Correlation of end-tidal and arterial carbon-dioxide levels in critically Ill neonates and children

Sir,

We thank Jindal and Singha for having a keen interest in our article "Correlation of end-tidal and arterial carbon-dioxide levels in critically Ill neonates and children" and bringing attention to a few important points.[1]

First, we would address the typographical error. We had excluded 21 patients, including 6 patients having gestational age <32 weeks, not 9, as reported in the flowchart. In addition, 2 pairs of measurements were excluded due to contamination of samples with the line fluid. We thank the authors for bringing this to our attention. Study was limited to duration of 6 months, rather than the number of patients for feasibility issues.

Jindal *et al.,* mentioned that a large surgical shunt is related to stiffer lungs, and higher respiratory resistance is makes end-tidal monitoring unreliable. Our 12% newborns had left-to-right shunts of varying size that may or may not have contributed to the severity of lung disease. It is a commonly encountered condition that we elected to include to represent the overall population of patients in neonatal intensive care unit for the purpose of generalizability of application of our results in this age group. We are looking at the impact of severity of lung disease on correlation of end-tidal and arterial carbon-dioxide levels irrespective of underlying etiology that may be cardiac or noncardiac. Jindal *et al.,* have also stated that decreased pulmonary-to-systemic shunt ratio increases arterial to end-tidal carbon-dioxide difference secondary to pulmonary hypoperfusion. We would like to emphasize that left-to-right shunts do not decrease, but increase pulmonary-to-systemic shunt ratio (Qp/Qs). Cyanotic lesions with low Qp/Qs were not part of our study.

Jindal *et al.,* had a concern that vasoactive therapy in 68% of newborns could have decreased the pulmonary blood flow and adversely affected the relationship of $EtCO_2$-$PaCO_2$. This was a reasonable assumption, since vasoactive agents may have differential influence on regional blood flows despite improvement in cardiac output, blood pressure and myocardial performance. However, authors did not provide us any evidence in favor of their assumption. Nevertheless, we would like to cite a study conducted in postoperative cardiac surgery patients to evaluate the stability of $PaCO_2$-$EtCO_2$ gradient during vasoactive therapy.[2] Investigators reported a normal population distribution of the *P* (A-a) CO_2 gradients. Thus, we do not concur with the idea that vasoactive agents could have adversely affected $PaCO_2$-$EtCO_2$ gradient.

The Jindal *et al.,* presume that low tidal volume strategy would have been used in ventilating the neonates, and dead space of 6 ml in mainstream end-tidal monitor would have been large enough to confound the study results. We ventilated all our newborns with pressure-controlled ventilation strategy as it allows a more reliable compensation of breathing circuit compressible volume; tidal volume does not remain constant for obvious reasons.[3,4] Moreover, the mainstream analyzer gives a more accurate representation of the expired CO_2 waveform in small children at rapid respiratory rates.[5]

We conclude that the inclusion of patients with left-to-right shunt was an appropriate methodological step; there is little evidence to suggest that vasoactive therapy could alter *P* (A-a)CO_2 gradient; and pressure-controlled ventilation with mainstream analyzer is a good option to monitor $EtCO_2$ in small subjects.

Hiren Mehta, Rahul Kashyap[1], Sangita Trivedi[2]

Clinical Fellow, Pediatric Intensive care, Royal Manchester Hospital, Manchester, UK, [1]Department of Anesthesia and Critical Care Medicine, [2]Fellow, Department of Pediatric Critical Care, Mayo Clinic, Rochester, Minnesota, USA

Correspondence:
Dr. Sangita Trivedi,
Department of Pediatric Critical Care, Mayo Clinic Children's Center, 200 First St., SW, Rochester, Minnesota 55901, USA.
E-mail: trivedi.sangita@mayo.edu

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Quick Response Code:	**Website:** www.ijccm.org
	DOI: 10.4103/0972-5229.146342