CASE REPORTS

SPONTANEOUS PNEUMOTHORAX OF THE NEWLY-BORN

BY

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Twenty-one cases of pneumothorax of the newly-born have been recorded in medical literature. A recent review by Glaser and Landau¹ emphasized the rarity of the condition, the high mortality, the greater incidence in the male sex, the predominance of left-sided involvement and the difficulties that arise in an attempt to explain satisfactorily the origin of this interesting and unusual complication. Willi recognized three groups of cases, classified according to their etiology, as mechanical, infectious, or congenital.

The first group includes such causative agents as forcible artificial respiration, the use of the Drinker respirator, obstruction of the air passages by aspirated amniotic material, enlargement of the thymus and collapse of the epiglottis. The exact significance of obstruction to the ingress of air in the production of a pneumothorax is by no means as obvious as certain writers suggest. Many cases of obstruction accompanied by the most laboured inspiratory efforts have been observed in cases of congenital laryngeal stridor, subglottic stenosis and other malformations of the upper respiratory passages without the escape of air into the pleural cavity. A recently observed case of obstruction due to aspirated amniotic material showed at autopsy a few marginal emphysematous bullae but not a pneumothorax notwithstanding very strenuous inspiratory efforts for several hours before death. It appears as a possibility at least, that histological studies of serial sections may disclose congenital defects in the lung tissue as the primary cause and mechanical obstruction to the entrance of air as a necessary but secondary factor in the development of this type of pneumothorax.

Three cases have been placed by Wilkinson² in the infectious group. He demonstrated conclusively in his own case that the formation of a pulmonary abscess which ruptured into the pleural cavity was caused by multiple septic pulmonary emboli and was not secondary to a low grade inhalation pneumonia starting at birth, a mechanism suggested by other investigators. The emboli were attributable to a septicaemia originating in all likelihood from an omphalitis or a circumcision wound. Unfortunately knowledge of neonatal septicaemias is still imperfect. Their occurrence has been noted in

the absence of any focus of infection and irrespective of the best obstetrical and nursing care.

The third group due to congenital pulmonary defects is best illustrated by Weiner's case. This child had a left pneumothorax, first noted fourteen days after birth. At autopsy a canal was found about the diameter of a pencil and communicating both with the pleural cavity and a bronchus. The canal opened a short distance from the surface of the lung within a fold of tissue which accounted for its valve-like action.

In approximately thirty per cent. of the cases recorded, no acceptable hypothesis could be advanced to explain the pneumothorax.

Clinical record

The following case observed at the Hospital of the Good Samaritan apparently belongs either to this group or to the cases with congenital defects. The mother was delivered by caesarean section and the baby

FIG. 1.—Left pneumothorax, heart and mediastinum are displaced to right.

weighed 8 lb. 1 oz. Pregnancy had been entirely normal and the operation under spinal anaesthesia was uncomplicated. Two siblings are living and well. A miscarriage preceded the last pregnancy. The infant's first inspiration was vigorous and his cry strong. His condition immediately after delivery presented nothing remarkable. A few minutes later respirations appeared slightly laboured and were accompanied by moderate cyanosis. It was thought that some mucus had been aspirated into the upper respiratory passages as a result of the initial inspiratory effort. The mucus present was

more than customary but not enough to cause obstruction. A five per cent. carbon-dioxide-oxygen mixture was administered through a funnel shaped mask in the usual way with a fairly satisfactory response. When the baby was stimulated the cry was lusty and the general condition at that time was not critical. Twenty hours after the birth, cyanosis had become marked and the dyspnoea moderately severe. Complete absence of breath sounds over the left lung was ascribed to atelectasis. Oxygen administration was begun and continued by nasal catheter. A second examination, five hours later, revealed dextrocardia in addition to the atelectasis. Since an uncomplicated true congenital dextrocardia is asymptomatic, the physical signs in this case were thought to be due either to a left diaphragmatic hernia with compression of the lung by intestine and consequent cardiac displacement, or to a left pneumothorax. The latter appeared more probable, as suppression of breath sounds was complete and the tinkling sound of air in the intestines was not elicited over the left thorax. X-ray examination confirmed the diagnosis of left pneumothorax and also showed a small pneumothorax on the right side. Oxygen was discontinued during the x-ray examination and the cyanosis immediately increased. The nasal catheter was re-inserted with obvious and convincing beneficial results. As the result of oxygen administration improvement was so satisfactory that aspiration of air did not seem warranted.

FIG. 2.—Complete disappearance of pneumothorax three days later.

Feeding was started and taken unusually well. The following day respirations were less laboured and at the end of the third day air was entering the left lung. On the fourth day complete expansion of the lung together with a normal position of the heart was verified by x-ray. At present the child, seven months old, is perfectly normal.

Summary

A case of pneumothorax of the newly-born with initial symptoms shortly after birth is presented. The cause could not be attributed to either a mechanical or an infectious factor. The administration of oxygen was the only therapeutic procedure used. A spontaneous recovery ensued.

REFERENCES

1. Glaser, J., & Landau, D. B., *Am. Dis. Child.*, Chicago, 1935, L, 986.
2. Wilkinson, S. J., *ibid.*, 1936, LII, 361.