CASE REPORT

ABNORMAL EXPANSION OF THE LUNGS IN AN INFANT

BY

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Clinical record

A male infant, aged five weeks, was admitted to the Children's Hospital owing to the onset, soon after birth, of attacks of dyspnoea and cyanosis. He was a full term child; birth had been normal. Examination revealed the heart in the right thorax; the lungs were normal except for fine crepitations at both bases. X-ray pictures following a barium feed confirmed the position of the heart in the right side of the thorax, with no transposition of other organs. A diagnosis was made of dextroversion of the heart associated with further serious congenital abnormalities.

From the date of admission the attacks of dyspnoea accompanied by cyanosis gradually became more frequent, and death occurred fifteen days after admission, following continuous attacks of dyspnoea and cyanosis. Fourteen days after admission a systolic bruit was noted for the first time in the third right intercostal space, two inches from the mid-sternal line. During the last week there was a mild degree of pyrexia; the pulse rate throughout varied from 160 to 180, the respiratory rate from 40 to 76 per minute. The cyanotic attacks were to some extent relieved by inhalation of oxygen.

Autopsy

On opening the thorax the upper lobe of the left lung, much enlarged, was found lying transversely, posterior to the upper part of the sternum; a well-marked groove in this portion of the lung had been produced by the pressure of the sternum. The anterior portion of the lobe was dull white in colour and obviously emphysematous, whilst the posterior portion was dark and of normal texture. It was now clear that the displacement of the heart was the result of the abnormally situated left upper lobe (see fig. 1).

The specimen was shown to Dr. John Beattie, Conservator of the Museum of the Royal College of Surgeons of England, who kindly supplied the following report:—

REPORT ON SPECIMEN OF THORACIC VISCERA OF CHILD H.

The specimen consists of the lungs, heart and great vessels of a child shortly after birth. The upper lobe of the left lung has expanded abnormally across the middle line and invaginated the right pleural sac, displacing the

thymus gland backwards and to the right. A well-marked groove on the anterior aspect of this lobe probably contained the sternum during life, and suggests that considerable pressure had been exerted on the great vessels. The upper lobe of the right lung had not expanded, and only partial expansion of the lower lobe on this side had taken place. The lower lobe of the left side was normal. Examination of the specimen revealed no embryological defect, but it was observed that the upper branch of the left pulmonary artery had been acutely kinked around the ductus arteriosus.

FIG. 1.—Line drawing showing transverse position of upper lobe of the left lung and displaced heart.

The abnormality seems to be best explained by a deficient expansion of the right lung, followed by a compensatory emphysema of the left lung. The kinking of the pulmonary artery and the trapping of the left upper lobe behind the sternum, may have contributed to some extent to the persistence of the emphysematous condition. The cause of death can only be guessed at, but it seems probable to me that interference with the great vessels due to pressure may have contributed if it was not the only cause of death. A histological section of the emphysematous lung demonstrates quite clearly that there was no inflammatory reaction within the lung itself.

Comment

From the clinical aspect the interest of the case centres on the diagnosis from congenital dextrocardia, of that type in which the heart is simply rotated and lies in the right side of the chest; is unassociated with the transposition of the cardiac chambers; and is almost invariably associated with some serious congenital anomaly. Such a diagnosis gave a satisfying explanation for the paroxysms of cyanosis and dyspnoea. The autopsy, however, revealed what so far as can be ascertained from a search of the literature is a hitherto unrecorded cause of extrinsic dextrocardia, and as such the case is published.

Etiologically the condition might be explained by atelectasis of the upper lobe of the right lung, followed by compensatory emphysema of the upper lobe of the left lung, and the occupation by the latter of space normally occupied by the former. Alternatively, it is recognized that expansion of the lungs in the newborn is a gradual process, taking place unequally throughout the lungs and requiring two to three days or longer for full inflation, and it seems feasible that expansion of the upper lobe of the right lung might be delayed to such an extent as to lead to compensatory emphysema of the corresponding lobe on the opposite side. The systolic bruit which developed during the course of the illness was no doubt the result of distortion of the great vessels, as described in Dr. Beattie's report.