A CASE OF PULMONARY STENOSIS

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T.G.C., was a girl of 19 when she was seen in August 1954 with a history of dyspnoea on exertion for 1 year. She came from a family of six and was the youngest in the family. She had a normal history of birth and development, and there was no history of any serious personal or familial disease

On examination she was of normal build and acyanotic. There was no finger clubbing and no sign of cardiac decompensation. Her blood pressure was 100/70, pulse 86/minute and regular, and she was dyspnoeic on moderate exercise. Her cardiac apex was 3" from the mid line in the left 5th intercostal space with a right ventricular heave, and there was a distinct systolic thrill in the parasternal border at left 2nd, 3rd and 4th intercostal spaces. On auscultation, the second sound was single and soft, and there was a rough loud systolic murmur maximal in the left 2nd space and audible all over the chest and back but not conducted to the neck.

A straight X-Ray of the chest showed a slight enlargement of the heart with prominent right ventricle, and screening showed a slight pulsation of hilar vessels. The Radiologist reported that the finding was compatible with a patent ductus arteriosus or a ventricular septal defect. Electrocardiograph tracing showed

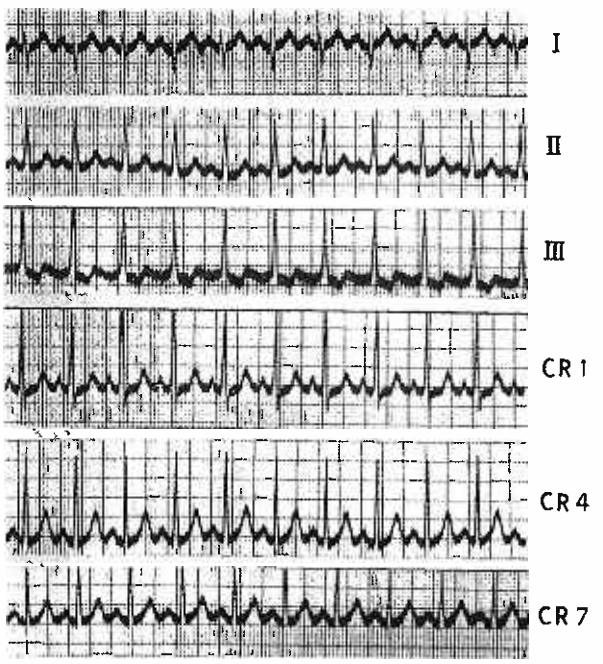

Fig. 1 E.C.G.

a right ventricular hypertrophy pattern with no bundle branch block (Fig. 1). A provisional diagnosis of pulmonary stenosis was made and the patient was kept under observation.

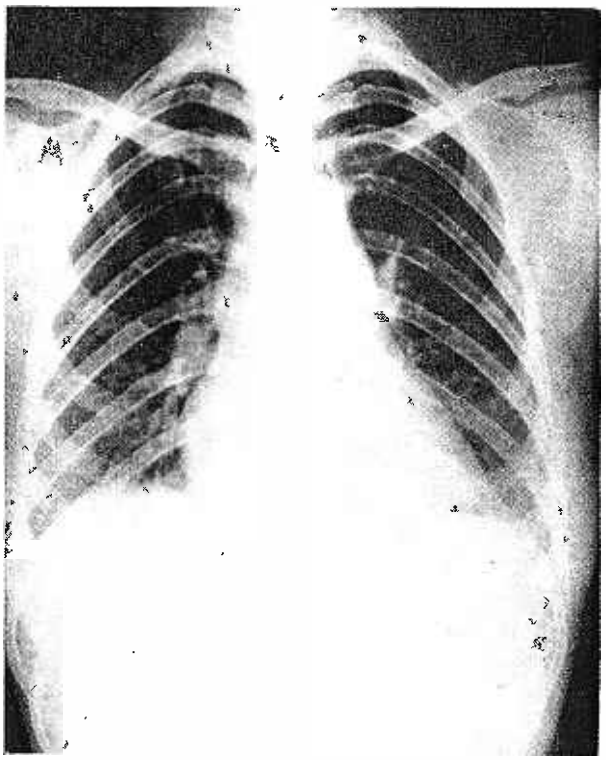

Fig. 2. At time of first visit.

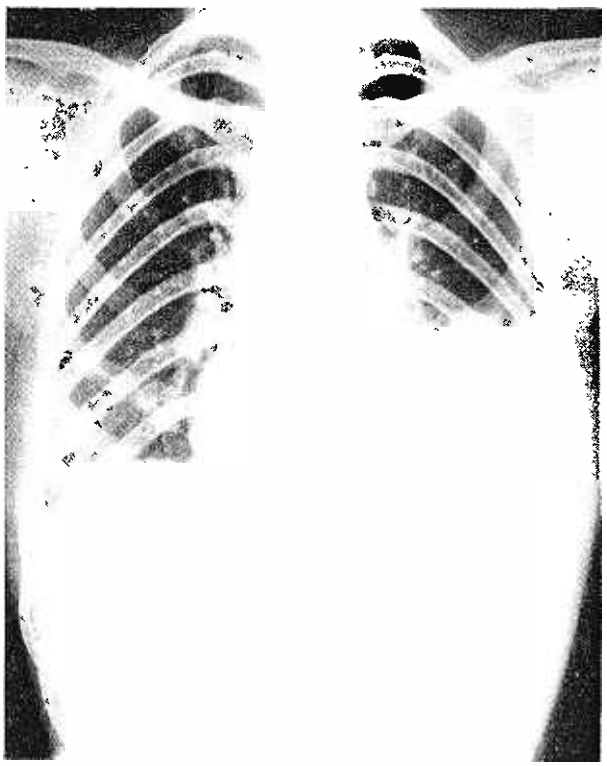

Fig. 3. Left pleural effusion.

PROGRESS

In November 1954, a left pleural effusion was found, with an acute history of chest pain and fever. The fluid was straw-coloured but did not clear up with penicillin. The sputum was negative, and the culture of the pleural fluid did not grow acid fast bacillus. Nevertheless, in view of the prevalence of tuberculosis locally, she was given streptomycin, isonicotinic acid and paraaminosalicylic acid as a case of tuberculosis. Two weeks of the treatment did not show any improvement as the fever continued unabated, and although her blood culture was repeatedly negative, she was in addition given 4 mega units of penicillin a day, and she began to improve, and after 4 weeks of penicillin, she was well and was continued on antitubercular therapy.

In April 1955, however, she developed a triangular opacity in the right base with no apparent change in her physical state apart from the fact that her heart was getting larger. It was then realised that she was most probably a case of subacute bacterial endocarditis with recurrent pulmonary infarction and she was put back on penicillin for 6 weeks with good results. An angiocardiogram was done, and the pulmonary trunk and the main arteries were grossly enlarged. Although a definite valvular defect was not seen, because of the dilatation of the pulmonary arteries conforming to that of post-stenotic dilatation, a valvular stenosis was suspected as an infundibular stenosis did not as a rule cause marked post-stenotic dilatation. Also the pulmonary second sound was single.

For the next year till 1956, she remained well, her exercise tolerance increased, and her radiological heart size also decreased slightly. It certainly appeared that the endocarditis had improved the condition perhaps by ulcerating away part of the stenosis thereby increasing the blood flow to the lungs.

She remained reasonably well, but her exercise tolerance was still limited and on 1st June 1960, as facilities for open heart operation under hypothermia were available, she was submitted to an operation. During the operation, she was found to have pulmonary valvular stenosis with a good deal of thickening and deformity of the valve which was still quite stenotic and resembled a diaphragm. The valvular diaphragm was slit with scissors, and the patient made an uninterrupted recovery.

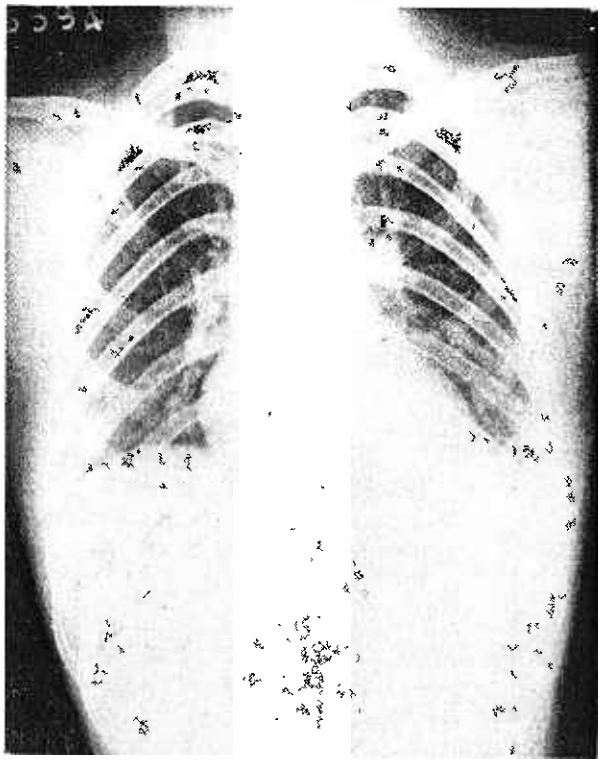

Fig. 4. Left effusion resolved.

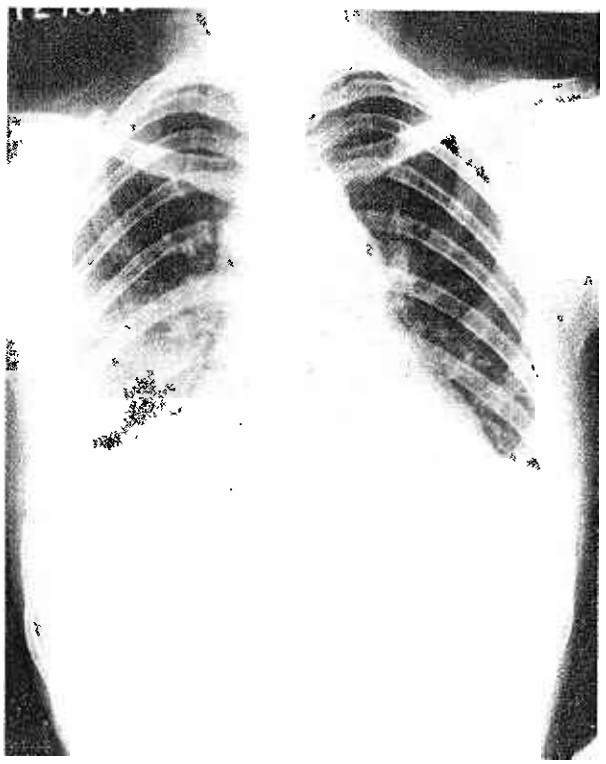

Fig. 5. Right pulmonary infarct

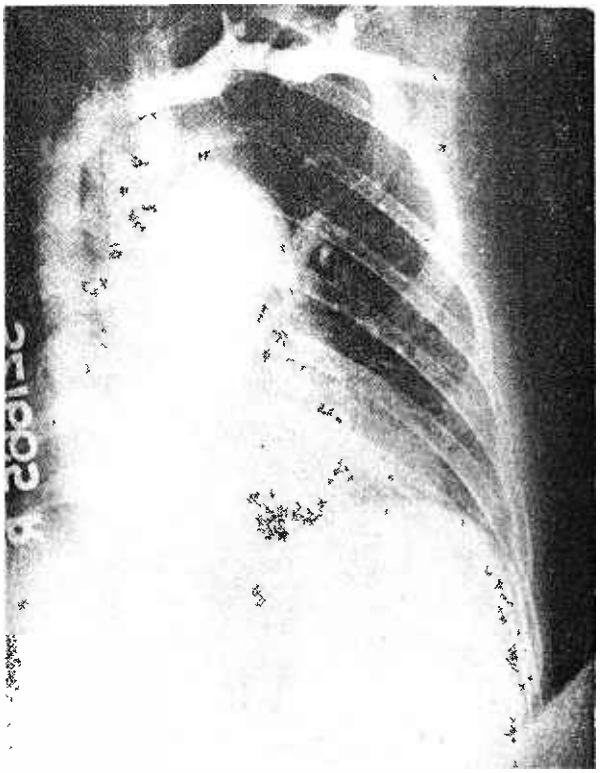

Fig. 6. Angiocardiogram showing enlargement of pulmonary arteries.

COMMENT

This case is reported for two reasons: firstly, this is the first case of pulmonary stenosis operated locally; and secondly, it was thought a rather interesting point that the patient had an ulcerating endocarditis which resulted in some measure of improvement. Had the ulceration been more thorough, she might in fact have obtained a natural cure. As it was, she actually improved in general condition so that when she went for surgery she was in a better state than when she first came under observation.

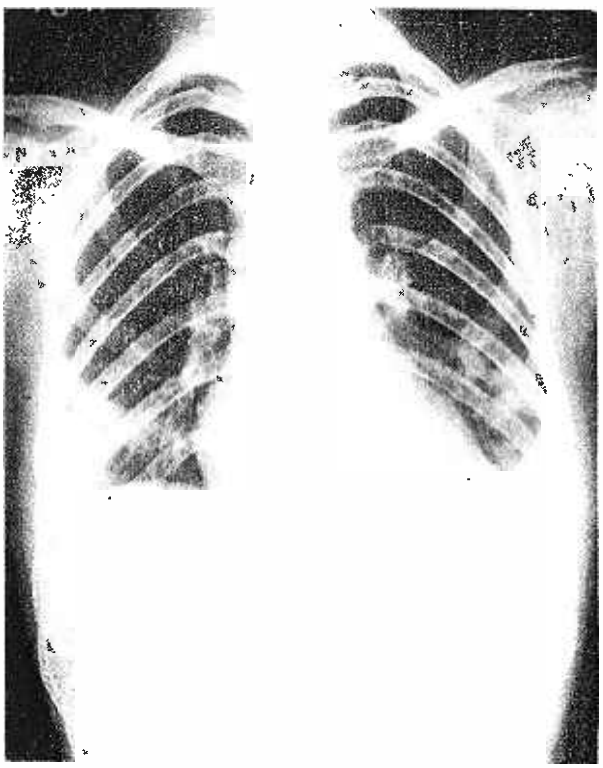

Fig. 7. Two years later with smaller heart.