Home monitoring of transcutaneous oxygen tension in the early detection of hypoxaemia in infants and young children

Christian F Poets, Martin P Samuels, Jane P Noyes, Kirsty A Jones, David P Southall

Abstract

Twenty three patients (age range 0.5-40 months) with recurrent cyanotic episodes underwent physiological recordings, including transcutaneous oxygen tension (TcPO₂) from a monitor modified for use at home (Kontron 821S). Of 69 episodes in which the arterial oxygen saturation (Sao₂, Nellcor N200) was ≤80% for ≥20 seconds and/or central cyanosis was present, the TcPO₂ monitor alarmed (≤20 mmHg or 2.67 kPa) in every episode. The pulse oximeter identified hypoxaemia in 62 out of 69 episodes, failing in seven episodes due to signal loss from movement artefact. In only seven of 69 episodes was there an accompanying apnoeic pause (≥20 seconds), and heart rate fell to ≤80 beats/minute in only five of 28 episodes in which an electrocardiogram was recorded. In 32 episodes in which Sao₂ fell to ≤60%, the TcPO₂ monitor alarmed after a median time interval of 16 seconds (maximum time interval 30 seconds).

The TcPO₂ monitor was then used in an uncontrolled trial at home in 350 patients at increased risk of sudden death and/or hypoxaemia. Indications for monitoring included apparent life threatening events or cyanotic episodes (n=163), prematurity and prematurity related disorders (n=86), and sudden unexpected death in one or more siblings (n=122).

The TcPO₂ monitor detected cyanotic episodes at home in 81 patients, 52 of whom received vigorous stimulation and/or mouth to mouth resuscitation. Twenty one of these 52 patients had further hypoxaemic episodes documented in hospital with pulse oximetry. In 30 patients, the TcPO₂ monitor also identified the gradual development of hypoxaemia, as confirmed by pulse oximetry. Twenty of these needed additional inspired oxygen and six subsequently needed ventilatory support in hospital.

This TcPO₂ monitor is a reliable detector of both sudden and gradual onset hypoxaemia and is able to be used by parents in the home.

Electronic monitors are widely used in the home on infants at increased risk of sudden death. Most monitors aim to sense an absence of breathing movements and/or a fall in heart rate. Unfortunately, a considerable number of infants have died while on such monitors.¹⁻³ One possible explanation, based on our investigations of cyanotic episodes, is that a prolonged absence of breathing movements or a

bradycardia is relatively infrequent or late in its timing compared with severe hypoxaemia.⁴

Although the final pathways leading to sudden infant death syndrome (SIDS) remain unknown, the sudden onset of severe hypoxaemia is one cause.⁴ Hypoxaemia may occur not only as a result of a cessation in breathing movements, but also as a consequence of upper airway obstruction, seizures, or intrapulmonary shunting.^{4, 5}

Oxygenation may be monitored continuously and non-invasively by using either pulse oximetry or a transcutaneous oxygen tension (TcPO₂) sensor. Pulse oximetry, if used correctly, provides an accurate measure of arterial oxygen saturation (Sao₂)⁶ and does not require heating of the skin. However, it has the major drawback of signal loss due to motion artefact. TcPO₂ monitoring is a well validated technique for the detection of hypoxaemia in preterm infants undergoing neonatal intensive care,⁷ but the sensor is more difficult to use, and measurements of oxygenation are substantially affected by skin perfusion (see below).

In this study we investigated a modified TcPO₂ monitor in its ability to detect sudden onset hypoxaemia and to be used for the home surveillance of infants at increased risk of sudden death. Three questions were addressed: (i) will the system detect episodes of hypoxaemia reliably and in time for potentially effective intervention; (ii) will the monitor show reproducible baseline readings; and (iii) is the system suitable for long term use by parents in the home in terms of ease of application, frequency of false alarms, and potentially harmful side effects?

Methods

APPARATUS

A standard neonatal TcPO₂ monitor was modified (Kontron 821S) to provide (i) a constant, single electrode temperature of 43°C to allow a longer interval between sensor resiting, (ii) an inability to be left in a calibration, non-alarm mode, and (iii) toddler proof alarm controls. The TcPO₂ values were displayed in mm Hg so that parents did not have to use decimals. The sensor was calibrated in air (160 mm Hg or 21.30 kPa) before each application. The value of TcPO₂ was continuously displayed. The high alarm was set 10 mm Hg (1.33 kPa) higher than the highest level of TcPO₂ found on the baby, so that in the event of even slight separation of the sensor from the skin a high alarm (approaching the oxygen tension of air) would sound. The low

Department of Paediatrics, National Heart and Lung Institute, Royal Brompton Hospital, Fulham Road, London SW3 6HP
Christian F Poets
Martin P Samuels
Jane P Noyes
Kirsty A Jones
David P Southall

Correspondence to: Dr Southall.

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alarm was usually set to 20 mm Hg (2.67 kPa). The monitor was powered by long life or rechargeable batteries, or through a mains adaptor. It was portable (76×205×104 mm) and provided with a carrying case.

VALIDATION STUDIES
Changes in physiological parameters during sudden onset hypoxaemic episodes
Twenty three infants and young children (median age 5 months, range 2 weeks–40 months) who were having recurrent cyanotic episodes underwent long term multichannel recordings of SaO₂ from a pulse oximeter (Nellcor N200, modified to give beat to beat averaging), photoplethysmographic waveforms for the validation of the SaO₂ signal, electrocardiography, abdominal wall movements (Graseby volume expansion capsule) and TcPo₂ (Kontron 821S). Recordings were performed in the intensive care or high dependency area of the Royal Brompton Hospital and trained cardiothoracic nurses were asked to note all episodes of clinically apparent central cyanosis. Analysis of the recordings included the identification of the number of episodes in which (i) SaO₂ fell to 80% or less for at least 20 seconds, and/or (ii) central cyanosis occurred. The association between changes in TcPo₂ and other physiological parameters and recorded or observed hypoxaemic episodes was analysed.

Response time
Recordings which showed one or more episodes of severe hypoxaemia (SaO₂ ≤60% for ≥10 seconds) underwent analysis to calculate the time interval for the TcPo₂ monitor to alarm (TcPo₂ ≤20 mm Hg or 2.67 kPa) after the SaO₂ reached 60%. A threshold of 60% SaO₂ was arbitrarily defined according to clinical experience: cyanosis is usually present at this level if the haemoglobin concentration is within the normal range. In addition, it has been shown that TcPo₂ equals arterial oxygen tension minus 14 mm Hg (1.87 kPa) at a sensor temperature of 43°C,⁸ and a SaO₂ of 60% approximately corresponds with an arterial oxygen tension of 35 mm Hg (4.67 kPa) on the oxygen dissociation curve, which in turn corresponds with a TcPo₂ of 20 mm Hg (2.67 kPa).

Baseline measurements, skin status, and false alarms
A sample of 43 parents consecutively entered into the monitoring programme was asked to keep a record of the time and the TcPo₂ values before each resiting of the sensor and 20 minutes after the resiting in order to assess the reproducibility of the baseline measurements, the time interval between resittings, and the degree with which readings changed or drifted during each resiting interval. They were also asked to keep a record of the skin status observed after each change of sensor site. This was estimated using a score, which included zero for no mark present, one and two for red marks of different intensities, and three for the

presence of a blister. Finally, they were asked to keep a record of alarms in which their baby appeared to be well (false positives) and the reasons for their occurrence, if evident (for example, the sensor lifted from the skin).

CLINICAL TRIAL
Patients
Between May 1988 and November 1990, 350 patients were referred either by their general practitioner (n=47), or community or hospital paediatrician (n=303). Their median age was 2.6 months (range 1 day–5 years). The clinical indications for referral are shown in table 1. Over 95% of patients, according to published information, were considered to be at increased risk of sudden death or hypoxaemia. The majority (93%) had experienced one or more episodes of cyanosis, were born prematurely, or had one or more siblings who died of SIDS. In 14 of the remaining 25 patients the TcPo₂ monitor was used to assist parents in the use of negative pressure ventilation at home.⁹
Before the onset of home monitoring, 253 patients (72%) underwent analog tape recordings of SaO₂ photoplethysmographic waveforms, breathing movements, electrocardiography, and airflow from either a thermistor (Yellow Spings Instruments) or expired carbon dioxide signals (Engström Eliza). Recordings were not performed in infants who had lost one sibling from SIDS.

Protocol for the use of the monitor at home
Parents and infants were discharged after a minimum of five hours' use of the monitor in hospital, in 82% involving at least one overnight stay. All parents were given detailed verbal and written instruction in the use of the TcPo₂ monitor including advice on troubleshooting. For example, in the event of a persistent fall in baseline TcPo₂, parents were taught the following sequence of checks to ascertain that this was not artefactual in origin. Firstly, the sensor was

Table 1 Clinical indications for home TcPo₂ monitoring in 350 patients

	No of patients*
ALTE or episodes of cyanosis	163
ALTE resolving with cardiopulmonary resuscitation	62
ALTE resolving with vigorous stimulation	72
Cyanotic episodes resolving spontaneously or with mild stimulation	18
Cyanotic breath holding spells	11
Prematurity and prematurity related disorders	86
Born ≤32 weeks' gestation	76
History of severe apnoea of prematurity	26
Bronchopulmonary dysplasia:	45
Receiving additional inspired oxygen	31
And/or negative pressure ventilation at home	11
SIDS in sibling(s)	122
SIDS in one sibling	100
SIDS in two siblings	11
SIDS in twin/triplet	11
Other medical diagnoses	25
Sleep related upper airway obstruction	8
Congenital alveolar hypoventilation syndrome	4
Respiratory problems after cardiothoracic surgery	8
Others (myopathy (n=2), parental anxiety, Joubert's syndrome, SVT)	5

*Some patients in more than one category. ALTE=apparent life threatening event, SIDS=sudden infant death syndrome, SVT=supraventricular tachycardia.

recalibrated in air and its site on the skin was changed. If the value remained low, the sensor was placed on the surface of the parents' own forearm. If the reading on the parent was 'normal' (usually >50 mm Hg or 6.67 kPa), the cause of the low baseline value was probably a low blood oxygen tension or a disturbed skin perfusion in the infant. In this event parents were asked to notify their family doctor or local hospital immediately. If the value remained low on the parent's skin the sensor was remembraned and resited first on their forearm and then on their baby.

In those infants with bronchopulmonary dysplasia receiving domiciliary oxygen it was recommended that a reduction in values of $TcPO_2$, which were not artefactual, were followed by an increase in the flow of inspired oxygen to bring the $TcPO_2$ concentration to that which was normal for the baby (depending on recommendations based on pulse oximetry). In this event parents were also asked to contact their local hospital immediately. In hospital the low $TcPO_2$ values were correlated with a pulse oximeter and if hypoxaemia was confirmed the causes for this were treated appropriately.

High alarms due to partial or complete separation of the sensor from the skin were minimised by modifications to the 'sensor to lead junction' and the use of adequate tape over the sensor itself and on the sensor lead. Care was taken not to compress the sensor onto the skin surface or place it over a bony surface since this could reduce skin blood flow and produce low $TcPO_2$ values.

Parents were given instructions on how to manage a severe episode of hypoxaemia needing immediate intervention. This training was provided by a clinical nurse specialist. Instruction included the use of a video, Resusci-doll (Laerdal) and a purpose written handbook on infant

resuscitation (available on request). A 24 hour, seven day a week on call service was provided by both clinical nurse specialists and the medical staff involved in the programme. A broken monitor would be replaced within 18 hours.

Parents were thereafter seen at regular visits in the outpatient clinic and either at home or in hospital by the clinical nurse specialist who provided advice and support.

A decision to discontinue monitoring was undertaken in full collaboration with parents and in circumstances appropriate to the medical problems involved. For example, in subsequent siblings of victims of SIDS a post-term age of 9 months was usually chosen ($>95\%$ of all cases of SIDS have occurred by this age¹⁰).

Results

VALIDATION STUDIES

TcPO₂ changes during severe hypoxaemic episodes

Of 23 infants with recurrent cyanotic episodes who underwent multichannel recordings (total duration of recordings 340 hours), 16 patients (median age 5 months, range 2 weeks–40 months) had a total of 69 prolonged hypoxaemic and/or cyanotic episodes. Fifty two episodes involved a desaturation on the tape recording; although cyanosis may have been present, it was not specifically documented by the nurses. In 10 episodes, cyanosis was reported by the nurses and confirmed by low SaO_2 readings (fig 1). In a further seven episodes with clinically apparent central cyanosis the SaO_2 signal was uninterpretable due to movement artefact (fig 2). All 69 episodes (100%) were associated with a fall in $TcPO_2$ to ≤ 20 mm Hg (2.67 kPa). Only seven of the 69 episodes were accompanied by a pause in breathing movements of ≥ 20 seconds. In two episodes bag and mask resuscitation was required to initiate recovery

Figure 1 A major hypoxaemic episode in a 2 year old child with recurrent cyanotic episodes. Breathing movements, although reduced in amplitude, continue throughout the hypoxaemic episode (from B to D). SaO_2 (Nellcor N200 in the beat to beat mode) starts to fall at B, and remains low for 150 seconds (to D). The onset of the dip in $TcPO_2$ (from A to C) occurs simultaneously with the onset of the fall in SaO_2 . The alarm on the $TcPO_2$ monitor was set at 20 mm Hg (2.67 kPa) and would have sounded 17 seconds after the point at which the SaO_2 reaches 60%. Examination of the arterial pulse (photoplethysmographic) waveforms shows no evidence of a bradycardia. The child, who was deeply cyanosed, was resuscitated with bag and mask ventilation. (1 kPa = 7.5 mm Hg.)

Figure 2 Recording on an infant (aged 2 months) during an episode of cyanosis, at the time of which there is a sudden fall in $TcPO_2$ lasting 90 seconds. This is accompanied by continuous breathing movements and a fall in heart rate to 65 beats/minute. The pulse oximeter signal is lost due to movement artefact and would not therefore have identified this event. (1 kPa = 7.5 mm Hg.)

(fig 1), one of these episodes was associated with a prolonged apnoeic pause. In 28 episodes in 10 patients where an electrocardiogram was recorded, bradycardia (heart rate ≤ 80 beats/minute) was present during just five episodes.

Response time

Thirty seven episodes with $SaO_2 \leq 60\%$ for ≥ 10 seconds were identified to have occurred in 16 patients. Five episodes in three patients showed a very slow onset of a fall in SaO_2 (time to reach $60\% > 1$ minute). In all these five episodes $TcPO_2$ reached the 20 mm Hg (2.67 kPa) threshold up to 108 seconds before SaO_2 fell to 60% . The median response time in the remaining 32 episodes with a fall in $TcPO_2$ after the onset of low SaO_2 readings was 16 seconds, the maximal time observed was 30 seconds. There was no consistent change in the response time with increasing age.

Baseline measurements

Forty one parents (95%) returned the questionnaire. They had been entered into the monitoring programme between five days to four months before receiving the questionnaire. They reported a total of 988 resiting episodes in their infants. The median of the individual mean resiting intervals in these 41 patients was 6.8 hours (interquartile range (IQR) 6.1 to 7.8 hours). The median of the individual mean baseline readings was 59.9 mm Hg (7.98 kPa) (IQR 54.9 to 65.8 mm Hg or 7.32 to 8.77 kPa). There were no differences between different age groups. The median of the individual mean changes in $TcPO_2$ values over an average resiting interval of 6.8 hours was -13 mm Hg

(-1.73 kPa) (IQR -8.1 to -17.1 mm Hg or -1.08 to -2.28 kPa).

In order to assess the variability of baseline values on any one infant, SDs were calculated for the individual baseline readings in each infant. The median of these SDs as an indicator for the reproducibility of the baseline readings was 6.9 mm Hg (0.92 kPa) (IQR 5.7 to 7.5 mm Hg or 0.76 to 1.00 kPa). This means that the average variability in two thirds of the baseline readings was within a range of ± 7 mm Hg (0.93 kPa).

False positive alarms

A total of 129 false positive alarms (73 high, 56 low) occurred in 30 of the 41 infants during a total monitoring period of 540 days, giving an average for the whole group of one false alarm every four days of monitoring. In the two patients with the highest numbers of false alarms (12 and 16, respectively), the monitor turned out to be faulty. In the remaining infants high alarms were all explained by loose sensor contact and occurred predominantly in the older, more active babies. False low alarms were reported in 20 infants, most of them were associated with a drift downwards in baseline readings or the infant rolling over on to the sensor.

Skin status

A red mark appeared on the skin in 872 (88%) of the 988 sitings. In 838 (96%) of these marks the mark had faded completely after 48 hours. Thirty four marks were reported in 11 infants to have persisted for more than 48 hours, all but two were stage 1 marks (slight redness). In one

infant one blister was reported, which occurred after the mother had not changed the sensor site for nine hours.

CLINICAL DATA

Duration of monitoring

Over a 30 month period, 201 infants completed monitoring. In 19 patients (10%) the parents decided to discontinue monitoring before medically advised. The mean (SD) duration of monitoring in patients who completed the programme according to medical advice was 6.9 (3.7) months.

Detection of hypoxaemia

Two patterns of low TcPO₂ readings were observed: acute falls in TcPO₂ and changes in baseline TcPO₂. Acute falls in TcPO₂ associated with cyanosis or pallor were observed in 81 patients (table 2). Fifty two of these patients had an apparent life threatening event, needing vigorous stimulation (n=38) or mouth to mouth resuscitation (n=14) for recovery. Thirty nine of these 52 patients had entered the programme because of a previous apparent life threatening event or cyanotic episode, seven were preterm (≤ 32 weeks' gestational age), and five were siblings of a SIDS victim. Twenty one of the 52 infants had a further hypoxaemic episode, confirmed in hospital by pulse oximetry to be due to arterial desaturation.

Seventy two patients had a persistent fall in the baseline concentrations of TcPO₂; in 30 patients these lower values were subsequently confirmed by pulse oximetry in hospital to indicate real hypoxaemia. Most of them were preterm infants (20/30) and 12 had bronchopulmonary dysplasia. Twenty five infants had a respiratory infection. In 20 infants (16 preterm) additional inspired oxygen was given, and in six of these further respiratory support was later necessary. One infant with low baseline readings during a bronchiolitis subsequently suffered an apparent life threatening event needing resuscitation while in hospital.

In the remaining 42 patients with a low baseline TcPO₂ identified by parents, this was either proved by pulse oximetry not to represent arterial hypoxaemia (n=13), or the infant was seen by the family doctor and the low readings were not checked against pulse oximetry (n=29). Intercurrent infections, usually respiratory but also gastrointestinal, were subsequently (within

one to two days) or simultaneously identified in 35 (83%) of these patients. For example, in one infant (a SIDS sibling) the monitor alarmed at 3 am, reading 13 mm Hg (1.73 kPa). The values remained low, and the family doctor called could not find anything wrong with the infant except for a rectal temperature of 38°C. She was admitted to hospital for observation. Within one hour of admission, three hours after the initial alarm on the monitor, the infant became drowsy and unresponsive. A lumbar puncture revealed *Haemophilus influenzae* meningitis.

Deaths during monitoring programme

Four infants have died suddenly despite the parents having been issued with a TcPO₂ monitor. The first infant referred at the beginning of the monitoring programme was the twin of a SIDS victim and lived in severely deprived home circumstances. Every effort was made to teach the parents how to use the monitor but on the night of her death it was connected to the infant in the calibration mode, which at that stage of the programme muted the monitor alarms (Kontron 820). The potential danger of this possibility had not been considered by us or the manufacturers but was then overcome by modifying the monitor so that this could not recur (Kontron 821S).

The second infant, who had bronchopulmonary dysplasia and was receiving additional inspired oxygen at home, had demonstrated a major fall in baseline TcPO₂ values for two days before his sudden death. His inspired oxygen concentration was progressively raised in an unsuccessful attempt to compensate for this but unfortunately no contact was made with the hospital. On the third day of this increased oxygen requirement he developed sudden and severe cyanosis while crying, progressed to prolonged apnoea,⁴ and could not be resuscitated. He was found to be dead on arrival at hospital. A postmortem examination showed bronchopneumonia and bronchopulmonary dysplasia.

A third infant with congenital myopathy died suddenly at home. A postmortem examination showed bronchopneumonia. Unfortunately, without informing us, his parents had substituted a breathing movement detector for TcPO₂ monitoring because of an inability to afford the batteries. We now supply rechargeable batteries on loan to financially compromised parents.

The fourth infant, aged 10 months, had suffered recurrent severe cyanotic episodes due to intrapulmonary shunting⁴ and requiring resuscitation on multiple occasions at home and in hospital. He died suddenly while asleep but not on the monitor. His mother was convinced despite our advice to the contrary that cyanotic episodes only occurred when he was awake. The certified cause of death was apnoea of infancy.

Additionally, two infants in the monitoring programme died in hospital during severe pneumonia.

Discussion

VALIDATION STUDIES

This TcPO₂ monitor showed a sensitivity of

Table 2 Outcome of 350 patients undergoing home TcPO₂ monitoring

	No of patients
Hypoxaemic episodes with cyanosis or pallor as reported by parents:	81
Needed vigorous stimulation or mouth to mouth resuscitation	52
Had further episode in hospital documented by pulse oximetry	21
Baseline decreases in TcPO₂ proved to be hypoxaemia (by pulse oximetry):	30
Had respiratory infection	25
Needed additional inspired oxygen	20
Needed subsequent respiratory support	6
Discontinued monitor prematurely	19
Sudden unexpected deaths	4

100% in the detection of sudden severe episodes of hypoxaemia or cyanosis. This contrasts with a sensitivity of 90% for pulse oximetry, 10% for breathing movement monitoring, and 18% for electrocardiographic monitoring. TcPo₂ measurements respond to changes in arterial oxygen tension and in the delivery of oxygen to the skin (skin perfusion). Perhaps because of associated reflex changes in the latter in response to sudden hypoxaemia,¹¹ the response time of the TcPo₂ signal is relatively short. The median delay when compared with the pulse oximeter in the beat to beat mode was 16 seconds.

As the association between SaO₂ and arterial oxygen tension is influenced by the many factors influencing the oxygen dissociation curve, pulse oximetry cannot serve as a gold standard for the validation of oxygen tension monitoring. In addition, the influence of skin perfusion and its effects on TcPo₂ can be considerable. We are careful therefore not to imply that absolute values relating TcPo₂ to SaO₂ can be given. This TcPo₂ monitor has already been extensively validated against arterial oxygen tension measurements.¹²⁻¹³ In this study, we set out to investigate the response of the monitor to sudden onset and rapidly developing hypoxaemia. As it is impractical and unethical to obtain repeated arterial blood gas measurements sequentially during the unpredictable and rapid development of the hypoxaemia that occurs during cyanotic episodes, recordings of pulse oximetry, using a well validated instrument in beat to beat averaging mode,⁶⁻¹⁴ were considered the most objective measure of clinically relevant changes in oxygenation.

Investigations performed in these patients and in other reported studies have shown that hypoxaemia is a better indicator of dangerous respiratory pathophysiology than is cessation of breathing movements or bradycardia.⁴⁻¹⁵ Severe hypoxaemia may also occur despite continued breathing movements and airflow.¹⁶ The use of pulse oximetry could provide a more rapid response time for hypoxaemia. Unfortunately, validated pulse oximetry, although excellent for the non-invasive measurement of oxygenation, is not yet suitable for long term monitoring because of movement artefact (fig 2).

TcPo₂ monitors measure skin oxygen, not arterial oxygen tension. Their mode of action results in a sensitive response to changes in peripheral circulation.¹¹ This response may be of value in identifying potentially dangerous causes of a low cardiac output (for example, cardiac arrhythmia), or of peripheral vasoconstriction (for example, metabolic acidaemia, hypoglycaemia). Hypoxaemia may also cause peripheral vasoconstriction (through sympathetic activity),¹¹ which may explain why the TcPo₂ monitor alarmed before the pulse oximeter readings reached equivalently low values during the relatively slow onset of hypoxaemia. However, further studies are required to investigate these other potentially important functions of the monitor.

The patients in whom our validation studies were performed are not representative of the total population of infants at risk of sudden

death in that many were suffering recurrent cyanotic episodes. Regardless of the primary cause of the pathways leading to death, however, it was our contention that TcPo₂ would fall early in sudden infant death at home because of arterial hypoxaemia, reduced skin perfusion (reduced cardiac output or peripheral vasoconstriction), or both. Because life threatening episodes are rare, even in epidemiologically defined high risk groups, we considered it was most practical to validate our TcPo₂ monitor in infants with recurrent cyanotic episodes.

The variability of the baseline measurements recorded in the questionnaires was considered small enough for parents to become aware of the normal range of oxygen tension values in their baby. There was, however, a downward drift in some patients. This drift usually began after four hours and has been previously reported by others, using the same electrode over a period of six hours.¹⁷

The high rate of false alarms with conventional home monitoring may lead to severe disturbances in family life.¹⁸ Weese-Mayer *et al* identified false alarms from cardiorespiratory monitors with an average frequency of six per day.¹⁹ In our study, including parents with different levels of technical experience with the monitor and different social backgrounds, false alarms occurred with an average frequency of one every four days of monitoring.

The temporary redness of the skin resulting from the heated sensor is undesirable. However, skin injury is minimised by strictly advising the parents to avoid resiting intervals of more than eight hours with more rapid resiting during fevers or after recent exposure to sunlight. Nevertheless, we are concerned about this skin effect and are investigating alternative ways of overcoming this problem.

CLINICAL DATA

The patients entered into this TcPo₂ monitoring programme had heterogeneous problems. However, the majority (93%) came from one of the three well recognised risk groups for sudden infant death (table 1). Nevertheless, it has been questioned whether the subsequent siblings of SIDS victims should be monitored.²⁰ However, in two recent large population based studies the SIDS incidence in this group was four and six times higher than in the normal population.²¹⁻²² In the latter study, the overall postneonatal mortality rate in the SIDS siblings was 20.8 per 1000.²²

The TcPo₂ monitor identified two kinds of hypoxaemia: sudden onset, short lived episodes and longer term, more gradual changes in baseline oxygenation. Acute, apparently life threatening episodes of hypoxaemia were predominantly found in infants referred because of a previous apparent life threatening event. In 73% of occasions the monitor appeared to identify sudden episodes in time to effectively resuscitate the infant by stimulation alone without the need for mouth to mouth or bag and face-mask ventilation (required in the remaining 27%).

Detection of changes in baseline TcPO₂ was particularly valuable in preterm patients; particularly in those with bronchopulmonary dysplasia receiving additional inspired oxygen. Our experience is thus similar to that of Lafeber *et al* who found that TcPO₂ monitoring was more sensitive than pulse oximetry to changes in oxygenation in infants with bronchopulmonary dysplasia.²³ Such baseline changes often accompanied intercurrent respiratory infections and frequently preceded the clinical symptoms and signs apparent to parents. Because of the propensity of airway hypoxia, hypoxaemia, and acidaemia (from tissue hypoxia) to induce pulmonary hypertension, the early detection of baseline hypoxaemia may avert rapid, life threatening falls in arterial oxygen tension due, for example, to sudden intrapulmonary right to left shunts.⁴ The fact that 20 of 30 infants with baseline falls in TcPO₂ confirmed to represent arterial hypoxaemia by pulse oximetry in hospital, needed additional inspired oxygen, and six subsequently needed ventilatory support, supports the idea that the early detection of hypoxaemia may prevent progression to life threatening events in the home; particularly in those infants with bronchopulmonary dysplasia.

The detection of baseline falls in TcPO₂ shown by pulse oximetry *not* to represent hypoxaemia and presumably therefore indicating changes in skin perfusion still deserves further investigation. As shown in the infant with meningitis, but also during gastrointestinal and respiratory infections, it appears that these low readings are a sensitive and early indicator of infection, probably due to peripheral vasoconstriction associated with the infection. We advise our parents to have their baby checked by their family doctor or local hospital if the low readings on their baby appear to be real (that is, the monitor shows normal readings on the parent).

Our data concerning clinical experiences with the monitor at home are limited in their potential accuracy by the lack of medical or nursing experience of most parents. In 26% of patients the occurrence of further proved hypoxaemic episodes in hospital validated parental observation. To date these can represent the only definitive events detected by the monitor. We are now, however, beginning to combine the TcPO₂ monitor with an event recorder. This device senses electrocardiogram, breathing movements, and SaO₂, and stores these data together with the TcPO₂ values onto computer disks before and during a low alarm on the TcPO₂ monitor. Thus recorded physiological data during apparent life threatening episodes at home will become available.

This is the first time that a monitor which has been used at home has been validated on infants in hospital with observed and documented episodes of severe hypoxaemia. Nevertheless there remains, at present, no proof that this monitor is superior to other monitoring devices in the prevention of SIDS. Moreover, as with all the

other existing home monitors, the data on clinical experience of the parents with this monitor at home are uncontrolled and anecdotal. There is absolutely no evidence as yet that this device will effectively reduce the incidence of sudden infant death.

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