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Hemodynamic changes during neural deactivation in human brain: A positron emission tomography study of crossed cerebellar diaschisis

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The mechanism of crossed cerebellar diaschisis (CCD) is considered to be secondary hypoperfusion due to neural deactivation. To elucidate the hemodynamics during neural deactivation, the hemodynamics of CCD was investigated. The cerebral blood flow (CBF), cerebral blood volume (CBV), cerebral oxygen extraction fraction (OEF), cerebral metabolic rate of oxygen ($CMRO_2$), and vascular responses to hypercapnia and acetazolamide stress for CCD were measured in 20 patients with cerebrovascular disease by positron emission tomography with $H_2{}^{15}O$, $C^{15}O$, and ${}^{15}O_2$. Vascular responses to hypercapnia and acetazolamide stress were almost the same between CCD side and unaffected side of the cerebellum, a finding that supports the idea that the mechanism of CCD is secondary hypoperfusion due to neural deactivation. The degree of decrease in CBF on the CCD side was almost the same as that in CBV, indicating that vascular blood velocity does not change during neural deactivation. The relation between CBF and CBV of the CCD and unaffected sides was $CBV = 0.29\ CBF^{0.56}$. On the CCD side, the degree of decrease in $CMRO_2$ was less than that in CBF, resulting in a significantly increased OEF. The increased OEF along with the decreased CBV on the CCD side might indicate that neural deactivation primarily causes vasoconstriction rather than a reduction of oxygen metabolism.

Key words: neural deactivation, hemodynamics, cerebellum, diaschisis, PET