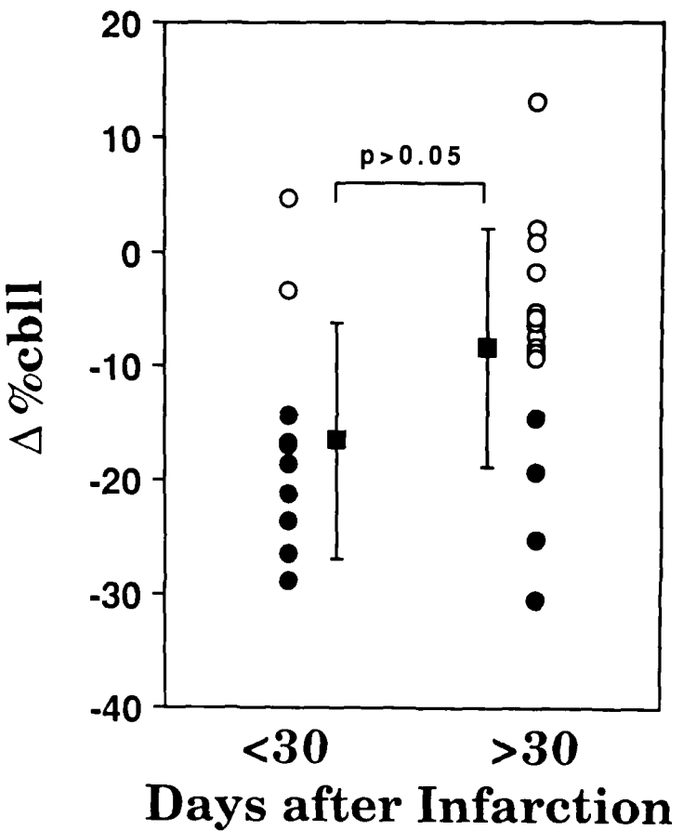

Fig. 4. Δ%cbll in the early (〈30 days after onset) and late (〉30 days) studies of the patients with cerebral infarction. Individual and mean (±SD) values are shown. Values for the patients with CCD are shown as closed circles. The incidence of CCD was greater during the initial 30 days (8/10) than for those cases studied one month ir after the clinical event (4/16) (p=0. 006).

p=0.06; Fig. 4).

As shown in Table 2, there was no significant correlation between either SVD and Δ %cbll or %SVD and Δ%cbll in both the 24 cerebral infarction patients and the 12 infarction patients with CCD. Even when the studies of the patients more than 30 days after stroke (i. e. beyond the period of the perfusion) were analysed separately, no correlation resulted. Also, neither MVD nor %MVD correlated significantly with Δ%cbll in the 22 patients with cerebral infarction. In addition, there were no differences between the patients with and without CCD, in terms of their SVD (71 ± 47ml and 70 ± 68 ml, respectively), %SVD (7.8 ± 4.9% and 7.7 ± 6.9%), MVD (90 ± 84ml and 67 ± 77ml) and %MVD (10 ± 8.7% and 7.7 ± 8. 9%) (Table 3). However, in 10 infarction patients with CCD, a significant inverse correlation was found between the MVD and the Δ %cbll (r=0.63, p=0.049). Also, the correlation between &MVD and Δ%cbll approached statistical significance (r= -0.60, p=0.070).

The frequency of CCD in patients showing a SVD larger than MVD (5/13) did not differ significantly from the incidence of CCD in patients showing a SVD that was nearly equal to or smaller than their MVD (5/7). Furthermore, both the difference between the SVD and MVD (SVD minus MVD) and the ratio of SVD to MVD showed no significant correlations to the Δ %cbll in the 12 stroke patients more than 30 days after the clinical event (r=0.22 and r=0. 06, respectively) (Table 2).

The frequency and severity of CCD according to the location of the supratentorial infarction are shown in Fig. 5. The frequency of CCD

Table 2. Correlation of Δ%cbll with SVD,%SVD, MVD, %MVD , SVD minus MVD, and SVD/MVD ratio in subgroups of patients

Correlation of Δ%cbll with	Total infarction patients			Patients〉30 days after infarction			Infarction patients with CCD		
	n	r	p	n	r	p	n	r	p
SVD	24	-0.18	〉0.05	15	-0.33	〉0.05	12	-0.37	〉0.05
%SVD	24	-0.17	〉0.05	15	-0.32	〉0.05	12	-0.34	〉0.05
MVD	22	-0.30	〉0.05	13	-0.36	〉0.05	10	-0.63	0.049
%MVD	22	-0.28	〉0.05	13	-0.37	〉0.05	10	-0.60	0.070
SVD-MVD				12	0.22	〉0.05			
BVD/MVD				12	0.06	〉0.05			

CCD, crossed cerebellar diaschisis: n, number of patients for whom the test was conducted: r, correlation coefficient: p, probability according to linear regression analysis: for other abbreviations, refer to Table 1.

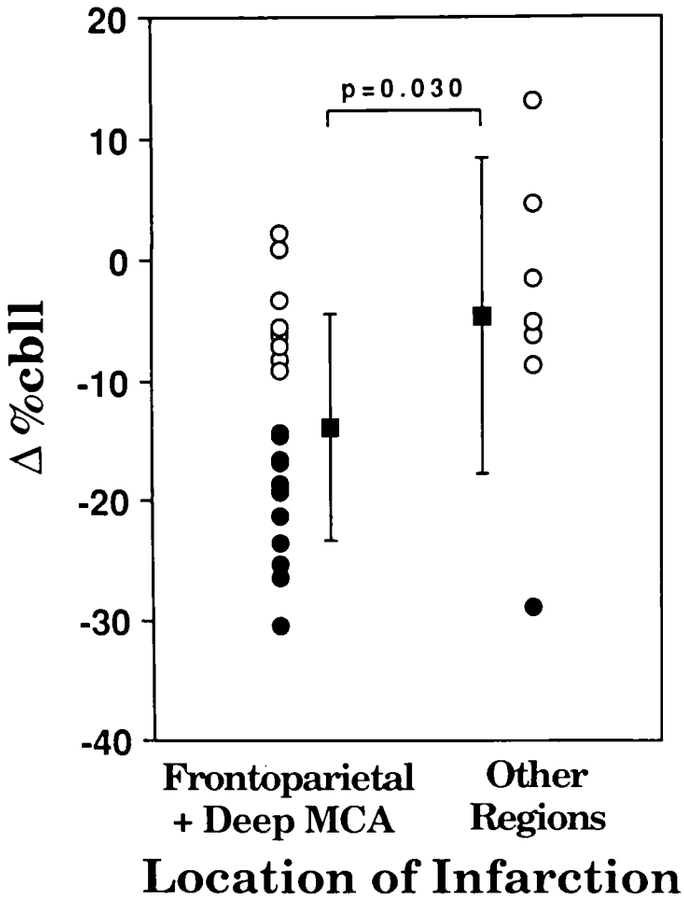

Fig. 5. Δ%cbll versus the location of the cerebral infarction. Individual and mean (±SD) values are shown. Values for the patients with CCD are shown as closed circles. The frequency of CCD was significantly higher in patients whose infarctions were in either the frontoparietal lobe or the deep MCA territory (11/19) than in those patients whose infarctions were in other regions (1/7) (p=0. 048).

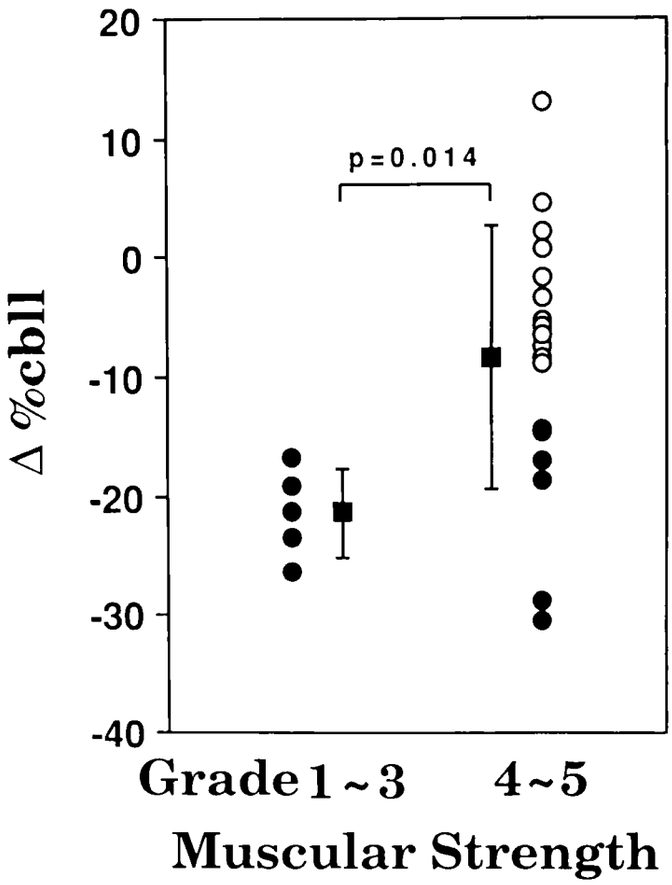

Fig. 6. Δ%cbll versus muscular strength in patients with cerebral infarction. Individual and mean (±SD) values are shown. Values for the patients with CCD are shown as closed circles. The frequency of CCD was significantly higher in patients having Grade 1,2 or 3 muscular strength (5/5) than in those patients with milder hemiparesis (6/18) (p=0. 008).

was significantly higher in patients whose infarctions were in either the frontoparietal lobe or the deep middle cerebral artery (MVD) territory, including the basal ganglia and internal capsule (11/19) than in those patients whose infarctions were in other regions (1/7) ($p=0.048$). Accordingly, the Δ%cbll was significantly lower in the former group (-13.9±9.4%) than in the latter (-4.7 ±13.0%) ($p=0.030$).

The relationship between motor deficit and CCD is shown in Fig. 6. Patients having Grade 1, 2 or 3 muscular strength had a significantly higher frequency of CCD and lower Δ%cbll than those patients with milder or no hemiparesis (frequency, 5/5 and 6/18, respectively, $p=0.008$;Δ%cbll, -21.4 ± 3.8% and -8.3 ± 11.1%, respectively, $p=0.014$). However,

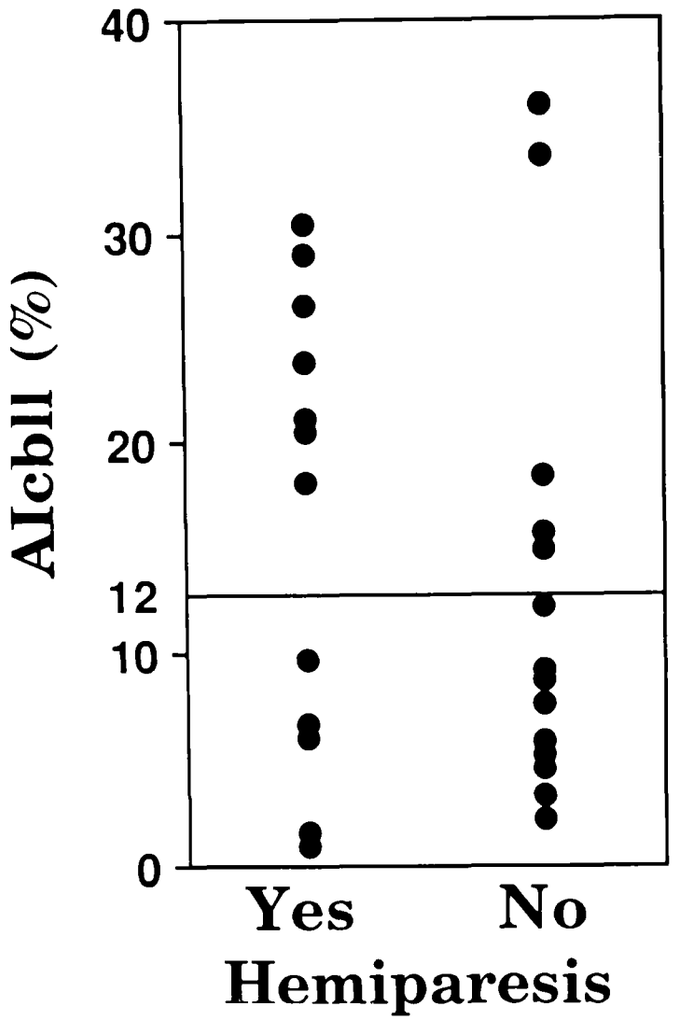

Fig. 7. Alcbll in infarction patients with and without hemiparesis. The solid line represents the upper limit of 95% confidence interval for Alcbll.

CCD also occurred in 5 of the 14 patients without hemiparesis, and was not seen in 5 of the 12 hemiparetic patients (Fig. 7). None of the 12 infarction patients with CCD showed the apparent clinical signs of cerebellar dysfunction.

DISCUSSION

We found that CCD is a frequent phenomenon after supratentorial infarction, since cerebellar perfusion was significantly asymmetric in 46.2% of the patients studied. Our findings agree with prior reports that showed CCD to be present in about 50% of patients after stroke or cerebral tumors, as determined by tomographic mapping of oxygen consumption, glucose utilization and perfusion (Lenzi *et al.* 1982; Meneghetti *et al.* 1983; Baron *et al.* 1984; Kushner *et al.* 1984; Biersack *et al.* 1984; Meneghetti *et al.* 1984; Patronas *et al.* 1984; Casaro *et al.* 1985; Brott *et al.* 1986; Pantano *et al.* 1986 & 1987; Holman *et al.* 1989).

The time course of CCD in ischemic stroke patients has been a matter of controversy with discrepant results. We found the incidence of CCD to be greater during the initial 30 days than those studied one month or more after the clinical event. However, CCD appears to be a relatively persistent phenomenon, since it remained in 30% of the patiients more than 10 months after the clinical event . Furthemore, the present study demonstrates that there is no significant correlation between the severity of CCD and the duration after infarction. This lack of recovery from crossed cerebellar diaschisis contrasts sharply with other examples of transynaptic depression. Similar to our results, Lenzi *et al.* (1982), Martin and Raichle (1983), Biersack *et al.* (1984), Meneghetti *et al.* (1984), Pantano *et al.* (1986 & 1987), Vorstrup (1988), and Bogsrud *et al.* (1990) reported unchanged or even exaggerated CCD in old stroke patients as compared with acute ones. Neverthless, recovery from CCD has been reported in a few stroke patients (Baron *et al.* 1980 B, 1981 b: Meneghetti *et al.* 1984; Kushner *et al.* 1984; pantano *et al.* 1986), suggesting that this pro-

Table 3. SVD, %SVD, MVD and %MVD in patients with and without CCD

	Patients with CCD	Patients without CCD
SVD (ml)	71 ± 47 (12)	70 ± 68 (12)
%SVD	7.8 ± 4.9 (12)	7.7 ± 6.9 (12)
MVD (ml)	93 ± 84 (10)	67 ± 77 (12)
%MVD	10 ± 8.7 (10)	7.7 ± 8.9 (12)

CCD, crossed cerebellar diaschisis; for other abbreviations, refer to Table 1. Values are mean ± SD. Number in parentheses after each value represents the number of patients for whom the measurement was made. Statistical analysis did not show significant differences in SVD, %SVD, MVD, and %MVD between the patients with and without CCD.

cess is not necessarily unrecoverable. While the mechanisms for recovery are unclear, they may represent a link between reversible diaschisis and irreversible degeneration (Pantano *et al.* 1986).

The relationship between CCD and the size and location of the primary supratentorial lesion is controversial. In most studies, the extent of the lesion was determined using qualitative or, at best, semiquantitative analysis. Baron *et al.* (1989 b) repoted that there was no correlation between the occurrence of CCD and the size of the supratentorial infarction. However, several studies have shown that CCD is more frequent and severe with large lesions invoving two or three cerebral lobes (Lenzi *et al.* 1982, Kushner *et al.* 1984; Feeney and Baron 1986; Pantano *et al.* 1986,1987). Lenzi *et al.* found CCD to be more evident with parietal cortex infarcts. Kushner *et al.* (1984) found that patients with cerebral infarcts or tumors showed profound cerebellar hypometabolism in association with lesions that produced multilobar involvement; however, CCD was highly associated with parietal lesions but not with frontal, thalamic, or basal ganglia lesions, supporting Lenzi's conclusion. An opposite observation was made by Martin and Raichle (1983) where asymmetry of the cerebellar blood flow and metabolic rate was found in patients with infarcts involving the frontal cortex, but not in those with parietal or parietooccipital infarcts. Meneghetti *et al.* (1984) did not find CCD in patients with medium sized or small infarcts involving the temporal or the parietal lobe. Pantano *et al.* (1986) found that CCD was most pronounced and most frequent with deep MCA territory infarcts that extensively infarcts that extensively involved the internal capsule and lentiform nucleus. Further, he found that multilobar infarcts induced more significant CCD than did unilobar infarcts. Recently, Primeau *et al.* (1990) reported that the CCD was associated most often with motor lesions.

Our study is the first to employ absolute quantification of the infarct size as well as the severity of the cerebral hypoperfusion to examine the correlations CCD. Neither the severity of the cerebral hypoperfusion (SVD, %SVD) nor the size of the cerebral infarct (MVD,%MVD) correlated with the occurrence of CCD in stroke patients. We also found no significant correlation in patients with CCD between the severity of the cerebral hypoperfusion and the magnitude of CCD. This finding is similar to the absence of a significant correlation between the degree of perfusion asymmetry in the hemispheres and the degree of CCD reported by Pantano *et al.* (1987). In contrast, a significant correlation was found between the severity of CCD and infarct size in patients with CCD. These results suggest that neither the size of the cerebral infarct nor the severity of the cerebral hypoperfusion is critical for the occurrence of CCD. However, once a CCD developes, infarct size may play a potential role in determining its severity.

The anatomical location of the lesion, rather than its size, may be the major determinant for the occurrence of CCD. We have shown that both the frequency and severity of CCD are greater in patients with an infarction in either the frontoparietal lobes or the deep MCA territory, as compared to patients with infarcts of similar or even larger size involving other cerebral lobes. These topographical correlates fit the widely held hypothesis that

CCD is a consequence of disrupted corticopontocerebellar pathways (Baron *et al.* 1980 a, b). Although the relative contributions from the different cortical areas have not been settled in man, a major part of the corticopontine system originates from the frontal and the cortices in monkeys (Beodal 1978, Gliskstein *et al.* 1985), and the corticopontine fibers converge toward the anterior and posterior limbs of the internal capsule as well as its retrolenticular and sublenticular portions (Carpenter 1976).

Our study shows that the discordance between the SPECT volume deficit and the magnetic resonance volume deficit is not associated with the occurrence of CCD. Contrary to our results, Holman *et al.* (1989) reported a higher incidence of CCD when there was a substantial difference between CT and SPECT in the extent of the cerebral abnormality. It seems that the discrepancy depends, at least in part, on whether the methods employ quantitative or visual analysis.

Bierasck *et al.* (1984) found that CCD was present only in patients with hemiparesis, while most patients without CCD did not suffer hemiplegia. They concluded that CCD is caused by the reduction of spinocerebellar stimuli due to the paresis of the respective extremities. Our results show that CCD is associated with the severity of hemiparesis. However, CCD was absent in many hemiparetic patients and could be found in a considerable proportion of the patients without hemiparesis. This finding agrees with several previous studies (Kushner *et al.* 1984; Pantano *et al.* 1986). This agreement helps to confirm that interruption of the corticopontocerebellar pathways is the most likely mechanism of CCD, as previously proposed by Baron *et al.* (1980 a, b), with spinocerebellar or corticospinal alterations playing a secondary role at best.

At present, there is no well established clinical expression of CCD. We found that the presence of a cerebellar abnormality was not related to the presence of clinical signs of cerebellar dysfunction. This finding is similar to Kushner *et al.* (1984), who observed a clear cerebellar syndrome in only one of 16 patients with CCD. We do not know why the clinical signs of cerebellar dysfunction were not apparent in our patients. It is possible that most cerebral lesions interrupt cerebrocerebellar connections widely, whereas more selective involvement, such as that seen in ataxic hemiparesis, is needed to produce clear-cut ataxia (Perman and Racy 1980; Kushner *et al.* 1984). The presence of severe weakness, aphasia, or other neurological deficits may interfere with the demonstration of ataxia by bedside testing methods (Kushner *et al.* 1984).

In conclusion, we have used quantitative methods which avoid the use of subjective visual impressions in order to demonstrate the correlation between CCD and the severity of cerebral hypoperfusion, infarct size, and the location of the supratentorial lesion. Once crossed cerebellar diaschisis occurs, the infarct size may provide a potential role in determining the magnitude of CCD. However, neither the size of the cerebral infarction nor the severity of the cerebral hypoperfusion may be critical for the occurrence of CCD. The location rather than the extent of the lesion appears to be the major determinant for the occurrence and magnitude of CCD in stroke patients. The very concept of CCD still remains largely unproven. Future investigations should be directed to understanding of the pathophysiology, clinical correlations, and therapeutic implications of crossed cerebellar diaschisis.

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