

Transcutaneous Po_2 measurements in seriously ill newborn infants

S. SWANSTRÖM, I. VILLA ELISAGA, L. CARDONA, A. CÁRDENES,
C. MÉNDEZ-BAUER, and G. ROTH

From the Maternity Hospital, Casa de Salud de Santa Christina, Madrid, Spain, and the Perinatal Research Unit, Department of Paediatrics, University Hospital Uppsala, Sweden

Swanström, S., Villa Elisaga, I., Cardona, L., Cárdenes, A., Méndez-Bauer, C., and Roth, G. (1975). *Archives of Disease in Childhood*, 50, 913. **Transcutaneous Po_2 measurements in seriously ill newborn infants.** Continuous monitoring of oxygen tension using the transcutaneous electrode developed by Huch and co-workers was studied in severely ill newborn infants. Acceptable results were obtained despite the theoretical possibility that vasoconstriction might interfere with the method when used in the very ill. The reliability and clinical usefulness of the method is illustrated in 4 cases of severely ill newborn infants.

Continuous monitoring of Po_2 is now accepted as important in the management of many sick newborn infants. By means of polarographic techniques, such measurements can be made using intra-arterial catheters with microelectrodes fitted into their tip (Parker, Key, and Davies, 1971; Huch *et al.*, 1972a, 1973b; Harris and Nugent, 1973) or using electrodes implanted in tissues (Strauss, Beran, and Baker, 1972).

A noninvasive and bloodless method using a heated skin electrode was devised by the Huchs and colleagues (Huch, Huch, and Lübbers, 1969; Huch, Lübbers, and Huch, 1972b, 1974; Huch *et al.*, 1973c; Huch, Huch, and Lübbers, 1973a). Earlier published results using this type of electrode were mainly obtained in healthy or moderately ill newborn babies. It was the aim of the present study to observe the validity and usefulness of the method in severely ill infants, in whom peripheral vasoconstriction might interfere with local blood supply at the electrode site.

Materials and methods

A Clark electrode with a 15 μm platinum cathode and a silver anode connected to a heating coil covered by 12 μm thick cuprophane and teflon membranes (from the laboratory of A. Huch and R. Huch, Marburg) was used. The electronic unit was built by the department for electronic developments, University of Marburg. New membranes were mounted at least every

morning. Calibration was undertaken before and after each recording. The tcPo_2 values given all refer to the initial calibration, as the recalibration after the measurements showed only negligible drift. Pure N_2 at 42°C gave a zero for oxygen tension, and room air of 42°C saturated with water vapour gave approximately 135 mmHg. The core temperature of the electrode was 43°C giving about 42°C on the surface of the skin (Huch *et al.*, 1973a). The electrode was placed on the abdomen below the umbilicus in order to get a reference to blood gases from umbilical artery catheters. Respiration was recorded by impedance equipment. A 3-channel Rikadenki Multi-pen recorder registered on a 25 cm wide chart.

The Clark electrode measures molecular oxygen changes with the polarographic technique. The 'Huch' electrode with a well regulated heating unit makes it possible to induce a cutaneous hyperaemia at a preselected temperature. With the core temperature of the electrode set at 43°C in newborn infants, the normal blood flow variations have negligible influence on the observed tcPo_2 . The decrease in the tcPo_2 level caused by the oxygen consumption of the skin and of the electrode itself is counteracted by the temperature displacement of the oxygen dissociation curve to the right. The net sum of the different factors acting on the tcPo_2 has been found empirically to give tcPo_2 values closely related to PaO_2 values in healthy newborn infants (Huch *et al.*, 1974).

Patients and results

Case 1. 5½-day-old boy. Birthweight 2260 g at 40 weeks' gestation, with signs of 'intrauterine malnutrition'. On day 1 he developed an aspiration syndrome,

necessitating oxygen. Respiratory difficulties increased so that on day 5 he had to be mechanically ventilated. The infant died on day 6. Necropsy showed cerebral oedema, with macroscopical signs of possible focal meningitis, and pulmonary haemorrhages.

Observations of tcPO₂ made in this case are shown in Fig. 1-5. With a Bennett respirator at the rate of 45 per minute (Fig. 1), a positive pressure of 27 cmH₂O,

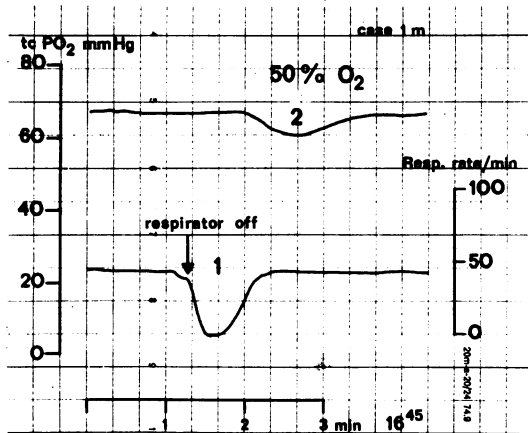


FIG. 1.—Case 1. Short disconnection of respirator decreased the respiratory rate (1) and induced a small tcPO₂ change (2).

and an oxygen concentration of 50%, tcPO₂ was maintained at a desired level of about 67 mmHg. A temporary disconnection of the tube system for 20 seconds in order to drain out water is indicated at (1) by a dip in respiratory rate down to zero, which nevertheless led to only a slight fall in tcPO₂ (2).

15 minutes later (Fig. 2) the rate of the respirator was decreased from 43 to 33 and then to 25 per minute in order to trigger spontaneous breathing. At (1) the first superimposed spontaneous breaths were seen as a change from an even to an irregular respiratory pattern. At (2) the respirator support was stopped and the oxygen concentration increased from 50 to 100%. The net effect of these procedures was a small increase in tcPO₂.

25 minutes later (Fig. 3) the same infant was still breathing 100% oxygen through an endotracheal tube, and maintaining an adequate tcPO₂ level (1). The respirator was then turned on with its previous setting in order to rest the infant's respiratory muscles (2). At first no marked difference in the tcPO₂ level was seen, but at (3) owing to a mechanical fault the respirator pressure fell from 27 to 10 cmH₂O. This caused an arrest of chest movements, respiration rate fell to zero (3), and tcPO₂ fell after about 40 seconds (4). As soon as the mechanical fault was recognized the tcPO₂ curve returned to its previous level.

10 minutes after the correct pressure of the respirator had been reset (Fig. 4) the tcPO₂ curve again fell (1), and this led to discovering that the endotracheal tube had become displaced. An Ambu bag connected to 100% oxygen was used in order to prepare the infant for intubation. The rapid rate of the manual ventilation was shown at (2) and its positive effect was noted by an increase in tcPO₂. After 5 minutes of oxygen priming reintubation was attempted but was not immediately successful, and the attempt was interrupted when the tcPO₂ had fallen to a critical level of about 30 mmHg (3). The infant was again reoxygenated by Ambu bag ventilation on mask and the rapid positive effect on oxygenation can be followed on the tcPO₂ curve. A second and third intubation were later attempted unsuccessfully (4) with the same fall in tcPO₂. Fig. 5 shows the fourth attempt to intubate. It was at first

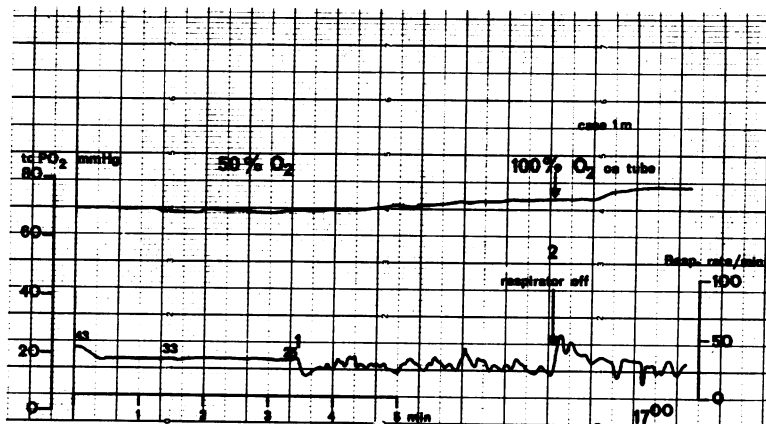


FIG. 2.—Case 1. 15 minutes after Fig. 1. Respirator rate decreased to 25 (1). Spontaneous breathing was triggered, and at (2) the respirator was switched off since the tcPO₂ curve did not decline.

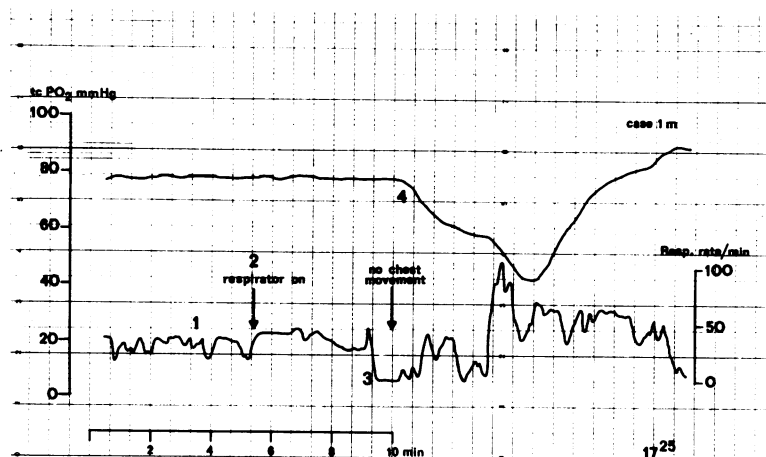


FIG. 3.—Case 1. 25 minutes after Fig. 2. Spontaneous breathing (1). Respirator support (2). Respirator failure (3). Subsequent drop of tcPO₂ (4) followed by an increase in tcPO₂ as the respirator was corrected and the lungs reventilated.

considered successful by auscultation signs but clearly was not so when the fall in tcPO₂ was apparent (1). When the endotracheal tube was correctly placed, the tcPO₂ rose (2). Five different attempts were made in all, each with a duration of about 1½ minutes, before the intubation was satisfactory. All attempts were interrupted by manual ventilation on mask, when tcPO₂ fell to 40–30 mmHg. A blood gas taken some hours earlier when the infant was on the respirator and the tcPO₂ was 55 mmHg, showed PaO₂ 48 mmHg, Pco₂ 42 mmHg, pH 7.38, and base deficit 1 mmol/l. After the successful fifth intubation, when the tcPO₂ was 54 mmHg, a blood gas analysis showed PaO₂

63 mmHg, Pco₂ 58 mmHg, pH 7.38, and base deficit 0 mmol/l. It is worth noting that despite the many difficulties encountered with attempted intubation, no hypoxic metabolic acidosis occurred because the attempts to intubate and their timings were guided by the tcPO₂ monitoring.

Case 2. Preterm male infant, birthweight 1600 g, gestational age 32 weeks. Soon after birth developed respiratory distress, metabolic acidosis, and an x-ray picture compatible with hyaline membrane disease.

At 12 hours (Fig. 6) the infant was maintaining an adequate tcPO₂ level of 50–60 mmHg in a Gregory box

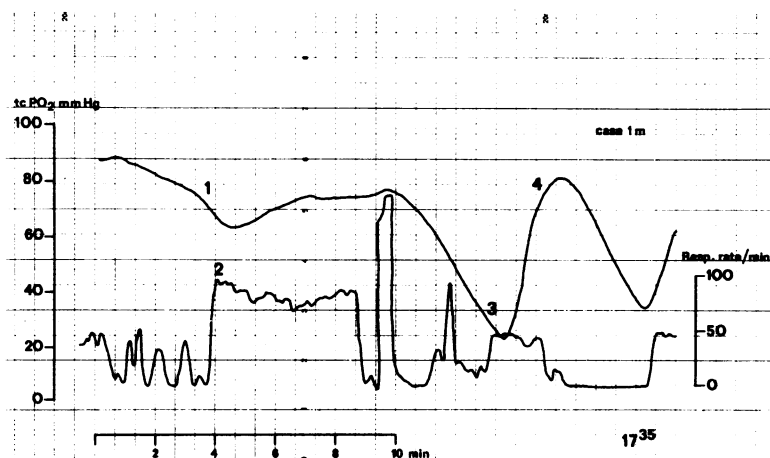


FIG. 4.—Case 1. 10 minutes after Fig. 3. Irregular ventilation with tcPO₂ decrease (1). Manual ventilation with Ambu bag started (2). Attempted reintubation interrupted because of low tcPO₂ (3). New intubation attempt (4).

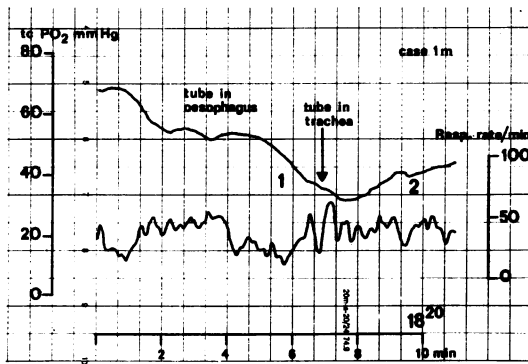


FIG. 5.—Case 1. The endotracheal tube had slipped into the oesophagus (1). $tcPO_2$ increased when the tube was back in the trachea (2). The irregular respiratory rate did not show this complication, but the fall in $tcPO_2$ gave an early indication of impaired ventilation.

with 5 cmH₂O positive airway pressure, and an oxygen concentration of 38%. In view of the low pressure in the Gregory box and the low oxygen admixture there was some doubt if the Gregory box was really essential, so the collar around the neck was opened and the positive pressure eliminated. The $tcPO_2$ curve began to drop rapidly and therefore the positive pressure was re-established within 20 seconds (1). In spite of this the $tcPO_2$ dropped to less than 20 mmHg before the $tcPO_2$ curve started to rise.

Some 30 minutes later (Fig. 7) still in the same oxygen concentration and with the same pressure in the box,

i.e. 5 cmH₂O, blood gases were checked; there was fair agreement between Pao_2 , 76 mmHg and $tcPO_2$, 60 mmHg. A gradual fall of the $tcPO_2$ a couple of minutes later was observed though initially no clinical deterioration was noted. Increase first in the oxygen concentration, then in the Gregory box pressure, and subsequently Ambu bag ventilation, intubation, and mechanical ventilation failed to prevent the persistent decline in $tcPO_2$. When $tcPO_2$ was 11 mmHg a blood gas analysis showed Pao_2 , 17 mmHg. Finally there was profound haemorrhage through the mouth and all subsequent resuscitation efforts were without results. A spinal tap showed massive haemorrhage and necropsy confirmed intracranial bleeding together with hyaline membrane disease.

Case 3. Female infant, delivered by caesarean section at 33 weeks, with a birthweight of 2700 g. Apgar score 9. The mother was diabetic with one previous abortion and one preterm infant of 2000 g that died at 36 hours. This infant was initially well with no respiratory problems, but after some days she developed pneumonia and in spite of vigorous antibiotic treatment she died aged 7 days. Necropsy confirmed pneumonia and there was also hyperplasia of the islets of Langerhans.

The skin electrode was placed on the skin of the infant at time 0 (Fig. 8). Normally this lends to the $tcPO_2$ registering 30–40 mmHg before a steady rise in the curve appears as thermal hyperaemia is achieved. Such a pattern has been seen in more than 200 healthy newborn infants studied (Huch, Huch, and Rooth, 1975). However, in this case $tcPO_2$ immediately fell to zero level (1) indicating severe peripheral vasoconstriction and/or a very low Pao_2 . Clinically there was a dusky colour and poor respiration. After 5 minutes of manual ventilation with an Ambu bag the short bursts of

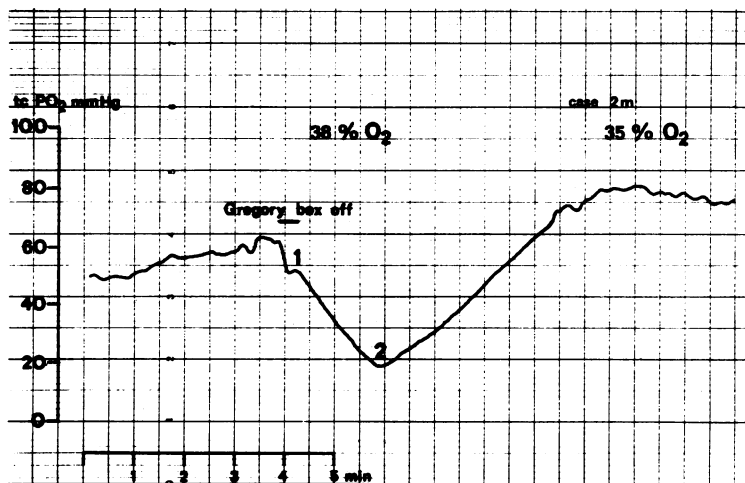


FIG. 6.—Case 2. Age 12 hours. In Gregory box with 38% oxygen and 5 cmH₂O positive airway pressure. The pressure was released for less than $\frac{1}{2}$ minute (1) with a subsequent drop in $tcPO_2$ (2).

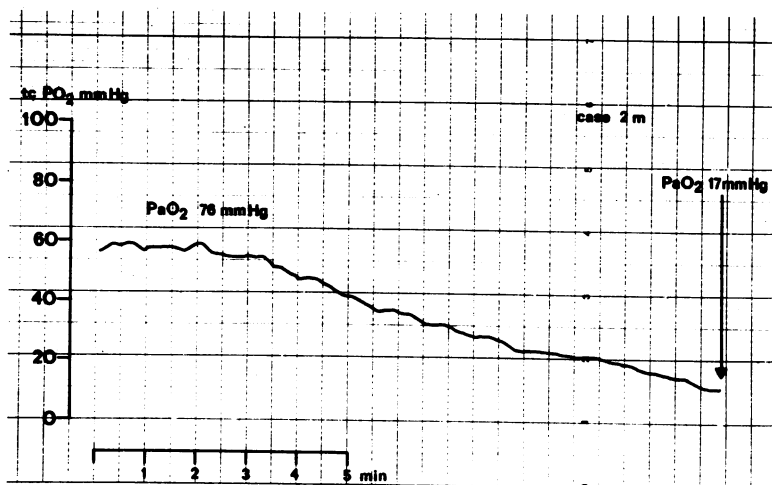


FIG. 7.—Case 2. Age $12\frac{1}{2}$ hours. A persistent $tcPO_2$ decline was the first sign of a complicating brain haemorrhage. In spite of resuscitation efforts, deterioration progressed and the low $tcPO_2$ was confirmed by a PaO_2 of 17 mmHg.

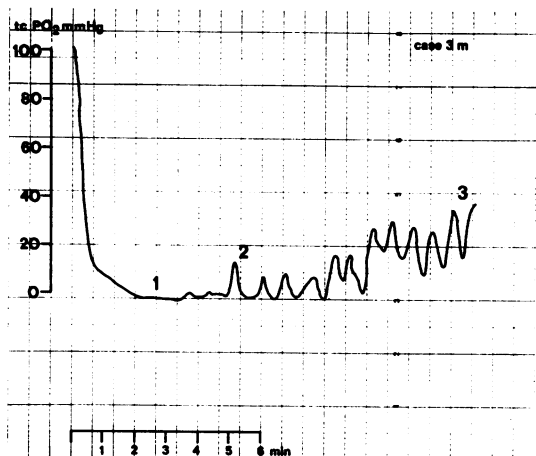


FIG. 8.—Case 3. After the application of the electrode $tcPO_2$ registered zero (1), indicating a serious clinical situation. Gasping after resuscitation (2) and gradual increase in $tcPO_2$ level as the thermal vasodilatation occurred (3).

spontaneous intensive breathing were reproduced by the $tcPO_2$ curve (2). (The $tcPO_2$ level in this figure would not be expected to represent the PaO_2 values since at least 15 minutes are needed for adequate vasodilatation.) The same initial fall in $tcPO_2$ to zero after the application of the electrode was seen 2 hours later in the same patient.

Three and a half hours later (Fig. 9) the infant's respiratory efforts were not sufficient to maintain a proper oxygen tension even in 100% oxygen. Two blood gas analyses from the umbilical artery catheter

when $tcPO_2$ was 27 and then 86 mmHg gave PaO_2 values of 45 and 80 mmHg respectively. The capacity of the lungs themselves for good oxygenation when the ventilation was adequate was proved by the sharp rise in $tcPO_2$ when the Ambu bag was used (1) and (2). Death occurred 4 hours after this recording.

Case 4. Male infant, birthweight 4000 g, gestational age 44 weeks. Delivered by caesarean section because of fetal stress indicated by fetal heart rate varying from 60 to 210 beats/minute. Apgar score 5. Maternal rubella infection probable in the second month of pregnancy. From the first day the neurology was abnormal and the infant was severely ill, cyanotic in high concentrations of O_2 , and with respiratory and metabolic acidosis. Death at 19 days. Necropsy showed renal dysplasia, with renal necrosis and infection. The lungs were congested and haemorrhagic. The heart was normal.

An effect of incubator and skin temperature on respiration and $tcPO_2$ was noted in this baby (Fig. 10). A fairly low temperature in the incubator ($36^\circ C$) and a low skin temperature of $31^\circ C$, as measured on the abdomen close to the electrode, were accompanied by an irregular breathing pattern with several deep gasps. When the incubator temperature was gradually increased to $36.9^\circ C$, skin temperature rose to $35^\circ C$ and the respiration pattern developed a cyclical pattern, a deep gasp being followed by rapid breathing of about 50/minute. Associated with the changed pattern of breathing was a fall in the $tcPO_2$ from about 100 to 85 mmHg, the cause of which was not clear. This was presumably due to a later drop in incubator temperature with a subsequent fall of skin temperature which reproduced the irregular type of respiration with its concomitant fall in $tcPO_2$.

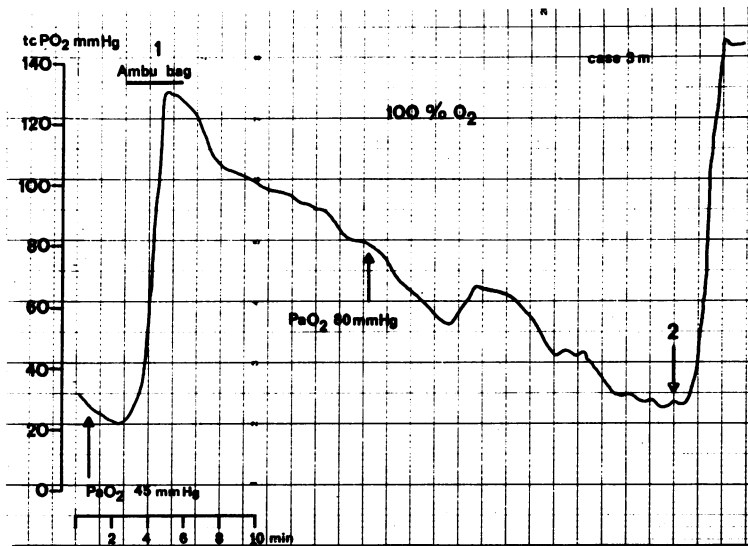


FIG. 9.—Case 3. $3\frac{1}{2}$ hours after Fig. 8. Intermittent manual ventilation with an Ambu bag was used (1), (2), in order to support the failing respiratory effort.

Discussion

These 4 cases of oxygen-dependent, severely ill newborn infants were monitored with a transcutaneous PO_2 electrode in order to see if the information received was acceptable for guiding intensive

care. A possible limitation to the use of the electrode in this type of case would be peripheral vasoconstriction. Although statistical analysis was not possible because of the small number of blood gases taken, we observed reasonably good agree-

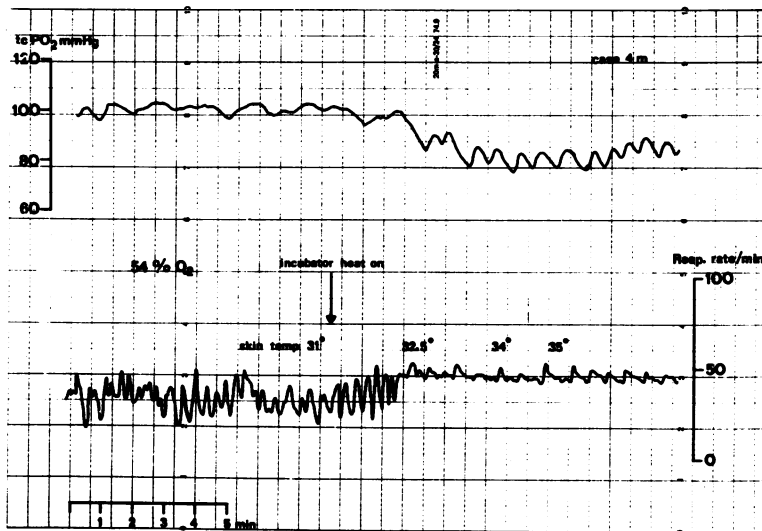


FIG. 10.—Case 4. The pattern of respiration was changed by variations in environmental temperature and a change both in configuration and level of the $tcPO_2$ curve was produced.

ment between tcPO₂ and PaO₂. Vasoconstriction in these infants therefore did not prevent tcPO₂ measurements from providing extremely valuable information. The pattern of the curve after application of the electrode (Fig. 8), or the absence of respiratory influence of the tcPO₂ pattern may suggest a profound vasoconstriction.

Continuous tcPO₂ monitoring of these infants was found to give more useful and reliable information of their oxygen status than merely looking for colour changes, or monitoring heart rate, respiratory rate, or impedance. It was possible to detect early and to evaluate events interfering with oxygen supply without disturbing the infant. The optimum moment to intubate, the necessary oxygen priming, and the time available before interrupting an intubation attempt could easily be decided by looking at the tcPO₂ curve (Fig. 4). The tcPO₂ provided a valuable early sign when complications in respirator treatment occurred enabling corrective measures to be applied without delay. When an infant was removed from the respirator the tcPO₂ values gave quick and reliable information on how the infant would fare. The delay in information on abrupt changes of PaO₂ was about the same as seen in healthy newborns, 20–30 seconds (Fig. 1, 3, 6).

Blood sampling in these infants is still necessary in order to monitor the acid-base balance, but their number can be reduced.

We conclude that even very ill newborn infants can usefully be monitored with the Huch tcPO₂ equipment with the object of keeping the oxygen tension at a normal level. Although severe

vasoconstriction may occasionally be a problem, in the present study this was not the case.

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Correspondence to Dr. G. Rooth, Perinatal Research Unit, Dept. of Paediatrics, Uppsala University Hospital, S-750 14 Uppsala, Sweden.