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To the Editor: In the January issue of this journal, Bassan and colleagues reported disturbed cerebral autoregulation in 5/ 43 (13%) critically ill infants with congenital heart disease who underwent cardiopulmonary bypass surgery (1). Spontaneous changes in MAP, NIRS, and CBFV were correlated to quantify the pressure passive index (PPI 0 = intact autoregulation). In this patient population, the task is technically challenging, and the investigators should be commended for studying cerebral autoregulation in this group of patients at risk of long-term neurologic sequelae. While the authors have correctly stated that doing tilt or static testing in these children is problematic, they should have discussed the limitations of the methodology used in this study to determine autoregulatory capacity. The difficulty in quantifying the incidence of impaired autoregulation using spontaneous correlation analysis of physiologic variables is that one cannot be sure that the autoregulatory system was indeed stimulated and that the changes in CBFV or NIRS were due to changes in MAP. Cerebral autoregulation is a homeostatic system controlled by a feedback loop with as yet undefined metabolic/neural mechanism. To trigger this homeostatic mechanism, a sufficient stimulus must be furnished. At the same time, the stimulus must not be coupled with other factors that may alter blood flow independently. For example, if cerebral metabolic rate is increased for whatever reason during the study, MAP and CBFV will all increase, and cerebral oxygen saturations may or may not increase due to flow metabolism coupling. Since cerebral metabolic rate was neither measured nor specifically controlled during the study period, one cannot be sure that the observed phenomenon represents impairment of the autoregulatory process rather than preservation of flow metabolism coupling. This criticism is not to detract the potential value of these autoregulation studies, but we must be cognizant of the limitations of the method, which in our opinion, at best provide a qualitative rather then quantitative assessment of cerebral autoregulation. The authors also report increased odds of impaired autoregulation with hypercapnea ($ET\text{-}CO_2 \geq 40$ mm Hg). However, in critically ill patients, $ET\text{-}CO_2$ may underestimate $PaCO_2$. An analysis of the relationship between $PaCO_2$ and autoregulatory capacity would provide more useful information.

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Response

To the Editor: We thank Drs. Vavilala and Lam for their interest in our recent report and for their insightful comments. However, there appears to be misunderstanding of the methodology used in our study. The authors are correct in stating that reliance upon spontaneously occurring blood pressure changes to assess the efficacy of cerebral pressure autoregulation leaves uncertainty about whether the cerebral autoregulatory system has been adequately tested. This is only true in situations where there is lack of concordance between blood pressure (BP) and cerebral blood flow (CBF). However, in situations when there is significant frequency-specific coherence between changes in BP and those in CBF, it is reasonable to infer that the changes in BP are causally related to the changes in CBF. It is for precisely this reason that we emphasized in this report (and in its title) that we were testing not for the presence of autoregulation but rather its absence, *i.e.*, the pressure-passive state. We agree with Drs. Vavilala and Lam that CBF is influenced by a host of other factors, including cerebral metabolic rate (CMR). It is likely that at any given time a variety of stimuli are exerting an influence on the regulation of CBF. For these reasons, we have used the systems analysis approach together with coherence and transfer function analysis to better discriminate between the effects on CBF of changes in BP and changes in other "input" stimuli. It is well known that under normal conditions, changes in CMR trigger changes in CBF. However, unless CMR and BP were changing at the exact same frequency, changes in CMR would not influence the coherence between BP and CBF.

Finally, Drs. Vavilala and Lam quite correctly state that the output of an end-tidal CO2 (ET-CO2) monitor may not correlate accurately with measured arterial PaCO2. As other techniques for continuous PaCO2 measurement are, unfortunately, not well established, this remains a reliable and useful trend monitor. As stated in the Methods section of our paper, we adjusted the ET-CO2 values to measured arterial PaCO2 levels at the beginning and end of the studies.

Thank you for the opportunity to clarify these issues.

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