Short reports

Pressure volume characteristics of the lungs in sudden infant death syndrome

D G FAGAN AND A D MILNER

Departments of Pathology and Child Health, University Hospital, Nottingham

SUMMARY Data on the pressure volume characteristics of left lungs obtained from 23 babies dying from sudden infant death syndrome were compared with results from 18 length-matched babies dying from established but primarily non-pulmonary causes. Volume distension at 30 cm of water and deflation flow volume characteristics were very similar in the two groups. These findings do not suggest that babies dying from sudden infant death syndrome have abnormally stiff lungs.

The aetiology of sudden infant death syndrome, now the third most important killer in infancy after prematurity and major congenital abnormality, continues to elude us. A recent study¹ has shown that the composition of surfactant in babies dying from sudden infant death syndrome differs strikingly from that of babies and infants dying of other causes. In particular, the lung washings from the victims of sudden infant death syndrome had relatively less phospholipid and dipalmitoylphosphatidylcholine compared with others, a pattern similar though not identical to preterm babies dying from the idiopathic respiratory distress syndrome. These changes, which were highly significant, suggested that although the appearances at necropsy do not show the alveolar collapse and hyaline membranes seen in idiopathic respiratory distress syndrome, a degree of alveolar instability may be important at times when the baby is compromised by an infection.

With this hypothesis in mind we considered it important to re-examine data² on the pressure volume characteristics of lungs from victims of sudden infant death syndrome and to compare the results with data from babies who had died of primarily non-respiratory causes.

Subjects and method

Method. The left lung was removed during routine

necropsy at the Children's Hospital, Sheffield. In every case the body had been stored at 4°C and the necropsy carried out within 48 hours of death. The lungs were floated in polythene bags in a water bath at either 18 or 37°C for two hours. The lungs were not voided of air but were weighed, and their volume measured by water displacement. The volume of air in the lungs was then calculated, assuming a specific gravity of 1.062 for lung tissue. The left main bronchus was then cannulated and the preparation mounted within a glass container, using the method described by Gribetz *et al.*³

The lung was then inflated up to a pressure of 30 cm of water which was maintained for at least 30 seconds. The volume changes were noted and added to the initial calculated lung volume. This volume was then reduced to 90, 60, and 50% of its original value and at each point the distending pressure was measured once the pressure manometer had stabilised. Finally, the distending pressure was reduced to 2.5 cm of water and the lung volume recorded. Thus it was possible to measure the maximum distending volume at 30 cm of water and to define the deflation curve of the left lung. Further details are published elsewhere.⁴

Subjects

Data were collected on the lungs of 23 babies who died suddenly and unexpectedly. Careful necropsy showed either no pathology (14 of 23) or evidence of only mild respiratory tract infection (9 of 22), representing groups D and C in the Sheffield classification.⁵ Their crown-heel length ranged from 50 to 88 cm (mean (SEM), 62.1 (1.86)). Information was available on 18 control babies whose crown-heel length lay approximately within the same range, that is from 52 to 88 cms (mean (SEM), 66.1 (2.91)). None of these children had died primarily as a result of pulmonary illness or had important lung pathology at necropsy (Table 1).

Table 1 *Causes of death in control group*

Hydrocephalus	5
Cerebral palsy	2
Gastroenteritis and dehydration	2
Ventriculitis	1
Uraemia	1
Hepatic failure	1
Neuroblastoma	1
Duodenal stenosis	1
Waterhouse-Friedricksen syndrome	1
Croup	1
Congenital heart disease	1
Bacterial meningitis	1

Results

The maximal inflation volume was slightly higher in the control group (Table 2). The distending pressure at 90, 60, and 50% of the maximum initial volume tended to be slightly lower in the victims of sudden infant death syndrome. All these differences were small and none reached statistical significance. When the babies were divided into 10 cm crown-heel length groups there were again no statistical differences between the index and control babies (Table 3). There was also no evidence that there was a baby or group of babies who had died as a result of sudden infant death syndrome who had abnormally stiff lungs as determined by the maximal inflation volume or deflation pressure volume characteristics.

Discussion

Although the distending volume at 30 cm of water was slightly higher in the control babies, this did not reach significance and was almost certainly due to an excess of babies with a crown-heel length of greater than 80 cm, compared with the study group (four compared with one). These findings indicate that babies dying of sudden infant death syndrome had lungs which did not differ in their mechanical recoil characteristics from those of babies dying of established but primarily non-pulmonary causes when studied 48 hours after death—a time interval which is similar to that occurring in Morley's surfactant studies.¹ There was no single victim of sudden infant death syndrome whose lung pressure volume results lay outside the range of the matched controls. It is of interest that James and his colleagues⁶ found virtually identical surfactant deficiency abnormalities in term neonates with congenital pneumonia and transient tachypnoea, again supporting the concept that these chemical findings represent secondary changes. Our findings and those of James *et al*⁶ suggest that the biochemical abnormalities which they and Morley's group¹ describe are not necessarily associated with changes in lung mechanics and thus conclusions on lung stiffness should not be deduced from biochemical assays. In summary, we are unable to find any evidence from our pressure volume studies to

Table 2 *Inflation volume at 30 cm H₂O (V_{max}) and 2.5 cm H₂O (V_{2.5}) and distending pressures at 90%, 60%, and 50% of V_{max} (P₉₀, P₆₀, and P₅₀) in babies dying from sudden infant death syndrome (SIDS) and from non-pulmonary pathology (controls). (Values are mean (SEM))*

	V _{max} (ml)	V _{2.5} (ml)	P ₉₀ (cm H ₂ O)	P ₆₀ (cm H ₂ O)	P ₅₀ (cm H ₂ O)
SIDS (n=23)					
Mean (SEM)	185.8 (24.9)	89.0 (8.7)	13.3 (0.6)	3.6 (0.2)	2.5 (0.2)
Controls (n=18)					
Mean (SEM)	231.0 (35.2)	101.0 (11.7)	13.7 (1.1)	3.8 (0.3)	2.6 (0.2)

Table 3 *Results of inflation volume at 30 cm H₂O for sudden infant death syndrome (SIDS) and control babies expressed in 10 cm crown-heel length groups. (Values are mean (SEM))*

Crown-heel lengths group (cm)		No	Mean crown-heel length (cm)	V _{max} (ml)
50-59	SIDS	10	55.6 (0.96)	112.0 (12.0)
	Controls	7	53.1 (0.7)	106.3 (19.7)
60-69	SIDS	9	62.3 (1.0)	180.7 (8.4)
	Controls	4	66.7 (1.4)	190.0 (22.4)
70-79	SIDS	3	74.2 (2.4)	301.7 (43.5)
	Controls	3	75.0 (2.0)	300.7 (35.0)
80-89	SIDS	1	88.0 (—)	621.0 (—)
	Controls	4	82.5 (1.9)	437.7 (64)

V_{max}=inflation volume at 30 cm H₂O.

confirm that babies dying from sudden infant death syndrome have increased alveolar instability as a result of surfactant abnormalities.

References

- ¹ Morley CJ, Hill CM, Brown BD, Barson AJ, David JA. Surfactant abnormalities in babies dying from sudden infant death syndrome. *Lancet* 1982;i:1320-3.
- ² Fagan DG. Developmental and pathological studies of the structure and function of the lung in children. (Thesis). Cambridge: Cambridge University, 1973.
- ³ Gribetz I, Frank WR, Avery ME. Static volume-pressure relationships of excised lungs of infants with hyaline membrane disease, newborn and stillborn infants. *J Clin Invest* 1959;38: 2168.
- ⁴ Fagan DG. Postmortem studies of the semistatic volume-pressure characteristics of infants' lungs. *Thorax* 1976;31: 534-43.
- ⁵ Emery JL. Unexpected death in infancy. In: Hull D, ed. *Recent advances in paediatrics* - 5. Edinburgh: Churchill Livingstone, 1976:203-20.
- ⁶ James DK, Chiswick ML, Harkes A, Williams M, Hallworth J. Non-specificity of surfactant deficiency in neonatal respiratory disorders. *Br Med J* 1984;288:1635-7.

Correspondence to Professor A D Milner, Department of Child Health, University Hospital, Queen's Medical Centre, Nottingham NG7 2UH.

Received 17 January 1985

Night cough counts and diary card scores in asthma

L N J ARCHER AND H SIMPSON

Department of Child Health, Leicester University Medical School, Leicester Royal Infirmary

SUMMARY A tape recording system for recording night cough in asthmatics at home is described. Objective cough counts and half hour periods containing cough did not correlate with diary card scores awarded to eight children on seven nights each. Night cough diary scores may mislead in the assessment of symptom severity.

The severity of childhood asthma may be difficult to assess at outpatient clinic attendance. Reliance on history taking and examination alone may lead to an underestimate of disability caused by asthma.¹ Symptom diary cards² give subjective information on severity, while peak flow readings in the home are of value only in children old enough to cooperate properly in the use of peak flow meters.³ Cough counting with tape recorders has been performed for 20 years,⁴ but is rarely reported in the study of night cough in asthmatic children.⁵ We set out to compare recorded night cough counts with diary card scores of symptom severity in children with asthma.

Patients and methods

Eight asthmatic children aged 3 years and 6 months to 9 years and 8 months were each studied for seven nights, in seven cases consecutively. The children were on regular prophylactic treatment and their parents were already familiar with the use of symptom diary cards. Studies were carried out between acute exacerbations of the disease. Scores were awarded according to severity of night cough,

thus: no cough 0, little cough 1, moderately bad cough 2, and severe cough 3. Scores were also awarded for severity of night-time wheeze, daytime wheeze, and daytime activity. A microphone placed near the child's head recorded cough during sleep with the recording level set to detect even the quietest cough. Sound recorded on channel I of the tape was also recorded on channel II one second later. The machine was switched on at the parents' bedtime and recorded continuously for six hours. Tapes were renewed daily without altering recording levels or the position of the microphone. They were later replayed through a compression unit triggered by the noise of a preset intensity on channel I so that the same noise was recorded from channel II onto tape 2. The compression unit was triggered by the quietest cough. The compressed tape (tape 2) was automatically switched off when signals from tape 1 ceased. The compression unit incorporated a time signal onto tape 2 every 30 minutes. The number of night coughs occurring during the original recording could then be counted by listening to the condensed tape with a time signal marking each half hour period.

Results

The Table summarises the results obtained from an analysis of 56 nights of recording in eight children. No child ever received a diary score of 3 for night cough and three children (cases 1, 2, and 4) always had a score of 0. Thus, the full range of diary card scores was not available for each child to test whether night cough counts and diary card scores