pecific factor in the asthmatic which converts changes in heat loss from the mucosa to a specific stimulus to bronchial smooth muscle contraction is not well understood. However, asthmatic airways are generally hyperreactive to a variety of nonimmunologic stimuli, including methacholine. Perhaps a similar degree of stimulation of nonspecific irritant receptors secondary to mucosal cooling provokes a more vigorous response in asthmatic subjects than in nonasthmatics. Alternatively, a difference in release of, or response to, local mediators might also be involved.

Neither of our patients was a known asthmatic. Although it might be argued that patient No. 2 was an asthmatic waiting for the appropriate challenge, prior to therapy with propranolol he had never experienced any episodes of dyspnea. He had done heavy work for several years in the meat cooler without any difficulty and returned to such work after propranolol was discontinued. Patient No. 1 was a smoker with chronic bronchitis who had had a chronic cough and mild dyspnea on exertion over a period of almost ten years without any episodic changes in function prior to therapy with propranolol. His clinical course followed that pattern once again after propranolol was discontinued.

Patients with chronic bronchitis have a tendency to have increased airway reactivity similar to that seen in asthmatic subjects. Perhaps in some of these patients (as in asthmatics) the ability to combat reflex bronchoconstriction or bronchoconstriction induced by local mediators may depend upon an intact adrenergic response, whereas in normal individuals the same stimuli may be much less potent and the effect of beta blockade inconsequential.

Regardless of the mechanism, it is important to recognize that propranolol can be associated with severe acute respiratory failure, even in patients without severe history of asthma. Exercise in cold air was the precipitating event in our patients. Each patient in retrospect complained of decreasing exercise tolerance since starting the drug.

Propranolol is tolerated without difficulty by most patients with chronic lung disease without a history of reversible airway obstruction. Nonetheless, patients with chronic bronchitis who are started on propranolol should be asked specifically whether they are having episodes of shortness of breath precipitated by exercise. The drug should be stopped in any patient who reports a history consistent with exercise-induced dyspnea.

REFERENCES


Surgical Treatment for Chest Pain in Mitral Valve Prolapse*

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The case of a 40-year-old woman with mitral valve prolapse and severe atypical chest pain is presented. The diagnosis was confirmed by phonocardiographic, echocardiographic, and angiographic studies. The electrocardiogram revealed an ischemic pattern of ST-T on the anterior and inferior wall. Coronary angiographic studies showed normal coronary arteries. The patient's long-standing, prolonged, disabling atypical chest pain could not be relieved with medical therapy, despite the administration of beta-adrenergic blocking agents, calcium antagonists, and short-acting nitrates during a 30-month period. Thus, the prolapsed mitral valve was replaced with a Hancock xenograft. After 12 months the patient is totally free of symptoms, without any treatment and with a normal ECG. This excellent surgical result could be explained on the basis of the valvular theory of chest pain in mitral valve prolapse, suggesting that pain is promoted probably by a regional imbalance between oxygen availability and consumption, because of the excessive papillary muscle stretching produced by the prolapse. To our knowledge, this is the first published report of successful surgical treatment of chest pain in mitral valve prolapse.

The spectrum of mitral valve prolapse varies greatly, from symptom-free patients, who represent approximately 20 percent of the total and who are discovered by means of typical auscultatory, echocardiographic, or angiographic findings, to patients who die suddenly, probably because of lethal arrhythmias, although the great majority of these arrhythmias are well tolerated. The most common symptom is an atypical and long-standing pain in the chest. It has already been demonstrated that this pain is generally controlled by therapy with propranolol, but not in all cases.

We have observed one case of mitral valve prolapse with ventricular arrhythmia and atypical and intractable pain in the chest, despite therapy with nearly all antianginal drugs. The persistence and severity of the pain in the chest led us to replace the mitral valve with a xenograft, on the basis of the most accepted theory about the mechanism of chest pain in mitral valve prolapse. We think this is the first successful report of this approach. Its potential consequences and mechanisms will be briefly discussed.

CASE REPORT

A 40-year-old woman without a history of rheumatism had

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had eight years of palpitations and exertional chest pain that was relieved by therapy with nitroglycerin. In 1976, she was admitted to our clinic because of severe and long-standing pain in the chest that could not be relieved by administration of nitroglycerin. Since then, the patient's almost continuous pain in the chest, even at rest, was so disabling that she was confined to bed. The response to therapy with nitroglycerin was variable. During the next 18 months, the pain could not be relieved despite the administration, alone or in combination, of propranolol (120 mg/day), oxprenolol (160 mg/day), perhexiline maleate (300 mg/day), diazepam (10 mg/day), and isosorbide dinitrate (5 mg sublingually every three to four hours).

Physical examination showed a mesosystolic click and a telesystolic murmur, with the presence of a third heart sound (Fig 1). The resting electrocardiogram showed ischemic inversion of the T waves in the apicolateral and inferior wall (Fig 2A) and occasional ventricular bigeminy. The results of an exercise stress test (bicycle ergometer; maximal effort) were normal. The mitral valve prolapse was documented by echocardiograms.

Cardiac catheterization revealed normal right-sided and left-sided pressures. Cardiac output and vascular resistances were also normal. Ventriculographic studies showed a grade-4 triscallop posterior mitral valve prolapse, with minimal mitral telesystolic regurgitation and normal ventricular performance (Fig 3).

![Figure 1. Phonocardiogram. Huge mesosystolic click (MC), followed by mesotelesystolic murmur (LSM). Splitting of first heart sound and a third heart sound are also recorded. HF, High frequency; and LF, low frequency.](image)

![Figure 2. A (top), Preoperative ECG. Ischemic T-wave inversion on lateral and inferior wall, without hypertrophy of chamber. B (bottom), Eight-month postoperative ECG, showing complete recovery of ventricular repolarization.](image)

![Figure 3. Left ventriculogram. A (top), Telediastolic frame. B (bottom), Telesystolic frame. Open arrows point out posterior mitral valve prolapse. Solid arrow indicates mild mitral regurgitation.](image)
Selective coronary arteriographic studies, performed with the Bourassa technique, revealed normal coronary arteries, with a right predominance and a short and thin circumflex artery. These studies were repeated at 18-month intervals, with identical results. Due to the failure of all previous therapeutic measures, after thorough consideration, surgical treatment was attempted in November 1977. The mitral valve and papillary muscles were excised, and a 29 Hancock xenograft was placed with interrupted sutures anchored in Teflon because of the fragility of the mitral annulus. The excised valve had much redundant tissue, especially in the posterior leaflet (Fig 4). There were several abnormal papillary muscles and direct insertion of the chordae tendineae in the posterior wall of the left ventricle. Pathologic study showed mild valvular myxomatous degeneration and slight hyperplasia of the papillary muscles. There were no postoperative complications.

During a follow-up period of 12 months, the patient's condition has remained stable, with no chest pain or cardiac medication. The ECG (Fig 2B) shows complete recovery, with normal ventricular repolarization.

**DISCUSSION**

Two main theories have been advanced to explain the etiology of mitral valve prolapse.6,7 The myocardial theory supposes that the disease is produced by a silent ischemic cardiomyopathy.8 It is believed that the alteration of coronary blood flow produces papillary muscle dysfunction and abdominal segmental contraction.9 This theory seems invalid to us because not all cases, including ours, have abnormal coronary arteries.6,10 and abnormal segmental contraction can be induced by ectopic beats or stretch-induced papillary muscle fibrosis.

The valvular theory ascribes the prolapse to myxomatous degeneration of the valve1-5 or to other associated defects, such as Marfan's syndrome or atrial septal defect. This valvular theory could explain thoroughly the various manifestations of this syndrome. Nutter et al12 proposed some physiopathologic mechanisms that could explain the clinical history of mitral valve prolapse.

![Figure 4. Mitral valve at surgery. Anterior (arrowhead) and posterior (arrow) leaflets have redundant tissue. Clamp pulls on anterior leaflet.](image)

We find their theory logical and probably right. According to these authors,12 the mitral valve prolapse is not due to abnormal contraction, nor to cardiomyopathy, nor to coronary heart disease, but to an abnormal mitral apparatus due to a heritable factor or to an acquired structural defect. In addition, a hyperkinetic ventricular contraction would enhance the prolapse. The mitral prolapse can stabilize or progress and produce excessive papillary muscle stretching, leading to pain in the chest, arrhythmias, and the other manifestations of the syndrome.

The different causes cited to explain the anginal pain in mitral valve prolapse are coronary atherosclerosis, circumflex coronary arterial compression in the atrioventricular groove by the prolapsing mitral cusps,8 congenital absence of the circumflex coronary artery, psychologic factors, and imbalance between the regional consumption of oxygen and the coronary blood flow.13 Of these theories the last is perhaps the most suitable. Because of mitral valve prolapse, the base of the papillary muscle suffers an abnormal stretching which, in turn, alters the balance between oxygen demand and coronary flow in the region of the mitral apparatus.

The mitral valve prolapse can produce mitral regurgitation, usually tellesystolic. Fortunately, this regurgitation is mild and usually well tolerated. Cardiac surgery with prosthetic valve replacement has been necessary in relatively few patients with mitral valve prolapse.1,11 As in our case, we recommend affixing the prosthesis with interrupted sutures.

There are a few reported cases14,15 of mitral replacement for controlling recurrent, refractory, and potentially lethal ventricular arrhythmias. In these cases the arrhythmias disappeared after valvular replacement. Assuming that the pain in the chest is produced by the imbalance between the regional demand for oxygen and the coronary flow, we undertook mitral valvular replacement for controlling the disabling pain in the chest of our patient. We thought that if we eliminated papillary muscle stretching, we could relieve the cause of the chest pain. So far, the assumption seems to be correct, since our patient is completely free of symptoms. We have found only one case (referred to by Jeresaty16 as a personal communication from Litwak) of mitral valvular replacement for testing pain in the chest.

Our data seem to indicate that the valvular anomaly of mitral valve prolapse is responsible for the onset of symptoms, which usually are mild. But when they become severe and disabling, with poor response to treatment, we think that it is wise to take into account the alternative of valvular replacement.

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**SURGERY FOR CHEST PAIN 103**
Respiratory Failure due to

**Strongyloides stercoralis** in a Patient with a Renal Transplant*

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We report a case of respiratory failure caused by **Strongyloides stercoralis** in a patient with a renal transplant; the respiratory failure showed dramatic response to therapy with thiabendazole. The clinical aspects of infestation with **S stercoralis** in the immunocompromised host are discussed, and features are demonstrated which may have significant implications concerning primary treatment and prophylaxis.

**Strongyloides stercoralis** is a helminth that can cause severe infestation in the immunosuppressed and malnourished host. We report a case of infestation with **S stercoralis** with severe, diffuse pulmonary involvement causing marked hypoxemia in a patient with a renal transplant. Although severe pulmonary infestation with this parasite has previously been reported in the immunocompromised host, this case displays some unique features, including antemortem demonstration of the organism in pulmonary tissue, a dramatic response to therapy with thiabendazole, later requiring administration of pyrimethamine pamoate to eradicate the organism, and the presence of an additional pulmonary infection.

**CASE REPORT**

A 47-year-old Puerto Rican male recipient of a renal transplant was admitted to the University of Illinois Hospital on Feb 11, 1979, with chief complaints of malaise, abdominal pain, and diarrhea. Renal failure secondary to hypertension had been noted three years earlier. In August 1978, a guaiac test of stools was noted to be positive for blood on a screening physical examination done in preparation for a renal transplant. The stools also revealed larval forms of **S stercoralis**, and the patient was treated with thiabendazole (25 mg/kg of body weight) daily for four days. One month after therapy, stools and duodenal aspirate were negative for organisms.

In November 1978, transplantation with a cadaveric kidney was successfully performed, and the patient received prednisone and azathioprine. He was readmitted one month later because of acute rejection, which was treated with

![Figure 1. Chest roentgenogram on second day of hospitalization. Diffuse nodular infiltrate resolved after five days of therapy with thiabendazole.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21153/)