

Letter to the Editor

Cerebral Vasoreactivity in Children with Sickle Cell Disease: A Transcranial Doppler Study

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I read with great interest the original article by Macedo-Campos et al¹ titled "Cerebral Vasoreactivity in Children with Sickle Cell Disease: A Transcranial Doppler Study." Authors have explored the cerebrovascular reactivity assessed by breath-holding index (BHI) in the children with sickle cell disease (SCD). They have reported the impaired cerebrovascular reactivity in the children with SCD as compared to that of control subjects. However, few potential methodological limitations need to be addressed regarding this study.

First flaw was in the assessment of BHI. It was calculated as the percentage change in cerebral blood flow velocity. However, it is now an established fact that cerebrovascular reactivity should be calculated as the percentage or absolute changes in cerebrovascular conductance rather than in the changes in cerebral blood flow velocity.^{2,3} Assessment of cerebrovascular reactivity by percentage change in cerebrovascular blood flow velocity may not nullify the confounding effect of systemic blood pressure. Breath holding manoeuvre itself is associated with significant changes in systemic blood pressure. This change in systemic blood pressure may affect cerebral blood flow velocity, in addition to that of caused by reactivity of cerebral vessels. Calculation of cerebrovascular reactivity by changes in cerebrovascular conductance minimizes the confounding effect of systemic blood pressure over cerebrovascular reactivity.

Also, the baseline cerebral blood flow velocities in SCD group were significantly higher than in the control group, calculation of BHI as percentage changes rather than absolute or delta change may underestimate the dilatory capacity of the cerebral vasculature. Therefore, it is suggested to estimate the cerebral vasodilatory capacity by calculating absolute or delta changes rather than the percentage changes, especially in the patient groups, where we assume that the cerebral vasodilatory reserves are being utilized even at the resting stage, and cerebral autoregulation is impaired.⁴

A third flaw, also mentioned by the authors that PaCO₂ was not measured. Measurement of PaCO₂ is essential to ascertain that the impaired cerebrovascular reactivity is whether because of impaired vasodilatory capacity of cerebral vessels or insufficient rise of PaCO₂ during breath holding manoeuvre.

A minor clarification is also required for Table 2 of the said article (1). It shows the "Comparison of the baseline velocities (V_{mean}), final velocities (V_{max}), and the calculated BHI following breath-holding test in the subjects and controls." Kindly clarify that whether the "Subjects" here, refers to all the participants of the study or only to those of the SCD-group.

I congratulate Macedo-Campos et al for this informative study which addresses the cerebrovascular reactivity in the children with SCD. However, health professional and clinicians should interpret this study with caution, because of its potential shortcomings.

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