Pneumothorax in the newborn

Changing pattern

V. Y. H. YU, S. W. LIEW, and N. R. C. ROBERTON*

From the Department of Paediatrics, University of Oxford, John Radcliffe Hospital

Yu, V. Y. H., Liew, S. W., and Robertson, N. R. C. (1975). *Archives of Disease in Childhood*, 50, 449. **Pneumothorax in the newborn: changing pattern.** The clinical course of pneumothorax and its allied conditions was studied in 34 newborn infants who presented over a 2½-year period. We found an overall incidence of 3/1000 live births. 11 term infants without obvious pulmonary pathology presented early (9 within minutes of birth); 6 of these had aspirated meconium or blood. The remaining 23 were preterm infants with hyaline membrane disease (HMD) and accounted for 68% of the infants in this series. In contrast, they presented late (mean 45 hours) and 16 were on continuous distending pressure (CDP) or intermittent positive pressure ventilation (IPPV) at the onset of pneumothorax. 15% of all infants with HMD who required CDP/IPPV developed pneumothorax; this increased incidence was most evident in infants who received CDP only.

All except 2 of the 11 term infants in the first group were managed conservatively and all survived. When pneumothorax occurred as a complication of HMD in preterm infants, 14 of the 16 infants required intrapleural drainage. Persistence or recurrence of pneumothorax occurred in 9 infants, 7 of whom were receiving CDP/IPPV at the time. Lung expansion was affected only after replacement with a patent chest drain through the same incision or insertion of a second drain on the same side of the chest.

All 5 deaths occurred in the group of preterm infants with HMD. 3 resulted directly from respiratory failure due to severe HMD complicated by pneumothorax. We emphasize the increasing importance of pneumothorax as a complication of HMD in preterm infants, particularly in those receiving CDP. Successful management depends on prompt diagnosis and treatment of pneumothorax, which may occur as unexplained sudden deterioration at any time during the course of illness in this group of high risk infants.

Pneumothorax may be associated with severe respiratory distress, and particularly in the presence of hyaline membrane disease (HMD) it is a frequent cause of sudden deterioration or collapse. Nevertheless, it is one of the few pulmonary diseases in the newborn period in which prompt treatment can be life saving. Detection in newborn infants depends as much on a high degree of awareness of its possibility as on the knowledge of its predisposing factors and clinical features. We present our experience of this condition over the last 2½ years and emphasize the increasing importance of its

occurrence in low birthweight infants with HMD during intermittent positive pressure ventilation (IPPV) and continuous distending pressure (CDP). Pneumomediastinum and pneumopericardium are included in the analysis as they are probably a variant of the same underlying pathology.

Clinical observations

The clinical course of pneumothorax and pneumomediastinum was studied in 34 newborn infants. These patients represent all recognized cases of pneumothoraces and allied conditions occurring in the special care nursery during a 2½ year period, January 1972 to July 1974, with the exception of 2 for which case notes were incomplete. 3 infants were referred from other hospitals on the first day of life.

Received 11 November 1974.

*Present address: Department of Paediatrics, Addenbrooke's Hospital, Cambridge.

The overall incidence of pneumothoraces and allied conditions was 3/1000 live births. This study includes 26 infants with pneumothoraces all of whom were symptomatic and in whom deterioration in respiratory symptoms had led to chest x-ray. 6 out of the 26 had coexisting pneumomediastinum (23%) and one had coexisting pneumopericardium. 6 other infants had isolated pneumomediastinum and 2 had severe pulmonary interstitial emphysema with lung cysts; these 8 were asymptomatic and were diagnosed on routine x-rays. Males were more than twice as common as females (24:10). Pneumothoraces were found nearly twice as frequently on the right as on the left (R:L = 14:8) and 4 patients (15%) had bilateral pneumothoraces.

The infants studied can be separated into two distinct groups depending on whether there was associated idiopathic respiratory distress syndrome—presumably HMD (Table I). The first group, that of infants

of apnoea is responsible for the pneumothoraces. 3 infants in this group had coexisting pneumomediastinum.

In the second group, pneumothorax and pneumomediastinum occurred as a complication of HMD in 23 infants, and these accounted for 68% of the infants in this series. The types of air leak developing in the 23 cases are shown in Table II. Their mean gestational

TABLE II

Types of pneumothoraces and allied conditions

	Infants without HMD (11)	Infants with HMD (23)
Pneumothorax alone	7	12
Pneumothorax with pneumomediastinum	3	3
Pneumothorax with pneumopericardium	0	1
Pneumomediastinum alone	1	5
Severe pulmonary interstitial emphysema with lung cysts	0	2

TABLE I

Clinical data of infants with pneumothoraces and allied conditions

	Infants without HMD	Infants with HMD
No.	11	23
Gestation (w)	40 (38–41)	34 (27–38)
Birthweight (g)	3453 (2710–4130)	2307 (990–3510)
Age of onset (h)	Birth (2 at 14 h)	45 (12–140)
Apgar score at birth 4 or less	4	7
Endotracheal intubation and IPPV at birth	4	8
Aspiration of meconium or blood	6	0
CDP or IPPV at onset of pneumothorax	—	16

HMD, hyaline membrane disease; CDP, continuous distending pressure; IPPV, intermittent positive pressure ventilation.

without obvious pulmonary disease, used to be the most common: they were usually diagnosed in the first hour of life, being mainly term or post-term infants presenting with respiratory difficulty at delivery. There was often a history of fetal distress, difficult delivery, or overzealous resuscitation, and evidence of aspiration of meconium, blood, or mucus. This group of 11 infants accounted for only 32% of pneumothoraces in Oxford. Their mean gestational age was 40 weeks (range 38 to 41 w) and mean birthweight 3453 g (range 2710–4130 g). In 9 infants the signs were evident at delivery, while in the remaining 2 pneumothorax was not obvious until 14 and 15 hours of age, respectively. There was a strong possibility of aspiration of meconium or blood as a predisposing event in 6 out of the 11 infants and the possible aspiration of mucus could not be ruled out in any infant. 4 of the pneumothoraces occurred in resuscitated infants, but it is not possible to ascertain to what extent overzealous resuscitative efforts or the underlying cause

age was 34 weeks (range 27–38 w) and mean birthweight was 2307 g (range 990–3510 g). All suffered from respiratory distress syndrome presumably due to HMD. Initial x-rays were compatible with HMD and did not show pneumothoraces. Onset of pneumothorax was later than in the first group and occurred at a mean of 45 hours postnatally (range 12 to 140 h). The diagnosis was strongly suspected in all 16 infants with pneumothorax in this group before x-ray confirmation. 4 infants presented with acute collapse and 12 with unexplained but more gradual clinical deterioration during the course of the respiratory distress syndrome so that repeat chest x-rays were taken to detect possible pneumothorax. The remaining 7 infants, 5 of whom had pneumomediastinum without pneumothorax and 2 with severe pulmonary interstitial emphysema and lung cysts were detected by routine chest x-ray without antecedent clinical suspicion. 12 of the 16 infants with pneumothorax were on either CDP (8 cases, mean duration 30 h, range 2–70 h) or IPPV (4 cases, mean duration 23 h, range 2–120 h) at the time of onset of pneumothorax. All infants received a maximum CDP of 10 cm H₂O except one early case in whom 15 cm H₂O was applied for a period of 9 hours. The 4 infants who developed pneumothorax during IPPV were being ventilated at a pressure of 40 cm H₂O. Only one infant was subjected to IPPV in association with positive end-expiratory pressure and this was discontinued 20 hours before the onset of pneumothorax. The overall incidence of pneumothorax and allied conditions occurring in infants with HMD was 11% (23 out of 216). 15% of infants with HMD on CDP or IPPV developed these complications (16 out of 106). This increased incidence was most evident in infants who had received CDP alone and was lower in infants on IPPV (Table III).

TABLE III

Pneumothoraces and allied conditions in infants with HMD on ventilation therapy

	Total no.	Total no. on CDP and/or IPPV	No. on CDP	No. on IPPV	No. on CDP and IPPV
HMD, all cases	216	106	29	34	43
HMD with pneumothorax and allied conditions	23	16	7	3	6
%	11%	15%	24%	9%	14%

TABLE IV

Treatment and outcome of pneumothorax

	No. of cases	Umbilical vessel catheterization	Intrapleural chest drain	Persistence or recurrence of pneumothorax	No. survived
Infants without HMD	11	4	2	0	11
Infants with HMD (total)	16	16	14	9	11
Infants breathing spontaneously	4	4	3	2	4
Infants on CDP/IPPV	12	12	11	7	7

Treatment

The method of treatment and outcome of therapy are shown in Table IV. In the group of infants without underlying HMD, 9 out of 11 pneumothoraces and pneumomediastina were managed conservatively and in only 4 were umbilical catheters inserted for monitoring blood gases. In an attempt to speed up the absorption of the pneumothorax, these infants were kept in the highest oxygen concentration considered to be safe in terms of lung and retinal toxicity. Deterioration in blood gas values associated with increasing respiratory difficulty in 2 infants led to insertion of chest drains at 4 and 14 hours, respectively. Radiological lung re-expansion in cases treated conservatively was achieved in a mean period of 48 hours (range 24–60 h). All infants in this group survived.

When pneumothorax complicated HMD, blood gases deteriorated sufficiently for insertion of chest drains in 14 of the 16 infants. Pneumothoraces were drained as quickly as possible after radiological confirmation in these cases through the second intercostal space just lateral to the midclavicular line. Underwater drainage with suction at –5 to –10 cm H₂O was always applied. There was, however, persistence or recurrence of pneumothorax in 9 out of 14 infants during chest drainage and 7 of these occurred in those infants receiving CDP or IPPV. In 4 infants the chest drain was blocked and re-expansion of the lung was achieved only after replacement of the drain through the same incision. In the other 3 the drain was patent but insertion of a second drain (on the same side in the 6th intercostal space at the midaxillary line) was needed to achieve re-expansion. One infant required both these procedures and another died before further treatment could be carried out. Intercostal drains were left *in situ* for a mean of 70 hours (range 24–120 h), usually 24 hours after the drainage

tube was clamped and subsequent confirmation of lung expansion by x-ray. None of the infants was still on CDP or IPPV at the time when the chest drains were removed.

The 5 deaths in this series occurred in this group; all 5 showed the pathological features of HMD, but one had in addition a massive pulmonary haemorrhage and one other renal agenesis with some pulmonary hypoplasia.

The use of high inspired oxygen concentration to accelerate nitrogen washout from interstitial sacs of air was the only possible therapy for pneumomediastinum. Surgical drainage was not attempted in any of our cases. All infants who developed a pneumomediastinum alone had spontaneous resolution and all infants with pneumothorax and coexisting pneumomediastinum also survived.

The 2 infants who developed severe pulmonary interstitial emphysema and lung cysts without pneumothorax were managed conservatively initially. The first infant in whom the lung cysts were detected at 4 days of age after having received 60 hours of CDP from birth had gradual radiological improvement with complete resolution by 3 weeks of age. The second who received in total 10 hours of IPPV at 40 cm H₂O pressure followed by 48 hours of CDP at 10–14 cm H₂O had severe pulmonary interstitial emphysema with one particular cyst in the right upper lobe which gradually increased in size and persisted until thoracotomy and excision was carried out at 12 weeks of age, after which the infant made an uneventful recovery.

Discussion

The incidence of pneumothorax is difficult to ascertain since many patients are asymptomatic (Chernick and Reed, 1970). Radiological surveys

have shown pneumothorax in 1 to 2% of all newborn infants (Davis and Stevens, 1930; Solis-Cohen and Bruck, 1934; Steele *et al.*, 1971) and the incidence of symptomatic cases was reported to be 0.05–0.07% (Harris and Steinberg, 1954; Howie and Weed, 1957; Chernick and Avery, 1963; Malan and de V. Heese, 1966). In some of these earlier reports on 'spontaneous' pneumothorax in the newborn, infants who had received oxygen under positive pressure (Chernick and Avery, 1963; Malan and de V. Heese, 1966) or who had underlying HMD (Malan and de V. Heese, 1966) were excluded. Reports of pneumomediastinum and pulmonary interstitial emphysema, when unaccompanied by pneumothorax were also not included in these case summaries (Harris and Steinberg, 1954; Howie and Weed, 1957), though they are probably related (Ovenfors, 1964; Caldwell, Powell, and Mullooly, 1970; Thibeault *et al.*, 1973).

In this series, a 0.3% incidence of pneumothorax and its allied conditions is reported. Low birthweight infants with HMD accounted for 68% of our cases. Previous studies have suggested that the term infant is most at risk (Peterson and Pendleton, 1955; Emery, 1956; Chernick and Avery, 1963), and it has even been suggested that in preterm infants HMD is not a common predisposing factor (Kirschner and Strauss, 1964; Grosfeld, Clatworthy, and Frye, 1970). More recently it has been recognized that HMD significantly increases the incidence of pulmonary interstitial emphysema and pneumothorax (Thibeault *et al.*, 1973). With the increasing use of CDP or IPPV in infants with HMD, pneumothorax has become even more frequent (Lancet, 1973; Baum and Robertson, 1974). Signs of air leak develop in as many as 30% of infants on positive pressure ventilation plus positive end-expiratory pressure (Blake *et al.*, 1973). In this series, 15% of infants with HMD requiring CDP and/or IPPV developed this complication and 24% of those treated with CDP alone. The detection of pneumothorax in this group of infants depended on a high degree of awareness that the diagnosis must always be considered when such infants have sudden or unexpected deterioration at any time during CDP or IPPV. Radiological confirmation may be difficult as an anteroposterior x-ray may not be sufficient. A horizontal beam, lateral ('cross-table') view of the chest with the patient in the supine position should always be done (Robertson, 1975). Pneumomediastinum as well as intrapleural air collecting at the top of the thoracic cavity can be seen better in such a view.

Pneumothorax as a result of overzealous positive pressure resuscitation of the apnoeic newborn (Chernick and Reed, 1970; Chernick and Avery, 1963) is a less frequent occurrence, and in 7 of our 11 early pneumothoraces no IPPV had been given. Neonatal resuscitation is safer today than in the past. It is known that the water manometer system in resuscitation apparatus can be unreliable when used with excessively applied oxygen flow (Mathias, 1966) and modification of such systems by the addition of a deadweight relief valve (Hey and Lenney, 1973) had been carried out in our delivery rooms.

Breathing 100% oxygen accelerates the resorption of a pneumothorax (Chernick and Avery, 1963; Northfield, 1971). This form of therapy has generally been successful in our group of infants without HMD who had only moderately severe respiratory difficulty from pneumothorax or who had a pneumomediastinum alone. In contrast, most of the infants in this series with underlying HMD who developed pneumothorax did require insertion of a chest drain. A continued air leak into the pleural space, particularly in conjunction with CDP or IPPV required application of suction to prevent reaccumulation of air. But, in spite of this, over half in this group had persistence or recurrence of pneumothorax: 5 of these 7 were on CDP or IPPV at the time. Such complications often occurred in situations where the chest drain became blocked or was clamped too soon. We found it useful to replace a new drain through the same incision or to insert a second one on the same side of the chest under such circumstances. We have maintained a policy of leaving the chest drain *in situ* during the period in which the infant was still receiving CDP or IPPV.

Overall mortality rate for pneumothorax in this series was 0.04% (5 in 11 169 live births). The prognosis for pneumothorax occurring in infants without HMD was uniformly good and all the deaths occurred in the preterm infants with HMD. Mortality rate in the infants with HMD developing pneumothorax was 31% (5 of 16), compared with a mortality rate of 14% in cases of HMD without pneumothorax (27 of 200). This difference is not statistically significant ($\chi^2 = 3.15$, $P > 0.05$) and clearly pneumothorax is likely to be more common in infants with severe HMD since pulmonary interstitial emphysema is more likely to occur and higher inflation pressures with IPPV are necessary. Thus the deaths in cases complicated by pneumothorax, when promptly and adequately treated, may be more indicative of the severity of the HMD than of the consequence of this complication.

We thank Professor J. P. M. Tizard for advice and help in the preparation of the manuscript.

REFERENCES

- Baum, J. D., and Robertson, N. R. C. (1974). Distending pressure in infants with respiratory distress syndrome. *Archives of Disease in Childhood*, **49**, 771.
- Blake, A. N., Collins, L. M., Durbin, G. M., Hunter, N. J., MacNab, A. J., Reynolds, E. O. R., and Sellens, G. (1973). Simplified mechanical ventilation for hyaline-membrane disease. *Lancet*, **2**, 1176.
- Caldwell, E. J., Powell, R. D., and Mullooly, J. P. (1970). Interstitial emphysema: a study of physiological factors involved in experimental induction of the lesion. *American Reviews of Respiratory Disease*, **102**, 516.
- Chernick, V., and Avery, M. E. (1963). Spontaneous alveolar rupture at birth. *Pediatrics*, **32**, 816.
- Chernick, V., and Reed, M. H. (1970). Pneumothorax and chylothorax in the neonatal period. *Journal of Pediatrics*, **76**, 624.
- Davis, C. H., and Stevens, G. W. (1930). Value of routine radiographic examinations of the newborn. *American Journal of Obstetrics and Gynaecology*, **20**, 73.
- Emery, J. L. (1956). Interstitial emphysema, pneumothorax, and air-block in the newborn. *Lancet*, **1**, 405.
- Grosfeld, J. L., Clatworthy, H. W., Jr., and Frye, T. R. (1970). Surgical therapy in neonatal air-block syndrome. *Journal of Thoracic and Cardiovascular Surgery*, **60**, 392.
- Harris, L. E., and Steinberg, A. G. (1954). Abnormalities observed during the first six days of life in 8716 live-born infants. *Pediatrics*, **14**, 314.
- Hey, E., and Lenney, W. (1973). Safe resuscitation at birth. *Lancet*, **2**, 103.
- Howie, V. M., and Weed, A. S. (1957). Spontaneous pneumothorax in the first 10 days of life. *Journal of Pediatrics*, **50**, 6.
- Kirschner, P. A., and Strauss, L. (1964). Pulmonary interstitial emphysema in the newborn infant, precursors and sequelae. A clinical and pathologic study. *Diseases of the Chest*, **46**, 417.
- Lancet (1973). Increasing the transpulmonary pressure in respiratory-distress syndrome, **2**, 244.
- Malan, A. F., and de V. Heese, H. (1966). Spontaneous pneumothorax in the newborn. *Acta Paediatrica Scandinavica*, **55**, 224.
- Mathias, J. (1966). Resuscitation of the newborn. *Lancet*, **1**, 262.
- Northfield, T. C. (1971). Oxygen therapy for spontaneous pneumothorax. *British Medical Journal*, **4**, 86.
- Ovenfors, C. O. (1964). Pulmonary interstitial emphysema: an experimental roentgen-diagnostic study. *Acta Radiologica*, **244**, 1.
- Peterson, H. G., and Pendleton, M. E. (1955). Contrasting roentgenographic pulmonary patterns of the hyaline membrane and fetal aspiration syndrome. *American Journal of Roentgenology*, **74**, 800.
- Robertson, N. R. C. (1975). *Recent Advances in Respiratory Diseases*. Ed. by T. H. Stretton. Churchill, London. (In the press.)
- Solis-Cohen, L., and Bruck, S. (1934). A roentgen examination of the chest of 500 newborn infants for pathology other than enlarged thymus. *Radiology*, **23**, 173.
- Steele, R. W., Metz, J. R., Bass, J. W., and Du Bois, J. J. (1971). Pneumothorax and pneumomediastinum in the newborn. *Radiology*, **98**, 629.
- Thiebaud, D. W., Lachman, R. S., Laul, V. R., and Kwong, M. S. (1973). Pulmonary interstitial emphysema, pneumomediastinum, and pneumothorax. *American Journal of Diseases of Children*, **126**, 611.

Correspondence to Dr. V. Y. H. Yu, Department of Paediatrics, John Radcliffe Hospital, Headington, Oxford OX3 9DU.