RUPTURE OF THE FOETAL HEART DURING LABOUR

BY

R. A. McINROY and ANNE L. M. GRAHAM

From the Department of Pathology, St. Luke's Hospital, and St. Luke's Maternity Hospital, Bradford

(RECEIVED FOR PUBLICATION FEBRUARY 9, 1953)

A case is reported in which haemopericardium, resulting from rupture of the foetal heart during labour, was the cause of death in a stillborn infant.

Case Report

Obstetrical History. The mother, aged 28 years, gave a history of two miscarriages about the eighth and tenth weeks of pregnancy in 1947 and 1951. During the present pregnancy she had been attending the ante-natal clinic and her general condition was considered satisfactory but, on June 12, 1952, about the thirty-ninth week of pregnancy, she was admitted to hospital on account of pitting oedema of the ankles, legs and abdominal wall. On admission, her blood pressure was 134/90 mm. Hg and the urine was free from protein.

History of Labour. The presentation was a vertex and clinically there was no cephalo-pelvic disproportion. Pelvimetry also showed the pelvis to be adequate. On June 12, 1952, a quinine induction (four doses of quinine hydrochloride, gr. 5, at four hourly intervals) was given, followed by 'pitocin' (six doses of 2 i.u. at half-hourly intervals) to induce labour. (The expected date of confinement was June 18, 1952.) On June 13, the membranes were ruptured surgically as labour had not begun. After this, labour proceeded normally; a bilateral sympathetic block was performed with good relief during the first stage. The first stage lasted 11 hours and 40 minutes. A local pudendal block was performed at the start of the second stage. The head advanced slowly and, because of delay in delivery of the head, an episiotomy was performed. Following delivery of the head, difficulty in delivering the shoulders was experienced: this was easily overcome by placing fingers in the axillae and exerting traction. No difficulty was encountered thereafter. The second stage lasted for one hour and 50 minutes.

A stillborn male infant weighing 8 lb. 6 oz. (3.8 kg.) was born. The foetal heart sounds had been regular during labour, the last recording being made 10 minutes before delivery. Although the baby was stillborn it was considered advisable, at the time, to attempt to establish respiration; this was done by intubation and oxygen insufflation. An intracardiac injection was not given.

Necropsy. The infant was well nourished and there were no external physical malformations. Oedema, jaundice and cyanosis were absent. A prominent caput was present over the vertex.

On opening the thorax the pericardial sac was seen to

be distended with blood. The sac was opened: fresh blood escaped and a blood clot, approximately 15 mm. in diameter, lay along the left border of the left ventricle. Carefully turning the apex of the heart upwards, the diaphragmatic surface and base of the heart were examined. A small hole, through which a clot of blood protruded, was observed in the lower and posterior part of the right atrium just anterior to the point of insertion of the inferior vena cava. This tear, approximately 6 mm. long $\times$ 1.2 mm. broad, involved mainly the membranous portion of the atrial wall but extended into the muscular part, and extravasation of blood within the wall could be seen over an area adjacent to the site of rupture (Fig. 1). The other cardiac chambers, valve orifices and cusps, and the myocardium presented no abnormal features.

The lungs showed evidence of very slight aeration. In the abdomen, apart from moderate congestion of the liver, no abnormality was seen. The brain was normal.

Discussion

Haemopericardium, resulting from rupture of the foetal heart, is extremely rare. Two cases in stillborn infants, in addition to the present one, have been reported, one by Longridge (1907) and one by Hunt (1952). One case of haemopericardium in a stillborn infant, not associated with rupture of the heart, was reported by Silbernagel and Fidler (1943) and another case of haemopericardium, again in a stillborn infant, in which the precise site of bleeding was not determined, was described by Potter (1952).

The case reported by Longridge (1907) was one in which a shoulder presentation at full term was admitted to hospital with a prolapsed and pulseless cord. After craniotomy and powerful traction a child, weighing 9 lb. 5 oz. (4.22 kg.), was born. Necropsy revealed a haemopericardium with rupture of the heart at the junction of the inferior vena cava and right atrium. In the case described by Hunt (1952) the foetus, weighing 5 lb. 11 oz. (2.58 kg.), was delivered as an assisted breech; the foetal heart sounds had been heard earlier in labour. In this case, a perforation, measuring less than half a millimetre in diameter, was found situated in the anterior wall of the right atrium close to the root of the atrial

FIG. 1.—Necropsy specimen showing the diaphragmatic surface and base of the heart with the rupture and adjacent area of bruising in the right atrial wall.

appendix. The perforation was attributed to a focal hyaline degeneration of the myocardium, of unknown aetiology, at the point of rupture. The cases described by Silbernagel and Fidler (1943) and Potter (1952) were similar to each other in that in each case the mother gave birth to a stillborn infant following an accident. In the former case the mother had been involved in a car accident in which she was thrown from the car and struck her abdomen on the kerb of a pavement, and in the latter the mother had fallen down a flight of stairs two days before delivery.

The tear in the atrial wall in our case was not an artefact produced at the time of necropsy. Histological examination of the atrial wall near the site of rupture shows extravasation of blood between muscle fibres, an ante-mortem change (Fig. 2). Moreover, comparison by histological examination of the atrial wall at the site of rupture with the corresponding part in normal foetal hearts has shown no evidence of any difference in thickness of the wall to suggest a congenital anomaly or any degenerative change in the muscle fibres. Assuming the tear to have been traumatic in origin, consideration has been given to the mechanics whereby this

might possibly have been produced. When it is recalled that in the foetus the liver at birth occupies the greater portion of the upper abdominal cavity and is relatively unprotected by the thoracic cage, it will be appreciated that considerable strain could be exerted on the inferior and superior venae cavae and the right atrial wall by pressure of the contracting uterus, either holding the liver fixed in position in the abdominal cavity or forcing it downwards towards the pelvis, and the tension, operating simultaneously and in the opposite direction, exerted on the mediastinum by traction on the head or shoulders. Attempts to reproduce this state of affairs have been made on the cadaver, and although rupture of the atrium has not been produced, considerable tension on the atrial wall can undoubtedly be exerted. During life an additional factor might well be a congested and tensely dilated right atrium with congested venae cavae such as might be the case during a labour more difficult than average. Under these conditions it is conceivable that the wall of the right atrium might tear about the site where the rupture occurred in the present case and in the case reported by Longridge (1907).

The possibility of an uterine contraction ring

FIG. 2.—Photomicrograph showing extravasation of red blood cells between muscle fibres in the atrial wall. Haematoxylin and eosin $\times 300$.

being responsible for the difficulty encountered in delivering the shoulders and exerting some unusually severe pressure on the viscera of the foetus is not considered likely, as, had it been so, one might have expected that further attempts to deliver the child without anaesthesia would have tended to aggravate the condition rather than prove beneficial.

In the case presented here, the delivery was in the hands of the nursing staff until one of us (A.G.) took over after difficulty with delivery of the shoulders had been encountered. There had been no undue torsion of the body. It is possible, although only conjectured, that the degree of traction exerted on the head, when difficulty in delivering the broad shoulders of the overweight infant was experienced, might have been considerably more powerful and protracted than would be exerted during the course of a normal labour. It is, however, difficult to envisage the precise aetiological factor responsible for this distinctly rare occurrence when the maternal pelvis, the foetus and course of labour were in no way widely divergent from normal.

This appears to be the third reported case of

haemopericardium associated with rupture of the heart in a stillborn infant.

Summary

A case is described in which haemopericardium, resulting from rupture of the right atrium during labour, was found on necropsy to be the cause of death in a stillborn infant.

The rupture is considered probably to have been traumatic in origin, and factors concerned in the possible mechanism leading to this rare occurrence are briefly considered.

We wish to thank Mr. G. A. Craig for his helpful advice and criticism during the preparation of this paper. We are also indebted to Mr. P. Harrison for the photographs, and to Messrs. E. F. Hill and J. D. Musgrave for the preparation of the histological material.

REFERENCES

- Hunt, W. E. (1952). *Archives of Disease in Childhood*, 27, 291.
Longridge, C. N. (1907). *Trans. obstet. Soc. Lond.*, 49, 214.
Potter, Edith L. (1952). *Pathology of the Fetus and the Newborn*, p. 96. Chicago: and personal communication.
Silbernagel, W. M. and Fidler, R. S. (1943). *Amer. Heart J.*, 26, 129.