

Crossed Cerebellar Diaschisis Due To Intracranial Hematoma in Basal Ganglia or Thalamus

Joon Seok Lim, Young Hoon Ryu, Byung Moon Kim and Jong Doo Lee

Division of Nuclear Medicine, Department of Diagnostic Radiology, Yonsei University Medical College, Seoul, Korea

The purpose of our study was to evaluate the remote effects on the cerebellum and cerebral cortex from subcortical hematoma without cortical structural abnormality. **Methods:** Our study included 23 patients with hematoma, strictly confined either to the basal ganglia ($n = 12$) or thalamus ($n = 11$) without cortical abnormality on CT or MRI. Twenty psychiatric patients without structural abnormality on MRI were selected as control subjects. Technetium-ethyl cysteinate dimer brain SPECT was performed in patients and control subjects. Regional cerebral blood flow (rCBF) was visually and semiquantitatively assessed. Asymmetry index (AI) was determined using data from regions of interest at the basal ganglia, thalamus, cerebellum, frontal, parietal and temporal cortex to support the semiquantitative analysis. The criteria for defining hypoperfusion that reflected diaschisis was based on an AI $>$ the mean $+ 2$ s.d. of AI in control subjects. **Results:** In the basal ganglia hematoma, rCBF was reduced significantly in the contralateral cerebellum (10/12), ipsilateral thalamus (12/12), ipsilateral frontal (6/12), parietal (12/12) and temporal cortex (10/12). As for thalamic hematoma, significantly reduced perfusion was seen in the contralateral cerebellum (10/11), ipsilateral basal ganglia (7/11), ipsilateral frontal (5/11), parietal (11/11) and temporal cortex (3/11). **Conclusion:** Crossed cerebellar diaschisis (CCD) and cortical diaschisis frequently were observed in patients with subcortical hematoma without cortical structural abnormality. This suggested that CCD can develop regardless of interruption of the corticopontocerebellar tract, which is the principal pathway of CCD.

Key Words: cerebellum; diaschisis; subcortical hematoma; brain SPECT

J Nucl Med 1998; 39:2044-2047

Diaschisis refers to matched depression of blood flow and metabolism occurring remotely from the responsible structural lesion. It has been suggested that this remote decreased neuronal activity results from an interruption of afferent or efferent fiber pathways (1-5). In particular, crossed cerebellar diaschisis (CCD) is known to develop in the cerebellar hemisphere contralateral to a focal supratentorial lesion, and it is a well-recognized phenomenon after a cerebral infarction (1,2,6-9).

Many previous studies using SPECT and PET have reported CCD predominantly in association with a cerebral infarction (1,2,6-9), and it has been described as a developed phenomenon after interruption of the corticopontocerebellar tract. Only a few articles (10,11) have described CCD after a subcortical hemorrhage, although the basal ganglia or thalamus would not be connected directly to the corticopontocerebellar tract.

The remote effect has been observed not only in the contralateral cerebellum but also in the cortex in patients with recent ischemic or hemorrhagic unilateral thalamic lesions (4,12). Ezzedine et al. (13) reported the occurrence of a significant reduction in cortical regional cerebral blood flow

(rCBF) in patients with unilateral capsulothalamolenticular lesions.

The purpose of our study was to evaluate how the cerebellum and cerebral cortex are affected by a deep-seated hematoma strictly confined either to the basal ganglia or thalamus without cortical structural abnormality.

MATERIALS AND METHODS

Patients and Control Subjects

Our study included 56 patients with hypertensive intracerebral hemorrhage. Patients with clinical symptoms of an ischemic episode before hemorrhagic attack or who had MRIs showing a cortical structural abnormality suggesting a previous ischemic episode were excluded. According to this criteria, 23 patients (7 women, 16 men; age range 30-67 yr; mean age 54.6 ± 9.2 yr) were included in our study. None of the selected patients had suffered a second symptomatic neurologic event since their hemorrhagic attack. All lesions were confined strictly to the deep-seated structures (basal ganglia, $n = 12$; thalamus, $n = 11$) and were confirmed by initial CT and follow-up MRI. In all patients, there was no structural abnormality in either the cerebellum or cerebral cortex.

The SPECT study also was conducted in 20 psychiatric patients without prior history of neurological deficits or vascular risk factors (8 women, 12 men; mean age 43.5 ± 11.4 yr), who were the control group. In all of these control subjects, MRI revealed no structural abnormalities in the brain.

Imaging Procedures

SPECT was performed after an intravenous injection of 740 MBq ^{99m}Tc -ethyl cysteinate dimer using a brain-dedicated annular crystal gamma camera (Digital Scintigraphic Inc., Waltham, MA) with low-energy, high-resolution, parallel-hole collimators. One hundred twenty projections were acquired with 3° angular increments. The matrix size was 128×128 . Transaxial images were obtained by the filtered backprojection method using a Butterworth filter (Nyquist frequency 1.1 cycle/cm at order no. 10). Attenuation correction of the transaxial images was performed by the Chang's method, and coronal and sagittal slices were calculated from the original transaxial images (parallel to the orbitomeatal line). The mean interval from onset of the symptoms to the SPECT examination was 53.6 ± 26.7 days.

Data Analysis

Assessment of rCBF was performed visually and semiquantitatively. In each subject, regions of interest (ROIs) were drawn over the bilateral thalami, basal ganglia, cerebellum, frontal, parietal and temporal cortex for semiquantitative analysis (Fig. 1). Asymmetry index (AI) was calculated as $200 \times (C_R - C_L) / (C_R + C_L)$, where C_R and C_L are, respectively, the mean reconstructed counts for the right and left ROIs in both the patient and control groups. The criteria for defining hypoperfusion that reflected diaschisis was based on an AI $>$ the mean $+ 2$ s.d. of AI in the control subjects.

Received Oct. 9, 1997; revision accepted Apr. 15, 1998.

For correspondence or reprints contact: Jong Doo Lee, MD, Division of Nuclear Medicine, Department of Diagnostic Radiology, Yonsei University Medical College, 134 Shinchondong, Seodaemun-Gu, Seoul, 120-752, Korea.

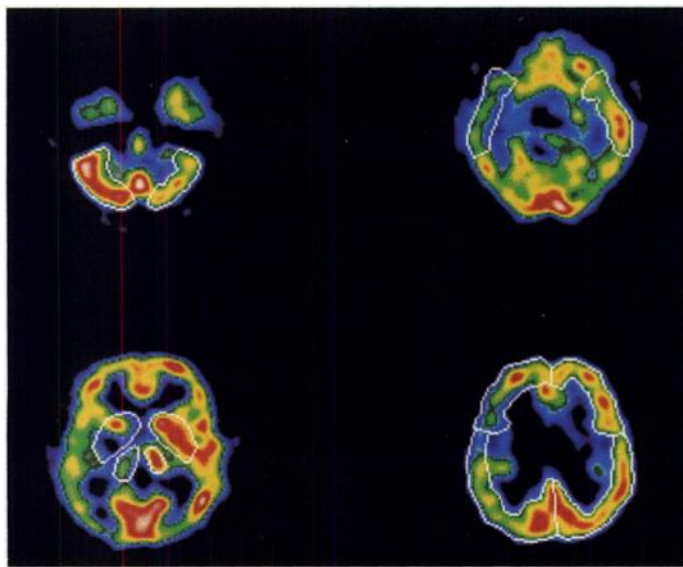


FIGURE 1. Drawing of regions of interest (ROIs) in patient with basal ganglia hematoma. ROIs were drawn manually along margin of basal ganglia, thalamus, cerebellum and frontal, parietal and temporal cortex. Mean pixel counts of ROIs were obtained, and asymmetry index values were calculated.

RESULTS

In the control group, mean \pm s.d. AI was 4.35 ± 0.94 in the cerebellum, 2.57 ± 1.45 in the thalamus, 4.34 ± 2.08 in the basal ganglia, 3.24 ± 0.87 in the parietal, 4.02 ± 2.04 in the frontal and 5.13 ± 1.69 in the temporal cortex. Significant hypoperfusion in patients was defined by an AI greater than the mean + 2 s.d. of AI in the control subjects (i.e., AI greater than 6.23 in the cerebellum, 5.47 in the thalamus, 8.50 in the basal

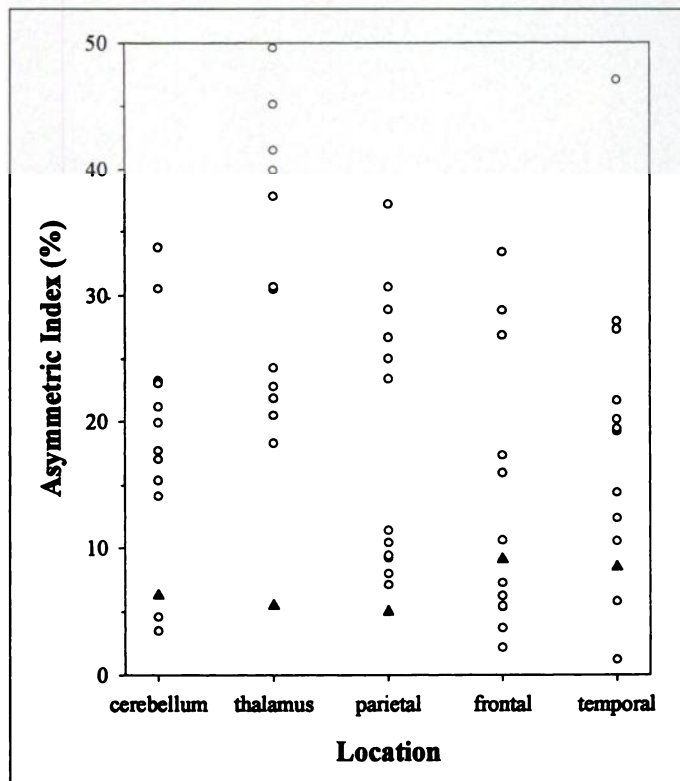


FIGURE 2. Values of asymmetry index (AI) in basal ganglia hematoma patients and mean + 2 s.d. of AI in each location of control subjects. AI values for patients are shown as open circles. Closed triangle indicates mean + 2 s.d. of AI in each location of control subjects. Open circle above closed triangle indicates significant hypoperfusion.

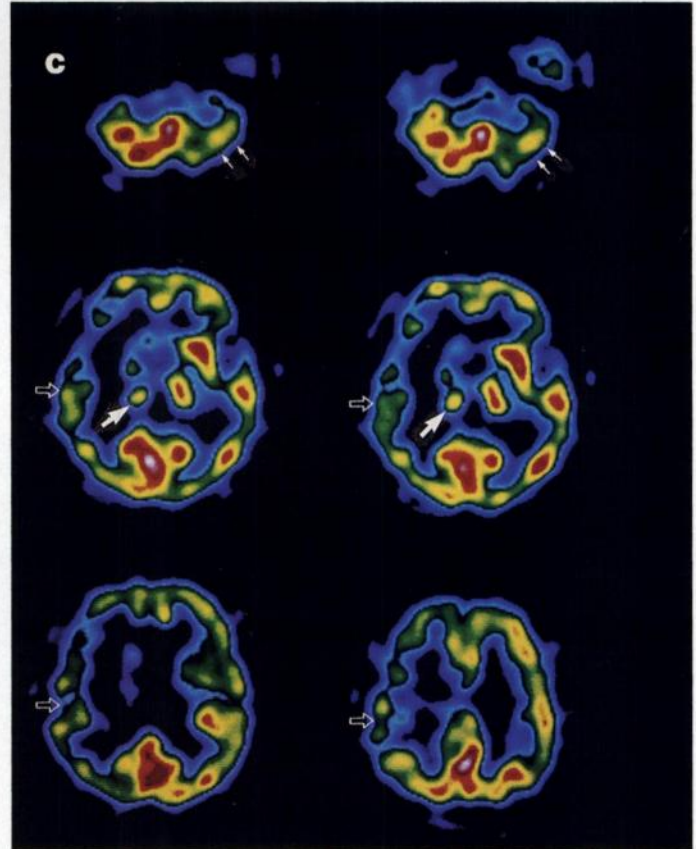
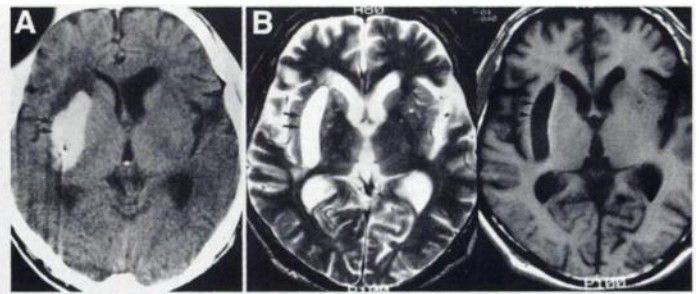


FIGURE 3. Diaschisis in patient with basal ganglia hematoma. (A) Initial CT shows intracranial hematoma in basal ganglia at hemorrhagic attack (arrows). (B) Follow-up MRI shows encephalomalacic change of affected area without cortical structural abnormality on T2- and T1-weighted images (arrows). (C) SPECT shows reduced regional cerebral blood flow in contralateral cerebellar hemisphere (small arrows), ipsilateral thalamus (closed arrow) and cerebral cortex (open arrow).

ganglia, 4.98 in parietal, 9.1 in the frontal and 8.51 in the temporal cortex).

In basal ganglia hematoma ($n = 12$), Figure 2 shows the individual AI value of patients according to each location, compared with mean + 2 s.d. of AI in the control subjects. In the cerebellum, 10 of 12 patients had higher AI values (mean \pm s.d. 21.61 ± 6.4) than 6.23, suggesting significant hypoperfusion in the contralateral cerebellum. Semiquantitative analysis also showed significant hypoperfusion in the ipsilateral thalamus ($n = 12$), the ipsilateral parietal ($n = 12$), the frontal ($n = 6$) and the temporal cortex ($n = 10$) (Fig. 2). SPECT images demonstrated decreased uptake within these areas (Fig. 3).

In thalamic hematoma ($n = 11$), as shown in Figure 4, hypoperfusion of the contralateral cerebellar hemisphere was observed in 10 of 11 patients (mean \pm s.d. of AIs 14.82 ± 5.29), compared with 6.23. Significant hypoperfusion also was seen in the ipsilateral basal ganglia ($n = 7$), the ipsilateral parietal ($n = 11$), the frontal ($n = 5$) and the temporal cortex

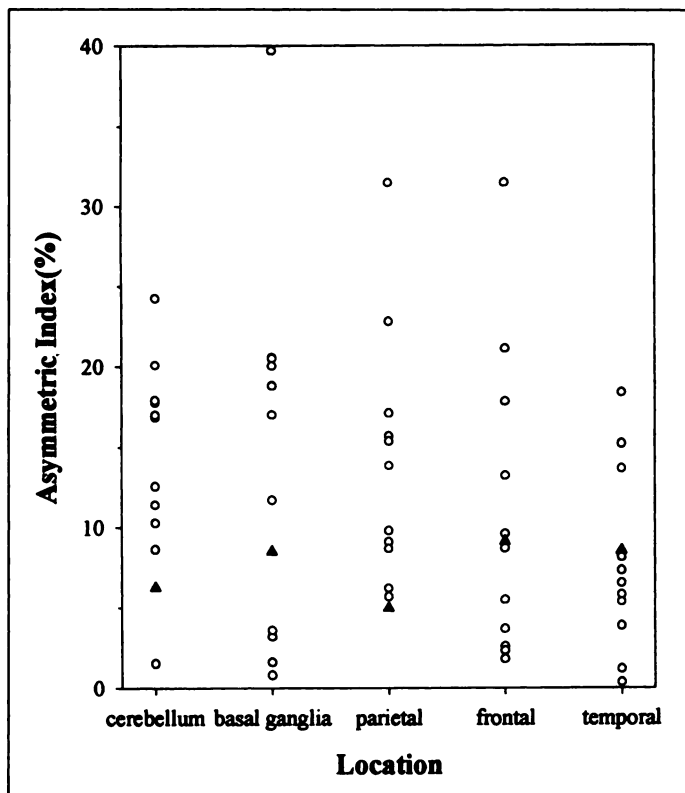


FIGURE 4. Values of asymmetric index in thalamic hematoma patients and mean + 2 s.d. of asymmetry index (AI) in each location of control subjects. AI values for patients are shown as open circles. Closed triangle indicates mean + 2 s.d. of AI in each location of control subjects. Open circle above closed triangle indicates significant hypoperfusion.

(n = 3) (Fig. 4). CCD and cortical diaschisis also were demonstrated in the SPECT images (Fig. 5).

DISCUSSION

In our study, brain SPECT of patients with hematoma strictly confined to the thalamus or basal ganglia showed decreased perfusion at the site of the anatomic lesion and in remote areas such as the ipsilateral cerebral cortex and contralateral cerebellum. On the basis of these results, CCD was a frequent phenomenon after hemorrhagic attack both in the basal ganglia and thalamus.

CCD has been described as being caused by disruption of the corticopontocerebellar pathway, which is cerebellar afferent from the pons to the cerebellum through the middle cerebellar peduncle. Many previous articles were concerned about CCD after cerebral infarction associated with a structural abnormality of the cerebral cortex (1,2,6-9). A few studies reported CCD in patients with a deep-seated infarction such as in the thalamus (11,14,15). Pappata et al. (11) reported that CCD was found in 2 of 6 patients with thalamic infarction. They explained that this phenomenon may have resulted either directly from damage to the cerebellar efferent pathway, (i.e., ascending cerebello-thalamocortical system) or indirectly from hypofunction of the cerebral cortex.

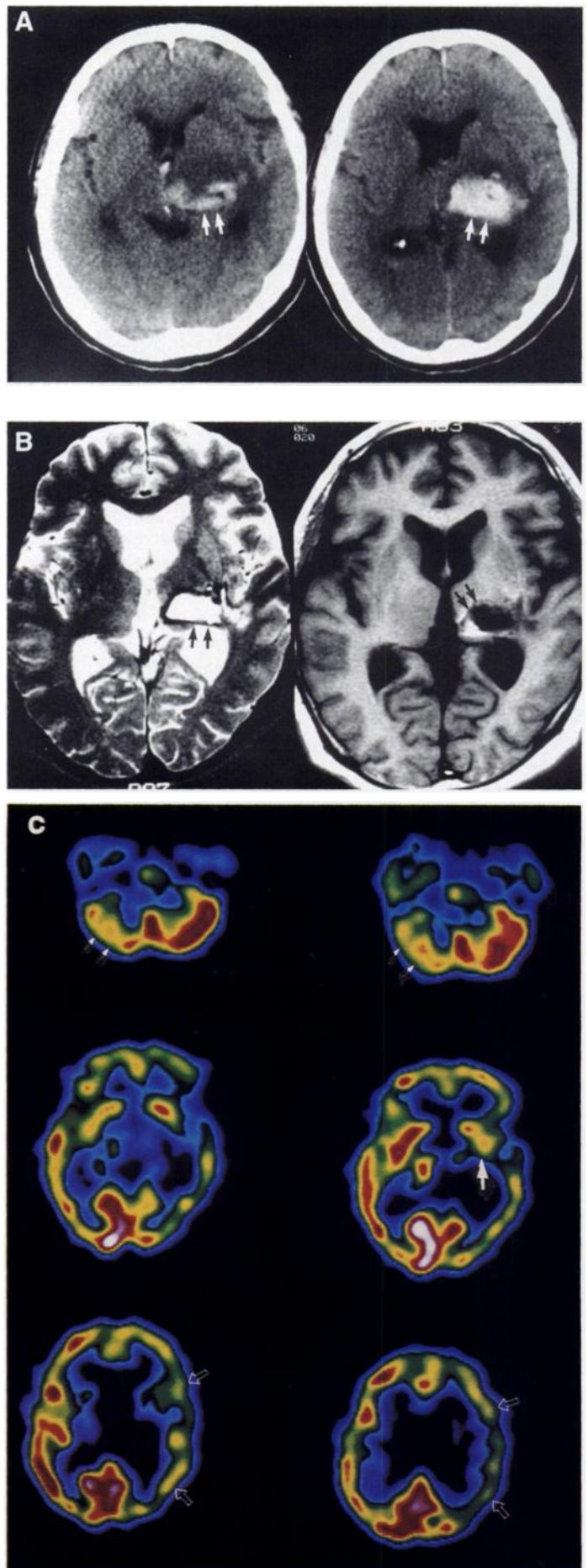


FIGURE 5. Diaschisis in patient with thalamic hematoma. (A) Initial CT shows intracranial hematoma in thalamus at hemorrhagic attack (arrows). (B) Follow-up MRI shows strictly confined hematoma in thalamus without cortical structural abnormality on T2- and T1-weighted images (arrows). (C) Reduction of regional cerebral blood flow in contralateral cerebellum (small arrows), affected thalamus, ipsilateral basal ganglia (closed arrow) and cerebral cortex (open arrow) are demonstrated.

In thalamic hematoma, there are at least three main anatomical pathways that can be associated with CCD. The first is the interruption of the efferent pathway from the cerebellum as described by Pappata et al. (11). Previous human postmortem studies (16) that have shown thalamic lesions may result in retrograde contralateral dentate nucleus atrophy may support our hypothesis. The second, hypoperfusion of the cerebral cortex, which can be produced by thalamocortical diaschisis, may lead to CCD. The third possible mechanism is the interruption of the corticopontocerebellar tract by either intrinsic occult injury or compression of the posterior limb of the internal capsule by an anatomically adjacent thalamic hematoma. In our patients, most SPECT images were obtained after the late subacute stage, in which surrounding edema usually disappears. MRI did not show any compression of the internal capsule by either hematoma or edema. Interruption of the corticopontocerebellar tract may not be a main mechanism of CCD according to our study.

In basal ganglia hematoma, Kanaya et al. (10) have described putaminal hemorrhage resulting in CCD. In terms of the anatomical connections between the basal ganglia and the cerebellum, there are at least three putative pathways that could be involved. First, the basal ganglia may have many neuronal connections with the thalamus. The striatum (caudate and putamen) receives inputs from the intralaminar thalamic nuclei and gives inhibitory axons (GABAergic) to the globus pallidus, which is the major outflow nucleus of the corpus striatum. The globus pallidus, in turn, gives inhibitory axons to the ventral nuclei (ventral anterior and ventral lateral) of the thalamus, which also receives input from the cerebellum (17). The interruption of this circuit in the region of the basal ganglia is assumed to be responsible for the rCBF reduction in the contralateral cerebellar hemisphere through the cerebellar efferent pathway. Second, the basal ganglia also have many neuronal connections with the cerebral cortex (18). Our study showed significant hypoperfusion in the ipsilateral cortex in all patients with basal ganglia hematoma. Hence, CCD in patients with basal ganglia hematoma, may result indirectly from hypoperfusion of the cerebral cortex. Finally, an anatomical neurochemical pathway (dopaminergic pathway) arises from the dentate nucleus of the cerebellum. It crosses the midline at the level of the brachium conjunctivum and sends terminals to the substantia nigra. The projections from the substantia nigra enter the neostriatum (19,20). Therefore, interruption of these neuronal circuits may be another possible explanation of CCD in patients with basal ganglia hematoma.

CONCLUSION

Crossed cerebellar diaschisis and cortical diaschisis can develop in the basal ganglia or thalamic hematoma without cortical structural abnormality. These data suggest that CCD may develop regardless of any interruption of the corticopontocerebellar pathway.

REFERENCES

1. Baron JC, Bousser MG, Comar D, Castaigne P. Crossed cerebellar diaschisis in human supratentorial brain infarction. *Trans Am Neurol Assoc* 1980;105:459-461.
2. Lenzi GL, Frankowiak RSJ, Jones T. Cerebral oxygen metabolism and blood flow in human cerebral ischemic infarction. *J Cereb Blood Flow Metab* 1982;2:321-335.
3. Metter EJ, Mazziotta JC, Itabashi HH, Mankovich NJ, Phelps ME, Kuhl DE. Comparison of glucose metabolism, X-ray CT, and postmortem data in a patient with multiple cerebral infarcts. *Neurology* 1985;35:1695-1701.
4. Pawlik G, Herholz K, Beil C, Wagner R, Wienhard K, Heiss WD. Remote effects of focal lesions on cerebral blood flow and metabolism. In: Heiss WD, ed. *Functional mapping of the brain in vascular disorders*. Berlin-Heidelberg: Springer-Verlag; 1985:59-83.
5. Martin WRW, Raichle ME. Cerebellar blood flow and metabolism in cerebral hemispheric infarction. *Ann Neurol* 1983;14:168-176.
6. Kushner M, Alavi A, Reivich M, Dann R, Burke, A, Robinson G. Contralateral cerebellar hypometabolism following cerebral insult: a positron emission tomographic study. *Ann Neurol* 1984;15:425-434.
7. Meneghetti G, Vorstrup S, Mickey B, Lindewald H, Lassen NA. Crossed cerebellar diaschisis in ischemic stroke; a study of regional cerebral blood flow by ¹³³Xe inhalation and single photon emission tomographic study. *J Cereb Blood Flow Metab* 1984;4:235-240.
8. Pantano P, Baron JC, Samson Y, Bousser MG, Derouesne C, Comar D. Crossed cerebellar diaschisis, further studies. *Brain* 1986;109:677-694.
9. Pantano P, Lenzi GL, Guidetti B, et al. Crossed cerebellar diaschisis in patients with cerebral ischemia assessed by SPECT and ¹²³I-HIPDM. *Eur Neurol* 1987;27:142-148.
10. Kanaya H, Endo H, Sugiyama T, Kuroda K. Crossed cerebellar diaschisis in patients with putaminal hemorrhage. *J Cereb Blood Flow Metab* 1983;3(suppl):S27-S28.
11. Pappata S, Mazoyer B, Tran DS, Cambon H, Levasseur M, Baron JC. Effect of capsular or thalamic stroke on metabolism in the cortex and cerebellum: a positron tomography study. *Stroke* 1990;21:519-524.
12. Baron JC, D'Antona R, Pantano P, Serdaru M, Samson Y, Bousser MG. Effects of thalamic stroke on energy metabolism of the cerebral cortex. *Brain* 1986;109:1243-1259.
13. Ezzedine A, Andre C, Guy D, Michel V. Remote effect of deep seated vascular brain lesions on cerebral blood flow. *Stroke* 1990;21:1555-1561.
14. Pawlik G, Beil C, Herholz K, Szeliess B, Wienhart K, Heiss WD. Comparative dynamic FDG-PET study of functional deactivation in thalamic versus extrathalamic focal ischemic brain lesions. *J Cereb Blood Flow Metab* 1985;5(suppl 1):S9-S10.
15. Kushner M, Kaasik AE, Nencini P, et al. Contralateral cerebellar hypometabolism following cerebral infarction: an acute and follow-up study [Abstract]. *Neurology* 1988;38(suppl 1):147.
16. Chung HD. Retrograde crossed cerebellar atrophy. *Brain* 1985;108:881-895.
17. Barbara FW, Eduardo EB, Jasper RD, Thomas JR, Burton AS. *Medical neurosciences, motor system*, 3rd ed. Boston, New York, Toronto, London: Little Brown & Co.; 1994:193-195.
18. Hoover JE, Strick PL. Multiple output channels in the basal ganglia. *Science* 1993;259:819-821.
19. Andre P, Lili NH. Functional anatomy of the basal ganglia. *Brain Res Rev* 1995;20:91-127.
20. Snider RS, Maiti A, Snider SR. Cerebellar pathways to ventral midbrain and nigra. *Exp Neurol* 1986;53:714-728.