COMMUNICATIONS TO THE EDITOR

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Normal Function of Parachute Mitral Valve
Association with Tetralogy of Fallot, Atrial Septal Defect, and Patent Ductus Arteriosus

To the Editor:

Parachute mitral valve is a rare congenital cardiac anomaly in which the chordae tendineae of both leaflets of the mitral valve insert into a single papillary muscle.1 The lesion is often associated with a supravalvular ring of the left atrium, subaortic stenosis, and coarctation of the aorta, forming a specific developmental complex ("parachute mitral valve complex"),1,2 but may also occur in isolation or in apparently incidental association with ventricular septal defect, patent ductus arteriosus, transposition of the great vessels, or other anomalies.3 Regardless of associated lesions, parachute mitral valve almost always causes either stenosis or incompetence of the valve, necessitating valve replacement.5

This report describes a case of parachute mitral valve occurring in association with tetralogy of Fallot, atrial septal defect, and patent ductus arteriosus. The mitral valve lesion was not hemodynamically significant and was only discovered at autopsy four years after total correction of the tetralogy of Fallot and other lesions.

CASE REPORT

A white boy was noted to have cyanosis at birth, and physical development in the first year of life was retarded. At the age of two years, diagnoses of tetralogy of Fallot, atrial septal defect, and a small patent ductus arteriosus were established by angiocardiographic studies. Aortopulmonary fenestration was performed at this time, and subsequent development through childhood and adolescence was satisfactory, despite persistent cyanosis, limited exercise tolerance, and two episodes of bacterial endocarditis.

At the age of 26 years, total correction of the tetralogy of Fallot and closure of the atrial septal defect and of the patent ductus arteriosus were performed, using a pericardial graft to reconstruct the right ventricular outflow tract and polyester (Dacron) grafts to close the atrial septal and ventricular septal defects. The mitral valve was inspected through the left atrium and appeared normal. After surgery, there was improvement in exercise tolerance and resolution of clubbing.

A persistent soft localized apical systolic murmur was heard, but there was no other evidence of mitral valvular disease. Four years after this operation, the patient died unexpectedly at home.

At autopsy, the heart weighed 450 gm, and both left and right ventricles were hypertrophic. The anterior papillary muscle of the left ventricle was absent (Fig 1), and all chordae tendineae of both leaflets of the mitral valve inserted into a single posterior papillary muscle. The leaflets of the valve were mildly thickened and opaque. Their free margins were slightly ballooned between the attachments of the chordae tendineae, but there were no clefts or other anomalies. The chordae tendineae themselves were normal in thickness and length and were not interadherent. The left atrium was not dilated and contained no thrombi. The grafts in the pulmonary conus and both atrial and ventricular septa were intact. Death was considered to be probably due to an arrhythmia.

DISCUSSION

In most reported cases of parachute mitral valve, the leaflets and commissures were normal, but the chordae tendineae were short and interadherent, resulting in functional stenosis of the valve; however, the mitral leaflets in other cases were either cleft or partially unsupported by chordae tendineae, resulting in mitral regurgitation.4 One previous case has been reported in which the parachute anomaly of the mitral valve was apparently functionally insignificant.4

In the present case the leaflets and chordae tendineae of the mitral valves were normal, and the parachute anomaly resulted in no significant hemodynamic abnormality of the valve. The soft localized systolic murmur heard after the correction of the coexistent tetralogy of Fallot may have been due either to trivial mitral regurgitation or to turbulence in the flow of blood about the abnormally inserted chordae tendineae.

This case demonstrates the association of a parachute deformity of the mitral valve and tetralogy of Fallot, together with other cardiac lesions, and shows that this mitral deform-

FIGURE 1. Left atrium and ventricle, showing parachute deformity, (insertion of chordae tendineae of both mitral valvular leaflets into single papillary muscle; arrow). LA, Left atrium; and LV, left ventricle.
ity may cause no significant functional abnormality of the valve.

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REFERENCES


The Importance of Impedance in Evaluating the Function of Pacemakers

To the Editor:

The article by Salem et al entitled "Fracture of Pacing Electrode Mimicking Failure of Pulse Generator" (Chest 74:673-674, 1978) is very useful. Now that pulse generators with longer lived batteries are available and now that more and more cardiothoracic surgeons are returning for replacement of pulse generators (the lead having been implanted for many years), chronic problems with electrodes are to become even more frequent.

In my recent series, 19 of 50 patients with delayed failure of the system predicted to be due to a faulty lead were found instead to have a correctable problem with the lead. Most of these were due to "exit block," but one was due to a fracture of the electrode with an intact sheath. Often such fractures will not be evident even upon close inspection of the x-ray films.

At a secondary operation, in virtually every such instance the use of one of the new analyzers will give appropriate electrical evidence of the correct diagnosis. In the case of Salem et al, there was no capture, even at the highest setting (10 v at 2.0 msec), but this can mean "exit block," displacement of the tip of the electrode, fracture of the length of the lead, or a problem at the lead terminal-generator connection (or some combination of these problems). Even though there was no capture, the "resultant current" on the display of the testing device should have been noted. If it were in the very low range (eg, 1.0 ma or less), then the "calculated impedance" could still be determined. In the cited hypothesis, it would be greater than 10,000 ohms, diagnostic of discontinuity somewhere along the lead system. It would also exclude "exit block" because in that circumstance, both the voltage and the current at threshold are proportionally high, so that the impedance is unchanged.

Intraoperative electrical testing at the time of all primary or secondary operations involving pacemakers is warranted, and the new analyzers are extremely useful.

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REFERENCE


To the Editor:

My coauthors and I wish to thank James W. Calvin, M.D., for his comments concerning our article. We agree with his observations that it is very common to find failure of the pacemaker system due to a correctable problem with the lead, rather than a faulty generator. His point is well taken that the measurement of the "resultant current" would have been very useful in assessing the impedance across the wire. Unfortunately, this measurement was not made, but judging from our success after repairing the broken pacing lead, we believe that it can safely be assumed that the impedance was quite high (ie, greater than 10,000 ohms).

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Bronchial Stenosis and Wegener's Granulomatosis

To the Editor:

We recently reported (Chest 74:690-691, 1978) the case of a young woman diagnosed as having idiopathic bronchial stenosis. Since acceptance of our manuscript for publication, the patient was hospitalized in April 1978 with a bilateral lower lobe pneumonia, which cleared in four weeks on therapy with erythromycin. The hematocrit reading, results of urinalysis, blood urea nitrogen level, and creatinine level were normal. A subsequent bronchoscopy revealed that the left main-stem stenotic lesion just allowed passage of the bronchoscope, which was 5.5 mm in diameter. The left upper lobe, lingula, and left lower lobe orifices were all narrowed but otherwise open. The right bronchus intermedius was unchanged.

In October 1978, the patient went to her college dispensary with a sore throat; she had a negative throat culture and hematuria. The hematuria did not resolve with therapy with a combination of sulfamethoxazole and trimethoprim (Septra). Subsequent evaluation at Walter Reed Army Medical Center disclosed a hematocrit reading of 31 percent, a blood urea nitrogen level of 67 mg/100 ml, a creatinine level of 6.5 mg/100 ml, and a urinalysis that revealed 3+ protein, red blood cells (RBCs) that were too numerous to count, and several RBC casts. A renal biopsy showed subacute glomerulonephritis with cellular infiltration, as well as focal glomerular sclerosis and tubular fibrosis.

The patient was referred by our renal department to Dr. Fauci at the National Institutes of Health. After reviewing her clinical course and biopsy, he believed that she most likely had Wegener's granulomatosis. In the light of her smoldering disease, she was treated with prednisone and cyclophosphamide, with stabilization of her renal function. Fauci stated (personal communication, January, 1979) that he had seen patients with Wegener's granulomatosis who had endobronchial disease and stenotic lesions; however, when biopsied, these lesions usually contained granulomas, unlike our patient, where two biopsies failed to reveal either granulomas or evidence of angitis.

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