Control of Intractable Ventricular Tachycardia by Coronary Revascularization

By Roger R. Ecker, M.D., Charles B. Mullins, M.D., John C. Grammer, M.D., William J. Rea, M.D., and James M. Atkins, M.D.

SUMMARY

Ventricular tachycardia (VT) is an arrhythmia that has an ischemic origin in up to 74% of cases and results in a 42 to 67% mortality when it is recurrent and paroxysmal. Present therapy is aimed at suppression of the abnormal rhythm but does not alter the prognosis of the underlying ischemic heart disease. A new concept of treatment of VT is introduced that is based on direct coronary revascularization by the aorta to coronary, saphenous vein-bypass technique. The method was successfully applied in a 61-year-old man who developed episodes of VT 2 months after myocardial infarction. Maximal medical therapy in a coronary care unit for 26 days did not abolish the arrhythmia which occurred as frequently as seven times per hour. Coronary angiography and aortocoronary bypass grafting were done when the patient developed electrocardiographic and enzyme evidence of subendocardial myocardial infarction and symptoms of cerebral ischemia. The patient remains free of arrhythmia 1 year later, and his exercise capability is now normal for his age. Follow-up coronary angiography is presented. Coronary revascularization has been shown to abolish angina pectoris. This report demonstrates that aortocoronary bypass grafting can abolish an arrhythmia of ischemic origin. When persistent or recurrent VT fails to respond to all medical therapy, direct coronary revascularization should be considered to control this ischemic arrhythmia.

Additional Indexing Words:
Aortocoronary-saphenous vein bypass
Coronary artery disease
Coronary artery surgery
Ventricular aneurysm
Myocardial infarction

Coronary arteriography
Myocardial ischemia

Ventricular tachycardia is a serious arrhythmia which is usually associated with ischemic heart disease. Antiarrhythmic drugs,1,2 electrical countershock,3 and electrical pacing4-6 are successful in controlling the majority of episodes, but recurrence is common, and the mortality is increased compared to patients with comparable conditions but without this arrhythmia.7 Conventional therapy is aimed at suppressing the abnormal rhythm and thereby preventing the adverse hemodynamic effects8,9 of ventricular tachycardia, but it does not correct the underlying ischemic heart disease which is responsible for the increased mortality.3,5,7,9

This is a report of a new method of treatment of refractory ventricular tachycardia based on surgical relief of the associated myocardial ischemia. Successful clinical application of the method in a patient is illustrated.

Report of Case

The patient, a 61-year-old white male, was in excellent health until November 1969, when he suffered an acute myocardial infarction and was hospitalized at another hospital. His course was...
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Figure 1

Synopsis of course in the coronary care unit and treatment for recurrent ventricular tachycardia. This represents a minimum frequency of episodes of VT since they were often too frequent to be recorded as individual arrhythmias.

complicated by thrombophlebitis and by frequent premature ventricular contractions (PVCs) requiring procainamide and diphenylhydantoin. After 6 weeks of hospitalization he was discharged with instructions to take procainamide 2 g/day and diphenylhydantoin 400 mg/day.

In January 1970 he had a syncopal episode and was readmitted to the coronary care unit when it was discovered that he had recurrent episodes of ventricular tachycardia (VT). This arrhythmia was treated with antiarrhythmic drugs, singly and in combinations, to the point of toxicity while overdrive transvenous temporary pacing was being used to correct the VT. While the number of episodes of VT was temporarily decreased, he would revert to his previous status in spite of continuation of the same regimen.

Episodes of VT continued; these sometimes lasted several minutes and occurred as often as seven times an hour. Serial electrocardiograms and serum enzyme levels did not show evidence of a myocardial infarction. A pulmonary angiogram was taken to rule out the possibility of pulmonary embolism, but no evidence of embolism was found.

On January 26 after 12 days of hospitalization in the coronary care unit, the patient underwent cardiac catheterization. A left ventriculogram revealed akinesia of the left ventricular apex. Left ventricular end-diastolic pressure was 12 mm Hg. Coronary angiography demonstrated 75% stenosis of the distal part of the right coronary artery and 90% stenosis of the marginal branch of the left circumflex artery.

On February 2, 1970, the patient was transferred to Parkland Memorial Hospital. Physical examination at that time revealed an apprehensive, alert white male with a tachycardia. The chest was clear, the heart rate rapid, but no gallops or murmurs were heard. Peripheral
pulses were present and equal. There was no edema or cyanosis. Chest X-rays revealed minimal cardiomegaly. Admission ECG revealed ventricular tachycardia, and the patient was given a bolus of lidocaine intravenously. Another temporary pacemaker electrode was placed percutaneously and pervenously in the right ventricle. Pacing at a rate of 110 to 130/min reduced the number of episodes of VT but did not abolish them. The pacemaker was also used to overdrive pace by exceeding the ventricular rate, sometimes requiring a pacer rate of 180 to capture the ventricle. The pacemaker was then gradually turned down to a rate of 100 to 110. Various combinations of drugs were pushed to toxicity without further improvement (fig. 1). On February 6, for the first time chest pain developed during an episode of tachycardia. The electrocardiogram revealed changes compatible with an inferior subendocardial infarction (fig. 2). By the following day the serum glutamic oxalacetic transaminase (SGOT) had risen to 86 (normal, 0–41 Karmen units) and lactic acid dehydrogenase (LDH) to 480 (normal, 42–130 milliunits/ml). Following this event, episodes of ventricular tachycardia became more refractory to treatment and the patient required direct-current shock cardioversion as well as pacing and drugs (fig. 3). The patient continued to have runs of ventricular tachycardia with a worsening of his general mental and physical status. He began to develop “graying-out spells” and slowed mental processes during his episodes of tachycardia, indicating cerebral ischemia. It was felt that the patient was deteriorating in spite of maximum medical therapy, and it was decided to submit him to operation for coronary revascularization.

On February 9, 1970, through a median sternotomy incision and while using total cardiopulmonary bypass, a saphenous vein graft was placed from the ascending aorta to the distal part of the right coronary artery and another saphenous vein graft was placed from the ascending aorta to the obtuse marginal branch of the left circumflex artery. The interior of both coronary vessels contained plaques, and palpation of the coronary arteries revealed extensive involvement of the proximal portion of the circumflex artery, an indurated lesion in the upper one third of the anterior descending coronary artery, and some beadlike thickenings of the posterior descending branch. There was a fibrotic, thinned-out area on the diaphragmatic surface of the right ventricle which bulged during systole. This was plicated with horizontal mattress sutures and oversewn but was not resected. Temporary epicardial atrial and ventricular pacing leads were placed.

Postoperatively the patient did well with only one unsustained episode of ventricular tachycardia 2 hours after operation. He had occasional PVCs which were easily controlled with diphenylhydantoin. He was discharged from the hospital 14 days after operation on the same dose of diphenylhydantoin. Postoperative electrocardiogram is demonstrated in figure 2.

Follow-up cardiac catheterization and coronary angiography were performed on April 20, 1970. The bypass graft to the right coronary artery was patent with good filling of the distal vessel. The left coronary artery was visualized with both the anterior descending and circumflex branches being seen, but the obtuse marginal branch did not fill. The aorto-left circumflex bypass graft could not be visualized, and it was presumed to be occluded. The left ventriculogram and left ventricular end-diastolic pressure were unchanged from preoperatively.

In February 1971, one year after operation, the patient had a graded bicycle ergometer test with loads of 300 kp-m, 450 kp-m, and 600 kp-m. The patient exercised for 5 min at 600 kp-m and attained a heart rate of 150 (resting 70) and a respiratory rate of 36; the test was discontinued because of fatigue. Results were interpreted as being at the lower limits of normal work capacity for the patient’s age and weight. The patient had

**Figure 2**

Electrocardiogram between episodes of VT immediately before and after operation.

**Figure 3**

Rhythm strips from a monitor lead showing ventricular tachycardia. Cardioversion and a paced rate of 120 did not entirely suppress the ventricular ectopic rhythm.
flat T waves at rest with 1 to 4 PVCs/min; there were no ST or T changes with exercise and the PVCs disappeared at the lowest workload. The patient was receiving no medication at this time.

Because of the PVCs noted at rest at the time of the exercise test, an 8-hour continuous electrocardiogram was performed using a Holter recorder. The continuous electrocardiogram revealed normal sinus rhythm with 1 to 4 PVCs/min when his heart rate was below 85 but no PVCs at heart rates above 90.

The patient was asymptomatic at this time and no further treatment or precautions were deemed necessary.

Discussion

Ventricular tachycardia is associated with ischemic heart disease in as high as 74% of cases.7,10 In early series before current therapy was developed, recurrent paroxysmal VT carried a 42% mortality when associated with coronary artery disease and a 67% mortality when myocardial infarction was associated.7 The impact of currently used medical therapy on mortality cannot be determined, but in a series of patients in a coronary care unit, 23% of patients with at least one episode of ventricular tachycardia developed ventricular fibrillation.9 There was a 57% mortality for ventricular fibrillation in this series in the hospital and on follow-up extending to 7 months, giving a calculated mortality of 13% for ventricular tachycardia in this group of patients. Of seven patients with refractory recurrent ventricular tachycardia recently reported,5 three died.

The reported incidence of ventricular tachycardia varies with the method of detection. Before the advent of modern coronary care, monitoring, and treatment, mortality was 80%,11,12 indicating that only prolonged or recurrent attacks were recognized. Using alarm-type monitors, the incidence was found to be 11%.2 When patients with acute myocardial infarction were continuously monitored, 62 of 273 patients developed ventricular tachycardia.9

A wide variety of therapeutic modalities have been reported. Lidocaine was effective in 19 of 29 patients with VT and other ventricular arrhythmias.1 Direct-current countershock,4 electrical pacing,5,15 propranolol,6,18 and other antiarrhythmic drugs have been successful in treating many cases of ventricular tachycardia. Drug therapy can be expected to convert 78% of cases of VT2 to normal rhythm and may prevent recurrence. Closed-chest external electrical shock may convert up to 97% of episodes but requires anesthesia for the conscious patient and does not prevent recurrent episodes. Hyperbaric oxygen was utilized in one case of refractory ventricular tachysystole with beneficial results; however, the patient died 3 months later of recurrent myocardial infarction. Two cases have been reported in which VT was treated by resection of a left ventricular aneurysm. In the first case4 an emergency resection for an aneurysm was performed on a 60-year-old man with intractable ventricular tachycardia, and the patient had no further episodes of VT. However, he had 18 episodes in the postoperative period requiring electrical countershock. He was reported free of arrhythmias 13 months after surgery. The second patient15 had an anterior ventricular aneurysm removed for ventricular tachycardia which was controlled with difficulty with quinidine. Postoperatively the quinidine therapy was discontinued without recurrence of the arrhythmia.

Ventricular tachycardia has been produced experimentally by ligation of a coronary artery and clinically is associated with ischemic heart disease. It would seem rational, therefore, to treat this arrhythmia by increasing the total blood flow to the heart. It has been speculated that abnormal rhythms arise in the ventricle at the junction between well-vascularized and poorly vascularized myocardium, but this has never been conclusively demonstrated.

The termination of multiple episodes of ventricular tachycardia in our patient could have been brought about by several mechanisms. The most likely explanation is that better blood flow to the myocardium and relief of ischemia resulted in termination of the episodes. Since the bypass graft to the marginal circumflex artery was apparently occluded at restudy 3 months later, it is possible that thrombosis occurred in this vessel and produced a myocardial infarction.
which resulted in abolition of the arrhythmia. To fulfill this hypothesis, this infarction would have had to occur during or immediately after operation. This possibility is not corroborated by electrocardiographic changes and is at variance with the patient's smooth postoperative clinical course. The small right ventricular aneurysm on the diaphragmatic surface of the heart was repaired by plication without removal, and since ventricular aneurysmectomy in one case has resulted in termination of VT, this could be a possible mechanism. Against this is the fact that the aneurysm was confined to the right ventricle, was not excised, and the patient did not have the gradual resolution of arrhythmia as in the patient whose case was previously reported. The patient's VT was of the right bundle-branch pattern suggesting that it arose from a focus in the left ventricle. Furthermore, during electrical pacing using a right ventricular electrode, the electrocardiographic pattern was of the left bundle type, which is further evidence that the irritable focus was in the left ventricle.

Myocardial revascularization achieved by means of the saphenous vein-bypass technique brings about the relief of angina pectoris and may improve ventricular function. It is hoped that it will also prevent recurrent myocardial infarction and prolong life expectancy. Our experience suggests that myocardial revascularization by the saphenous vein-bypass technique is a rational and effective method of relieving ventricular arrhythmias of ischemic origin in patients unresponsive to prolonged medical therapy. The transition of our patient from multiple life-threatening episodes of ventricular tachycardia before operation to complete freedom from this rhythm after operation was very dramatic. Further evidence for the validity of this approach is needed, and the ability of this method of treatment to terminate this arrhythmia deserves further clinical trial. An aorta to coronary artery shunt made with a saphenous vein graft might also be of value in selected patients with demonstrated important stenoses of major coronary arteries when medical therapy fails to control dangerous ventricular arrhythmias of ischemic origin such as recurrent ventricular fibrillation and frequent multifocal ventricular premature contractions in the absence of acute transmural infarction.

References
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