The Functional Nature of Cerebellar Diaschisis

Vittorio Di Piero, MD, François Chollet, MD, Ray J. Dolan, MD, David J. Thomas, MD, and Richard Frackowiak, MD

We report a patient who presented with transient clumsiness of his right hand due to a small hemorrhage in the left globus pallidus. Ten days later, positron emission tomography performed at rest showed decreased oxygen metabolism and blood flow at the site of the anatomic lesion and in remote areas such as the ipsilateral frontotemporoparietal cortex and the contralateral cerebellar hemisphere. Cerebellar hypometabolism has been ascribed to functional disconnection of the contralateral hemisphere from the cerebral cortex and has been termed crossed cerebellar diaschisis. One month later, positron emission tomography performed during unilateral motor activation (finger opposition) showed increased blood flow in the sensorimotor and supplementary motor areas contralateral to the hand engaged in the motor task. An at-rest study at this time showed resolution of the crossed cerebellar diaschisis observed acutely, but cerebellar asymmetry was demonstrated during performance of the motor task with the normal as well as with the previously paretic hand. Our activation study demonstrated cerebellar asymmetry in the chronic phase during a motor task, even though resting cerebellar blood flow was symmetrical. This observation reveals the dynamic, functional nature of crossed cerebellar diaschisis and may partially explain the lack of any clinical counterpart in functional studies of the cerebellum performed with the patient at rest. (Stroke 1990;21:1365-1369)

Cerebellar diaschisis contralateral to a supratentorial lesion was first described by Baron et al. in 1980 using positron emission tomography (PET) in a stroke patient. Interruption of cerebrocerebellar pathways, probably due to damage of the predominantly excitatory corticopontine projections, is considered to be the principal mechanism of this transneuronal cerebellar metabolic depression, although interruption of spinocerebellar pathways has also been implicated.

Despite its name, crossed cerebellar diaschisis has characteristics that do not fit with the classical definition of diaschisis, which implies a functional phenomenon without structural change and which is reversible. Crossed cerebellar diaschisis has been observed in diseases of slow onset, such as progressively enlarging tumors, or may persist or even worsen with time. In this regard, it has been suggested that anterograde transneuronal degeneration is responsible for the irreversible component of the phenomenon, which then reflects secondary morphologic change. Nevertheless, recovery from crossed cerebellar diaschisis has been described in patients with cerebrovascular diseases, and the relative contributions of the two components, functional and structural, are not always obvious from static imaging. There are no clinical clues to the presence of secondary, irreversible cerebellar atrophy following cerebral hemispheric infarction.

Case Report

We studied a 52-year-old man who presented with clumsiness of his right hand, followed by complete recovery the next day. Magnetic resonance imaging showed a small hemorrhagic lesion of the left globus pallidus (Figure 1). Cerebral angiography was normal. To evaluate the cerebral metabolic rate for oxygen (CMRO₂) and the cerebral blood flow (CBF), a dynamic PET oxygen-15 steady-state study was performed 10 days after the onset of symptoms. The study was carried out with an ECAT 931/8/12 PET...
scanner (CTI Inc., Knoxville, Tenn.) with an intrinsic spatial resolution of $6.5 \times 6.5 \times 7.0$ mm full width at half-maximum (FWHM), which allowed simultaneous collection of 15 contiguous transaxial planes. Reconstruction, attenuation correction (by measurement), and filtering resulted in an image with a resolution of $8.5 \times 8.5 \times 7.0$ mm FWHM. The data set was then expanded by linear interpolation in the axial dimension to produce 43 transaxial slices with cubic voxels allowing three-dimensional visual inspection of the scans. The scans were analyzed on a computer (SUN 3/60, Mountain View, Calif.) with image analysis software (ANALYZE; Biodynamic Research Unit, Mayo Clinic, Rochester, Minn.) that allowed them to be scaled to standard stereotactic coordinates based on the atlas of Talairach et al. At the cerebellar level, two irregular regions of interest comprising the entire cerebellar hemisphere were chosen. At the cerebral level, irregular regions of interest were drawn interactively on the areas of visually observed marked reductions of regional CBF. Values averaged from all planes were assigned to lobes and expressed relative to the global CBF obtained at each brain level from the remaining, normal brain areas to normalize the values and allow comparison with subsequent measurements of CBF.

Performed with the patient at rest in a quiet, dimmed room, the dynamic PET study showed reduced CMRO$_2$ and CBF at the site of the anatomic lesion, in the ipsilateral frontotemporoparietal cortex, and in the contralateral cerebellar hemisphere (Figure 2). The asymmetries of CBF and CMRO$_2$ between the affected region and the homologous region in the unaffected hemisphere were $-38\%$ and $-35\%$ in the frontal cortex, $-25\%$ and $-20\%$ in the temporal cortex, and $-30\%$ and $-20\%$ in the parietal cortex, respectively. Our mean±SD values for right/left asymmetries in six normal volunteers in these three regions are $-2.3\pm3.3\%$, $0\pm4.4\%$, and $-2.3\pm3.9\%$ for CBF and $-1.9\pm3.6\%$, $0\pm3.2\%$, and $-1.5\pm2.4\%$ for CMRO$_2$, respectively. In the cerebellum, the left/right asymmetry was $27\%$ for CBF and $19\%$ for CMRO$_2$. The study included acquisition of data at six 10-minute intervals during various states of physiological activation: A) at rest, B) during opposition of the right (recovered) fingers, and C) during opposition of the left (normal) fingers. The tasks were performed in the order ABCBBA to control for habituation and time-dependent effects. Thus, we compared the average of the two CBF measurements during each physiological state. During motor performance, CBF increased an average of $11.3\%$ over that during the resting state in the sensorimotor and supplementary motor areas contralateral to the hand engaged in the motor task (Figure 3). The two at-rest measurements showed no significant cerebellar CBF asymmetry ($5.8\%$). The cerebral cortical asymmetries seen in the 10-day study were also no longer apparent, with right/left ratios of $-2.7\%$, $-4.1\%$, and $-3.3\%$ in the frontal, temporal, and parietal cortices, respectively. However, a marked left/right cerebellar asymmetry, characterized by an increase in CBF in the left hemisphere, was revealed during the performance of the motor task with the normal (left, $19\%$) as well as with the previously paretic (right, $14\%$) hand (Figure 4).

**Discussion**

Our results suggest that crossed cerebellar diaschisis is a complex phenomenon. At rest, it frequently occurs during the early phase of stroke, probably due to acute cerebrocerebellar disconnection. The functional nature of crossed cerebellar diaschisis is underlined by its occurrence in patients with transient ischemic attacks, without any evident supraten-
Crossed cerebellar diaschisis often resolves with time, but the mechanisms of returning cerebellar metabolism to normal are still unknown.

Our activation study revealed the presence of cerebellar asymmetry of regional CBF during later stages when resting cerebellar CBF was symmetrical. This may partially explain reported findings of the lack of a clinical counterpart or an association with abnormal function in the cerebellum when the patient is studied at rest.

Our patient raises questions about the nature of "transient" crossed cerebellar diaschisis, which could merely reflect the lack of cerebellar hemispheric...
Cerebellar asymmetry during motor performance by the affected limb supports the notion that spino-
cerebellar input to the ipsilateral cerebellum plays a
minor, if any, role in evoking a cerebellar functional
response.

In conclusion, our observation confirms that crossed
cerebellar diaschisis is mainly due to a disturbance of
corticocerebellar input, that spino-cerebellar input is
of little significance in the mechanisms underlying the
phenomenon, and that recovery following stroke may
be accompanied by functional rearrangements of neu-
ronal connectivity that may compensate more or less
adequately for the motor deficit. The correlation
between crossed cerebellar diaschisis and clinical
prognosis has been very poor; we suggest that PET
scanning during motor activation will result in a better
understanding of the significance of the phenomenon
in relation to clinical outcome.

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Figure 4. Bar graph summarizing cerebral blood flow (CBF)
asymmetries (filled bars, left; shaded bars, right) at cerebellar
level in first positron emission tomographic study at rest (1) and
in three physiological states during activation study (at rest, 2;
with movement of normal left fingers, 3; and with the movement
of recovered previously paretic right fingers, 4).

Asymmetry since the functional studies were performed
at rest. We show that cerebellar asymmetry can be
demonstrated in patients with lesions of the
motor pathways by engaging the patient in motor
tasks.

Hand movement increases regional CBF in the
ipsilateral cerebellar hemisphere.14–16 During focal
ictal activity, there is transient cerebellar hyperper-
fusion contralateral to the cerebral epileptic focus.17
Studies in normal and diseased humans indicate that
the cerebellum responds to activation of the motor
cortex in a very precise lateralized manner. In our
patient, cerebellar asymmetry was elicited on the
same side of the cerebellum (the left) by motor
activity in the previously paretic, as well as in the
contralateral unaffected, hand. Our findings suggest
bilateral activation of the cerebellum during a later-
alized motor task. The occurrence of reversed cere-
bellar asymmetry during performance of the task
with the previously paretic, now recovered, hand
indicates that the phenomenon might reflect the
emergence of new, functional neuronal connections
and/or cross-talk between the affected and unaf-
fected cerebral hemispheres (at whatever anatomic
level), causing bilateral cerebellar activation. Such a
mechanism would result in a relative state of unbal-
anced afferent input to the cerebellar hemispheres,
thus revealing the functional incompetence of cerebral
efferent projections to the contralateral cerebellar
hemisphere.

KEY WORDS: cerebral blood flow • diaschisis • tomography, emission computed
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