

c. Four patients with hemodynamic disorders in whom corrected cpCO<sub>2</sub> was consistently higher than p<sub>a</sub>CO<sub>2</sub>. This difference disappeared once the hemodynamic disorders had been corrected.

### Conclusions

These findings recorded in a small number of patients must be interpreted with caution, particularly in view of the fact that, in a few cases, two types of traces were observed in the same patient at different times. Nevertheless, they do reveal the advantages and limitations of cpCO<sub>2</sub> monitoring:

1. In most cases cpCO<sub>2</sub> makes it possible to monitor pCO<sub>2</sub> since it is significantly correlated with p<sub>a</sub>CO<sub>2</sub>.
2. It ceases to be of value when collapse has occurred. Its deviation from p<sub>a</sub>CO<sub>2</sub> is an indirect reflection of hemodynamics and could contribute to its monitoring.
3. In general it can be said that these variations are much smaller than those of cpO<sub>2</sub> and there are relatively few situations in which they can be used as a guide in ventilation.

4. Studies of cpCO<sub>2</sub> and cpO<sub>2</sub> traces could perhaps provide better understanding of various pathological respiratory conditions but we feel that it should be linked to that of other parameters, such as alveolar pCO<sub>2</sub>.

### References

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## Transcutaneous pO<sub>2</sub> and pCO<sub>2</sub> Monitoring With a Single Electrochemical Sensor; Its Clinical Use and Advantages in Neonatal Intensive Care

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Our medical Physics Department has designed a sensor which combines into a single device a Clark polarographic O<sub>2</sub> electrode and a Stow Severinghaus CO<sub>2</sub> electrode. Details of the probe's construction have been given [1]. The accuracy of simultaneous estimation of p<sub>a</sub>O<sub>2</sub> and p<sub>a</sub>CO<sub>2</sub> has been investigated in sick infants [2].

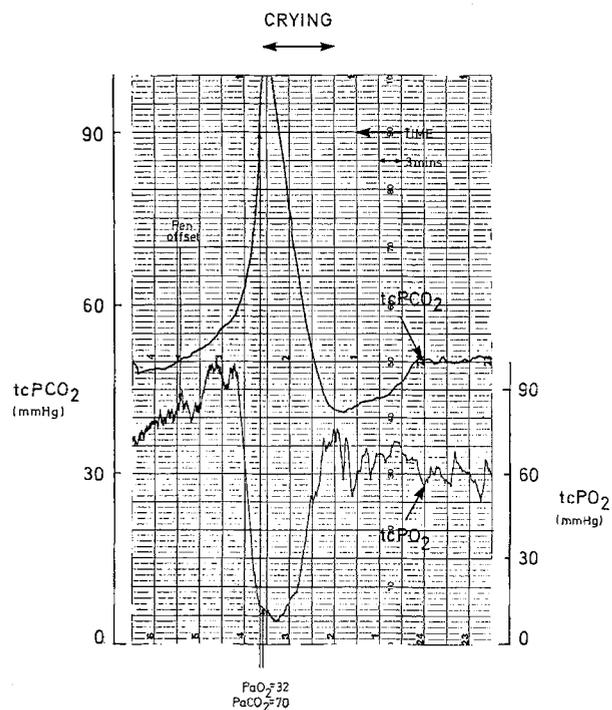
Twenty-two studies were performed on ten newborn infants with respiratory illnesses. All had umbilical artery catheters in situ. The sensor was heated to 44°C and attached to the infant's abdomen for 3½ to 6 h. p<sub>a</sub>O<sub>2</sub> was measured continuously by a Searle intravascular oxygen electrode or estimated by a Dräger transcutaneous oxygen electrode. Several samples of arterial blood were taken through the umbilical artery catheter during the course of each study and their p<sub>a</sub>O<sub>2</sub> and p<sub>a</sub>CO<sub>2</sub> measured. The initial blood gas values for p<sub>a</sub>O<sub>2</sub> and p<sub>a</sub>CO<sub>2</sub> were used as in vivo calibration points for tcPO<sub>2</sub>, as previously described [3] and for tcPCO<sub>2</sub>.

tcPO<sub>2</sub> was compared with p<sub>a</sub>O<sub>2</sub> recorded by the intravascular oxygen electrode or by p<sub>a</sub>O<sub>2</sub> estimated by the Dräger transcutaneous electrode. tcPCO<sub>2</sub> was compared with the p<sub>a</sub>CO<sub>2</sub> of samples of arterial blood. The relation between tcPO<sub>2</sub> and p<sub>a</sub>O<sub>2</sub> after in vitro calibration was tcPO<sub>2</sub> = 0.95 p<sub>a</sub>O<sub>2</sub> + 11.93 mmHg (*r* = 0.69, *p* < 0.001) and after in vivo

calibration tcPO<sub>2</sub> = 1.00 p<sub>a</sub>O<sub>2</sub> + 1.27 mmHg (*r* = 0.89, *p* < 0.001). After in vitro calibration tcPCO<sub>2</sub> = 0.99 p<sub>a</sub>CO<sub>2</sub> + 9.88 mmHg (*r* = 0.89, *p* < 0.001) and after in vivo calibration tcPCO<sub>2</sub> = 0.98 p<sub>a</sub>CO<sub>2</sub> - 2.33 mmHg (*r* = 0.97, *p* < 0.001.)

The results showed that the sensor gave an estimate of p<sub>a</sub>O<sub>2</sub> and p<sub>a</sub>CO<sub>2</sub> which was sufficiently accurate for use in the clinical management of newborn infants. The combined tcPO<sub>2</sub>/tcPCO<sub>2</sub> sensor is now being used routinely in our neonatal intensive care unit. The importance of continuous p<sub>a</sub>O<sub>2</sub> or tcPO<sub>2</sub> monitoring is already well established. Arterial pCO<sub>2</sub> has often been thought to remain fairly stable, with gradual changes in p<sub>a</sub>CO<sub>2</sub> requiring intermittent blood gas monitoring only. However, we have found that tcPCO<sub>2</sub> can fluctuate rapidly and unexpectedly.

Due to the profound effect that p<sub>a</sub>CO<sub>2</sub> has on cerebral blood flow, continuous monitoring of tcPCO<sub>2</sub> has proved invaluable in the clinical management of sick infants. In respiratory illnesses carbon dioxide retention occurs and mechanical ventilation may be required to reduce it. The combined tcPO<sub>2</sub>/tcPCO<sub>2</sub> sensor enables us to intervene at the appropriate time, preventing p<sub>a</sub>CO<sub>2</sub> from reaching dangerous levels, whilst ensuring the blood is still kept well

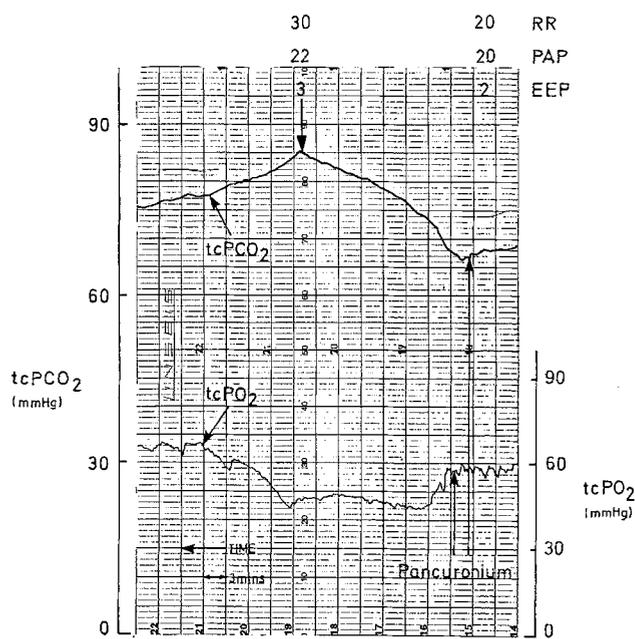


**Fig. 1.** Simultaneous records of tcPO<sub>2</sub> and tcPCO<sub>2</sub> recorded by the combined sensor. The infant weighed 2,465 g at 32 weeks of gestation and had severe rhesus haemolytic disease. He was 46 h old at the time of study and was being treated by intermittent mandatory ventilation with 40% O<sub>2</sub>. The effect of crying on tcPO<sub>2</sub> and tcPCO<sub>2</sub> is readily apparent

oxygenated. The level of arterial pO<sub>2</sub> and pCO<sub>2</sub> can then be monitored until suitable ventilator settings have been selected. If an infant's arterial pCO<sub>2</sub> is too low, it can be raised by alterations in ventilator settings, or by introducing a dead space. The effectiveness of such treatment can then be observed. Due to the hazards of prolonged ventilation, it is desirable to wean babies from ventilatory support as soon as possible. Continuous tcPO<sub>2</sub> and tcPCO<sub>2</sub> monitoring provides useful information in determining when the infant no longer requires ventilation.

Crying and fighting the ventilator are common causes of fluctuating high pCO<sub>2</sub> with low pO<sub>2</sub> (Fig. 1). This behaviour can cause a pneumothorax to occur, which may be followed by intraventricular haemorrhage. The administration of a muscle relaxing drug prevents this struggling but the infant's own respiratory efforts are often reduced, causing arterial pO<sub>2</sub> to fall and pCO<sub>2</sub> to rise. It is therefore important, at this time, to be able to monitor both tcPO<sub>2</sub> and tcPCO<sub>2</sub> continuously so that ventilation can be increased as necessary to bring arterial pO<sub>2</sub> and pCO<sub>2</sub> back to normal (Fig. 2).

Continuous pO<sub>2</sub> and pCO<sub>2</sub> monitoring is proving extremely valuable for the clinical management of very sick infants.



**Fig. 2.** Simultaneous records of tcPO<sub>2</sub> and tcPCO<sub>2</sub> recorded by the combined sensor. The records should be read from right to left. The infant weighed 1,070 g at 28 weeks of gestation and had hyaline membrane disease. He was 48 h old at the time of study and was being treated by intermittent mandatory ventilation with 90% O<sub>2</sub>. The effect of pancuronium and changes in ventilator settings can be seen.

## References

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