Monitoring for central apnoea in infancy—limitations of single channel recordings

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SUMMARY Apparent central apnoea (absent breathing movements) detected by monitoring movement of the thoracic wall was compared with simultaneous detection by abdominal wall movement. Eighteen infants provided one or more 24 hour recording of heart rate (electrocardiography), thoracic respiration (transthoracic impedance), and abdominal wall movement (pressure sensitive capsule distortion). Detection of true apnoea, recognition of artefact, and measurement of the duration of true apnoea were all improved when two channels of respiratory monitoring were used in combination. We recommend that any study purporting to observe breathing patterns by indirect recording of respiratory movement will be more reliable if more than one channel of respiratory movements is recorded simultaneously. Further, in infants no estimation of duration of central apnoea can be made on the basis of either a transthoracic impedance record alone or an abdominal wall movement sensor alone. Comparison of findings among studies using different single channel recordings are unlikely to be meaningful.

Several reports on the occurrence of central apnoea in infancy have been based on information provided by a single respiratory channel that detects cessation of either chest or abdominal movements.^{1–6} The work of Henderson-Smart and Read,⁷ Curzi-Dascalova,⁸ and our own observations of polygraphic recordings, however, led us to question the reliability of this approach as the relative contributions of chest and abdominal movements to respiratory excursion may vary considerably among infants and, over a short time period, in the same infant. As a preliminary to studying patterns of central apnoea and heart rate in infants in the home, we recorded simultaneously chest and abdominal movements on independent channels and compared information obtained regarding central apnoea for each channel with that derived from the two channels combined. We made no attempt to detect obstructive apnoea—cessation of airflow at the mouth and nostrils with continuing respiratory movements²—as the use of airflow sensors is impracticable in the home setting. Our recording system also included heart rate. We present evidence that the recording of both chest and abdominal movements simultaneously improves the detection and quantitation of central apnoea.

Subjects and methods

The subjects, 11 boys and seven girls, were a subgroup from 50 infants being studied prospectively for respiratory pauses and heart rate during sleep at different ages through infancy. Five had been born prematurely but had recovered from all neonatal problems by the time of discharge home. Each was clinically normal and apparently healthy. The infants had at least two 24 hour recordings in the home between 46 and 60 weeks postconception.

Simultaneous recordings of the electrocardiographic and respiratory signals from the thorax were obtained using a single pair of paediatric adhesive electrocardiographic electrodes applied on opposite sides of the chest on the anterior axillary lines at the level of the nipples. The initial adequacy of the respiration signal was confirmed by the doctor who prepared the infant for recording either by checking a visual oscilloscope signal during quiet breathing or including a calibration period at the start of the tape for later inspection. The electrocardiographic and transthoracic impedance signals from these electrodes were recorded on a Healthdyne infant monitor incorporating a Medilog I 24 hour four channel ambulatory taperecorder. The abdominal

movement signal was obtained using a Graseby MR10 respiration monitor. This device has as its sensor for respiration a soft plastic capsule taped firmly to the anterior abdominal wall. Air pressure variations within the capsule associated with abdominal wall movement are transmitted along a fine plastic tube to the monitor.⁹ This instrument was modified to provide a continuous analogue electrical signal representing the pressure waves within the capsule that was recorded on a third channel of the tape recorder in the Healthdyne monitor in addition to the electrocardiographic and thoracic impedance signals. The alarm facilities of the monitors were not used in the present study.

A specially trained senior physiological measurement technician (GB) continuously monitored the signals on a large screen oscilloscope during the playback. The tapes were replayed at a speed 60 times faster than the original recording speed and the electrocardiographic and the two respiratory signals fed to solid state electronic buffer memories that allowed signals to be captured at any time during analysis. The two respiratory signals were also fed to a purpose built apnoea analyser that compared the amplitude of each respiratory waveform with a threshold that had been adjusted by the operator to 25% of the amplitude of the respiratory signal during a calibration period in which the infant was at rest and breathing quietly. This 25% rule was more stringent than either an arbitrary visual threshold or both monitors' own breath detection threshold. Movements with amplitudes less than a quarter of this were considered not to be associated with appreciable ventilation and could be discounted as effective breaths.

The analyser could be switched to measure the time between successive breath detections in (a) the thoracic impedance channel alone, (b) the abdomi-

nal pressure channel alone, or (c) the two channels taken together. In this last mode the duration of an apnoeic pause was thus the period during which neither the thoracic nor abdominal channel detected a breath.

An adjustable 'apnoea duration threshold' was provided in the analyser; this could be set to generate an output automatically whenever the breath-to-breath interval as defined by one of the above detection modes exceeded a chosen value. An electronic progress clock within the analyser accumulated the total time during which analysis was in progress.

Each time an episode of apnoea was detected the analyser output was used to 'freeze' the whole system: this stopped the tape replay and the progress clock and captured in the buffer memories the last 40 seconds of the electrocardiographic and respiratory waveforms. The signals could then be further examined on the oscilloscope screen and both respiratory traces printed out with the corresponding electrocardiographic signal (fig 1).

The operator was provided with a hand held push button control box from which the system could be 'frozen' manually for detailed inspection of disturbances noticed on the monitor screen. Another push button on the control box was used to disable analysis and stop the progress clock while allowing the tape replay to continue past any periods in which the quality of the recorded signals was inadequate because of recording artefacts. At the end of the tape the progress clock was read to obtain the total record duration actually analysed for that patient.

We elected to analyse 100 pauses >10 seconds for each respiratory channel (chest and abdomen). To ensure that a wide range of different recordings was included we selected by random numbers 27 tapes for scrutiny. As a single tape with frequent apnoeic

Fig 1 Corresponding heart rate, transthoracic impedance, and abdominal movement during normal quiet breathing.

pauses could easily have resulted in 100 pauses from the same recording, we used a sequential sampling technique whereby the first three pauses of each tape were studied and analysis continued using the next tape in the series. Once all 27 tapes had been sampled for the first three pauses >10 seconds the process was continued cycling through the parts of each tape not yet analysed. This resulted in all 27 tapes contributing to the documented results. The procedure was then repeated with judgments on the presence of central apnoea in each channel being made by visual inspection.

We defined bradycardia as a heart rate of <80 beats/minute for >10 seconds measured as sequential R-R intervals. We noted its occurrence in relation to central apnoea but made no attempt to quantify it in the tapes studied.

Results

The results were analysed using two criteria for absence of breaths on individual channels—one based on operator judgment and the other on the threshold for absent breaths as <25% of the amplitude seen in quiet breathing. There were no significant differences in the results obtained using these methods ($p>0.05$); findings for the former method are reported here. One hundred periods of absent chest signals (>10 seconds) were identified from a total of 559 hours of recording. The abdominal signal yielded 100 pauses from 266 hours of recording. The table gives the results obtained. On 30 occasions visual inspection suggested that transient poor lead contact was the cause of the sub-threshold signal (straight line appearance of apparent apnoea), while normal breathing movements were detected on the other channel. In the remaining 70 occasions where there were breathing pauses >10 seconds by transthoracic impedance, 33 (47%) were confirmed as true central apnoea of this duration after inspection of the second channel. In 13 (19%) instances breathing movements continued normally

on the second channel ('false' apnoea identified), while in a further 24 (34%) instances, the pause in respiratory movements in the abdominal channel was less than 10 seconds ('false' apnoea duration identified). Thus in 67 of 100 electronically detected episodes of apnoea and in 37 of 70 (53%) episodes of combined electronically detected and visually detected apnoea based on single channel transthoracic impedance data, the use of a second respiratory channel altered the interpretation in qualitative or quantitative terms.

When the abdominal respiratory movement channel was inspected a similar discrepancy was seen between single channel and dual channel findings. The number of signal failures interpreted as apnoea was much smaller, only 2%, but in 59 instances (60%) the abdominal channel was silent for 10 seconds or more while the thoracic channel continued to show normal breathing movement. On 15 (15%) occasions the duration of the pause in abdominal movement was up to 5 seconds longer than that in thoracic movement, altering the quantitative assessment of the apparent central apnoea. Figs 2–4 show patterns observed in simultaneous recordings of chest and abdominal movements.

Apparent apnoea on one respiratory channel and normal respiratory movements on the other was not associated with bradycardia. Like other investigators, however, we found that prolonged central apnoea was sometimes accompanied by bradycardia after a variable period of up to 15 seconds.¹⁰ Bradycardia occurring in association with apnoea may indicate arterial hypoxaemia, although this postulate has been challenged by Peabody *et al.*¹¹

Discussion

Both chest and abdominal movements contribute to the normal breathing movement patterns of infants with their relative contributions in individual infants changing with sleep state and body position.⁸ We have shown that this variation may also be relevant

Table Results of recordings when <25% of the amplitude seen in quiet breathing was used as the respiratory threshold

Interpretation of both channels combined	100 pauses >10 seconds in thoracic movement	100 pauses >10 seconds in abdominal movement
Channels agree: 'true' apnoea	33	10
Channels disagree:	67	90
Normal breathing movement on other channels ('false' apnoea)	13	73
Duration of pause <10 seconds on other channel	24	15
Suspected signal failure (brief loss of background 'noise')	30	2

Fig 2 True central apnoea with absence of respiratory movement.

Fig 3 Absence of chest movement while abdominal movement continues—that is, adequate breathing appearing as apnoea when single channel only is observed.

Fig 4 Absence of abdominal movement while chest movement continues.

to the interpretation of central apnoea when a respiration monitor relies on only one component of respiratory movement. Transthoracic impedance changes as the chest circumference changes and gas content of the chest alters with breathing.¹² Although the theory of the method has been disputed,^{13 14} it is accepted as a useful measure of thoracic respiratory movement (or its absence) given appropriate lead siting and attachment. The brief signal failures detected in the present study, and which accounted for 30 discrepant results, commonly occurred during gross body movements when impedance signals could exceed maximum threshold. The remaining discrepancies between chest and abdominal signals were probably caused by the relative paucity of chest movements when breathing was predominantly abdominal.

Sensor or electrode detachment can contribute to observed errors using any method of monitoring. Faults in sensor attachment and signal production from the abdominal pressure capsule (for example, insecure taping to the skin) may be difficult to

identify. This highlights the need for a second method of respiration detection as a failsafe against over diagnosis of apnoea or of its duration. The initial studies describing the use of abdominal sensor capsule were performed in the neonatal period when spontaneous changes in body position (for example, rolling over) are less often seen than in older infants.⁹ Variability of total abdominal wall excursion with each breath or in the site of greatest movement are evident when observing sleeping infants. Thus in studies that use the volume capsule to indicate cessation of respiratory movements any apparent absence of movement must be confirmed by an alternative method and the duration of pauses specified with reference to all breathing movements. Diagnosis or quantitation of central apnoea based on a single channel of respiration, using either of the monitoring systems described, is liable to lead to errors in the detection of apnoea, measurement of its duration, and analysis of prognostic risks.¹⁵

The possibility exists that our threshold for detection of apnoea was inappropriate. No pub-

Fig 5 Typical 24 hour printout of heart rate, pauses >3 seconds/hour and apnoea >3 seconds frequency and duration in a normal infant. The apnoea duration trace represents each pause of 3 seconds or more as a vertical line whose height corresponds to length of apnoea as indicated on vertical axis.

lished report on single channel recording of central apnoea gives precise guidance on detection criteria. We found that visual interpretation of cessation of movements on either channel resulted in as discrepant findings for chest and abdominal movements as the fixed threshold chosen. It is clearly impossible to be absolutely sure that apparent respiratory pauses on a single channel do indicate movement cessation with absent or ineffective breathing. The cessation of movement on both channels at the same time diminishes the likelihood of error.

Inclusion of heart rate is useful as an indication of adequate impedance electrode contact and in the detection of changes in heart rate, but it does not necessarily overcome the difficulty inherent in interpreting the results of a single channel recording. A normal heart rate allows no conclusions to be drawn about occurrence or duration of central apnoea. On the other hand, absence of respiratory movement on one channel with bradycardia is likely to confirm true central apnoea, though of uncertain duration. The occurrence of bradycardia may also relate to obstructive apnoea with continuing respiratory movements. Theoretically absence of movement on one channel, even when associated with bradycardia, does not preclude the occurrence of movements on the second respiratory channel although, in practice, this situation was not observed in the course of these recordings.

Our results indicate that interpretation of coincidental respiratory pauses of the duration observed (up to 20 seconds) by the methods outlined demands two channel respiratory monitoring. This does not infer that chest or abdominal movements are silent during periods of normal breathing but rather that breathing movement detection was imperfect using either of two commonly used monitoring systems. As a result of our findings on two channel monitoring we have studied patterns of respiratory pauses in infancy prospectively using two channels for respiration and one for heart rate (fig 5). This system detects the occurrence and duration of cessation of movements associated with respiration (central apnoea) and allows more precise interpretation of respiratory events associated with bradycardia than is possible with a single channel system.

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References

- Hoppenbrowers T, Hodgman JE, Harper RM, Hofmann E, Sterman MB, McGinty DJ. Polygraphic studies of normal infants during the first six months of life. III Incidence of apnea and periodic breathing. *Pediatrics* 1977;**60**:418-25.
- Guilleminault C, Ariagno T, Korobkin R, et al. Mixed and obstructive sleep apnea and near miss for sudden infant death syndrome. Comparisons of near miss and normal control infants by age. *Pediatrics* 1979;**64**:882-91.
- Stein IM, White A, Kennedy JL, Merisalo RL, Chernoff H, Gould JB. Apnea recordings of healthy infants at 40, 44 and 52 weeks post conception. *Pediatrics* 1979;**63**:724-30.
- Southall DP, Richards J, Brown DJ, Johnston PGB, De Swiet M, Shinebourne EA. 24-hour tape recordings of ECG and respiration in the newborn infant with findings related to sudden death and unexplained brain damage in infancy. *Arch Dis Child* 1980;**55**:7-16.
- Stein IM. Patterns of the pediatric pneumogram. *Medical Instrumentation* 1979;**13**:177-80.
- Mitchell I, Barclay RP, Railton R, Fisher J, Conely J. Frequency and severity of apnoea in lower respiratory tract infection in infancy. *Arch Dis Child* 1983;**58**:497-9.
- Henderson-Smart DJ, Read DJC. Depression of respiratory muscles and defective responses to nasal obstruction during active sleep in the newborn. *Aust Paediatr J* 1976;**12**:261-6.
- Curzi-Dascalova L. Thoraco-abdominal respiratory correlations in infants; constancy and variability in different sleep states. *Early Hum Dev* 1978;**2**:25-8.
- Valman HB, Wright BM, Lawrence E. Measurement of respiratory rate in the newborn. *Br Med J* 1983;**286**:1783-4.
- Southall DP, Richards JM, Lau KC, et al. An explanation for failure of impedance apnoea alarm systems. *Arch Dis Child* 1980;**55**:63-5.
- Peabody JL, Philip AGS, Lucey JF. 'Disorganised breathing'—an important form of apnoea and cause of hypoxia. *Pediatr Res* 1977;**11**:540.
- Farman JV, Juett DA. Impedance spirometry in clinical monitoring. *Br Med J* 1967;**iv**:27-9.
- Victorin L, Daily W, Olsson T. Transthoracic impedance methodological studies in newborn infants. *Acta Paediatr Scand [Suppl]* 1970;**207**:37-47.
- Kira S, Hukushima Y, Kitamura S. Transthoracic electrical impedance variations associated with respiration. *J Appl Physiol* 1971;**30**:820-6.
- Southall DP, Richards J, Rhoden KJ, et al. Prolonged apnea 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.

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