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Crossed Cerebellar Diaschisis

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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

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(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 ascending pathways are relayed by the thalamus, reticular formation, and the red nucleus (32). Interruption of this anatomic substrate connecting the cerebral hemispheres and the cerebellum provides 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 demonstrated in the cerebellum of rats, contralateral to a focus of cortical seizure activity (34).

In humans, the presence of contralateral cerebellar 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 hypometabolism. 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 appear 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 appear to be associated more often with contralateral 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 oxygen consumption in the contralateral cerebellum in patients with acute stroke, the effect was not seen in patients studied more than 2 months beyond the ictus (35). Similar findings were observed 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 hypometabolism 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 disappeared. Two patients restudied more than 3 months after an acute stroke by Kushner et al (12) no longer demonstrated contralateral cerebellar hypometabolism that had been present earlier. The mechanism by which cerebellar hypometabolism returns to normal is uncertain. Restoration of function in reversibly affected cerebral areas is one possibility. A more speculative hypothesis may be an increase in activity in alternate or subordinate cerebro-cerebellar pathways to compensate for the primarily affected cortical area.

The present issue of this journal contains two case reports of crossed cerebellar diaschisis that shed additional light on the time course of this phenomenon (37, 38). Both reports describe the prompt occurrence (within 1 min in one case and 10 sec in the other) of decreased blood flow in the contralateral cerebellum following temporary balloon occlusion of the internal carotid artery in patients being evaluated for the treatment of cerebral aneurysms. These studies confirm the rapidity of development of crossed cerebellar diaschisis previously observed in patients undergoing intracarotid sodium Amytal injection (17, 39, 40). In one report (37), a repeat flow study 15 minutes after deflation of the balloon revealed flow had returned to normal. The prompt onset and resolution of crossed cerebellar diaschisis lends support for the theory that this phenomenon is due to transneuronal loss of excitatory input to the cerebellum and that this functional alteration need not result in pathologic alterations in the cerebellum for crossed cerebellar diaschisis to be manifest. Crossed cerebellar diaschisis can produce clinical findings of cerebellar dysfunction, although this is often difficult to assess, since there is often limb dysfunction associated with the primary lesion. These studies lend support to the concept that improvement in neurologic function associated with cerebral insult may in part be due to the resolution of diaschisis as suggested by Feeney and Baron (41).

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