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Letter to the Editor

Which partial pressure of carbon dioxide during extracorporeal cardiopulmonary resuscitation (ECPR)?

Dear Sir,

We read with interest the paper by Kilgannon et al.[1] on the association between partial pressure of arterial carbon dioxide and neurological outcome following cardiac arrest. Although their study focusses on patients achieving recovery of spontaneous circulation we are seeing greater use of extracorporeal life support (ECLS) in patients with refractory cardiac arrest,[2] with the term ECPR used. We believe that the question of how best to manage CO_2 is also important in this population of patients, with further evidence linking a reduction in arterial CO_2 and mortality.[3] As blood is often sampled from the ECLS circuit as well as from the patient's arterial line we sought to investigate the importance of blood sampling site on manipulation of partial pressure of CO_2 in patients on ECLS.

During veno-arterial ECLS blood drains via a large central vein into an extra-corporeal circuit, passes through an oxygenator and is pumped back into the aorta. A flow of gas across the oxygenator (sweep) results in CO_2 clearance, as well as oxygenation.

We prospectively evaluated CO_2 values in a series of children undergoing ECPR following in-PICU cardiac arrest. 23 patients (mean (SD) age 1.9 (2.9) years) underwent 24 runs of ECPR. Underlying diagnoses were congenital cardiac disease (85%), drowning, myocarditis, and sepsis. Blood-gas sampling was undertaken at two circuit points – pre-oxygenator ($Ppre\text{-}oxyCO_2$), post-oxygenator ($Ppost\text{-}oxyCO_2$) – and from the patient's arterial line ($PaCO_2$). Our local ECPR protocol instructs changes to sweep gas flow based on $Ppre\text{-}oxyCO_2$, with a target of 4.5–5.0 kPa.

$PaCO_2$ recorded before ECPR was generally high (median 8.0 kPa, range 1.2–20.0). Within 6 h of ECPR this had fallen to median 4.5 kPa (range 3.1–7.3). Median reduction in $PaCO_2$ following initiation of ECPR was 3.7 kPa, 42% experienced a reduction >5 kPa in the first hour (maximum 12.7 kPa).

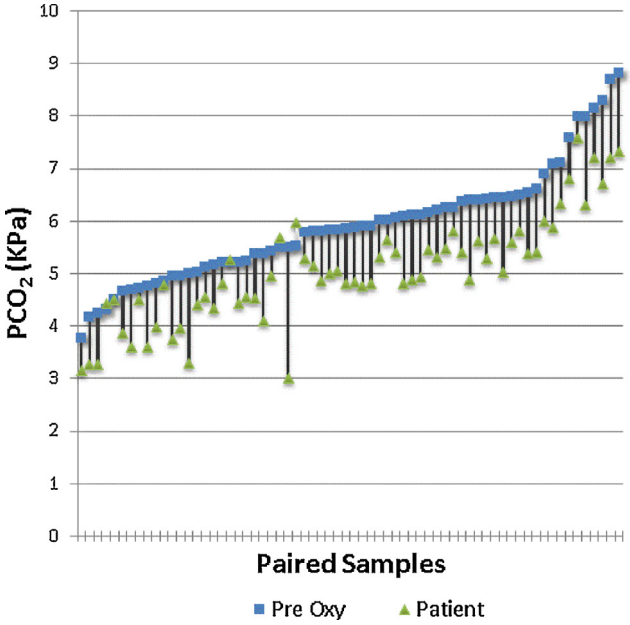

Fig. 1 – Comparison of PCO_2 from pre-oxygenator ($Ppre\text{-}oxyCO_2$) blood with paired patient arterial blood ($PaCO_2$) in samples taken in first 24 h of ECPR. The data are presented in ascending order of $Ppre\text{-}oxyCO_2$).

The relationship between P*pre-oxy*CO_2, P*post-oxy*CO_2 and P*a*CO_2 was not consistent or predictable. The use of P*pre-oxy*CO_2 values to guide sweep flow was frequently associated with low P*a*CO_2 values (Fig. 1); over the first 24 h 38% of P*a*CO_2 samples were below 4 kPa, and 25% of patients experienced a P*a*CO_2 below 3.5 kPa.

A rapid reduction in P*a*CO_2 may occur soon after initiation of ECPR. P*pre-oxy*CO_2 and P*post-oxy*CO_2 should not be used as surrogates of P*a*CO_2 and sweep gas flow should be adjusted based on P*a*CO_2. The implications of rapid changes in P*a*CO_2 for ischaemia-reperfusion and the cerebral circulation are unknown and require further study.

Conflict of interest

We can declare that we do not have conflicts of interest.

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