BRIEF COMMUNICATIONS

CARDIOMYOPLASTY COMBINED WITH IMPLANTATION OF A CARDIOVERTER DEFIBRILLATOR

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In the United States alone, approximately 2 to 3 million patients generally are affected by heart failure, and this number is likely to increase.¹ Survival of patients with advanced ventricular dysfunction is limited not only by progressive pump dysfunction but also by the risk of sudden cardiac death. The 1-year mortality risk exceeds 50% for patients with class IV symptoms.² Dynamic cardiomyoplasty is emerging as a promising form of surgical therapy for patients with advanced ventricular dysfunction, but sudden cardiac death remains a major factor in decreased long-term survival among patients who have undergone cardiomyoplasty.³ Among the various available therapeutic strategies for patients at high risk for sudden cardiac death, the implantable cardioverter defibrillator (ICD) has been shown to be effective. Adjunctive use of the ICD may therefore provide an optimal outcome for patients undergoing cardiomyoplasty.⁴

The following clinical summary describes the course of a patient who underwent cardiomyoplasty with concomitant ICD implantation. We believe that this combined procedure may improve survival among cardiomyoplasty candidates who are deemed to be at high risk for sudden cardiac death.

Clinical summary. A 65-year-old retired physician with advanced ventricular dysfunction related to coronary artery disease was evaluated for latissimus dorsi cardiomyoplasty. In the months before evaluation, the patient had two episodes of unexplained syncope. He had an out-of-hospital cardiac arrest, from which he was successfully resuscitated; monomorphic ventricular tachycardia (VT) was documented as the initial rhythm.

The baseline 12-lead electrocardiogram showed sinus bradycardia with a first degree atrioventricular block, left bundle-branch block, and a rightward axis. During the electrophysiology study, sustained monomorphic VT of at least three morphologies and rates were easily inducible and pace terminable. The patient was started on a regimen of oral amiodarone loading for suppression of VT. Cardiac hemodynamic evaluation revealed evidence of pulmonary hypertension (right pulmonary artery pressure

58/25 mm Hg and mean 36 mm Hg), elevated pulmonary capillary wedge pressure (an atrial wave of 28 mm Hg, ventricular wave of 42 mm Hg, and mean of 32 mm Hg), and reduced cardiac index (2.12 L/min/m²). Coronary angiography showed severe three-vessel coronary artery disease. The left ventricle was severely hypocontractile, with an ejection fraction of 10%.

Latissimus dorsi cardiomyoplasty was chosen instead of heart transplantation because the patient did not have end-stage heart failure. Cardiomyoplasty was performed through a medial sternotomy and a posterior wrap was performed. A Medtronic cardiostimulator (Medtronic, Inc., Minneapolis, Minn.) was implanted with the myocardial and skeletal electrodes placed in the conventional manner. The operation was completed without complications.

After satisfactory postoperative recovery, the electrophysiologic study was repeated to evaluate the efficacy of drug therapy. Monomorphic VT of two hemodynamically compromising morphologies remained persistently inducible. Because of the patient's history of advanced ventricular dysfunction and cardiac arrest and the electrophysiologic indications of suppressive drug therapy failure, we elected to proceed with the implantation of a cardioverter defibrillator for prophylactic management of VT and sudden cardiac death. Implantation of an ICD with a transvenous lead system was performed 2 weeks after cardiomyoplasty. Defibrillation testing was performed with step-down energy levels. The lead system used was a Medtronic Transvene right ventricular transvenous defibrillation- and rate-sensing electrode with a second defibrillation electrode in the superior vena cava-innominate vein junction and a subcutaneous patch placed along the left chest wall. A Medtronic pulse generator (model 7219D) was used in conjunction with this lead system, and a defibrillation threshold of 24 J with a biphasic waveform was obtained with this configuration. During defibrillation testing, the cardiomyoplasty stimulator was temporarily programmed to deliver six pulses at the maximum amplitude to evaluate any potential interaction between the cardiomyoplasty device and the ICD. No interaction was noted during testing, and all ventricular fibrillation episodes were appropriately detected by the ICD. The patient had an excellent postoperative recovery after this procedure.

A follow-up electrophysiologic evaluation of the ICD and lead system was performed 3 days later. The cardiomyoplasty generator was programmed to deliver six pulses at maximum amplitude and pulse width during this testing, and the ICD was programmed at the maximum sensitivity to determine whether the ICD sensed any signals from the cardiomyoplasty generator. Inappropri-

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ate sensing was not observed despite these maneuvers, and satisfactory defibrillation was obtained at 24 J in a biphasic waveform. The cardiomyoplasty generator was reprogrammed to its original settings after this procedure, and the ICD also remained active.

The standard Medtronic cardiomyoplasty electrical stimulation protocol was started 2 weeks after cardiomyoplasty, and the final phase of training was reached 70 days after the initial cardiomyoplasty operation. During the follow-up period, the patient had two episodes of VT. Both were detected by the ICD, and appropriate therapy was delivered with consequent conversion to sinus rhythm. Interrogation and analysis of the stored electrograms revealed no interaction between the cardiomyoplasty generator and the ICD during these clinical events. VT was appropriately detected by the ICD without oversensing of the cardiomyoplasty generator during both events.

The patient has continued to report subjective improvement in functional class. He has had steady progress during 1 year of follow-up, although hypothyroidism resulting from amiodarone therapy has complicated his clinical course. Amiodarone therapy was initiated to reduce the frequency of VT, and consequently the number of ICD shocks. The patient has had seven ICD shocks for monomorphic VT since the initial ICD implantation, with five of these shocks occurring before and two after initiation of amiodarone therapy.

Discussion. Survival of patients with advanced ventricular dysfunction is limited both by progressive pump dysfunction and by the risk of sudden cardiac death. The principal mechanism of sudden cardiac death in this population remains arrhythmia, with VT that degenerates into ventricular fibrillation being the most common. The development of VT, usually as a result of reentry, may be modulated by a variety of triggers, such as by alteration in myocardial wall stress from changing preload or afterload and by myocardial ischemia.⁵ Additionally, it has been postulated that placement of the paced skeletal muscle onto a diseased cardiac muscle may create substrates for reentry as a result of scar and fibrous tissue development.

Four groups worldwide have published long-term results of clinical dynamic cardiomyoplasty. In each series, one of the major causes of late mortality was arrhythmic sudden cardiac death. Nine of 38 patients in the Allegheny General Hospital, Pittsburgh series died of arrhythmia,³ and six of 33 in the Heart Institute, São Paulo, Brazil, series died suddenly as a result of sudden cardiac death.⁶ All of these patients who died suddenly as a result of sudden cardiac death were receiving antiarrhythmic therapy for ventricular arrhythmias or atrial fibrillation. The Hospital Broussais, Paris, France, data confirmed deaths of 31 of 52 patients from sudden cardiac death.⁷ The data from Bakulev Institute for Cardiovascular Surgery in Moscow, Russia, are notable for one death from VT and one from sudden cardiac death out of a total of 25 patients.⁸ The overall incidence of sudden cardiac death related to VT was 13.5% (20/148) after dynamic cardiomyoplasty.

It is therefore obvious that patients with advanced ventricular dysfunction who undergo dynamic cardiomyo-

plasty remain at high risk for sudden cardiac death. Improvement in hemodynamic state and functional class does not appear to eliminate this risk. Both primary and secondary prevention of sudden cardiac death in this population must be addressed.

In contrast with empirical or electrophysiologically guided antiarrhythmic therapy, the ICD appears to be associated with the best reduction in sudden cardiac death risk in this population.⁴ Sudden cardiac death rates at 5-year follow-up with the ICD have been reported to be impressively low (4.5%).⁹ The ICD thus appears to be the optimal form of therapy for patients undergoing dynamic cardiomyoplasty.

The timing of ICD implantation with respect to dynamic cardiomyoplasty needs to individually determined. Cardiomyoplasty may be performed in patients who have already undergone ICD implantation. Alternatively, ICD implantation may be performed concomitantly with or soon after the cardiomyoplasty procedure. In some patients, VT and cardiac arrest have not recurred after cardiomyoplasty, perhaps as a result of improvement in hemodynamics, ventricular stretch, or autonomic imbalance. Implantation of a defibrillator should not be deferred in these cases, however, because the antiarrhythmic effect of cardiomyoplasty cannot be accurately predicted.

At present, little is known about the potential interactions between the ICD and cardiomyoplasty systems. It is necessary to perform careful intraoperative and postoperative testing to ensure that the cardiomyoplasty generator and the ICD do not interfere with one another. As long as care is taken during implantation to ensure that such interaction does not occur, long-term follow-up results should parallel the results of concomitant ICD and pacemaker implantation. In our case, episodes of VT treated by the ICD without compromising the operation of the cardiomyoplasty generator confirm that there was no interaction between the two devices.

Although dynamic cardiomyoplasty may provide improvement in hemodynamic state and functional class, the risk of sudden cardiac death remains a vexing problem. Antiarrhythmic drug therapy alone does not appear to extend a favorable outcome because of several limitations. We anticipate that the ICD combined with dynamic cardiomyoplasty will favorably affect survival by reducing the risk of arrhythmic death among patients with advanced ventricular dysfunction.

Since the initial writing of this case report, a second patient at our institution has undergone cardiomyoplasty with concomitant ICD implantation. After the cardiomyoplasty procedure, the patient had symptomatic, nonsustained VT. This was documented by Holter monitoring 3 months after the cardiomyoplasty operation. Subsequent complex electrophysiologic studies documented sustained monomorphic VT that was hemodynamically compromising and could not be suppressed with procainamide. The patient underwent ICD implantation without complication. No antiarrhythmic drug therapy was initiated. He has had no ICD shocks for the 2-month duration of the treatment and has continued to have steady progress. No adverse interactions between the two devices were noted during testing.

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CALCIFIED PEDICLED THROMBUS IN THE LEFT VENTRICLE: THE FINE ART OF NATURE

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Intracavitary cardiac thrombi are infrequent and usually found in patients with concomitant heart disease. These thrombi have a rubbery or malleable consistency, and calcification is rare.¹ Cases of calcified ball thrombus of the fixed type (attached to the atrial wall or septum) have occasionally been published. To our knowledge no case of calcified pedicled thrombus in the left ventricle has been previously reported.

A 46-year-old man with a recent history of thromboangiitis obliterans and characteristic nodules on the legs was found to have a calcified cardiac image on a chest roentgenogram while undergoing a work-up for a 6-month history of nonspecific discomfort and generalized asthenia. He was a heavy smoker with moderately elevated serum cholesterol levels. He had no symptoms of cardiac disease. Doppler echocardiography showed enlargement of the left ventricle with a decreased ejection fraction and apical akinesia. An elongated mass was fixed to the apex of the left ventricle, moving through the cavity, but no mitral valve abnormality was observed. Cardiac catheterization showed two-vessel disease with significant obstructions in the anterior descending and right coronary arter-

ies. The left ventriculogram showed apical akinesia in the area in which a pedicled and elongated mass originated (Fig. 1). The operation was performed with the use of standard cardiopulmonary bypass.

With the heart arrested with retrograde cardioplegia, the left ventricle was opened through the apex and an elongated mass attached to the apical endocardium by thin trabeculae was removed. The mass had a smooth, hard surface and its color was pearly. The apical opening in the left ventricle was repaired with a round Teflon patch and autologous pericardium implanted endocardially. Coronary artery bypass grafting was done concomitantly. The resected mass was ovoid and measured 5 cm in length by 1.8 cm in diameter in the central area (proximal

Fig. 1. Left ventriculogram showing the calcified pedicled mass attached to the apex. A significant lesion is present in the proximal anterior descending artery.

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