Correspondence

Pressure volume characteristics of the lungs in sudden infant death syndrome

Sir,

We share the disappointment of Fagan and Milner\(^1\) in their inability to find any difference in the pressure/volume relations of lungs from SIDS victims and controls, particularly as we have recently published\(^2\) a hypothesis on Sudden Infant Lung Collapse (SILC) syndrome in which surfactant malfunction is an important component! We offer the following comments, which may have some bearing on the problem.

Fagan\(^3\) pointed out in 1976 that the recoil of the lung is dominated by tissue forces at high lung volume and by surface tension at low volume. Therefore, if some examples of SIDS are related to abnormal surface tension rather than abnormal tissue forces maximal inflation volume need not concern us further.

Moreover, the lowest volume for which figures are given would approximate in vivo to functional residual capacity, a volume that is too high for initiation of collapse phenomena. The low pressure at which this volume remained, however, may be explained by previous studies showing that the rate of lung collapse is very temperature sensitive near body temperature below which the film is much more rigid and collapse rate considerably reduced. Fagan\(^4\) originally reported that their measurements were made at room temperature, and consequently any defect in performance of surfactant in SIDS cases (should it exist) could have been hidden by their methodology. Should Fagan and Milner extend their technique further, observations of opening behaviour at 37\(^\circ\)C may be more revealing.

In conclusion, the analysis presented by Fagan and Milner shows that there is probably no difference in tissue mechanics between SIDS and controls (high volume results), but it is not possible to judge the situation regarding a possible defect in lung surfactant, particularly if, as described in the original methodology,\(^4\) 'any lung that failed to open completely... was excluded from the series'. Further postmortem investigations at body temperature and at lower lung volumes must be performed to resolve the important questions raised as a result of this present study.

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Dr Fagan and Professor Milner comment:

We are grateful for the interest of Drs Soutllall and Talbert in our paper.\(^1\) We have re-examined our data in the light of their letter.

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<table>
<thead>
<tr>
<th>SIDS babies</th>
<th>No</th>
<th>Mean (SD)</th>
<th>Control babies</th>
<th>No</th>
<th>Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air volume at distending pressure of 1 cm(\text{H}_2\text{O}) as % of volume at a pressure of 30 cm(\text{H}_2\text{O})</td>
<td>20/23</td>
<td>35.2 (7.9)</td>
<td>8/17</td>
<td>36.6 (5.7)</td>
<td></td>
</tr>
<tr>
<td>Air volume at zero distending pressure as % of volume at a pressure of 30 cm(\text{H}_2\text{O})</td>
<td>17/23</td>
<td>22.2 (7.9)</td>
<td>13/17</td>
<td>22.0 (6.2)</td>
<td></td>
</tr>
</tbody>
</table>

Thus there is clearly no significant difference between the two groups and one third of the total lung volume remains with a distending pressure of only 1 cm\(\text{H}_2\text{O}\). We consider these data provide no evidence to suggest alveolar collapse is occurring at low lung volumes.

(2) Time and temperature effects: Time and temperature controls were carried out,\(^5\) and it was found that the alteration between successive inflations carried out at +37\(^\circ\)C or room temperature, or room temperature and +37\(^\circ\)C, were the same as between two successive inflations at either temperature. That is, the major source of variation between inflation cycles was whether it was the 1st, 2nd, or 3rd cycle rather than whether at room temperature or +37\(^\circ\)C. In this study the 2nd cycle at room temperature was taken as the index cycle.

For this letter, the differences between 1st and 2nd cycle were re-examined at a distending pressure of 5 cm\(\text{H}_2\text{O}\). Cot deaths: No=23 Variation between 1st and 2nd cycle=2.3\% (SD 1.1\%)
Normals: No=17 Variation between 1st and 2nd cycle=2.3\% (SD 3.6\%).

Gruenwald\(^6\) suggested that repeated cycling of the lung could be a method of assessing the amount/efficacy of the surfactant reservoir, and in the course of this investigation several lungs were repeatedly cycled on two or three occasions after several days storage at +4\(^\circ\)C between cycles. The effects shown,\(^7\) and current re-examination, were identical between SIDS and others. These data show that the results were not biased by temperature factors.

(3) Lung opening pressures. In our hands this was not a viable experimental procedure using postmortem material as it was found that attempts to degas a lung of infants beyond the neonatal period led to lung rupture. All the SIDS lungs contained air at the outset of the 1st volume-pressure loop and so, as expected, all opened at far lower pressures than degassed neonatal lungs.

Finally, we are sceptical that the information published by Soutllall et al\(^8\) supports the concept that alveolar collapse is occurring in babies with recurrent cyanotic attacks.

References

1. Fagan DG, Milner AD. Pressure volume characteristics of the
Pressure volume characteristics of the lungs in sudden infant death syndrome.
D P Southall and D G Talbert

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