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

Sir,

We thank Jindal and Singh 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 EtCO₂-PaCO₂. 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 PaCO₂-EtCO₂ gradient during vasoactive therapy.2 Investigators reported a normal population distribution of the PaCO₂-EtCO₂ gradients. Thus, we do not concur with the idea that vasoactive agents could have adversely affected PaCO₂-EtCO₂ 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 CO₂ 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 PaCO₂-EtCO₂ gradient; and pressure-controlled ventilation with mainstream analyzer is a good option to monitor EtCO₂ in small subjects.

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Successful management of acute kidney injury in severe acute pancreatitis with intra-abdominal hypertension using peritoneal dialysis

Sir,

This letter is written to emphasize the edge of acute peritoneal dialysis (APD) over hemodialysis in the management of acute kidney injury (AKI) in severe acute pancreatitis (SAP) with intra-abdominal hypertension (IAH) through a case we encountered recently.

The 35-year-old chronic alcoholic male, presented with acute abdominal pain and repeated episodes of nonbilious vomiting, following an alcohol binge. He was diagnosed to have hemorrhagic SAP by clinical features, lab investigations and imaging. He developed severe AKI with anuria within 48 h of admission and was initiated on hemodialysis. Hypotension was not recorded throughout the course of illness. He developed tense ascites by the end of 1st week and was deteriorating with persistent systemic inflammatory response syndrome, and anuria despite hemodialysis and paracentesis. His intra-abdominal pressure (IAP) was recorded to be 26 mmHg.

He was shifted to APD using rigid catheter mainly to decompress the abdomen gradually using tidal exchanges as well as based on the fact that peritoneal dialysis helps in the removal of bioactive substances presumed to be responsible for systemic inflammation associated with acute pancreatitis. [1] IAH is known to reduce the blood supply to the abdominal organs, including the kidneys and is a common cause of AKI.[2] Exact incidence of IAH is not clearly known as IAP is not measured routinely. IAP >15 mmHg is associated with higher mortality.[3] Endotoxins and reactive oxygen species also play an important role in the pathophysiology of alkaline phosphatase and AKI.[4] Percutaneous drainage is the preferred initial therapy in the presence of ascites or pseudocyst. Decompressive laparotomy is the most effective way of relieving IAP. In stable patients with infected necrosis, surgical, radiologic, and/or endoscopic drainage should be delayed, preferably for 4 weeks, to allow the development of a wall around the necrosis.[5] We suggest APD as the preferred modality of renal replacement therapy in patients with SAP with AKI and IAH.

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