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Autoregulation in the vertebrobasilar system using transcranial Doppler and CO₂ inhalation

Olga Noskin¹, Samuel Trocio Jr¹, Roque Sia¹, Emmanuel Carrera², and Randolph S. Marshall¹

¹Department of Neurology, Columbia University, New York, NY ²Department of Neurology, University Hospital, Lausanne, Switzerland

Loss of cerebral hemodynamic autoregulation plays an important pathophysiological role in brain injury such as stroke.¹ It has been shown that the magnitude of vasodilation in the anterior circulation serves as an indirect measure of cerebral autoregulation and that degree of vasoreactivity loss correlates with subsequent risk of stroke in symptomatic and asymptomatic carotid occlusive disease.^{2–6} Similar data for the posterior circulation have not been consistent. Some evidence suggests that vertebrobasilar circulation may harbor significantly different autoregulative characteristics which could explain its susceptibility to the posterior reversible leukoencephalopathies, hypertensive encephalopathy, immunosuppressive vasculopathy, and pre-/eclampsia.⁷ Our goal was to better characterize the autoregulatory profile of the vertebrobasilar circulation in healthy controls. By simultaneously assessing vasodilatory capacity (CVC) in anterior and posterior circulation, we aimed to establish normative data for the vertebrobasilar system, to compare the two circulations, and to assess for differential contributions of age, gender or blood pressure.

Methods

We recruited healthy adult controls with no history of cerebrovascular disease, hemodynamically significant occlusive disease, or risk factors such as use of vasoactive substances. Continuous transcranial Doppler ultrasound measurements before and after CO₂ inhalation were conducted and mean flow velocities (MFV) were recorded in an MCA on one side and a PCA on the other then switched in a pseudorandomized manner to control for possible CO₂-related potentiation effects. Left and right values were averaged. End-tidal pCO₂ (mm Hg) was measured continuously by a capnometer integrated into the respiratory circuit to capture exhaled breath. After two minutes of baseline monitoring, 5% CO₂ was administered at a flow rate of 15ml/min for each trial to induce hypercapnia, lasting 2 minutes. We measured CVC by the following formula:

$$CVC = \frac{\% \Delta MFV}{\Delta pCO_2}$$

We ran a paired samples t-test comparing anterior and posterior circulation CVC. We also performed Spearman's correlations for the variables of age, gender, baseline MAP, followed by a multiple regression analysis to look for independent contribution of these factors to the CVC.

Results

Thirty two subjects (14 females) were enrolled (mean age = 45, with four subjects over 60). The average CVC in the posterior circulation (2.50 ± 0.90) was not significantly different from that in the anterior circulation (2.69 ± 0.98 ; paired t-test = 0.81, $p=0.43$). No effect of age, gender, or baseline MAP was identified by multiple regression analysis in the anterior ($F 3, 22 = 0.201$ $p=0.90$) or posterior circulation ($F 3, 21 = 0.756$ $p=0.53$).

Conclusion

We demonstrated no significant difference in vasoreactivity profiles of the anterior and posterior circulations in healthy controls, and no variability by age, gender or blood pressure. Despite a relatively young cohort, our study provides normative data from which to investigate vasoreactivity profiles in diseases affecting the posterior circulation, such as atherosclerosis and the PRES spectrum. Future studies may reveal that in older patients or in patients with cerebrovascular conditions, autoregulatory processes may differ in the posterior circulation, supporting the hypothesis that a differential susceptibility of this vascular territory to certain diseases may be mediated by a physiological difference in autoregulatory responses.

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