Crossed cerebellar diaschisis.

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Crossed Cerebellar Diaschisis

The term "diaschisis," as originally used by Von-Monakow (1), implies an immediate decrease in neuronal activity in a region due to an interruption of its afferent axonal supply. Experimental studies, in animals, of unilateral ischemia in which neuronal function in the contralateral hemisphere was demonstrated to be impaired, provided evidence for such a mechanism (2). The corpus callosum appeared to play a role in this process. Changes in cerebral blood flow in the hemisphere opposite a cerebral infarction were first demonstrated by Kempinsky et al in 1961 (3), and Hoedt-Rasmussen and Skinhoj in 1964 (4). Several theories have been proposed to explain these remote alterations in blood flow, postulating neurogenic, vasogenic, and chemical mechanisms (3–10).

The remote blood flow changes observed, according to the neurogenic theory, are presumed to be secondary to metabolic alterations produced by the decreased axonal input to the region. Such decreased metabolism in the nonischemic hemisphere and in the contralateral cerebellum of patients with infarcts has been documented (8, 11–13).

Little data is available concerning the time course of these cerebral blood flow changes in the nonischemic hemisphere. A progressive decline in cerebral blood flow in the nonischemic hemisphere has been observed during the first week after an acute infarction (14). This decline in flow could be partially explained by a loss of autoregulation, but suggested a process more complex than destruction of axonal afferents to the nonischemic region.

Vasoactive substances released from ischemic tissue have been postulated to play a role in these remote effects (15). Experimental data have demonstrated the release of vasoactive substances and neurotransmitters from infarcted brain (16).

While the release of such vasoactive substances might play a role in a more generalized depression of flow and metabolism, the more focal effects seen in the cerebellar hemisphere contralateral to various types of lesions make this less likely to be an important factor in the production of crossed cerebellar diaschisis.

Crossed cerebellar diaschisis has been observed not only following cerebral ischemic events (12, 13), but in patients with brain tumors (13, 17) and following the injection of intracarotid sodium Amytal (18).

One of the earliest studies demonstrating the importance of the pathways between the cerebrum and cerebellum on the function of the latter was that of von Monakow in 1885 in which he noted hypoplasia of the contralateral cerebellar hemisphere following experimental cortical ablation (19). In the clinical literature, one of the earliest reports of similar findings was that of Hassin in 1935, in which atrophy of the contralateral cerebellum was observed in a patient with cerebral atrophy secondary to presumed birth injury (20). Ataxic hemiparesis (21) and other ataxic syndromes have been noted secondary to cortical insults (22, 23). False localized limb ataxia has been found to occur contralateral to cerebral tumors located either anteriorly or posteriorly in the cerebral hemispheres (24). Although such syndromes have been considered to occur mainly from contralateral frontal lesions (25), they also occur with lesions limited to the parietal lobe (22).

The cerebellum has a significant input from the cerebral hemispheres, the bulk of which is contained in the cortico-ponto-cerebellar pathway (26). This pathway arises in the motor, premotor, parietal association, and occipital cortices (27). Other cerebro-cerebellar connections are relayed through the inferior olive and reticular formation (28). The contribution of different regions of the cortex to the cortico-ponto-cerebellar pathway varies in different species. Parietal areas project most heavily to the cerebellum in the cat.
(29), whereas in the monkey, the majority of the 
descending input to the cerebellum is from the 
motor and premotor areas (30). Connections from 
the cerebellum to the cerebral cortex are also 
widespread and include the motor, premotor, and 
parietal association cortices (31). These ascend-
ing pathways are relayed by the thalamus, retic-
ular formation, and the red nucleus (32). Interru-
tion of this anatomic substrate connecting the 
cerebral hemispheres and the cerebellum pro-
vides a likely mechanism for the production of 
crossed cerebellar hypometabolism, which has 
been described with such lesions in humans (12, 
13, 17). Animal studies have also demonstrated 
reduced blood flow in the cerebellum of gerbils 
within hours of acute cerebral ischemia (33). In 
addition, increased metabolism has been dem-
onstrated in the cerebellum of rats, contralateral 
to a focus of cortical seizure activity (34).

In humans, the presence of contralateral cere-
bellar hypometabolism does not appear to be 
dependent on a single factor, such as the type, 
size, or severity of the lesion. It is equally likely 
to occur in cases of tumor and infarction (12). In 
general, however, widespread cerebral lesions, 
with marked hypometabolism, are more likely to 
be accompanied by contralateral cerebellar hy-
ppometabolism. Exceptions to this, however, are 
not uncommon. Relatively small lesions with mild 
metabolic depression also have been noted to 
produce contralateral cerebellar hypometabolism. 
The type of neurologic deficit also does not ap-
pear to be related to the presence of contralateral 
cerebellar diaschisis. Pure sensory syndromes are 
accompanied by cerebellar hypometabolism and, 
conversely, this metabolic abnormality may not 
be present in some patients with hemiplegia. The 
presence of a parietal lesion, however, does ap-
pear to be associated more often with contralat-
eral cerebellar hypometabolism. This may be the 
pathophysiologic correlate of the ataxia that can 
arise from lesions of the parietal lobe. It also 
suggests the prominence of the parietal cortex in 
the cortico-ponto-cerebellar pathway in humans.

There is little information available concerning 
the time course of crossed cerebellar diaschisis. In 
one report of reduced cerebral blood flow and 
oxxygen consumption in the contralateral cerebel-
lum in patients with acute stroke, the effect was 
not seen in patients studied more than 2 months 
beyond the ictus (35). Similar findings were ob-
erved in another report, in which cerebral blood 
flow and oxygen metabolism were studied in 
patients with cerebral infarctions (13). It was also 

noted that the most intense cerebellar hypomet-
abolism was produced with parietal infarction. 
Martin and Raichle (36) studied a patient both 
less than and more than 3 months after an acute 
stroke and found that the contralateral cerebellar 
hypometabolism initially present had disap-
peared. Two patients restudied more than 3 
months after an acute stroke by Kushner et al 
(12) no longer demonstrated contralateral cere-
bellar hypometabolism that had been present 
earlier. The mechanism by which cerebellar hy-
pometabolism returns to normal is uncertain. 
Restoration of function in reversibly affected cer-
ebral areas is one possibility. A more speculative 
hypothesis may be an increase in activity in 
alternate or subordinate cerebro-cerebellar path-
ways to compensate for the primarily affected 
cortical area.

The present issue of this journal contains two 

References

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