Surgical treatment of symptomatic, drug-resistant ventricular bigeminy and other forms of complex ventricular ectopy (ventricular allorhythmias)

This article describes 18 patients with drug-resistant ventricular bigeminy and other forms of complex ventricular ectopy (allorhythmias) associated with recurrent syncope. Particular emphasis is placed on the electrophysiologic methods used to characterize the basis of these arrhythmias and to accomplish isochronous mapping to identify their site of origin. A malignant course of drug-resistant bigeminy in combination with other rhythm disorders was accepted as indications for surgical intervention. Cryosurgery was the method used most frequently for the ablation of arrhythmogenic myocardium. All 18 patients underwent postoperative electrophysiologic studies and Holter monitoring to determine the efficacy of the operation. Fifteen of the 18 patients were cured of their symptomatic arrhythmia. Thus surgical intervention is an effective method for the treatment of patients with symptomatic, drug-resistant ventricular allorhythmias.

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The regular alternation of ventricular premature beats with basic normal sinus rhythm may lead to ventricular bigeminy, trigeminy, or more complex forms of ventricular ectopy. These different variants of ventricular ectopy, exclusive of ventricular tachycardia and ventricular fibrillation, have been termed allorhythmias.' Although allorhythmias are frequently asymptomatic and benign, in some patients they may cause a critical reduction in the number of effective heart contractions with resultant syncope or presyncope. In addition, ventricular bigeminy may be associated with ventricular tachycardia. Symptomatic ventricular allorhythmias require medical therapy, but unfortunately, many of the antiarrhythmic medications that are effective in suppressing the allorhythmias also have the side effect of decreasing the intrinsic heart rate. Because this drug-induced bradycardia may counterbalance any beneficial effects from suppression of the ventricular allorhythmias, surgical intervention must be considered in such patients. The probability of being able to ablate ventricular allorhythmias by surgical means has been enhanced by the demonstration that many types of ventricular premature beats occur on the basis of a reentrant loop, which should be capable of being interrupted by surgical means.1,2 This article describes our experience with the surgical treatment of 18 patients with symptomatic, drug-resistant ventricular allorhythmias.

Patient population

This study comprised a group of 10 female patients and eight male patients whose ages ranged from 16 to 52 years (mean 37.4 ± 2.7 years). The duration of symptomatic allorhythmias ranged from 6 months to 20 years before operation. Associated abnormalities included chronic myocarditis in two patients, congestive cardiomyopathy in two patients, arrhythmogenic right ventricular dysplasia in two patients, atrial septal defect in two patients, and mitral valve prolapse in one patient. The other nine patients were considered to have idiopathic ventricular allorhythmias (Table I).

Thirteen patients experienced a critical reduction in the effective heart rate (hemodynamic bradycardia of 28 to 30 beats/min) that led to syncope and presyncope. In five patients the ventricular allorhythmias were associated with episodes of ventricular tachycardia with cycle lengths varying from 260 to 420 msec. During ventricular tachycardia the QRS morphology was the same as that of the associated ventricular allorhythmia. Two patients had only classic ventricular bigeminy associated with episodes of reciprocating tachycardia as a result of the Wolff-Parkinson-White syndrome.

The number of antiarrhythmic drugs administered to these
patients varied from one to six, with amiodarone the most commonly used agent. Thus the only indication for surgical intervention in these patients was failure of medical therapy or intolerable drug-induced hemodynamic bradycardia.

**Methods**

Holter monitoring for 24 hours with modified leads V1 and V5, was performed in all patients. In all cases we found a sustained ventricular ectopic activity, such as ventricular bigeminy, ventricular trigeminy, ventricular quadrigeminy, frequent ventricular premature beats, and/or paired ventricular premature beats with a constant coupling interval of 440 to 550 msec. Of the five patients demonstrating ventricular tachycardia, the arrhythmia was sustained (greater than six successive beats) in two patients and not sustained in three patients.

All patients underwent two-dimensional echocardiography, cardiac catheterization, and an endocardial catheter electrophysiologic study before operation. Standard 6F or 7F USCI electrode catheters (Division of C. R. Bard, Inc., Billerica, Mass.) were used to stimulate and record electrophysiologic data during sinus rhythm, atrial and ventricular ramp pacing, programmed electrical stimulation (S1 to S5), and rapid burst pacing. Endocardial catheter mapping according to the method of Josephson and colleagues was performed in all patients before operation.

Intraoperative epicardial ventricular mapping was performed with a hand-held, roving electrode and fixed reference electrodes on the right ventricular outflow tract and the right and left atrial appendages. When necessary, endocardial and transmural mapping of the ventricle were performed with plunge needle electrodes. The site of origin of the ventricular arrhythmia was identified in all 18 patients by intraoperative mapping techniques and was found to be in the right ventricle in 13 of 18 patients. Three patients demonstrated arrhythmogenic regions in both the left and right ventricles, and in two patients (both with congestive cardiomyopathy) ventricular arrhythmias arose from the left ventricle only. In all patients with right ventricular arrhythmias, the earliest ventricular activation site during the arrhythmia was in the anterior septal portion of the right ventricular outflow tract.

**Operation.** The heart was exposed by median sternotomy, and all patients were placed on normothermic cardiopulmonary bypass for performance of the operation. After completion of invasive mapping, a ventriculotomy was performed either in the right ventricular outflow tract or at the border of the outflow and/or inflow tracts of the right ventricle, depending on the intraoperative maps. Any endocardial fibrosis that was detected after the ventriculotomy was excised. Multiple cryolesions (-100°C for 2 minutes) were applied to the site of arrhythmogenesis. Associated operations included closure of an atrial septal defect in two patients and ablation of an accessory pathway associated with the Wolff-Parkinson-White syndrome in two patients.

At the completion of the operation, all patients were weaned successfully from cardiopulmonary bypass (average bypass time 40 minutes). Multiple attempts to reinduce the ventricular arrhythmias by programmed electrical stimulation and burst pacing were unsuccessful.

**Results**

There were no operative deaths in this group of patients. Holter monitoring for 24 hours was performed in all patients within the first week after operation, and all patients underwent a postoperative endocardial catheter electrophysiologic study. These studies documented that 16 patients were free of any type of ventricular arrhythmia, whereas two patients continued to have arrhythmias after operation. During the postoperative follow-up period of 6 months to 6 years, 15 patients showed no evidence of recurrent ventricular arrhythmias. Eleven of these 15 patients required no antiarrhythmic medications. In four patients frequent ventricular premature beats occurred during the postoperative period, but they were responsive to antiarrhythmic medications postoperatively, despite the fact that the same drugs had been ineffective preoperatively. Ventricular arrhythmias recurred in three patients, including both patients with congestive cardiomyopathy and one
Fig. 1. Extrasystolic ventricular bigeminy and episode of self-terminated multiform ventricular tachycardia (torsades de pointes) in the patient with mitral valve prolapse. These are simultaneous leads I, II, III, and arterial pressure.

Fig. 2. Left ventriculogram (the same patient). The wall of the left ventricle contains the anatomic basis of arrhythmia dysplasia (D). Ao, Aorta; LV, left ventricle; MV, mitral valve.

The patient in whom ventricular allorhythms were associated with mitral valve prolapse warrants further comment. The patient was a 23-year-old woman who was initially hospitalized because of recurrent palpitations associated with syncope. At the time of admission her pulse was grossly irregular with an effective heart rate of 30 to 34 beats/min. Preoperative Holter monitoring demonstrated ventricular bigeminy and ventricular trigeminy associated with one self-terminating episode of torsades de pointes ventricular tachycardia (Fig. 1). Both the preoperative and intraoperative electrophysiologic studies demonstrated the earliest site of activation during ventricular allorhythmia to be at the base of the posterior papillary muscle (Figs. 2 and 3). As a result, the papillary muscle was resected, the left ventricular wall at the site of papillary muscle attachment was cryoablated, and mitral valve replacement was performed (Fig. 4). Holter monitoring during the immediate postoperative period showed no ventricular allorhythms, and none could be induced at the time of the
Discussion

Recent advances in programmed electrical stimulation techniques to induce and study the underlying mechanisms of arrhythmias and in preoperative and intraoperative mapping techniques have provided a more scientific basis for evaluating the efficacy of therapeutic interventions for the treatment of all types of ventricular arrhythmias. Ischemic ventricular arrhythmias, which constitute approximately 75% of all ventricular arrhythmias, are associated with anatomic lesions (endocardial fibrosis) that can be correlated with the electrophysiologic results of preoperative and intraoperative mapping techniques. Ischemic ventricular arrhythmias occur most commonly on the basis of a reentrant mechanism. Although associated anatomic lesions are not usually present in nonischemic ventricular arrhythmias, it is believed that they also occur on the basis of reentry. One of the best clinical models for the study of chronic nonischemic ventricular arrhythmias is arrhythmogenic right ventricular dysplasia. In this abnormality a partial degeneration of the myocardial wall of the right ventricle occurs in which most of the muscle fibers are replaced by fatty tissue with the survival of only a few healthy myocardial fibers. The propagation of electrical activation is delayed as it passes through this plexiform structure and in the zones adjacent to healthy muscle where reentrant phenomena may arise.

It is our hypothesis that the same pathophysiologic mechanisms may be responsible for ventricular tachy-
cardia, complicating some types of nonischemic ventricular arrhythmias in which there is no gross evidence of organic heart disease, the so-called idiopathic ventricular tachycardia. Recent studies of endomyocardial biopsies demonstrate that a majority of patients with severe ventricular tachycardia and normal gross cardiac anatomy have histologic abnormalities; thus the possibility of primary electrical heart disease or idiopathic ventricular tachycardia is doubtful.

Cardiac arrhythmias are now recognized to be one of the associated complications of the mitral valve prolapse syndrome. Premature ventricular contractions are the most common abnormality associated with prolapse of the mitral valve, but many different forms of rhythm disturbances may occur with this syndrome. In this study no correlation was found between the presence of arrhythmias and the various clinical features of the patients including age, mitral regurgitation, pansystolic murmur, or resting ST-T wave abnormalities. Relatively little is known about the precise pathogenesis of ventricular arrhythmias associated with mitral valve prolapse. It is our opinion that most ventricular arrhythmias associated with mitral valve prolapse originate at the base of the papillary muscle, as it did in our patient. The suggested mechanism is one of endocardial stress caused by mechanical tugging by the chordae tendineae during ventricular systole or ventricular ischemia resulting from papillary muscle tension.

In the spectrum of malignant nonischemic ventricular arrhythmias, we have made a distinction between ventricular tachycardia and ventricular arrhythmias, particularly ventricular arrhythmias that result in a critical reduction in the number of effective heartbeats. If these arrhythmias are symptomatic, they require special treatment, but conventional antiarrhythmic drug therapy frequently fails to suppress the arrhythmias. In addition, even if the arrhythmias can be suppressed, the antiarrhythmic drugs may cause such a profound bradycardia that the actual effect of the drug is to further reduce the number of effective heartbeats. In addition, some of these drugs may prolong the QT interval and result in torsades de pointes ventricular tachycardia. Because of these problems, ventricular mapping with surgical intervention may be the only approach capable of controlling these arrhythmias.

Different surgical concepts may be applied to the surgical treatment of ventricular arrhythmias. The choice of the surgical procedure to be used is based on the preoperative and intraoperative mapping studies and on the anatomic findings at the time of operation. The objective of the surgical technique described in this study is to destroy the tissue underlying the site of epicardial breakthrough of the tachycardia. The ventriculotomy may interrupt the reentrant loop. The concept of endocardial resection can be applied when an arrhythmogenic zone is located over an area that can be resected without impairing ventricular function. The cryoablative technique has the advantage of electively destroying a more limited amount of myocardium and thereby interfering less with myocardial function.

This study demonstrates that surgical ablation of arrhythmogenic areas is an effective procedure for the treatment of drug-resistant ventricular arrhythmias associated with syncope or ventricular tachycardia. Surgery can also be applied for the treatment of complex arrhythmias and arrhythmias associated with other heart disease, whether congenital or acquired, that require an operation.

REFERENCES