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Electrode Catheter Ablation of Refractory Focal Ventricular Tachycardia

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Localized, high energy, direct current intracardiac shocks effectively prevented recurrent ventricular tachycardia in one patient whose arrhythmia originated in the right ventricular outflow tract, and in two patients with ventricular septal tachycardia after myocardial infarction.

Recurrent ventricular tachycardia may prove refractory to conventional drug therapy, requiring aggressive management with investigational antiarrhythmic drugs, pacing techniques or attempted operative ablation (1-4). An alternative therapy of localized tissue destruction by focal intracardiac direct current shocks was successfully performed in three patients with life-threatening, recurrent ventricular tachycardia.

Case Descriptions

Case 1

A 32 year old woman first presented during pregnancy at age 25 with recurrent, symptomatic sustained and nonsustained ventricular tachycardia. Multiple daily episodes of ventricular tachycardia at a rate of 140 to 200 beats/min refractory to conventional antiarrhythmic drugs given alone and in combination continued after delivery. In 1979, trials of encainide and aprindine administered at a university center proved ineffective. During subsequent hospitalization, ventricular tachycardia recorded by telemetry degenerated to ventricular fibrillation requiring resuscitation with direct current countershock.

Electrophysiologic study and cardiac catheterization. An invasive electrophysiologic study including acute drug testing was performed at a second referral center identifying “focal” ventricular tachycardia that appeared to originate from within the right ventricular outflow tract as determined by right and left ventricular electrode-catheter mapping. The electrophysiologic features were unusual.

Several nonsustained episodes of ventricular tachycardia lasting a few seconds to several minutes occurred spontaneously, but they could never be induced by programmed stimulation or burst pacing of the right ventricular apex, outflow tract or left ventricle. All episodes were initiated with a QRS complex identical to subsequent QRS complexes during ventricular tachycardia.

Isoproterenol (Isuprel) infusion clearly increased the frequency of episodes of spontaneous tachycardia. Somewhat unexpectedly, programmed ventricular stimulation of the right ventricular apex repeatedly terminated episodes of ventricular tachycardia. Consequently, it was concluded that this predominantly automatic ventricular tachycardia had “mixed features of automaticity and reentry.”

An M-mode echocardiogram suggested mild mitral valve prolapse. However, two-dimensional echocardiography and right and left heart catheterization including biventricular angiography revealed no specific abnormality. The coronary arteries were normal. An endomyocardial biopsy revealed no evidence of occult myocarditis or other diagnostic abnormality.

Medical and surgical treatment. Subsequent trials of digoxin, verapamil, mexiletine, tocainide and multiple beta-receptor blocking agents in combination with conventional drugs failed to prevent repeated daily episodes of ventricular tachycardia, some associated with syncope and near syncope. After a second electrophysiologic study with identical findings, surgical exploration and intraoperative mapping of both right and left ventricles were performed with localization of the earliest epicardial and endocardial activation to the right ventricular outflow tract. Consequently, adjacent myocardium and subendocardium were resected from the right ventricular outflow tract. Left ventricular epicardial mapping immediately to the left of the ventricular septum but adjacent to the right ventricular outflow tract also identified relatively early activation, occurring within...
5 ms of the QRS onset and electrocardiogram from the right ventricular outflow site. Because of these mapping uncertainties, it was elected to also resect an area of high septal endocardium through a paraseptal left ventriculotomy. The procedure failed to prevent morphologically identical recurrent ventricular tachycardia and resulted in surgically acquired right bundle branch block with left anterior fascicular block. A late postoperative electrophysiologic study identified a normal HV interval and other findings during ventricular tachycardia identical to those observed preoperatively. The late postoperative course was complicated by recurrent pericarditis that was unresponsive to nonsteroidal antiinflammatory drugs and ultimately required prednisone therapy. Resultant labile blood potassium levels appeared to increase the frequency and severity of recurrent ventricular tachycardia with multiple daily episodes lasting 30 seconds to several minutes.

**Electrode catheter ablation.** In 1981, an episode of collapse and shock secondary to ventricular flutter at a rate of 250 to 300 beats/min required outpatient resuscitation with direct current countershock. Subsequent trials of oral bretylium tosylate, amiodarone and intramuscular lidocaine were ineffective in suppressing recurrent arrhythmia. In August 1982, an invasive electrophysiologic study including right and left ventricular endocavitary mapping was repeated. Bipolar and unipolar electrograms recorded during spontaneous ventricular tachycardia again localized the site of earliest activation to the right ventricular outflow tract (Fig. 1 and 2). Utilizing pentothal anesthesia and a standard defibrillator, three 300 J shocks were delivered through the tip electrode of a standard 7 French quadripolar electrode catheter (U. S. Catheter and Instrument) positioned adjacent to the site of earliest endocardial activation (Fig. 3 and 4). The discharges were successively directed toward a paddle applied to the back, lateral chest wall and anterior chest wall in a fashion similar to that initially described by Gonzales and Scheinman et al. (5,6) for His bundle ablation. Sinus rhythm continued without ventricular arrhythmia immediately after shocks, and initial marked ST segment elevation gradually returned to baseline over a few minutes.

**Follow-up.** The postprocedure course was uneventful and free of ventricular arrhythmia. The total serum creatine kinase reached 1,194 mlU/ml with MB fraction reported as "present" but not quantitated. While receiving no antiarrhythmic drugs, the patient remained asymptomatic and free of ventricular arrhythmia and tachycardia for 3 months. During this time, a general anesthetic was administered for emergency appendectomy that was uncomplicated. During the fourth postprocedure month, occasional premature ventricular complexes were documented by ambulatory monitoring but the patient remained asymptomatic. Disopyramide phosphate was prescribed with complete suppression of arrhythmia. The patient has remained free of sustained ventricular arrhythmia or tachycardia for a 9 month follow-

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**Figure 1.** Case 1. Heart rhythm at rest immediately before ablation therapy. Two *top tracings* are intracardiac recordings. F, I and V1 = surface electrocardiographic leads aVF, I and V1; HBE = recording from region of His bundle; HRA = high right atrium.

**Figure 2.** Composite recording of three beats of ventricular tachycardia with comparative unipolar electrograms from the right ventricular apex, midseptum and outflow tract. Reference tracings are from the His bundle region (HBE) and surface electrocardiographic leads V1, I and aVF. Note the onset of the right ventricular outflow electrogram 50 ms before the onset of the surface QRS electrogram and that from the His bundle region.
Figure 3. Case 1. Cinefluoroscopic frame illustrating electrode catheter positioning and back paddle from conventional defibrillator. Arrow at tip of quadripolar catheter indicates electrode of discharge. A bipolar catheter is positioned near the right ventricular apex for temporary pacing and a tripolar catheter is positioned across the tricuspid valve for recording from the region of the His bundle.

up period. A recent outpatient ambulatory electrocardiographic recording documented the presence of a single premature ventricular complex during a 24 hour period.

Case 2

A 65 year old man with prior anteroseptal myocardial infarction complicated by congestive heart failure and recurrent ventricular tachycardia underwent operative mapping with myocardial and endomyocardial resection in 1980. He remained asymptomatic for approximately 12 months followed by recurrent sustained ventricular tachycardia requiring rehospitalization. Episodes of ventricular tachycardia continued with increasing frequency including multiple daily episodes of sustained arrhythmia producing syncope and near syncope despite drug therapy with combinations of procainamide, quinidine, disopyramide, phenytoin, mexiletine and amiodarone.

Repeat left ventricular angiography showed a severely dilated and hypocontractile left ventricle with monoplane-derived ejection fraction of 18%. The left anterior descending coronary artery was completely occluded. The right and circumflex coronary arteries were normal. At repeat electrophysiologic study, burst pacing unreliably terminated the tachycardia with acceleration of ventricular tachycardia on some occasions and induction of ventricular fibrillation on others.

Eight weeks after initiation of amiodarone therapy, this patient was readmitted with incessant ventricular tachycardia and hypotension. Over the course of 1 week, combinations of intravenous lidocaine and bretylium reduced the frequency of ventricular tachycardia episodes but direct current shock and burst pacing were required repeatedly.

Catheter ablation procedure. In November 1982, right and left ventricular mapping was performed using multiple electrode catheters. The earliest site of endocardial activation during induced ventricular tachycardia was localized to a high left ventricular septal position using bipolar recordings (Fig. 5). Pace-mapping also confirmed the septal location by reproducing a paced QRS configuration similar to that of spontaneous ventricular tachycardia (Fig. 6). Pentothal anesthesia was administered. Because of uncertainties about which electrode of the bipolar pair was more closely adjacent to the site of origin of ventricular tachycardia, three 300 J shocks were delivered through one electrode and two 300 J shocks were delivered through the second electrode, discharging to an anterior chest paddle (Fig. 7). Immediately after the procedure, it was not possible to reinduce sustained ventricular tachycardia by pacing or programmed ventricular stimulation from the right or left ventricle.

Follow-up and repeat catheter ablation procedure. The postprocedure total serum creatine kinase reached 6,000
Figure 5. Case 2. Simultaneous recording from four ventricular sites during induced sustained ventricular tachycardia. Sites A, B and D are from the left ventricle; RV indicates a site near the right ventricular apex. The arrow in D indicates early baseline deflection of bipolar electrogram recorded from the high left ventricular septal position. This corresponds with electrode pair three and four of the quadripolar electrode catheter as depicted by arrows on Figure 7. Note the comparable early activity from site B, which represents the closely adjacent proximal pair of electrodes from the second quadripolar left ventricular catheter.

miU/ml, but creatine kinase MB fraction was reported as absent. The subsequent course was uncomplicated, although single ventricular ectopic beats continued without sustained repetitive beats. However, in consideration of the patient’s extremely poor left ventricular function, frequent ventricular arrhythmia and previous clinical course, it was elected to continue a relatively modest antiarrhythmic regimen at discharge including phenytoin, 100 mg three times a day, plus mexiletine, 100 mg four times a day. His clinical course changed dramatically and he remained asymptomatic and free of sustained ventricular tachycardia for 2 1/2 months. However, at that time ventricular tachycardia recurred, requiring hospitalization for repeat direct current countershock and drug regimen adjustment. The arrhythmia again proved refractory, requiring transfer to our institution where an electrophysiologic study and focal catheter ablation procedures. An invasive electrophysiologic study was performed during which ventricular tachycardia at a rate of 150 beats/min recurrently required direct current countershock. With these exceptions, the patient remained remarkably free of ventricular arrhythmia or tachycardia during several days of observation before his death from low output congestive heart failure and anuria.

At autopsy, the left ventricle and septum were extensively scarred, compatible with the patient’s prior myocardial infarction and surgical procedure. No additional specific abnormalities were observed and, in particular, there were no gross or microscopic findings tending to localize a site of electrical injury.

Case 3

A 60 year old man underwent placement of multiple saphenous vein bypass grafts to the coronary arteries and an apical aneurysmectomy in 1978. He remained well until February 1983 when there was onset of sustained ventricular tachycardia with near syncope requiring transthoracic direct current cardioversion. Subsequently hospitalized for 3 weeks, he had multiple episodes of sustained ventricular tachycardia despite treatment with procainamide, quinidine, verapamil, digoxin and mexiletine, given alone and in combination. A radionuclide-determined ejection fraction of 26% was reported.

After transfer to our institution, sustained ventricular tachycardia at a rate of 150 beats/min recurred despite a combination of oral antiarrhythmic drugs plus intravenous procainamide and lidocaine infusions. Transthoracic countershock was again required. Subsequent left heart catheterization recorded a mildly elevated left ventricular end-diastolic pressure. The left ventricle was severely dilated with akinesia of the entire anterolateral wall and apex. The proximal left anterior descending coronary artery, proximal circumflex coronary artery and distal right coronary artery were totally occluded. A vein graft to the left anterior descending artery was totally occluded, but a patent vein graft perfused the posterior descending branch of the right coronary artery. Two additional grafts provided flow to two obtuse marginal branches of the circumflex circulation.

The patient was not considered a candidate for an operative intervention because of extremely poor left ventricular function in the setting of earlier coronary bypass surgery and the absence of currently significant coronary obstructive lesions. The patient declined to receive further investigational antiarrhythmic drugs.

Electrophysiologic study and focal catheter ablation procedures. An invasive electrophysiologic study was performed during which ventricular tachycardia at a rate of 180 beats/min with left bundle branch block surface QRS configuration was easily induced. This tachycardia was morphologically identical to all recorded episodes of spontaneous ventricular tachycardia and could be terminated by...
burst ventricular pacing. Polymorphic ventricular tachycardia was never identified. Extensive right and left ventricular endocavitary mapping was performed during induced ventricular tachycardia (Fig. 8). All left ventricular sites were activated late relative to the onset of the ventricular tachycardia QRS complex. However, the midright ventricular septum was activated early with endocardial depolarization occurring 10 to 20 ms before the onset of the surface QRS complex (Fig. 9). Mapping of adjacent septal sites always recorded later activation.

With pentothal anesthesia, two 300 J shocks were delivered from the tip electrode and one 300 J shock was delivered from the second electrode of the tip pair directed to a posterolateral chest paddle. Sinus pauses resulted exceeding 3 seconds but were terminated by temporary ventricular pacing. On one occasion, spontaneously terminating ventricular fibrillation occurred (Fig. 10). After recovery of regular sinus rhythm, it was apparent that complete right bundle branch block had been acquired. At that time, extensive programmed right ventricular stimulation was again performed using single and paired stimuli coupled to sinus rhythm and multiple paced ventricular rates without induction of repetitive ventricular beats.

After the procedure, the patient’s 12 lead electrocardiogram continued to demonstrate right bundle branch block. His total serum creatine kinase reached 1,455 mIU/ml with MB band reported as present but not quantitated. The serum glutamic oxaloacetic transaminase reached 200 mIU/ml (normal 40). He remained free of arrhythmia and was discharged 48 hours later, taking quinidine sulfate as his only medication.

**Follow-up.** In a subsequent 7 week follow-up period, the patient remained asymptomatic and free of recurrent ventricular tachycardia, representing a dramatic change compared with his preablation course. At that time, he was electively readmitted to the hospital for a second invasive electrophysiologic study. Right bundle branch block was now absent on the electrocardiogram, which was identical to the preablation recording. Extensive programmed stimulation of the right ventricular apex and outflow tract was

**Figure 6.** Case 2. Comparison of configuration of spontaneous ventricular tachycardia (VT) (left side of tracing) with paced QRS configuration from left ventricular septal site D.

**Figure 7.** Case 2. Cinefluoroscopic frame in the left anterior oblique projection illustrating electrode catheter positioning and presence of chest paddle. A bipolar right ventricular apex catheter is present (below). Arrows indicate electrode pair D, utilized for intracardiac discharge (see Fig. 5).
performed utilizing single and paired stimuli coupled with intrinsic and multiple paced ventricular rhythms. Except for a rare $V_3/V_4$ phenomenon, it proved impossible to induce repetitive beats and no sustained arrhythmias resulted.

**Discussion**

Ablation of the His bundle by high energy intracardiac direct current shocks has been reported in animals (5) and more recently in patients (6,7). Animal pathologic studies and initial clinical observations suggest that localized and permanent tissue destruction results. At our institution, a 2 year experience with His bundle ablation served as the precedent for attempted ablation of focal arrhythmia.

**Application of electrode catheter ablation of focal ventricular tachycardia.** Prevention of recurrent ventricular tachycardia with its potential for electrophysiologic deterioration, hemodynamic collapse and sudden death remains a major clinical challenge. Consequently, the therapeutic potential of electrode catheter ablation of arrhythmogenic foci seems great. Its attraction lies in procedural simplicity, relative independence of drug therapy and avoidance of a major surgical intervention.

Theoretically, a focal tachycardia arising from a small and circumscribed area in the absence of extensive myocardial scarring would be most susceptible to ablation techniques. The first patient appears to represent successful management of such an arrhythmia. However, her clinical and electrophysiologic features are distinctly uncommon and are observed in a small minority of patients referred to an elec-

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**Figure 8.** Case 3. Cinefluoroscopic frame illustrating electrode catheter position for ablation of ventricular septal tachycardia. The large bipolar catheter with the tip positioned in the right ventricular apex (below) is a specialized catheter (Medtronic model 6880) utilized for intracardiac cardioversion of ventricular tachycardia by low energy discharges. A tripolar catheter is positioned adjacent to the His bundle. Two quadripolar catheters have been passed in a retrograde manner across the aortic valve and are positioned at dissimilar sites in the left ventricle for endocavitary mapping. A single quadripolar electrode catheter is positioned within the right ventricle with the tip electrode against the midseptum immediately adjacent to the earliest site of endocardial activation during ventricular tachycardia (arrow). Shocks were delivered from this and the adjacent electrode directed to a posterolateral chest paddle.

**Figure 9.** Case 3. Simultaneous bipolar intracardiac recordings from four different locations during sustained ventricular tachycardia. The earliest endocardial activation recorded (arrow) was at a mid-right ventricular septal (RVS) location preceding all other right and left ventricular sites. LV = electrograms from two dissimilar left ventricular sites; RVA = right ventricular apex; $V_1$, I, F are standard surface electrocardiographic recordings from leads $V_1$, I and aVF.
trophysiologic center for investigation of ventricular tachy­
cardia.

Recurrent sustained reentrant ventricular tachycardia oc­
curring after myocardial infarction is a much more frequent
clinical problem, but one that may be less suitable for cath­
er ablation techniques. Although localized microreentrant
circuits might be injured by intracardiac shocks, the pres­
ence of diffuse myocardial scarring may also provide an
anatomic substrate for multiple and larger reentrant circuits
less suited for focal ablation. Additionally, left ventricular
mapping and catheter stabilization using currently available
electrode catheters are more technically difficult than right
ventricular catheterization. Despite these considerations, the
effective ablation of ventricular tachycardia in Cases 2 and
3 does suggest that the technique can be applied to theo­
retically less focal and more complex tachycardias. Clearly,
this concept merits further controlled investigation.

Many important questions remain unanswered by these
three cases. The absence of creatine kinase MB fraction in
Patient 2 probably represents laboratory inaccuracy but does
suggest that a fairly limited myocardial injury resulted. The
extent of tissue destruction inflicted by or required for focal
ablation remains uncertain and merits further investigation.
Many technical concerns also require evaluation, including
energy characteristics and electrode configuration required
to safely create a controlled injury.

Implication. Until the safety, efficacy and short- and
long-term benefits of this new and novel therapy can be
further characterized, its performance should be limited to
a small number of centers with experienced electrophys­

Figure 10. Case 3. A 300 J shock is delivered during ventricular
tachycardia with discharge from the tip electrode of a quadripolar
electrode catheter positioned against the midright ventricular sep­
tum. Induced ventricular fibrillation terminated spontaneously after
5 seconds. RV2 = right ventricular electrogram from an additional
right ventricular site. Other abbreviations as in Figure 9.

ology. However, the potential impact of this technique
for management of complex and otherwise refractory ven­
tricular arrhythmias appears great.

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