Simplified pneumographic monitoring of infants at risk from sudden infant death syndrome

P M RAHILLY AND P F SYMONDS

Departments of Neonatal Medicine and Biomedical Engineering, Royal Alexandra Hospital for Children, Camperdown, Australia

SUMMARY A system of pneumographic study of infants considered at increased risk from the sudden infant death syndrome is described. It is simple for nursing staff to use and for clinicians to interpret. A total of 166 'at risk' infants were studied and 85 were subsequently monitored at home. Seventeen of these infants, 16 of whom had had abnormal pneumograms, subsequently suffered significant apnoea. Four of 20 babies who had abnormal pneumograms did not have subsequent episodes of apnoea. Two babies died; the first was on a monitor but the second, despite having had an abnormal pneumogram, was not.

Sudden infant death syndrome (SIDS) is the most common cause of death among children aged between 1 month and 1 year. Although, by definition, SIDS happens without warning, there are groups of babies who are epidemiologically recognised to be at an increased risk. These include twins and subsequent siblings of SIDS victims,^{1,2} babies suffering a near miss SIDS,^{3,4} and preterm babies.^{5,6} Methods of studying these babies vary but the necropsy findings of Naeye⁷⁻⁹ have led many workers to study respiratory control by complex polygraphic methods.^{3,10-13} Although these polygraphic methods are of great interest physiologically, they are expensive both in manpower and financial terms and they are also 'invasive', which has the double disadvantage of making them unpopular with mother and baby and altering perhaps the very physiological phenomena being studied.

We considered that it would be useful to develop more simple methods of studying breathing patterns that could be used in the clinical setting. We also believed that it would be advantageous to have a system for studying babies over a longer period of time compared with in depth but short term polygraphic studies. Such a system would have to be capable of: (1) detecting episodes of apnoea, periodic breathing, and bradycardia; (2) giving compressed (trend) recordings interpretable by interested clinicians and 'capturing' important episodes during prolonged studies; and (3) being used routinely by nursing sisters. The system would also have to be acceptable to both the baby and parents. We describe such a monitor and results from the first 166 babies studied.

Methods

Pneumographic monitor. The monitor consists of a modified cardiorespiratory monitor (Medtel HS7) and a two channel oscillographic chart recorder (MFE 1200). The unmodified monitor has a rolling display of four seconds duration of one trace electrocardiogram and one trace impedance pneumogram together with apnoea, tachycardia, and bradycardia alarms.

Two modifications were carried out to the monitor to allow trend recordings:

(1) The rolling display memory was slowed to extend the storage time (on screen) to 128 seconds.

(2) The electrocardiographic waveform on the upper trace was replaced by a heart rate signal; the higher the heart rate, the higher the vertical displacement on the screen. Additional markers were included on the screen indicating heart rates of 100 and 200 bpm. The monitor can be operated in the normal (unmodified) or trend (modified) mode at the turn of a switch.

Heart rate and pneumogram signals delayed by 128 seconds are taken from the monitor to the chart recorder via a control unit. The control unit allows the delayed signals to pass directly to the chart ('continuous recording' mode) or to be gated by the monitor's apnoea, tachycardia, or bradycardia alarm circuits before passing to the chart recorder, ('data capturing' mode).

In the 'data capturing' mode the chart recorder is switched on by any one of the alarms and runs for 150 seconds, allowing all 128 seconds of stored information on screen plus approximately 40

Fig. 1 *Pneumographic trace showing central apnoea of duration 22 seconds. The monitor was in the 'data capturing' mode; the write out was triggered by the apnoea and shows preceding events. On this occasion the apnoea alarm was set on 15 rather than the normal 10 seconds for clerical reasons.*

Fig. 2 *Pneumographic trace showing periodic breathing.*

seconds after the episodes to be written. Chart speed is 1 mm/second allowing easy measurement of episodes of apnoea. To minimise chart paper use in continuous operation a chart speed of 25 mm/minute is used.

In normal use, the equipment is wheeled to the bedside on a trolley, standard disposable electrodes are attached to the patient, and the monitor is adjusted (in normal mode) to obtain good electrocardiographic and pneumographic traces. The monitor is then set to the trend mode and recording to either 'continuous recording' or 'data capturing' mode.

In the 'continuous recording' mode central apnoea episodes of any length (Fig. 1) and periodic breathing (Fig. 2) may be detected as well as heart rate changes. In the 'data capturing' mode only those episodes of apnoea and heart rate changes

Fig. 3 *Pneumographic trace showing disorganised breathing associated with a slight rise in heart rate followed by a bradycardia of 68 bpm. This trace suggests obstructive apnoea and subsequent bronchoscopy showed tracheomalacia.*

which sound the monitor's alarm will be recorded. On the HS7 a 10 second episode of apnoea will sound the alarm and although this self cancels if the breathing starts before a total of 15 seconds has elapsed, the recording continues. Periodic breathing will not be recorded in 'data capturing' mode unless the associated episodes of apnoea are of more than 10 seconds.

Obstructive apnoea is difficult to document conclusively without complex screening systems such as nasal thermisters. With this system one relies on detection of increased respiratory excursions associated with tachycardia followed by bradycardia (Fig. 3). For the purpose of scoring (see below) we have

Table 1(a) Normal values for episodes of apnoea/100 minutes of sleep related to age (mean SD)*

Age (weeks)	Episodes of apnoea	
	11-12 sec	13-15 sec
0-4	1.9 (1.2)	0.3 (1.2)
4-8	1.2 (0.9)	0.2 (0.9)
8-20	1.4 (0.2)	0.2 (0.2)
20-52	1.6 (0.9)	0.6 (0.9)

Table 1(b) Range of periodic breathing for normal infants and for siblings of victims of sudden infant death syndrome (SIDS) related to age*

Age weeks	Range of percentage of time in periodic breathing	
	Normal infants	SIDS siblings
0-4	0-3.5	0-5.0
4-8	0-2.5	0-3.5
8-20	0-1.5	0-2.5
20-52	0-1.0	0-1.5

*Determined by Shannon and Kelly^{4,14} and in personal communication.

taken bradycardia of below 80 bpm as 'abnormal' and when this is associated with the above breathing pattern we investigate further to confirm or exclude obstructive apnoea.

Scoring of the pneumogram

Pneumograms were scored using the normal values of Shannon and Kelly (Table 1).^{4,14} These were obtained using similar non-invasive impedance monitors on infants of the relevant age range. For the purpose of this study babies falling outside one standard deviation for apnoea and the normal range for periodic breathing were considered 'abnormal'. Babies having episodes of disorganised breathing associated with bradycardia of below 80 bpm were also considered 'abnormal'.

Patients

A total of 166 babies had pneumographic studies performed. Ninety two (mean age 5 weeks, range 1 to 10 weeks) suffered from what was labelled 'near miss' SIDS, in that when thought to be asleep, they had been discovered to be very pale or blue and apparently not breathing. All these babies had needed vigorous shaking or mouth to mouth resuscitation. Pneumographic studies included continuous recordings of one or more nights and between three and 7 nights' data capturing. In addition the infants were investigated with a protocol (Table 2) to exclude medical conditions that

Table 2 Investigations performed on infants suffering 'near miss' SIDS

Full blood count and film	Urea and electrolytes
Calcium	Magnesium
Fasting blood sugar	Electroencephalogram with eye ball pressure
Chest radiograph	Lateral airways radiograph
Radionuclide oesophogram	Auditory evoked potentials of the brain stem

could be treated. Sixty seven subsequent siblings of SIDS victims (mean age 3.6 weeks, range 1 to 10 weeks) and 7 children (mean age 6 weeks, range 3 to 8 weeks) with anxious parents were studied with single overnight pneumograms. Management and follow up of these babies is discussed below.

Results

Medical investigations. Sixty six of the babies had moderate to gross reflux, 7 had abnormal electroencephalograms, and 11 had notable bradycardia on eyeball pressure. Six infants had upper airways abnormalities found on bronchoscopy. These medical findings are part of another study and will be discussed in a separate paper.

Pneumograms. Twenty seven had abnormal pneumograms, 11 of these were siblings of SIDS victims, and 16 were 'near miss' SIDS. Three of the 'near miss' SIDS babies had several episodes of apnoea of 15 seconds or greater. One SIDS sibling and one 'near miss' SIDS infant had increased episodes of apnoea of between 11 and 15 seconds duration. One SIDS sibling and two 'near miss' SIDS babies had disorganised breathing and a considerable number of episodes of bradycardia. Ten infants (four SIDS siblings and 6 'near miss' SIDS) showed excessive periodic breathing, and another five (all SIDS siblings) showed a percentage of time in periodic breathing which was above normal but within Shannon's range for SIDS siblings.¹¹ Four 'near miss' SIDS infants had pneumograms suggestive of an obstruction, and bronchoscopy confirmed airway abnormalities (one tracheomalacia, one subglottic stenosis, one laryngomalacia, and one laryngeal web). Two babies with normal pneumograms were considered to have abnormal airways and subsequent bronchoscopy showed left vocal cord palsy in one and tracheomalacia in the second.

Management of patients. Patients found to have gastroesophageal reflux, fits, or airway abnormalities were treated appropriately. Two infants who had presented with severe apnoea and had excessive

periodic breathing were treated with theophylline. Their subsequent pneumograms were normal, and for the purpose of the follow up studies they were classified as 'normal'. The babies with airway abnormalities were treated with a variety of surgical and conservative regimens and were excluded from the follow up study. Of the babies without airway pathology, 65 'normal' and 20 'abnormal' babies were monitored at home. Thus, in only one 'abnormal' baby was monitoring declined. Monitors mainly consisted of two types of mattress monitors; Tenby, EMI and in 11 cases Health-Dyne Impedance Cardio Respiratory Monitors. Monitoring was continued at home for a mean of 7.2 months (range 4 to 18 months).

Follow up studies. All home monitored babies were followed by one of the authors (PR) for a mean of 7 months (range 4 to 28 months) and the incidence of subsequent apnoea was documented. In addition, follow up data was obtained from 74 of the 75 unmonitored babies, one being lost to follow up after returning to England.

Results of follow up studies. One of the 65 'normal' babies monitored suffered from serious apnoea. A second 'normal' baby died, despite the monitor sounding, when the parents were unable to resuscitate him successfully. Sixteen of the 20 'abnormal' babies had appreciable episodes of apnoea. These are therefore significantly different populations (χ^2 , 67.7; $P < 0.001$) and the pneumogram as a predictor of future apnoea has both a specificity and sensitivity of 94%.

All the 'normal' unmonitored babies survived with no evidence of apnoea. One 'abnormal' baby, a 'near miss' SIDS with an excessive number of episodes of apnoea of 11 to 15 seconds duration died at home at the age of 16 months and was diagnosed at necropsy as SIDS.

Discussion

Judgement on how easy it is to use a piece of equipment is, to a great extent, subjective. The unmodified cardiorespiratory monitor is of a design and type often used in children's hospitals and it is therefore not surprising that skilled but not specifically trained or delegated nursing staff could come to terms with its use even when modified. Thus, nursing sisters were soon able to set up the monitor and run it overnight making appropriate observations.

The scoring of the pneumogram takes about 10 to 15 minutes in the case of a normal pneumogram of several days data capturing recording and about 20

to 30 minutes in the case of an abnormal pneumogram. In all cases scoring was carried out by one of the authors (PR), a paediatrician with previous experience in experimental neonatal physiology, who found it simple to score the pneumogram as part of the routine ward round.

Considering that all these infants were from a selected 'at risk' population (apart from the infants of anxious parents) the range of apnoea and of periodic breathing compares reasonably closely to that of Shannon and Kelly,¹¹ in that only 27 of the babies fell outside their normal range. The low incidence of periodic breathing compared with that found by some authors is of interest.^{15 16} The variation in normal values found by different authors may be explained in part by different monitoring techniques and the time of day or night at which the recordings were made.

There are several reasons why these studies are useful as part of the investigation of 'near miss' SIDS and babies being considered for home monitoring. They give useful insight into the cause of a 'near miss' episode and they show periods of shallow breathing which, although normal, may well cause false alarms at home. These periods can be demonstrated to the parents along with other normal variants and so used as part of the training programme in the difficult task they have ahead of them. Probably most important of all, these studies show appreciable episodes of bradycardia which would be undetected by simple mattress monitors as chest wall movement continues (for instance in obstructive apnoea).

There is some evidence that future SIDS victims have abnormal respiratory patterns during the first days of life,¹⁷ although studies are hard to conduct as abnormal respiratory patterns are often treated.¹⁸ Again, with a normal incidence of about 1/800 sudden infant deaths huge studies have to be carried out before any system may be proposed as a true 'screening test' for SIDS. More complex polygraphic studies have shown that babies with abnormal respiratory patterns often suffer future episodes of apnoea.¹⁹ Our follow up studies were designed to test whether the simple pneumograms predicted future apnoea. The results indicate that babies with an abnormal pneumogram in the first month of life do come from a significantly different population than those with normal pneumograms, and that the test has an acceptably high specificity and sensitivity.

It is instructive to consider the cases where the pneumogram failed to predict the actual outcome. Two of the four babies with abnormal pneumograms who did not go on to have apnoea had excessive periodic breathing. One of these was a SIDS sibling and the other was a 'near miss' SIDS who also had

fairly notable reflux, and in whom treatment for reflux may have removed the precipitating factor of the original apnoea. The other two infants with abnormal pneumograms who had no subsequent apnoea were two SIDS siblings in whom the incidence of periodic breathing was above normal but within the range noted by Shannon in his SIDS siblings.¹⁴ In other words, of these four, two had only minor abnormalities and the third had a treatable precipitating factor of apnoea.

One baby had a notable episode of apnoea after a normal pneumogram. This baby was interesting in that his original pneumogram at 10 days of age was abnormal and he subsequently had several episodes of apnoea. The second pneumogram was carried out because the parents felt the baby had recovered and were trying to decide whether or not to stop monitoring.

The baby with a normal pneumogram who died is obviously of concern. This SIDS sibling died at the age of 4 months, three months after his normal pneumogram, and no episodes of apnoea were monitored. The baby slept beside the parents and one morning they were awoken by the alarm and found the baby warm but not breathing and the mother (a trained nurse) was unable to resuscitate the baby using mouth to mouth respiration and cardiac massage. Necropsy showed no abnormalities. It is arguable on the basis that the baby had no previous episodes of apnoea and was impossible to resuscitate that he did not in fact die from primary apnoea as such but from one of the other even less well understood causes of cot death.

Our experiences of the impact of home monitoring on the parents are similar to those of other workers^{20, 21} and despite the relatively high incidence of false alarms not one of the parents regretted the decision of having a home monitor. It has to be remembered that parents who have found their baby dead or apparently not breathing while thought to be sleeping will have a very different outlook on the problems of monitoring than most other people.

In conclusion, we have presented a modification of the cardiorespiratory monitor which allows screening of breathing patterns in a fashion acceptable to the babies, the parents, the nursing staff of a busy general ward, and the fulltime clinician undertaking the programme. The system gives information helpful in the management of 'near miss' SIDS and in any baby who is to undergo home monitoring.

We thank Professor D C Shannon and Dr D Kelly for permission to use and quote their normal values, and are grateful to Professor D Read for his encouragement. We also thank the nursing staff of Faithful Ward.

References

- 1 Froggatt P, Lynas MA, MacKenzie G. Epidemiology of sudden unexpected death in infants (cot death) in Northern Ireland. *Br J Prev Soc Med* 1971;119:25.
- 2 Peterson DR, Chin NM, Fisher LD. The sudden infant death syndrome: repetitions in families. *J Pediatr* 1980;97:265-7.
- 3 Guilleminault C, Ariagno R, Souquet M, et al. Abnormal polygraphic findings in near-miss sudden infant death. *Lancet* 1976;i:1326-7.
- 4 Kelly DH, Shannon DC. Periodic breathing in infants with near-miss sudden infant death syndrome. *Pediatrics* 1979;63:255-60.
- 5 Kulkarni P, Hall RT, Rhones PG, Sheehan MB. Postneonatal infant mortality in infants admitted to a neonatal intensive care unit. *Pediatrics* 1978;62:178-83.
- 6 Spiers PS, Wang L. Short pregnancy interval, low birth weight and the sudden infant death syndrome. *Am J Epidemiol* 1976;104:15-21.
- 7 Naeye RL. Pulmonary arterial abnormalities in the sudden infant death syndrome. *N Engl J Med* 1973;289:167-70.
- 8 Naeye RL. Hypoxemia and the sudden infant death syndrome. *Science* 1974;186:837-8.
- 9 Naeye RL. Brain stem and adrenal abnormalities in the sudden infant death syndrome. *Am J Clin Pathol* 1976;66:526-30.
- 10 Steinschneider A. Prolonged apnea and the sudden infant death syndrome: clinical and laboratory observations. *Pediatrics* 1972;50:646-54.
- 11 Shannon DC, Kelly DH, O'Connell K. Abnormal regulation of ventilation in infants at risk for sudden infant death syndrome. *N Engl J Med* 1977;297:747-50.
- 12 Guilleminault C. State of sleep and apnoea in infants (near miss for SIDS and controls). Mead Johnson Symp. *Perinat Dev Med* 1977;12:25-31.
- 13 Guilleminault C, Peraita R, Souquet M, et al. Apneas during sleep in infants: possible relationship with sudden infant death syndrome. *Science* 1975;190:677-9.
- 14 Kelly DH, Walker AM, Cahen L, Shannon DC. Periodic breathing in siblings of sudden infant death syndrome victims. *Pediatrics* 1980;66:515-20.
- 15 Adamson TM, Grange S, Maloney JE, Wilkinson MH, Wilson FE, Yu VYH. The maturation of respiratory patterns in normal fullterm infants during the first six postnatal months. I: Sleep states and respiratory variability. *Aust Paediatr J* 1981;17:250-56.
- 16 Adamson TM, Cranage S, Maloney JE, Wilkinson MH, Wilson FE, Yu VYH. The maturation of respiratory patterns in normal fullterm infants during the first six postnatal months. II: Sleep states and apnoea. *Aust Paediatr J* 1981;17:257-61.
- 17 Steinschneider A, Weinstein SL, Fiamond E. The sudden infant death syndrome and apnea obstruction during neonatal sleep and feeding. *Pediatrics* 1982;70:858-63.
- 18 Southall DP, Richards JM, Rhodes KH, et al. Prolonged apneas and cardiac arrhythmias in infants discharged from neonatal intensive care units—failure to predict an increased risk for sudden infant death syndrome. *Pediatrics* 1982;70:844-51.
- 19 Kahn A, Blum D. Home monitoring of infants considered at risk from the sudden infant death syndrome, four years experience (1977-1981). *Eur J Pediatr* 1983;139:94-100.
- 20 Black L, Hersher L, Steinschneider A. Impact of the apnea monitor on family life. *Pediatrics* 1978;62:681-5.
- 21 Cain LP, Kelly DH, Shannon DC. Parents perceptions of the psychological and social impact of home monitoring. *Pediatrics* 1980;66:37-41.

Correspondence to Dr P M Rahilly, Wade House Consulting Rooms, Royal Alexandra Hospital for Children, Camperdown, NSW 2050, Australia.

Received 17 November 1983