Correspondence


Oxygenator exhaust capnography as an index of arterial carbon dioxide tension during cardiopulmonary bypass using a membrane oxygenator

Editor—We were interested in the paper by O’Leary and colleagues1 who found oxygenator exhaust capnography during cardiopulmonary bypass was not useful for estimating arterial carbon dioxide tension ($P_{a\text{CO}_2}$).

The authors hypothesized that it would be important to temperature correct their data. Unfortunately, they used nasopharyngeal temperature which may not be the most appropriate; the temperature of the blood at the point that carbon dioxide is being evolved may be more logical. This is not difficult in practice, the temperature of arterial blood at the oxygenator outlet is (or should be) routinely measured during cardiopulmonary bypass.

To test our hypothesis we observed 19 patients undergoing non-pulsatile hypothermic (28°C) cardiopulmonary bypass using a Terumo Capiox SX membrane oxygenator and a Criticare POET IQ capnograph, taking 38 samples during cooling and rewarming, and using Dr O’Leary’s method with one modification—blood gases were corrected to the temperature of arterial blood rather than nasopharyngeal temperature.

The 95% limits of agreement between temperature corrected $P_{a\text{CO}_2}$ and partial pressure of CO$_2$ in the oxygenator exhaust gas ($P_{E\text{CO}_2}$) was $-0.54$ kPa to $+0.78$ kPa. The coefficient of determination between temperature corrected $P_{a\text{CO}_2}$ and $P_{E\text{CO}_2}$ was 0.95 (Fig. 1). There was a tendency for $P_{E\text{CO}_2}$ to under-read during hypothermia (Fig. 2). Of interest, when blood gases were corrected to nasopharyngeal temperature, the 95% limits of agreement were $-2.97$ kPa to $+3.0$ kPa which is an inaccuracy similar to that described in O’Leary’s paper.

These observations suggest oxygenator exhaust capnography may allow real-time, continuous estimation of $P_{a\text{CO}_2}$ with clinically useful accuracy, provided that it is corrected to arterial blood temperature, not the temperature of the nasopharynx.

W. M. Weightman
M. R. Sheminant
Department of Anaesthesia
Sir Charles Gairdner Hospital
Perth, Australia

Editor—Thank you for the opportunity to respond to the letter of Weightman and Sheminant, who should be congratulated for rapidly performing their study in response to our paper. As discussed in the opening paragraphs of our paper, we set out to determine whether monitoring exhaust gas carbon dioxide partial pressure ($P_{E\text{CO}_2}$) would be clinically useful during cardiopulmonary bypass; that is to say could measuring $P_{E\text{CO}_2}$ take the place of blood gas monitoring? We did not hypothesize, however, that temperature correction of arterial blood gases would improve the agreement between the two measures ($P_{a\text{CO}_2}$ vs $P_{E\text{CO}_2}$). Having noted that there was poor agreement with $P_{a\text{CO}_2}$ uncorrected to nasopharyngeal temperature, we then went on to investigate whether temperature correction might improve the level of agreement. We agree that measuring temperature at the point where gas exchange is occurring, rather than in the nasopharynx, is a logical step, and Weightman and Sheminant claim to have obtained reasonable agreement between the two measures when this technique is employed. Although this is clearly of interest, we would disagree with their statement that ‘oxygenator exhaust capnography may allow real-time, continuous estimation of $P_{a\text{CO}_2}$ with clinically useful accuracy, . . .’ In modern perfusion practice, using alpha stat blood gas correction, the uncorrected $P_{E\text{CO}_2}$ is the variable of interest and other measurement methods are only useful if they reflect this value accurately.

We do have a few concerns with the data presented by Weightman and Sheminant, however. Only the relationship between $P_{a\text{CO}_2}$ corrected to either nasopharyngeal or arterial blood temperature and $P_{E\text{CO}_2}$ are reported. We would be interested to know the relationship between uncorrected $P_{a\text{CO}_2}$ and $P_{E\text{CO}_2}$ in their study—was the agreement between these measures similar to our findings? Plots of the difference between the two measures vs the average of the two measures for corrected and uncorrected blood gases using both nasopharyngeal and arterial blood temperature measurements would be informative in a way that Figures 1 and 2 are not. In Figure 2, the relationship between the difference in the two measures (corrected $P_{a\text{CO}_2}$ and $P_{E\text{CO}_2}$) and temperature is shown; why are the hypothermic measures all at

---

Fig 1 Relationship between the partial pressure of carbon dioxide in arterial blood ($P_{a\text{CO}_2}$) and the partial pressure of carbon dioxide in oxygenator outlet exhaust ($P_{E\text{CO}_2}$).

Fig 2 Relationship between arterial blood temperature and the difference between the partial pressure of carbon dioxide in arterial blood ($P_{a\text{CO}_2}$) and the partial pressure of carbon dioxide in oxygenator outlet exhaust ($P_{E\text{CO}_2}$).
very low temperatures (20–27°C) when in the text the cooling temperature is stated at 28°C? Furthermore, why are there apparently 18 measurements in the upper temperature range and 20 in the lower range when 19 patients were studied? The absence of any measurements between 27°C and 34°C, where the majority of our measurements were made, makes comparison with our data, presented in the same format, impossible.

M. J. O’Leary
Intensive Care
St George Hospital
New South Wales, Australia

S. P. MacDonnell
Anaesthesia and Intensive Care
Colchester Hospital
Colchester, UK

C. N. Ferguson
Anaesthesia and Intensive Care
Derriford Hospital
Plymouth, UK

Perioperative hypotension following plasma volume expansion with albumin in an angiotensin-converting enzyme inhibited infant

Editor—Perioperative hypotension is usually due to hypovolaemia and often requires treatment with plasma expanders. Unfortunately, plasma expanders may themselves cause hypotension.1 Which, when and how to use plasma expanders is a matter of a continued debate. Historically, the plasma expander of choice in paediatric practice is albumin, and in particular in paediatric renal transplant patients where a poorer graft survival has been demonstrated with synthetic colloids.2,3

We wish to report a case of severe hypotension associated with a bolus dose of 4% albumin (Albumex 4% – Commonwealth Serum Laboratories) during anaesthesia in an infant scheduled for a living-related donor kidney transplant. The infant was a 9.8 kg 20-month-old female with chronic renal failure due to tubular interstitial nephritis. She was on peritoneal dialysis and her medication included captopril 2.5 mg tds, sodium carbonate, calcitriol, pentavite, iron, folate, recombinant erythropoietin, prednisolone, cyclosporin A and mycophenolate. General anaesthesia was induced with thiopental 50 mg, atracurium 8 mg and fentanyl 20 µg. Anaesthesia was maintained with isoflurane 1% in N2O 50%/O2. A double-lumen 5 FrG central venous catheter, a radial intra-arterial catheter and a low thoracic epidural (T11/T12) were then inserted. Arterial pressure remained stable at 90–100 mm Hg systolic, with a central venous pressure of 7 mm Hg and a heart rate of 110 beats min⁻¹. The arterial pressure fell to 85–90 mm Hg after establishment of the epidural block with 7 ml of 0.25% bupivacaine over 15 min, with no change in heart rate or CVP. For 35 min while awaiting the arrival of the donor kidney, the patient remained haemodynamically stable. Preoperatively, it had been decided to increase her plasma volume in anticipation of surgery, which involved anastomosis of the donor renal artery and vein to the aorta and inferior vena cava respectively. A bolus dose of 20 ml Albumex 4% was given via the central line over a 2-min period and the arterial pressure fell within 1 min to 50–55 mm Hg systolic with no change in heart rate or CVP.

While excluding other causes of hypotension, 250 ml of normal saline was rapidly infused over 10 min resulting in a slight increase in systolic arterial pressure to 70–75 mm Hg and a rise in CVP to 10 mm Hg. A dopamine infusion of 5–10 µg kg⁻¹ min⁻¹ was commenced with a return of systolic arterial pressure to 90 mm Hg 10 min later. During the remainder of the operation, fluid management consisted of crystalloids and packed red blood cells neither of which elicited any hypotensive episodes. A further epidural top-up of 0.25% bupivacaine 3 ml at the end of surgery did not result in any change in blood pressure. The child was extubated and transferred to the paediatric intensive care unit for further management. A postoperative chest x-ray did not show any sign of a pneumothorax.

A sample was taken from the albumin bottle (lot no. 3420000083) and analysed for prekallikrein activating factor (PKA) levels at CSL Bioplasma Ltd. The PKA concentration in the albumin solution was found to be 10 IU ml⁻¹, which is less than the highest required limit for release in Australia (<28.6 IU ml⁻¹).

This case report demonstrates that the administration of albumin in children receiving angiotensin converting enzyme inhibitors may produce unwanted hypotension, despite albumin solution PKA levels within manufacturer specifications. It was fortuitous that the albumin administration occurred during haemodynamic stability rather than during a period of significant intravascular fluid shifts in which distinguishing the cause of the persistent hypotension may have been difficult.

ACE inhibitors prevent the conversion of angiotensin I to angiotensin II as well as the biodegradation of bradykinin. Albumin solutions contain low levels of prekallikrein activating factor, which activates bradykinin. In the presence of ACE inhibition, the plasma half-life of bradykinin is significantly prolonged, thus administration of prekallikrein activator may result in significant and prolonged hypotension.4 This effect is, however, unpredictable as plasma ACE levels are highly variable and the amount of bradykinin produced from ACE inhibition may not cause significant hypotension. It has been suggested that a rapid rate of infusion (30–40 ml min⁻¹) of albumin 5%, for example in plasmapheresis, can cause a significant rise in bradykinin levels to produce flushing and hypotension in adult patients taking ACE inhibitors.5

Our patient was given her normal dose of captopril the night before surgery. As renal failure can significantly prolong the half-life of captopril, from 2 h up to 40 h,6 plasma ACE would have been inhibited at the time of surgery. The albumin was also administered rapidly (1 ml kg⁻¹ min⁻¹) via a central route and in combination with ACE inhibition may have produced a clinically significant rise in bradykinin level to account for the hypotension.

As albumin solutions are often used to maintain intravascular volume in renal transplantation, it may be prudent to stop the use of ACE inhibitors 24 h prior to surgery (if possible)—or to avoid the use of albumin in patients on ACE inhibitors in order to avoid such adverse reactions.

S. Y. Fong
T. G. Hansen
Department of Paediatric Anaesthesia
Princess Margaret Hospital for Children
Subiaco, Perth, Western Australia

1 Walker SR, MacSweeney ST. Plasma expanders used to treat or prevent hypotension can themselves cause hypotension. Postgrad Med J 1998; 74: 492–4