CASE REPORTS

LONGEVITY IN PATENT DUCTUS ARTERIOSUS

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Persistence of the ductus arteriosus is not usually associated with a normal life expectancy. All but three of 62 patients were under 40 when they died (Shapiro, 1944). Three patients, all women, have lived to over 70 years. One who died at 79 is mentioned briefly as having a small opening and great dilatation of the pulmonary arteries with general cardiac hypertrophy (Holman *et al.*, 1953). Another who lived to 75 had auricular fibrillation and congestive failure: the ductus measured 3 mm. across (Fishman and Silverthorne, 1951). Cardiac catheterization established the diagnosis in a woman aged 72 years with auricular fibrillation and congestive failure (Storstein *et al.*, 1952). Two cases are now reported, one of whom died at 85 years, and never suffered from congestive failure, while the other is living at 63.

Case 1. This patient was born in 1870 and spent her active life as a school-mistress. She was seen first in 1944, when she was 74, suffering from short paroxysms of tachycardia. The blood pressure was 180/80. The apex impulse was heaving and outside the nipple line. A continuous murmur with systolic accentuation was present in the pulmonary area, but there was no thrill. The pulmonary second sound was loud and clear. The electrocardiogram showed left ventricular hypertrophy. Screen examination showed some enlargement of the heart to the left, and the aorta was much unfolded with calcification in the aortic knob.

Later that year she was seen by Dr. James Brown who agreed with the diagnosis of a patent ductus. In 1949 she was shown to Sir John Parkinson who pointed out the calcified ductus in the left oblique view (Fig. 1). Subsequently the paroxysms of tachycardia became more frequent and lasted longer. The ductus murmur became less obvious and in 1955 only the systolic component was present. Finally in 1956, she became anæmic from gastro-intestinal bleeding and died, aged 85. She never had cardiac failure.

Necropsy (Dr. J. V. Wilson). The body was of normal development and nutrition. The heart weighed 270 g. The walls of the ventricles were of normal thickness and there was neither hypertrophy nor dilatation of any chamber. The heart valves were normal. The aorta showed extensive areas of degeneration and calcification and the ascending aorta and the arch were slightly dilated due to this degeneration. The coronary arteries were thickened and sclerosed but were nowhere occluded. The myocardium was healthy apart from some fibrosis.

The ductus arteriosus was patent. The aortic opening (Fig. 3) was funnel-shaped due to extensive calcification of the wall of the aorta. This calcification extended for a radius of about 1 cm. around the opening so that the wall of the opening itself was firm and largely calcified: it was 4 mm. in diameter. The total length of the ductus was 6.5 mm. and it was calcified for about 3.5 mm. distal to the aortic opening forming a rigid channel. The pulmonary opening (Fig. 4) was surrounded by a thin fringe of endothelium and was freely mobile on its attachment to the pulmonary artery. The main pulmonary artery showed no evidence of calcification and the pulmonary branches were not thickened.

The kidneys were contracted and granular. The bowel mucosa showed considerable congestion and the contents of the lumen were extensively blood-stained, especially in the upper part of the small intestine. There was no visible intestinal ulceration and the blood appeared to be oozing from the congested mucous membrane. Death was due to uræmia. A remarkable feature of the necropsy was the absence of evidence or circulatory embarrassment from the ductus arteriosus.

FIG. 1.—Left oblique film, showing calcified ductus. Case 1.

FIG. 2.—Generalized cardiac enlargement with prominence of pulmonary arc. Case 2.

FIG. 3.—Aortic opening, showing the areas of calcification and the funnel-shaped opening. Case 1.

Case 2 was born in 1892. At the age of 16 while staying with relatives in Colne she developed appendicitis and was attended by Dr. James Mackenzie who did not advise an operation on account of her heart. When seen in 1952 the pulse was collapsing and the blood pressure was 180/70. The apex impulse was heaving and 13.5 cm. to the left of mid-sternum. A continuous Gibson murmur with systolic accentuation was audible in the pulmonary area. There was no thrill. The pulmonary second sound was not audible.

FIG. 4.—Pulmonary opening with surrounding membranous fringe. Case 1.

By 1956 auricular fibrillation had supervened, but the murmur was unchanged. She was dyspnoeic on exertion but was complaining chiefly of pain down her legs due to a prolapsed disc. The electrocardiogram showed auricular fibrillation and left ventricular hypertrophy. Chest films showed considerable enlargement of the heart to the left, and some prominence of the pulmonary arc (Fig. 2). The lung roots were large and some pulsation could be seen on screening.

Cardiac catheterization (Dr. E. J. L. Pearce) disclosed a moderate rise of pulmonary arterial pressure, this being 50/15 in the right pulmonary artery and 50/0 mm. in the right ventricle: the left branch of the pulmonary artery could not be entered. There was also evidence of a left-to-right shunt into the pulmonary artery, probably with some regurgitation into the right ventricle. The oxygen saturation was 89 per cent in the pulmonary artery sample, 72 per cent in the high and 56 per cent in the low right ventricular sample, and 51 per cent in the right atrial sample. In view of the absence of the pulmonary second sound it seems likely that this increased oxygenation of the right ventricle was due to pulmonary incompetence. The systemic flow was calculated as 3.4 litres a minute and the pulmonary flow (20.9 litres) and shunt were very large but difficult to assess accurately as the high saturation of the pulmonary artery suggested this had been sampled near the ductus before there was much mixing.

Summary

Two cases of patent ductus arteriosus are described. One survived to the age of 85 years and never suffered from cardiac embarrassment. The other, with some rise of pulmonary arterial pressure and incompetence, is still active at 63 years.

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References

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