CONGENITAL MALFORMATION OF THE HEART IN ONE OF IDENTICAL TWINS

BY

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On June 12, 1944, Mrs. F. was delivered in her home of female twins. I had supervised her antenatally in this her second pregnancy, but the confinement was attended by the midwife only. There was a history of uniovular twins in both the patient's family and that of her husband. The pregnancy was normal except that at the third month she had a mild vaginal haemorrhage which was treated as a threatened abortion. Twins were suspected from this time owing to the size of the uterus, and confirmed later by x-ray examination. She became very distended in late pregnancy. The first stage of labour was long. The first child was delivered as an occipito posterior, weighing 5½ lb. The second child followed in five minutes and weighed 4½ lb. The single placenta followed: there was a small placenta succenturiata. The two cords were equal in size and attached to the main placenta.

The infants both appeared normal and breathed spontaneously, remaining a good colour. They were breast-fed six-hourly the first day for five minutes, four-hourly the second day, and three-hourly the third. Sterile water was given between feeds if required. Both sucked well at first, but at the end of the second day the smaller twin was noticed to be pale and she refused her feeds. The third morning she had two attacks of vomiting, with cyanosis of her lips only.

I saw her the third morning. Her breathing was shallow, with periods of apnoea. Her hands and feet were never cyanosed, her lips and face only at intervals. The chest moved poorly and breath sounds were almost entirely absent. The heart did not appear to be enlarged. There was a harsh systolic murmur over the base of the heart and tricuspid area. It was loudest in the second left interspace. The apical sounds were clear. The diagnosis of atelectasis with patent ductus arteriosus was made. The infant was given oxygen and two-hourly breast feeds from a pipette. She did not want her feeds and vomited some of them. The cyanotic attacks continued but were somewhat relieved by oxygen. She died early on the fourth day during an attack.

The first and larger twin seemed entirely normal in every respect. Recent x-ray examination including screening at four months of age shewed no radiographic abnormality.

Autopsy on the smaller twin showed almost complete atelectasis of both lungs. The heart was not enlarged, the pericardial sac and fluid were normal. The chambers and septa of the heart were normal. The ductus arteriosus was widely patent, being of the same calibre as the pulmonary artery. The aorta showed a marked constriction at the point of entry of the ductus arteriosus. Below this point it was of normal calibre. There were no other abnormalities.

Discussion

The rare occurrence of a congenital malformation in one of identical twins may throw light on pathological or physiological processes. I can find no other incidence of congenital defect in one of monovular twins, although occasionally abnormalities have been reported in both twins.

With regard to cardiac defects, Shirley Smith (1929) reported the occurrence of patent ductus arteriosus in both of monovular twins. Debreuil Chambarde (1927) reported transposition of the viscera in one of twins. Both had other abnormalities, one a mirror image of the other. Giustra (1939) found a case of cor bilocular in both twins.

According to Maude Abbott congenital cardiac abnormalities are generally due either to arrest of growth caused by disease in the foetal membranes, mother or germ plasm, or by disease in the foetus itself. Cardiac abnormalities are associated with other anomalies in 11 to 30 per cent. of cases. Rarely there is a family history of cardiac anomalies.

If the germ plasm itself is affected by any of these causes it is difficult to see how only one twin could be affected. The larger twin may have had the better nutrition throughout and the smaller twin suffered by an arrest of cardiac growth. The cords were both the same size, however, and the sizes of the twins were not grossly different. Regarding the other possibility of disease of the maternal tissue or foetal membranes, one of these may have been the cause in this case as shown by the vaginal haemorrhage in the third month. By that time the development of the heart is far advanced. Maude Abbott thinks that a patent ductus arteriosus may be caused by an acute infective process with primary involvement of the pulmonary trunk. It is often associated with other abnormalities, chiefly coarctation of the aorta. This represents an arrested foetal condition, possibly owing to a lowered state
of nutrition. The haemorrhage in this case may have brought about the appropriate conditions which only affected one of the twins, the smaller of the two.

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REFERENCES