

Transcutaneous monitoring: back to the future - An important adjunct to care during high frequency oscillatory ventilation

High frequency oscillatory ventilation (HFOV) is often used in neonatal intensive care. HFOV has been shown to decrease bronchopulmonary dysplasia [1,2] in preterm infants and to be very effective in the treatment of persistent pulmonary hypertension of the newborn when used in conjunction with inhaled nitric oxide [3]. Other uses include pulmonary hypoplasia, air leak, and ventilation after abdominal surgeries such as gastroschisis closure.

Effective use of HFOV requires close attention to lung volume, with use of an “optimal volume” strategy to open the lung and maintain it open [4]. Mean airway pressure is adjusted to minimize $FO_2(I)$ requirement without evidence of under- or overdistention on chest X-ray. Continuous pulse oximetry assists with adjustments of mean airway pressure and $FO_2(I)$.

Continuous assessment of CO_2 is also very important during HFOV. The oscillator is a powerful machine that can quickly drive arterial CO_2 to unsafe levels. Evidence is accumulating that suggests cerebral damage may result from hypocarbia [5,6]. In addition, hypercarbia may likewise be damaging to the preterm brain, causing disruption in cerebral autoregulation and increased incidence of intraventricular hemorrhage [7,8]. Many infants on HFOV have indwelling arterial lines; however, very frequent blood draws may be necessary to appropriately monitor CO_2 changes, leading to increased infection risk and/or anemia. A non-invasive, continuous estimate of pCO_2 during HFOV would be safer and more effective. Transcutaneous monitoring can provide this estimate.

Transcutaneous (tc) monitoring is not new; it has been available for well over twenty years [9]. Early machines were cumbersome and difficult to use. Accurate $tcPO_2$ assessment necessitated heating the skin to 43 °C, which often led to skin burns in small preterm infants. After the advent of pulse oximetry, use of tc monitoring faded in most NICUs. Unfortunately, this led to “throwing the baby out with the bathwater”, as $tcPCO_2$ monitoring also dramatically decreased despite the lack of a replacement for CO_2 monitoring such as pulse oximetry.

Currently available tc monitors are small and easy to use. Importantly, they can be used to monitor both $tcPO_2$ and $tcPCO_2$, or either one separately. Even more importantly, use of $tcPCO_2$ alone can accurately be done at a monitor temperature of 40 °C or less, thus not causing skin burns [10], and site changes can be done as infrequently as every six to eight hours. The machine must simply be calibrated at the appropriate temperature.

The $tcPCO_2$ will correlate with the $pCO_2(aB)$ – that is, as one goes up the other goes up; as one goes down the other goes down. The “closeness” of the numbers will depend on the thickness of the skin and the perfusion of the site. The numbers are seldom identical, as they measure different things: one measures the $pCO_2(aB)$ of arterial blood and the other the CO_2

diffusing from the cutaneous tissue. The numbers, however, will correlate (trend together).

It is important to periodically correlate tcP_{CO_2} values with arterial blood gas samples or well-done capillary samples. Correlation need not be done with each site change if the tcP_{CO_2} value is similar to that obtained prior to the site change. However, perfusion will vary somewhat from site to site, and thus the "closeness" of the numbers may also vary. A value widely discrepant from previous values should prompt a blood gas.

A slowly rising or decreasing tcP_{CO_2} should always be considered a patient problem until proven otherwise. Something often forgotten is that an increasing tcP_{CO_2} may of and by itself indicate decreasing perfusion in the patient – perhaps sepsis or impending shock. Though the tcP_{CO_2} will still trend correctly, the tcP_{CO_2} will be considerably higher than the $p_{CO_2}(aB)$ in a patient with significant circulatory compromise. In these cases the underlying cause of the problem must be treated. A decreasing tcP_{CO_2} indicates improving ventilation. Changes to ventilator settings can be easily made based on tcP_{CO_2} alone; blood gases can be done much less frequently.

"Something is wrong with the machine" is unfortunately often heard before evaluation of the patient has been done. A recent article, for example, documented the value of a rising tcP_{CO_2} in alerting staff to a pneumothorax well before acute decompensation of the patient [11]. A steadily rising or falling tcP_{CO_2} should prompt careful attention to reasons for under- or overventilation, not an immediate recalibration of the monitor or, worse, turning a blind eye to the readouts because "the machine is not working".

Troubleshooting the tc monitor is relatively easy. The calibration cylinder must contain sufficient gas and must be turned on during calibration. The cable must be intact. The sensor must be remembraned as per the manufacturer's recommendations. Sufficient contact fluid must be placed between the skin and the sensor. Recalibration should be done every six to eight hours if only tcP_{CO_2} is being used. We have found every six hours to be best in this circumstance; towards eight hours the contact fluid tends to evaporate, leading to spurious values. The sensor site should be changed every three to four hours if both tcP_{O_2} and tcP_{CO_2} are utilized. Heating of the sensor to 43 °C is needed if the tcP_{O_2} is employed, and the site must be changed more frequently to avoid skin burns. A tcP_{CO_2} value of 0 or tcP_{O_2} of about 150 means the sensor has dislodged or an air bubble is under the sensor. These are the values expected for room air. TcP_{CO_2} values that jump about wildly indicate need for recalibration/remembraning. Steadily rising or falling values reflect patient status.

Though pulse oximetry has largely replaced the need for tcP_{O_2} monitoring, tcP_{O_2} monitoring can provide useful and complimentary information should the practitioner choose to use it. High $p_{O_2}(aB)$ should be avoided in most cases [12, 13]. Because of the shape of the oxygen-hemoglobin dissociation curve, an oxygen saturation in an acceptable range could be associated with a $p_{O_2}(aB)$ that is unnecessarily high. By the same token, a low or borderline saturation might be associated with an acceptable $p_{O_2}(aB)$ because of shifts in the oxygen-hemoglobin dissociation curve and varying amounts of fetal hemoglobin. TcP_{O_2} monitoring can be very useful in titrating the $F_{O_2}(I)$. Use of tcP_{O_2} monitoring requires more frequent site changes and close attention to the baby's skin to avoid burns. In most cases, however, the "burn" is a reddened area just under the sensor that heals without residua. Occasionally, tiny scars can result if the sensor temperature is too high or the sensor is left on the skin for too

long.

For a patient being started on HFOV, the $tc\dot{p}CO_2$ monitor should be placed on the patient prior to instituting HFOV. Once the $tc\dot{p}CO_2$ is stable and a correlating ABG has been obtained, HFOV can be started and the amplitude adjusted using the tc monitor. Severe hypocapnia, such as can occur with an inadvertently high amplitude or postsurfactant can thus be entirely avoided. Hypercapnia from tube secretions, tube malposition, accidental extubation, pneumothorax or insufficient amplitude can also be quickly noted and appropriate interventions given. The $tc\dot{p}CO_2$ monitor is also very valuable as the patient begins to wean, avoiding hypocarbia and allowing the staff to pace the wean appropriately.

In infants, optimal use of HFOV, and in fact any form of full respiratory support, should include concurrent use of $tc\dot{p}CO_2$ monitoring to ensure prevention of hypo- and hypercarbia and timely interventions for both complications of therapy and patient weaning.

References

1. Gerstmann DR, Minton SD, Stoddard RA *et al*: The Provo multicenter early high-frequency oscillatory ventilation trial: Improved pulmonary and clinical outcome in respiratory distress syndrome. *Pediatrics* 1996; 98: 1044-57.
2. Courtney SE, Durand DJ, Asselin JM *et al*: High-frequency oscillatory ventilation versus conventional mechanical ventilation for very low birth weight infants. *N Engl J Med* 2002; 347: 643-52.
3. Kinsella JP, Truog WE, Walsh WF *et al*: Randomized, multicenter trial of inhaled nitric oxide and high-frequency oscillatory ventilation in severe, persistent pulmonary hypertension of the newborn. *J Pediatr* 1997; 131: 55-62.
4. Froese AB: Role of lung volume in lung injury: HFO in the atelectasis-prone lung. *Acta Anaesthesiol Scand* 1989; 33: Suppl. 90: 126-30.
5. Fujimoto S, Togari H, Yamaguchi N *et al*: Hypocarbia and cystic periventricular leukomalacia in premature infants. *Arch Dis Child* 1994; 71: F107-110.
6. Okumura A, Hayakawa F, Kato T *et al*: Hypocarbia in preterm infants with periventricular leukomalacia: The relation between hypocarbia and mechanical ventilation. *Pediatrics* 2001; 107: 469-75.
7. Kaiser JR, Gauss CH, Williams DK. The effects of hypercapnia on cerebral autoregulation in ventilated very low birth weight infants. *Pediatr Res* 2005;58:931-935.
8. Kaiser JR, Gauss CH, Pont MM, Williams DK. Hypercapnia during the first 3 days of life is associated with severe intraventricular hemorrhage in very low birth weight infants. *J Perinatol* 2006;26:279-285.
9. Pollitzer MJ, Whitehead MD, Reynolds EOR *et al*: Effect of electrode temperature and in vivo calibration on accuracy of transcutaneous estimation of arterial oxygen tension in infants. *Pediatrics* 1980; 65: 515-20.

10. Binder N, Atherton H, Thorkelsson T *et al*: Measurement of transcutaneous carbon dioxide in low birthweight infants during the first two weeks of life. *Am J Perinatology* 1994; 11: 237-41.
11. McIntosh N, Becher JC, Cunningham S *et al*: Clinical diagnosis of pneumothorax is late: Use of trend data and decision support might allow preclinical detection. *Pediatr Res* 2000; 48: 408-15.
12. Chow LC, Wright KW, Sola A *et al*: Can changes in clinical practice decrease the incidence of severe retinopathy of prematurity in very low birth weight infants? *Pediatrics* 2003; 111: 339-45.
13. Askie LM, Henderson-Smart DJ, Irwig L *et al*: Oxygen-saturation targets and outcomes in extremely preterm infants. *N Engl J Med* 2003; 349: 959-67.

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