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

Sir,

We read with great interest the study titled "Correlation of end tidal and arterial carbon dioxide levels in critically ill neonates and children" by Mehta *et al*.[1] We wish to highlight certain important issues.

It is not clear how the sample size was calculated for this study to reach to a fruitful conclusion as this was probably not a pilot study. We believe that without *a priori* calculation of sample size, it would be really impossible to draw conclusions.

We observed a major discrepancy regarding the number of patients being enrolled and excluded from the study as evident from the flow chart in Figure 1 of the article published. In the neonatal arm, authors have excluded 21 patients, whereas the sum of excluded patients is 24. From the data provided by you, the number of patients would be 63 instead of 66, which would change the statistics as reported. Similarly, in the infant and children arm, of 10 pairs that were excluded, the cause of exclusion of two pairs is missing.

In the neonatal arm, there were eight newborns (12%) suffering from congenital heart disease with left to right shunt. A large surgical shunt size is related to stiffer lungs and a large heart is associated with a higher respiratory resistance. During mechanical ventilation of patients, the end-tidal CO_2 may be an unreliable substitute for arterial CO_2. Studies have found a relationship between a decreased pulmonary to systemic shunt ratio and an increased arterial to end-tidal CO_2 difference. This may indicate that a reason for the unreliability of end-tidal CO_2 is an impaired gas exchange partially due to pulmonary hypoperfusion.[2]

In the neonatal group, 68% of newborns were on vasoactive therapy. It is well established that the vasopressors actually decrease the pulmonary blood flow (PBF), which would have an adverse effect on the $PaCO_2$ and $PEtCO_2$ relationship. Also, it would have been more appropriate if the authors could have shown the dosage of vasopressors in the presence of shock/chronic heart failure in the severe acute respiratory distress syndrome group as it would have a significant bearing on the PBF and thereby on the ventilation.

In newborns, mainstream sampling of the exhaled gas to monitor the $EtCO_2$ was used, which has a dead space of around 6 mL. It is noteworthy that the mean weight of the neonate arm is 2.1 kg only as mentioned. In all the patients, we presume that a low tidal volume approach would have been applied as the ventilation strategy. Thus, the dead space would account for almost 40–50% of the exhaled air being

analyzed by a mainstream sampler. We feel that low flow side-stream sampling would have been a more appropriate sampling tool in the neonate arm of this study as the significant dead space would be a confounding factor with regard to the result of the study.[2-7]

Almost all the limitations highlighted are independent variables known to disrupt the positive correlation between $PaCO_2$ and $EtCO_2$. Hence, in our opinion, it would not be rational to conclude strongly that increased severity of lung disease as defined by P/F ratio < 200 negatively affects $EtCO_2$ values in all age groups.

Atul Jindal, Subrata Kumar Singha[1]
Departments of Pediatrics and [1]Anaesthesia and Critical Care, AIIMS, Raipur, India

Correspondence:
Dr. Subrata Kumar Singha,
Department of Anaesthesia and Critical Care, AIIMS, Raipur - 492 099, India.
E-mail: subratsingh@gmail.com

References

1. Mehta H, Kashyap R, Trivedi S. Correlation of end tidal and arterial carbon dioxide levels in critically ill neonates and children. Indian J Crit Care Med 2014;18:348-53.
2. Matthews IL, Bjornstad PG, Kaldestad RH, Heiberg L, Thaulow E, Gronn M. The impact of shunt size on lung function in infants with univentricular heart physiology. Pediatr Crit Care Med 2009;10:60-5.
3. Badgwell JM, Heavner JE, May WS, Goldthorn JF, Lerman J. End-tidal PCO_2 monitoring in infants and children ventilated with either a partial rebreathing or a non-rebreathing circuit. Anesthesiology 1987;66:405-10.
4. Kirpalani H, Kechagias S, Lerman J. Technical and clinical aspects of capnography in neonates. J Med Eng Technol 1991;15:154-61.
5. Gravenstein N. Capnometry in infants should not be done at lower sampling flow rates. J Clin Monit 1989;5:63-4.
6. Sasse FJ. Can we trust end-tidal carbon dioxide measurements in infants? J Clin Monit 1985;1:147-8.
7. Gravenstein JS, Paulus DA, Hayes TJ, editors. *Capnography in clinical practice* Stoneham, MA: Butterworth Publishers; 1989. p. 43-9.
8. Schieber RA, Namnoum A, Sugden A, Saville AL, Orr RA. Accuracy of expiratory carbon dioxide measurements using the coaxial and circle breathing circuits in small subjects. J Clin Monit 1985;1:149-55.

Access this article online	
Quick Response Code:	Website: www.ijccm.org
	DOI: 10.4103/0972-5229.142183