Emergency Intracardiac Defibrillation for Refractory Ventricular Fibrillation During Routine Electrophysiologic Study

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Ventricular fibrillation refractory to cardiopulmonary resuscitation including multiple transthoracic defibrillations occurred in four patients during 1,215 consecutive ventricular tachycardia induction studies. A technique of emergency intracardiac defibrillation for management of refractory ventricular fibrillation is described. In four patients, stable monomorphic ventricular tachycardia (320 to 570 ms cycle length) was induced during the study and overdrive ventricular pacing resulted in ventricular fibrillation. These patients did not respond to prompt transthoracic defibrillations (5 to 15 attempts/patient) and cardiopulmonary resuscitation, including antiarrhythmic therapy. As a last resort, intracardiac defibrillation was performed with use of a previously inserted standard right ventricular quadripolar catheter as cathode and a posterior skin patch as anode. High energy intracardiac defibrillation pulses (100 to 500 J) delivered from a standard defibrillator successfully terminated each arrhythmia.

Intracardiac defibrillation is technically simple and appears effective in terminating refractory ventricular fibrillation in the electrophysiology laboratory. However, further research is necessary to determine the safety and efficacy of this technique, as well as potential applications in other emergency settings.

Methods

Study patients. As part of a routine ventricular tachycardia induction study, two 6F standard quadripolar catheters (USCI) were inserted into the right femoral vein and positioned in the right atrium and right ventricle. From January 1, 1987 to November 1, 1990, ventricular fibrillation refractory to cardiopulmonary resuscitation and at least four transthoracic shocks occurred in four patients during 1,215 consecutive VT induction studies. As a last resort, intracardiac defibrillation was performed in accordance with our standard written informed consent protocol, which permits the use of "any other operation(s) or procedure(s) during the specified operation that his/her [in the physician's] judgement may dictate for my [the patient's] well being."

Intracardiac defibrillation. This was performed with the distal pole of a standard 6F right ventricular quadripolar catheter as cathode and a posterior skin patch as anode. The distal electrode and patch were attached (with additional connecting cables) to a standard defibrillator (Physio-Control, LifePak 6). The intracardiac defibrillation pulses delivered were not synchronized. Figure 1 shows a schematic of this method, which is identical to that used during transcatheter DC ablation at our institution.

Results

Patient characteristics (Table 1). The four patients who underwent intracardiac defibrillation had a mean age of 65 ± 5 years (range 54 to 79) and all had severe coronary artery disease with left ventricular dysfunction (mean ejection fraction 29 ± 2%). Two of these patients had chronic obstructive pulmonary disease and all were obese (92 to 130 kg). All were treated with antiarrhythmic agents (amio-
darone plus mexiletine in two patients, procainamide alone in one and procainamide and mexiletine in one). In all four patients a stable, monomorphic ventricular tachycardia (320 to 570 ms cycle length) was initially induced with programmed electric stimulation. In all cases overdrive right ventricular pacing accelerated the stable ventricular tachycardia to ventricular fibrillation.

Traditional management. This included multiple transthoracic defibrillations, the anterior patch is promptly disconnected and the distal pole of a right ventricular catheter is attached to the defibrillator. High energy defibrillation pulses are then delivered from the right ventricular catheter to the posterior patch to terminate ventricular fibrillation. RV = right ventricular.

Intracardiac shocks for defibrillation. Patient 1 received intracardiac shocks approximately 45 min after onset of cardiopulmonary resuscitation. He required two intracardiac shocks (200 and 500 J, respectively) to terminate ventricular fibrillation. Although this patient remained in sinus rhythm, he died 1 h later of cardiac failure (despite intraaortic balloon counterpulsation and pressor support). After 10 min of ventricular fibrillation Patient 2 received a single intracardiac shock (300 J) that successfully terminated the arrhythmia.

Figure 2 shows the electrocardiographic (ECG) sequence of events leading to intracardiac defibrillation in Patient 3. After seven transthoracic defibrillation attempts proved unsuccessful, a single 100 J intracardiac shock converted ventricular fibrillation to asystole. Right ventricular pacing was performed from the same catheter, and normal arterial pressure was maintained until sinus rhythm returned 1 min later. Patient 4 required two intracardiac shocks (100 and 200 J, respectively) delivered 10 min after the onset of ventricular fibrillation to terminate the arrhythmia.

Serial ECGs, creatine kinase (CK) and CK isoenzymes were obtained during the 24 h after the procedure in the three survivors. The total CK levels were all markedly elevated (4,110 to 6,520 IU/liter), with CK MB fractions of 44 to 86 IU/liter. Serial ECGs demonstrated nonspecific ST-T wave changes after intracardiac defibrillation. There were no evolutional ECG findings of acute transmural myocardial infarction.

At a mean follow-up period of 9.3 ± 6.8 months (23, 3 and 2 months, respectively), the three survivors of the procedure are alive and well and without apparent adverse sequelae from their intracardiac defibrillation.

**Discussion**

Ventricular fibrillation during electrophysiologic study. Patients undergoing electrophysiologic study for ventricular arrhythmias rarely develop ventricular fibrillation refractory to multiple transthoracic defibrillations. The risk of death during ventricular tachycardia induction studies is very low because prompt termination of the arrhythmia by pacing or DC shock is almost always successful (3). Horowitz (4)

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**Table 1. Patient Characteristics**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (yr)</th>
<th>Gender</th>
<th>Diagnosis</th>
<th>Weight (kg)</th>
<th>EF (%)</th>
<th>Critical Medication</th>
<th>VT Cl. (ms)</th>
<th>Shocks no.:</th>
<th>Time to Intracardiac Shock (min)</th>
<th>CK (IU/liter)</th>
<th>CK MB (IU/liter)</th>
<th>Follow-Up</th>
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<tr>
<td>1</td>
<td>54/M</td>
<td></td>
<td>COPD/IMI</td>
<td>120</td>
<td>30</td>
<td>P, Mex, A, Brei</td>
<td>350</td>
<td>15</td>
<td>2</td>
<td></td>
<td></td>
<td>Later died of heart failure</td>
</tr>
<tr>
<td>2</td>
<td>64/F</td>
<td></td>
<td>ASMI</td>
<td>118</td>
<td>35</td>
<td>A, Mex, Lido</td>
<td>570</td>
<td>10</td>
<td>1</td>
<td>4,110</td>
<td>49</td>
<td>A/W, 23 mo</td>
</tr>
<tr>
<td>3</td>
<td>79/M</td>
<td></td>
<td>COPD/ASMI</td>
<td>92</td>
<td>25</td>
<td>A, P</td>
<td>120</td>
<td>7</td>
<td>1</td>
<td>6,520</td>
<td>65</td>
<td>A/W, 3 mo</td>
</tr>
<tr>
<td>4</td>
<td>63/M</td>
<td></td>
<td>ASMI</td>
<td>128</td>
<td>25</td>
<td>A, Mex, None</td>
<td>320</td>
<td>5</td>
<td>2</td>
<td>5,740</td>
<td>86</td>
<td>A/W, 2 mo</td>
</tr>
</tbody>
</table>

A = amiodarone; ASMI = anteroseptal myocardial infarction; A/W = alive and well; Brei = bretylium; CK = creatine kinase; Cl. = cycle length; COPD = chronic obstructive pulmonary disease; EF = ejection fraction; F = female; IMI = inferior myocardial infarction; IV = intravenous; Lido = lidocaine; M = male; Mex = mexiletine; P = procainamide; Trans = transthoracic; VT = ventricular tachycardia; — = determination not performed.
Figure 2. Patient 3. A, Simultaneous recordings of surface leads V₁, I, II, aVF and intracardiac recordings from the high right atrium (HRA). A hemodynamically stable ventricular tachycardia at a cycle length of 320 ms was induced by two extrastimuli from the right ventricular apex. Ventricular overdrive pacing accelerated this tachycardia to ventricular fibrillation. After two unsuccessful transthoracic defibrillation attempts (100 and 300 J, respectively), cardiopulmonary resuscitation was initiated. Five more attempts at transthoracic defibrillation (each at 500 J) during the next 10 min failed to terminate ventricular fibrillation. B, A single 100 J intracardiac shock delivered 15 min after the onset of tachyarrhythmia altered ventricular fibrillation to asystole and temporary right ventricular (RV) pacing from the same catheter maintained a normal arterial pressure. Sinus rhythm supervened 1 min later.

reported five deaths in 8,545 clinical electrophysiologic studies. Two of these deaths occurred after induction of ventricular tachycardia/ventricular fibrillation refractory to transthoracic defibrillation. The following factors were present in our patients and have been reported to decrease transthoracic defibrillation efficacy. These factors include 1) an ischemic or infarcted myocardium (5,6), 2) type I and type III antiarrhythmic drug therapy (7–10), and 3) increased transthoracic impedance caused by obesity or chronic obstructive pulmonary disease (11). In addition, a long duration of ventricular fibrillation may also raise defibrillation thresholds (12,13).

Open chest cardiopulmonary resuscitation with internal defibrillation is technically difficult and associated with high morbidity and mortality rates. In 1985 Mann et al. (14) described a case in which ventricular fibrillation developed during electrophysiologic testing and failed to respond to cardiopulmonary resuscitation and multiple high-energy transthoracic shocks. After 50 min, the right ventricular pacing lead was placed in contact with the anterior defibrillator paddle and the posterior paddle was placed under the left scapula. Administration of serial shocks (right ventricular catheter to posterior paddle) of 300 J was unsuccessful, but shocks of 360 J twice resulted in defibrillation. On follow-up at 10 months the patient was alive and well (on procainamide therapy) without arrhythmia recurrence (14).

In this study we assessed a closed chest technique of intracardiac defibrillation. This technique, which requires only standard equipment readily available in any electrophysiology laboratory, proved successful in terminating refractory ventricular fibrillation in four consecutive patients.

Efficacy and safety of catheter-delivered Intracardiac shocks. Three of the four patients in whom intracardiac defibrillation was attempted (as a last resort) are alive and well without sequelae from the procedure. Serial CK MB determinations and ECGs showed only minimal evidence of myocardial injury after intracardiac defibrillation in these three patients. A fourth patient underwent a prolonged resuscitation effort (including 15 unsuccessful transthoracic defibrillations) and after 45 min ventricular fibrillation was terminated with intracardiac defibrillation. Despite these efforts, this patient died of severe cardiac failure. In this patient it is not known whether the length of cardiopulmonary resuscitation or the magnitude of internal shock produced the severe heart failure.

The histologic effects of catheter-delivered shocks, used for ablation of supraventricular and ventricular arrhythmias, have been well described (15). Two hundred to 400 J of DC energy delivered from the distal pole of a right ventricular catheter to a large posterior skin patch results in a discrete region of myocardial necrosis (15). The size of this region is related to the magnitude of energy delivered and the degree of electrode contact (16–18). Inflammation and eventual fibrosis ensue. This technique was applied for ventricular tachycardia ablation in 164 patients (19), and seven procedural deaths were reported. These included electromechanical dissociation, intractable ventricular fibrillation and severe cardiac failure. Other infrequent complications included pericardial effusion, with or without cardiac tamponade, and pulmonary edema (19).

Precise right ventricular catheter location was unknown at the time of intracardiac defibrillation (during ventricular...
fibrillation arrest). In fact, the catheter might not have been in direct contact with the ventricular wall as a result of cardiopulmonary resuscitation. Therefore, the safety and effectiveness of this technique, with a variety of defibrillation energies and degrees of lead contact with the myocardium, require further investigation.

Prior investigation of low energy transvenous defibrillation. Previous studies have demonstrated the feasibility of low energy transvenous defibrillation with use of specially designed large surface area electrodes. Zipes et al. (20) used a custom-made catheter and in one patient successfully terminated ventricular fibrillation on three occasions with 25 J shocks. Waspe et al. (21) used a similar catheter in 13 patients and demonstrated that intracardiac defibrillation was uniformly effective in terminating induced ventricular fibrillation when adequate energy was utilized (up to 30 J) with no evidence of myocardial injury at these levels. The very large size of this experimental catheter precludes its routine use for invasive electrophysiologic studies.

Several investigators subsequently demonstrated the effectiveness of right ventricular catheter-chest wall patch (submuscular) lead configurations in terminating ventricular fibrillation. A similar configuration has been adapted to automatic cardioverter-defibrillators for implantation without thoracotomy (22-24). This configuration has a similar defibrillation efficacy to the catheter-patch configuration used in our study (25). Other lead configurations, delivery of sequential pulses and biphasic defibrillation waveforms might also improve intracardiac defibrillation efficacy (26-29).

Limitations. This study reports a new method of intracardiac defibrillation. We emphasize that this technique is still experimental with a risk of myocardial injury and should only be used as a last resort for treating refractory ventricular fibrillation. Clearly, prospective assessment of this technique is warranted to determine 1) the optimal timing of intracardiac defibrillation; 2) ideal lead-patch configurations for optimizing defibrillation thresholds; 3) the safety and efficacy of different energy levels and degrees of lead contact with myocardium (including intracardiac defibrillation from more proximal electrodes); 4) the utility of concomitant intravenous antiarrhythmic therapy; and 5) the long-term sequelae from this procedure.

Potential implications. This technique uses standard equipment available in most electrophysiologic and catheterization laboratories or intensive care units. Patients undergoing electrophysiologic testing or those with a temporary pacemaker are appropriate candidates for this still experimental technique should refractory ventricular fibrillation develop. We suspect that, if ventricular fibrillation fails to respond to at least three high energy transthoracic defibrillations, intracardiac defibrillation might be attempted beginning with 100 J. If ventricular fibrillation fails to terminate, attempts at higher energy levels are warranted. We believe that intracardiac defibrillation should be performed promptly without concomitant administration of intravenous antiarrhythmic drugs because of their potential proarrhythmic and negative inotropic effects. This technique may have important applications in other emergency settings.

References
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