Commentary

In this interesting article, Menon describes a case of crossed cerebellar atrophy.[1] Since the very first article of Baron *et al.*,[2] it is well known that supratentorial lesions can entail metabolic changes in the contralateral cerebellar hemisphere. Such a phenomenon, usually revealed by positron emission tomographic (PET) scans, has been ascribed to functional disconnection of the contralateral cerebellar hemisphere from cerebral cortex. The notion that an area remote from the site of brain lesion can show a transient impairment of its function was not new, since it was already described in 1914 by von Monakow,[3] who coined the term diaschisis to define this phenomenon. More recently, Tien and Ashdown[4] reported that a consistent percentage of patients who presented clear signs of cerebellar diaschisis, also showed hemiatrophy of the cerebellum in Magnetic Rseonance Imaging (MRI) scans. As a matter of fact, they studied 26 patients of cerebellar diaschisis demonstrated by PET scans, and in eight of them, MRI showed signs of hemiatrophy of the cerebellum. Interruption of cortico-cerebellar fibers sparing the neurons has been claimed to explain diaschisis; however,,the presence of cerebellar morphological changes obviously requires other explanations, such as, for example, anterograde transneuronal degeneration. In my opinion, this as

well as further studies on cerebro-cerebellar diaschisis and atrophy show two main reasons of interest. First of all, the relative rarity of similar reports might depend on an underestimation of the real extent of the phenomenon.Patients with crossed cerebellar atrophy show only few, if any, clinical signs of cerebellum impairment, and hence mild cerebellar atrophy might be underestimated by the radiologist. Furthermore, the relationship between cerebral cortex and cerebellum in crossed cerebellar diaschisis as well as crossed cerebellar atrophy is a hot topic for discussion. PET scan studies have also revealed remote involvement of the cerebral cortex following cerebellar lesion, which can be considered as the reverse of the cerebro-cerebellar diaschisis.[5,6] The metabolic involvement of the somatosensory postcentral cortex fitted well with some of the neurophysiological findings, suggesting a functional involvement of the primary somatosensory cortex.[7] In the last few years, it has become more and more evident that the cerebellar functions are not only limited to the fine tuning of motor control, but also involve the sensory processing and cognitive functions.[8] With this view, neuroradiological studies could be extremely relevant in disclosing metabolic as well as morphologic abnormalities of the cerebral cortex subsequent to cerebellar damage.

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