

## Editorial

**Crossed Cerebro-Cerebellar Diaschisis\***Andrea Poretti<sup>1,2</sup> Eugen Boltshauser<sup>2</sup><sup>1</sup> Division of Pediatric Radiology, Russell H. Morgan Department of Radiology and Radiological Science, The Johns Hopkins School of Medicine, Baltimore, Maryland, United States<sup>2</sup> Department of Pediatric Neurology, University Children's Hospital, Zurich, Switzerland

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Each cerebellar hemisphere is connected with the contralateral cerebral hemisphere through the cortico-ponto-cerebellar projecting fibers.<sup>1</sup> The densest cortico-ponto-cerebellar projections arise from the precentral and prefrontal cortical areas. Connections between the cerebellar hemispheres and other areas within the contralateral frontal, parietal, and temporal lobes have also been reported. A functional disruption of the cortico-ponto-cerebellar pathways is the underlying pathomechanism of the crossed cerebro-cerebellar diaschisis (CCCD).<sup>2</sup> In neurology, diaschisis refers to the reduction of function of a part of the brain following the interruption of an afferent pathway at a remote site. In rats, reduced neuronal activity in the cerebral cortex was shown to lead to a decrease in Purkinje cell spike activity in the contralateral cerebellar hemisphere.<sup>2</sup> This is explained by deactivation resulting from reduced efferent, excitatory activity in crossed projections from the cerebral cortex to Purkinje cells. This deactivation underlies a depression of neuronal metabolism and blood flow in the affected cerebellar hemisphere as demonstrated using perfusion-weighted imaging and single-photon emission computed tomography.<sup>3,4</sup> CCCD has been reported several times in adults with large hemispheric stroke, epileptic seizures, migraine, encephalitis, and brain tumors. Pediatric reports on CCCD are less common and include children with stroke,<sup>5</sup> seizures,<sup>6–8</sup> Rasmussen's encephalitis,<sup>9</sup> familial hemiplegic migraine,<sup>10</sup> and chronic unilateral cerebral injuries.<sup>11</sup>

In this issue, Koy et al report on a 3.8-year-old girl who developed CCCD of the left cerebellar hemisphere most likely after a non-convulsive status epilepticus involving the right cerebral hemisphere.<sup>12</sup> This case report does not only describe another child who developed CCCD after epileptic

seizures, but a rather unique case of CCCD. Indeed, before the non-convulsive status epilepticus, atrophy of the left cerebellar hemisphere was already present. This is most likely due to CCCD secondary to disruptive lesions related to prematurity within the right cerebral hemisphere. After status epilepticus at the age of 3.8 years, atrophy of the left cerebellar hemisphere increased significantly. This may be explained by the development of CCCD of the left cerebellar hemisphere twice: the first time due to neonatal brain injury, and the second due to status epilepticus.

Seizures, stroke, and chronic unilateral injuries are not uncommon in children. However, CCCD only occurs in few of them. An intriguing question addresses possible determinants of CCCD such as location, size, or severity of the cerebral injury. In adults with stroke, the occurrence of CCCD was shown to correlate with the involvement of the motor (precentral gyrus), premotor, and supplementary motor cortices (superior, middle, and inferior frontal gyri), primary (postcentral gyrus) and secondary (parietal operculum of inferior parietal gyrus) somatosensory cortices, auditory cortex (transverse and superior temporal gyri), temporal association cortex (middle and inferior temporal gyri and fusiform gyrus), basal ganglia, and limbic system (prefrontal cortex, amygdala, hypothalamus, hippocampus, and parahippocampal gyrus).<sup>13</sup> Additionally, a correlation between involvement of different areas of the cerebral cortex and three longitudinal cerebellar zones (medial, intermediate, and lateral) was reported.<sup>13</sup> On the other hand, there is no evidence for a direct relationship between size and severity of the cerebral lesion itself and the occurrence of CCCD. However, of course, a large infarction is more likely to include cortical areas associated with the development of CCCD.<sup>13</sup>

Interestingly, in some patients acute CCCD after stroke or status epilepticus was reversible.<sup>13,14</sup> Indeed, it should be differentiated between acute and chronic CCCD. Acute CCCD

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results from the deactivation of Purkinje cells caused by reduced excitatory input from the cerebral cortex and occurs immediately as response to reduced afferent input. Acute CCCD is a functional process and may be rapidly reversible when the excitatory input to the cerebellum returns (e.g., in the case of supratentorial reperfusion in patients with stroke).<sup>2,13</sup> Persistence of CCCD, however, is an irreversible process leading to transneuronal degeneration and usually resulting in atrophy of the affected cerebellar hemisphere.

Finally, CCCD is not only a remarkable neurophysiological phenomenon elucidating the cortico-cerebellar system, but seems to have a clinical significance. In adult patients with stroke, the degree of acute CCCD was shown to be a quantitative indicator of the long-term functional impairment.<sup>15</sup> In children, however, CCCD may be less important than in adults in terms of biomarker for functional outcome. The age at the time of cerebral injury was shown to be an important factor in the pattern of reorganizational changes within the cerebellum.<sup>11</sup> Damages to the immature brain are in general less disruptive to the overall function of the individual than comparable lesions in adults.

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