Upper airways obstruction and apnoea in preterm babies

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SUMMARY Measurements of tidal flow, volume, and oesophageal pressure were recorded on a group of 8 severely preterm infants with periodic respiration. Analysis of the flow trace for evidence of cardiac artefact, and the tidal volume trace to identify at what point of the tidal cycle apnoea starts, indicated that upper airways obstruction was associated with about half these attacks. It is surmised that this is produced by glottic closure.

Much has already been written on the aetiology and management of apnoea of prematurity. Whatever the initiating factor responsible for the attack, it is generally assumed that there is a cessation of active respiratory effort so that the respiratory system adopts a volume, the functional residual capacity (FRC), which is determined by the compliance characteristics of the lung chest wall and respiratory muscles. There is no direct evidence that this is so, since such studies as have been carried out on the pattern of respiration in preterm babies with apnoea have depended on devices, such as the impedance technique or paired magnetometers. Unfortunately these systems provide useful information on the rate and some information on the relative tidal volume, both produce signals which are out of phase with tidal breaths and are prone to a variety of artefacts.

Preliminary analysis of records of tidal flow, volume, and oesophageal pressure obtained on babies with periodic respiration and apnoea indicated that it was by no means true that the lung volume is always at FRC during apnoeic attacks. We therefore set out to analyse data on a group of very preterm babies who had recurrent apnoea, in an attempt to identify at what point in the respiratory cycle apnoea began, and how the lung could remain above the resting FRC during a period of apnoea.

Method

The babies were studied in the right lateral position in a cylindrical total body plethysmograph with a servo-controlled radiant heater. A soft, latex-coated flanged face mask mounted on a shutter system was advanced in order to obtain an airtight seal around the baby's nose and mouth. A bias flow of air (0.05 l/s) was fed to the face mask by tube to prevent rebreathing. Effarent flow, modified by the baby's respiratory efforts, was recorded by the pressure gradient across a gauze pneumotachograph mounted at the point where the face mask was attached to the shutter. Expired gases were conducted across the shutter and voided to the atmosphere by a further tube. The baby's tidal volume was measured by integration of the flow signal. Intrathoracic pressure changes were monitored by a soft latex balloon (3 by 0.8 cm) mounted on the tip of a 6 FG nasogastric tube, passed orally and positioned so that the balloon lay in the lower third of the oesophagus. The pneumotachograph was calibrated against a rotameter and the integrated volume calibrated against a syringe at the end of each study. The oesophageal pressure transducer was calibrated against a water manometer. All data were collected on magnetic tape. Further details of the system are given elsewhere. The data were subsequently played on to a UV recorder and examined for the following information: (1) the number and length of apnoeic attacks, (2) the point of tidal volume at which the apnoea began, (3) the effect of any intrathoracic pressure change on lung volume during apnoea, (4) the presence or absence of
cardiac artefact on the flow trace during apnoea. As the cardiac impulse routinely produces an artefact superimposed on the flow trace, the absence of this artefact would indicate that the respiratory tract was obstructed.

Subjects

The records of 8 babies studied on 13 occasions were examined. All were born at a gestational age < 30 weeks (mean 28.6, range 26–30) and had a mean birthweight of 1.02 kg (range 0.74–1.24). The age at the time of study ranged from 3 to 65 days (mean 30.3) and the mean weight at the time of study was 1.40 kg (range 0.72–2.34). All had had recurrent apnoeic attacks persisting for at least 30 seconds requiring stimulation, and 5 had required intubation to terminate an attack on at least one occasion. Seven had received oral theophylline and 3 had at least one period of continuous positive airways pressure. Two babies had required supportive ventilation, one for 2 days and the other for 7. At the time of study, 3 babies were on theophylline. Readings were continued for between 2 and 16 minutes (mean 7.6).

No sedation was given before any of these studies. The project was passed by the local ethical committee before starting the studies.

Results

Data were obtained on 79 apnoeic episodes lasting for at least 5 seconds. During 10 of the studies at least 3 apnoeic episodes were observed, and on two occasions more than 15. 42 persisted for 5 to 10 seconds, 28 for between 10 and 30, and 9 lasted for longer than 30 seconds—one persisting for 57 seconds.

The lung volume stabilised at the end-tidal point on 59 of the 79 occasions (Fig. 1), during the inspiratory phase in 5 (Fig. 2), at full inspiration in 4, and during the expiratory phase in 4 (Fig. 3). Respiratory efforts were seen during 26 apnoeic episodes. On only three occasions was there any associated tidal volume change (Figs 1 and 4). Evidence of cardiac artefact on the flow trace was present on 39 (Fig. 1) and absent during 39 of the apnoeic attacks (Fig. 4), even though the artefact was always present during tidal breathing. On one occasion the cardiac artefact was visible at the beginning of the apnoeic attack and then disappeared (Fig. 5). The relationship between the presence and absence of cardiac artefact, the point of the tidal volume at which apnoea begins, and the response to respiratory effort during apnoea are shown in Fig. 6.
The findings indicate that when apnoea began at a point other than end-tidal, the cardiac artefact was always absent from the flow trace. Respiratory efforts in the presence of the cardiac artefact always produced a volume change when the cardiac artefact was present but never when the artefact was absent.

**Discussion**

These data indicate that although during apnoea the respiratory system may fall to the FRC this does not always happen, and in this series occurred only in 59 of 79 apnoeic attacks. The mechanism for the failure to reach FRC appears to be that there is sometimes an obstruction in the upper airways throughout the period of apnoea. The cardiac impulse always produces an artefact on the flow trace during tidal breathing. In approximately half the apnoeic attacks this artefact disappeared, providing further strong evidence that upper airways obstruction is taking place. The absence of tidal exchange in the presence of vigorous respiratory efforts provides further strong support for this conclusion. The cause of the obstruction remains conjectural, but we would suggest that glottic closure is by far the most likely reason. It is a common clinical observation that babies requiring intubation for apnoea often have adducted vocal cords. Whether upper airways obstruction is important in initiating apnoeic attacks can only be surmised. Previously reported data showed that the same babies responded quite unlike their term counterparts to an externally imposed airways obstruction. Shutter closure during the measurement of thoracic gas volume resulted in a cessation of all respiratory effort, unlike the

![Graph](image1)

**Fig. 3** Trace of apnoeic attack starting during expiration. Lung volume does not fall to FRC until respiration restarts when the initial flow and volume changes are expiratory. Cardiac artefact is absent from the flow trace during apnoea. Tidal flow, volume, and oesophageal pressure traces show the pattern associated with grunting respiration.⁴

![Graph](image2)

**Fig. 4** Trace of prolonged apnoeic attack starting at FRC. Cardiac artefact is absent from the flow trace apart from a very small tidal exchange occurring 5 seconds after the onset of apnoea. No lung volume changes are seen, even in the presence of large intrathoracic pressure swings, until the attack terminated. This presumably results from airways obstruction.
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healthy, term babies in whom respiratory activity continued. The data indicate, however, that even in this group of very preterm babies, about half the attacks were not associated with air-flow obstruction, and so this mechanism cannot be regarded as the sole factor initiating the attacks. Indeed on one occasion airways obstruction started after the onset of apnoea (Fig. 5). Nevertheless it is interesting that babies with upper respiratory tract infection who adopt a periodic respiratory pattern similar to that seen in preterm babies do appear to be prone to sudden infant death. It is undoubtedly true that even a mild upper respiratory tract infection can produce considerable upper airways obstruction.

Finally it is tempting to draw a parallel between apnoea in neonates with the sleep apnoea syndromes in adults and older children, especially as the latter have also been subdivided into obstructive and central apnoea. Unfortunately we were unable to record the periods of REM sleep and relate this to the baby's apnoeic attacks.

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References


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