Outcomes of Cardiac Perforation Complicating Catheter Ablation of Ventricular Arrhythmias

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Background—Cardiac perforation is a recognized complication of catheter ablation procedures, most commonly encountered during ablation of atrial fibrillation. The study aims to investigate the incidence, management, and hospital outcomes of cardiac perforation complicating catheter ablation for ventricular arrhythmias.

Methods and Results—Consecutive patients undergoing catheter ablation for ventricular arrhythmias at a tertiary referral center were included in this retrospective analysis. Of 1152 consecutive catheter ablation procedures in 892 patients over 12 years, 11 procedures (1.0%) were complicated by ventricular perforation. Emergent pericardial drainage and surgical repair were required in 10 (91%) and 6 (55%) cases, respectively. No perforation was apparent in patients with prior cardiac surgery. More than half of the perforations (6 of 11) occurred in the context of steam pops during radiofrequency ablation and were more likely to require surgical repair ($P=0.07$). Intra-aortic balloon counterpulsation, left ventricular assist device, and biventricular assist device were used in 2 patients, 1 patient, and 1 patient, respectively. Of 6 cases in which the site of perforation could be determined at cardiac surgery, 5 were in the right ventricle (4 outflow tract, 1 free wall) and only 1 was located in the left ventricle. All patients survived to discharge.

Conclusions—Ventricular perforation and tamponade occurs in 1% of ventricular ablation procedures and in this series, occurred only in patients without a history of prior cardiac surgery. More than half the patients required surgical repair. Perforation is often associated with steam pops and emergent surgical repair is often required when perforation occurs after a steam pop. (Circ Arrhythm Electrophysiol. 2011;4:660-666.)

Key Words: ablation ♦ complications ♦ ventricle

Catheter ablation is an important therapeutic option for management of recurrent ventricular tachycardia (VT).1 The benefits and success rates of VT ablation have been largely established in reports from high-volume centers on patient cohorts selected for either having 1 predominant ECG morphology of VT or VT associated with a single underlying cause and from a small number of multicenter registries and randomized studies.2–6 It is commonly perceived that the risk of cardiac perforation is relatively small compared with atrial ablation due to the thicker ventricular walls, but there are no large series specifically evaluating this risk in the population of patients with different causes of VT. The objectives of this study were to evaluate the incidence, possible risk factors, management, and outcomes for cardiac perforation resulting in tamponade during catheter ablation for VT.

Clinical Perspective on p 666

Methods

Patient Selection

From January 1999 to August 2010, 892 consecutive patients (72.3% men, 57.3±15.3 years) who underwent 1152 VT ablations at our institution were included in this analysis, after excluding 13 surgical VT ablation using epicardial cryotherapy and 15 procedures with transcoronary ethanol ablation only. Cardiac perforation was defined as development of a new pericardial effusion recognized during or immediately after the procedure, associated with hemodynamic compromise.

In all patients, at least 1 episode of VT or frequent monomorphic premature ventricular contractions (PVC) was recorded during monitoring by using Holter, implantable cardioverter-defibrillator logs, or 12-lead ECG. VT storm was defined as more than 3 separate VT episodes in a 24-hour period before ablation. Patients were studied in a postabsorptive state, with all antiarrhythmics except amiodarone stopped for more than 5 half-lives unless incessant VT was present. Oral anticoagulants were discontinued or transitioned to heparin, which was then discontinued 6 hours before arrival in the electrophysiology laboratory. Written informed consent was obtained from all patients. Procedures and review of medical records were conducted under protocols approved by the institutional review committee.

VT Ablation

After venous access was attained from both femoral veins, standard quadripolar catheters were positioned to record electrograms at the distal right ventricular (RV) apex and bundle of His. Femoral arterial access was obtained to record arterial blood pressure and provide
access for ablation catheters introduced retrogradely into the left ventricle (LV). Induction of VT was achieved by programmed stimulation of the RV apex, using a combination of extrastimuli or burst pacing, both with and without pharmacological adjuncts such as isoproterenol or epinephrine. In VTs of LV origin, LV access was achieved by a retrograde approach in 85% and through a transseptal sheath in 15%. Transseptal puncture was performed from the right femoral vein under fluoroscopic and in some cases intracardiac echocardiography (ICE) with a Brockenbrough needle (BRK1, St Jude Medical). For LV endocardial procedures, intravenous heparin was titrated to achieve an activated clotting time between 250 to 350 seconds throughout the procedure.

The methods used for mapping and ablation were those previously reported. During the study period, ablation was performed with an assortment of 7Fr to 8Fr steerable catheters that were either irrigated (externally and internally) or solid tipped (3.5 to 8 mm) and had either a unidirectional or bidirectional deflection mechanism. Bipolar electrograms were recorded on the CARTO electroanatomic (filtered at 10 to 40 Hz) (Biosense-Webster Inc) or NavX (St Jude Medical) mapping systems and a separate digital system (filtered at 30 to 500 Hz; Prucka Engineering Inc). Ablation was aided by electrogram characteristics, pace mapping, entrainment mapping, and establishing electric inexcitability. After delivery of radiofrequency energy, unipolar pacing from the distal electrode of the mapping catheter with an initial current strength of 10 mA and pulse width of 2 ms was performed to establish electric inexcitability. Catheter stability during pacing was validated by observing the tip position on both fluoroscopy and the 3-dimensional mapping system and by monitoring the stability of local electrogram morphology. If ventricular tissue was still excitable to pacing at the predetermined output, additional ablation lesions were delivered until loss of pacing capture was achieved at that location.

If VT was not incessant, the ventricle of interest was mapped during sinus or paced rhythm to identify areas with abnormal electrograms and low-voltage regions (<1.5 mV) consistent with scar. If abnormal areas were present, the mapping catheter was placed at an abnormal site that had pace-mapping characteristics of an exit or potential isthmus site and VT was initiated to assess electrograms, perform entrainment mapping, and potentially deliver radiofrequency current to assess for VT termination. If VT was stable, hemodynamically tolerated, and did not terminate with ablation, mapping was continued during VT. If the circuit could not be identified or multiple morphologies of hemodynamically unstable VT were induced, ablation was performed through the presumptive exit and potential isthmus sites and VT was initiated to assess electrograms, perform entrainment mapping, and potentially deliver radiofrequency current to assess for VT termination. If VT was stable, hemodynamically tolerated, and did not terminate with ablation, mapping was continued during VT. If the circuit could not be identified or multiple morphologies of hemodynamically unstable VT were induced, ablation was performed through the presumptive exit and potential isthmus sites and VT was initiated to assess electrograms, perform entrainment mapping, and potentially deliver radiofrequency current to assess for VT termination. If VT was stable, hemodynamically tolerated, and did not terminate with ablation, mapping was continued during VT. If the circuit could not be identified or multiple morphologies of hemodynamically unstable VT were induced, ablation was performed through the presumptive exit and potential isthmus sites and VT was initiated to assess electrograms, perform entrainment mapping, and potentially deliver radiofrequency current to assess for VT termination. If VT was stable, hemodynamically tolerated, and did not terminate with ablation, mapping was continued during VT. If the circuit could not be identified or multiple morphologies of hemodynamically unstable VT were induced, ablation was performed through the presumptive exit and potential isthmus sites and VT was initiated to assess electrograms, perform entrainment mapping, and potentially deliver radiofrequency current to assess for VT termination. If VT was stable, hemodynamically tolerated, and did not terminate with ablation, mapping was continued during VT. If the circuit could not be identified or multiple morphologies of hemodynamically unstable VT were induced, ablation was performed through the presumptive exit and potential isthmus sites and VT was initiated to assess electrograms, perform entrainment mapping, and potentially deliver radiofrequency current to assess for VT termination. If VT was stable, hemodynamically tolerated, and did not terminate with ablation, mapping was continued during VT. If the circuit could not be identified or multiple morphologies of hemodynamically unstable VT were induced, ablation was performed through the presumptive exit and potential isthmus sites and VT was initiated to assess electrograms, perform entrainment mapping, and potentially deliver radiofrequency current to assess for VT termination. If VT was stable, hemodynamically tolerated, and did not terminate with ablation, mapping was continued during VT. If the circuit could not be identified or multiple morphologies of hemodynamically unstable VT were induced, ablation was performed through the presumptive exit and potential isthmus sites and VT was initiated to assess electrograms, perform entrainment mapping, and potentially deliver radiofrequency current to assess for VT termination. If VT was stable, hemodynamically tolerated, and did not terminate with ablation, mapping was continued during VT.

Management of Cardiac Tamponade

Once cardiac perforation and ensuing tamponade were suspected, the cardiac silhouette was examined in left anterior oblique and right anterior oblique projections for lack of motion, or ICE images were inspected for evidence of pericardial effusion and prompt pericardioce- tocentesis was performed as detailed below. When possible and if hemodynamic status allowed, echocardiography images to confirm effusion were obtained either by ICE or 2-dimensional echocardiog- raphy before pericardioce tocentesis. Cardiopulmonary resuscitation was commenced immediately in the presence of circulatory arrest. Volume expansion and intravenous administration of inotropic and vasoconstrictive agents was commenced. If intravenous heparin had been used during the procedure, protamine was administered intra- venously to reverse anticoagulation (0.5 to 0.75 mg of protamine for every 100 U of heparin, not to exceed 50 mg per dose). All patients had been typed and crossed, and packed red blood cells were available if needed. The emergency cardiac surgical team was usually called to the electrophysiological laboratory. Percardioce- tocentesis was performed using the subsaphoid approach. After local infiltration with 1% lidocaine, a 17-gauge Tuohy epidural needle, (Havel’s Inc, Cincinnati, OH) is introduced into the pericardial space. On reaching the pericardial fluid, a guide wire is inserted, ensuring that the course of the guide wire extended throughout the pericardial space, to avoid inadvertent passage of the sheath into a cardiac chamber. Over the guide wire, a standard dilator followed by a pericardial drainage catheter or pigtail catheter (Cook Medical) was introduced into the pericardial space. The pericardial fluid was initially drained completely by aspiration and once completely evacuated, the drain was connected to a closed negative pressure system for continuous drainage. In some cases of continued bleeding, autotransfusion was performed. Blood was continuously evacuated from the pericardial space through the indwelling sheath and then immediately autotransfused into the femoral vein.

If pericardial bleeding was persistent, the patient was intubated and transferred to the operating room for further surgical manage- ment. If bleeding stopped shortly after complete drainage of pericar- dial fluid, the pericardial catheter was left in place for a variable period and removed only when the patient was completely stabilized without evidence of further intrapericardial bleeding and follow-up echocardiography showed no significant residual effusion. For pa- tients who ultimately required surgery, the exact site of perforation could be identified. For patients not requiring surgery, the most likely site of perforation was determined based on abrupt onset of hypotension occurring after radiofrequency ablation particularly when a steam pop was heard, migration of a catheter beyond the cardiac silhouette, sudden loss or change in a previously stable pacing thresh- olds, or new appearance of fresh blood draining from a preexisting pericardial sheath inserted for epicardial mapping purposes.

Data Collection and Follow-Up

Data were collected from a centralized system that continued complete records of all patients treated and followed at Brigham and Women’s Hospital and all associated Partners Healthcare sites. These records provide a detailed history and diagnosis of all patients including ablation and operative reports, emergency department visits, outpatient visits, data recorded during inpatient care, as well as scanned follow-up progress notes from referring physicians moni- toring out-of-area patients. In addition, referring cardiologists and primary care physicians were contacted for clinical follow-up of their patients if necessary.

Statistical Analysis

Baseline characteristics of the patients were compared by means of the χ² test for the categorical variable s and a t test for quantitative variable s. A 2-sided p value of <0.05 was considered statistically significant. The authors had full access to the data and take full responsibility for the integrity of the data.

Results

Of 892 patients, 236 (26.5%) had no structural heart disease; 403 (45.2%) had ischemic cardiomyopathy (ICM) with cor- onary artery disease defined by a history of prior myocardial infarction or documented obstructive coronary artery disease. The remaining 253 (28.3%) had nonischemic cardiomyopa- thy (NICM) (Table 1). Prior cardiac surgery had been performed on 53.5% of ICM, 19.6% of NICM, and none of patients without structural heart disease. Catheter ablation...
Table 1. Baseline Characteristics

<table>
<thead>
<tr>
<th>Substrate</th>
<th>None (n=881)</th>
<th>Perforation (n=11)</th>
<th>ρ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>57.2±15.2</td>
<td>60.6±19.4</td>
<td>0.47</td>
</tr>
<tr>
<td>Sex, male</td>
<td>639 (72.5%)</td>
<td>6 (55%)</td>
<td>0.19</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>40.3±17.9</td>
<td>38.6±17.5</td>
<td>0.76</td>
</tr>
<tr>
<td>History of cardiac surgery</td>
<td>258 (29.2%)</td>
<td>0 (0%)</td>
<td>0.04</td>
</tr>
<tr>
<td>Failed anti-arrhythmic agents</td>
<td>2.2±1.32</td>
<td>2.7±1.35</td>
<td>0.20</td>
</tr>
<tr>
<td>Targeted arrhythmia</td>
<td></td>
<td></td>
<td>0.65</td>
</tr>
<tr>
<td>Premature ventricular contraction</td>
<td>168 (19.1%)</td>
<td>1 (9.1%)</td>
<td></td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>702 (80.8%)</td>
<td>10 (91.9%)</td>
<td></td>
</tr>
<tr>
<td>Ventricular fibrillation</td>
<td>10 (1.1%)</td>
<td>0 (0%)</td>
<td></td>
</tr>
<tr>
<td>Prior ablation</td>
<td>323 (36.7%)</td>
<td>5 (45.4%)</td>
<td>0.55</td>
</tr>
<tr>
<td>Substrate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic cardiomyopathy</td>
<td>400 (45.4%)</td>
<td>3 (27.3%)</td>
<td>0.30</td>
</tr>
<tr>
<td>Nonischemic cardiomyopathy</td>
<td>248 (28.1%)</td>
<td>5 (45.5%)</td>
<td>0.35</td>
</tr>
<tr>
<td>No structural heart disease</td>
<td>233 (26.5%)</td>
<td>3 (27.3%)</td>
<td>0.78</td>
</tr>
</tbody>
</table>

LVEF indicates left ventricular ejection fraction.

had been attempted in 36.7% of these patients before referral to our institution.

Substrate and Clinical Presentation

Of 1152 VT ablation procedures, 11 (1.0%) procedures in 11 patients were complicated by symptomatic cardiac tamponade. The patient demographics are listed in Table 1. Ten of the 11 subjects were undergoing ablation for sustained VT episodes and the remaining 1 for frequent symptomatic monomorphic PVCs. Eight (72.7%) had structural heart disease (3 ICM, 5 NICM). None of these patients had undergone prior cardiac surgery, although 5 (45.4%) had undergone a prior ablation procedure. Baseline patient characteristics were not significantly different between patients who developed perforation versus those who did not, except for absence of prior cardiac surgery in all tamponade patients (P=0.04). The same proportion of patients in both groups had previously undergone unsuccessful ablation procedures before transfer to our institution.

Table 2. Patient and Procedural Characteristics

<table>
<thead>
<tr>
<th>Age, y/Sex</th>
<th>Chambers</th>
<th>Mapped/Ablated</th>
<th>Ablation Location</th>
<th>Cardiac Disease</th>
<th>Catheter Tip Size, mm</th>
<th>Steering Mechanism</th>
<th>Irrigation</th>
<th>Pop/Site/Impedance</th>
<th>Fluid Removed, mL</th>
<th>CPR</th>
<th>Surgical Repair</th>
<th>Laceration</th>
<th>Length of Stay, d</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 49/F</td>
<td>RV/RV</td>
<td>RVOT</td>
<td>Idiopathic</td>
<td>4/Uni</td>
<td>No</td>
<td>Yes/RVOT/impedance rise</td>
<td>Yes</td>
<td>100</td>
<td>Yes</td>
<td>Yes</td>
<td>RVOT</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>2 41/M</td>
<td>RV/RV</td>
<td>RVOT</td>
<td>NICM</td>
<td>4/Uni</td>
<td>Int</td>
<td>Yes/RVOT/23</td>
<td>Yes</td>
<td>100</td>
<td>Yes</td>
<td>Yes</td>
<td>RVOT</td>
<td>92</td>
<td></td>
</tr>
<tr>
<td>3 82/F</td>
<td>LV/LV</td>
<td>LV apex</td>
<td>ICM</td>
<td>4/Uni</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>470</td>
<td>No</td>
<td>No</td>
<td>…</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>4 86/M</td>
<td>LV, RV/LV, RV</td>
<td>LVOT, RVOT</td>
<td>NICM</td>
<td>3.5/Uni</td>
<td>Ext</td>
<td>No</td>
<td>No</td>
<td>None</td>
<td>No</td>
<td>No</td>
<td>…</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>5 67/F</td>
<td>LV, RV/LV, RV</td>
<td>RVOT</td>
<td>NICM</td>
<td>4/Uni</td>
<td>No</td>
<td>Yes/RVOT/30</td>
<td>Yes</td>
<td>560</td>
<td>No</td>
<td>Yes</td>
<td>RVOT</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>6 52/M</td>
<td>LV, RV/LV, LV septum</td>
<td>RVOT</td>
<td>Idiopathic</td>
<td>4/Uni</td>
<td>Int</td>
<td>No</td>
<td>Yes</td>
<td>350</td>
<td>No</td>
<td>No</td>
<td>…</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>7 62/M</td>
<td>RV, LV, Epi/RV Epi</td>
<td>RVOT</td>
<td>ARVC</td>
<td>3.5/Uni</td>
<td>Ext</td>
<td>Yes/RVOT/13</td>
<td>Yes</td>
<td>500</td>
<td>No</td>
<td>No</td>
<td>…</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>8 28/F</td>
<td>LV/None</td>
<td>No Abl</td>
<td>Idiopathic</td>
<td>3/Uni</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>300</td>
<td>No</td>
<td>No</td>
<td>…</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>9 45/F</td>
<td>LV, RV/LV, RV</td>
<td>RVOT</td>
<td>NICM</td>
<td>3.5/Bl</td>
<td>Ext</td>
<td>Yes/RVOT/28</td>
<td>Existing sheath</td>
<td>650</td>
<td>Yes</td>
<td>Yes</td>
<td>RVOT</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>10 87/M</td>
<td>LV, RV/LV, RV</td>
<td>RV free wall</td>
<td>NICM</td>
<td>3.5/Bl</td>
<td>Ext</td>
<td>No</td>
<td>Yes</td>
<td>1400</td>
<td>Yes</td>
<td>Yes</td>
<td>RV free wall</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>11 67/M</td>
<td>LV/LV</td>
<td>PL basal LV</td>
<td>NICM</td>
<td>3.5/Bl</td>
<td>Ext</td>
<td>Yes/PL LV/19</td>
<td>Yes</td>
<td>1000</td>
<td>No</td>
<td>Yes</td>
<td>PL LV</td>
<td>43</td>
<td></td>
</tr>
</tbody>
</table>

CPR indicates cardiopulmonary resuscitation; RV, right ventricular; RVOT, RV outflow tract; LV, left ventricular; EPI, epicardium; PL, posterior-lateral; ICM, ischemic cardiomyopathy; NICM, nonischemic cardiomyopathy; ARVC, arrhythmogenic RV cardiomyopathy; Uni, unidirectional deflection mechanism; Bi, bidirectional deflection mechanism; Int, internally irrigated; and Ext, externally irrigated.

Procedural Data

Individual procedural data in 11 subjects with ventricular perforation are shown in Table 2. Electroanatomic mapping, irrigated-tipped catheter, and ICE were used in 8 (72.7%), 7 (63.6%), and 3 (27.3%) of these 11 subjects, respectively. No sheaths were used for RV-only ablations. Transeptal catheterization was only performed in 1 patient, and the cause of the perforation was confirmed at the time to be unrelated to transseptal access (case 11 in Table 2). In 1 case (case 9), a pericardial sheath was inserted at the beginning of the procedure to allow for epicardial mapping and potentially ablation. Before cardiac perforation occurred, there was no bleeding within the pericardial space. Intravenous heparin had been administered in 10 of 11 cases.

Mapping was performed in the RV alone in 2 of 11 cases. In 1 case (case 8), tamponade occurred before any ablation was performed, probably caused by puncture of the RV apex during positioning of the diagnostic catheter. Recognized steam pops occurred during endocardial radiofrequency ablation in 6 of 10 cases and heralded the onset of tamponade. During these 6 radiofrequency applications that resulted in steam pops, the max power and temperature were 43.6±4.8 W and 39.0±4.1°C, respectively, with an impedance drop of 22.5±7.9 Ω, excluding 1 patient with impedance rise.

Management of Perforation

In all cases, a drop in blood pressure to <90 mm Hg was observed, and new pericardial effusions were seen by trans-
thoracic echocardiography in 8 cases and by ICE in the remaining 3. In 7 of 11 cases, a precipitous fall in blood pressure within 2 to 3 minutes was seen. However, in a single patient (case 5), blood pressure only fell 20 minutes after a pop prompted the cessation of radiofrequency ablation in the RV outflow tract. Protamine was administered in 7 patients (5 of 6 patients who underwent surgical repair). Emergent periprocedural pericardiocentesis was required in 9 (82%) cases, and in a single subject (case 9), a pericardial sheath was already present when perforation after endocardial radiofrequency ablation occurred. Mean duration from steam pop to pericardiocentesis was 22 ± 15 minutes. In 1 subject (case 4), cardiac tamponade was not recognized until 1 hour after the end of the procedure, but the patient was treated conservatively with restoration of blood pressure by volume expansion alone. A mean of 543 ± 401 mL of blood was drained from the pericardium. In 4 of 10 patients, pericardial bleeding ceased after draining an average of 405 ± 95 mL. In the remaining 6 patients, hemodynamic stability could not be restored despite pericardiocentesis (mean drainage of 635 ± 509 mL) and volume expansion. Four patients received red blood cell transfusion before cardiac surgery. Chest compressions and assisted ventilation were performed in 4 patients. They were emergently taken to the operating room for surgical repair. In 1 subject (case 2), tamponade occurred after a pop in the RV outflow tract, and despite successful insertion of a pericardial drain, only 100 mL of blood could be aspirated without any improvement in hemodynamics, and ventricular fibrillation remained refractory to external cardioversion. An emergency subxiphoid dissection and creation of a pericardial window was performed by the surgical team in the electrophysiology laboratory to evacuate a significant amount of thrombus that had prevented further evacuation of the pericardial space. The patient was then transferred to the operating room with ongoing cardiopulmonary resuscitation and spontaneously defibrillated once sternotomy was performed and internal cardiac massage instituted. Five of 6 patients who had steam pops during their last radiofrequency energy application required surgical repair and were more likely to require surgical repair (P = 0.07).

Surgical Repair
The duration from detection of hypotension related to pericardial bleed, to commencement of surgery, was 48 ± 22 minutes. All 6 patients taken to the operating room underwent emergency sternotomy. Pericardiotomy combined with evacuation of pericardial blood improved blood pressure or allowed for the cessation of cardiopulmonary resuscitation in all patients. Lacerations were identified and closed by sutures.

For 3 subjects (cases 1, 5, and 9), repair of RV outflow tract lacerations with sutures was performed without the need for cardiopulmonary bypass. The other 3 subjects required cardiopulmonary bypass, of which 2 (cases 1 and 10) received intra-aortic balloon pump counterpulsation (Datascope Corp) to aid weaning of bypass. One patient (case 1) could not be weaned off cardiopulmonary bypass despite intra-aortic balloon pump counterpulsation, and a biventricular assist device (Thoratec) was placed. He had been previously been accepted for transplantation, and he underwent orthotopic cardiac transplantation 74 days later.

In case 11 (Table 2), a large dissecting intramural hematoma formed within posterior-lateral LV after a steam pop during ablation in a posterior basal aneurysm (Figures 1 and 2). Because of the extensive myocardial injury and coexisting moderate aortic stenosis, aortic valve replacement and placement of a LV assist device (TandemHeart, CardiacAssist Inc) was performed. The LV assist device was explanted 23 days later. In a single patient, additive epicardial cryoablation (case 10) was applied with a surgical probe around the epicardial border of an anterior wall infarct, due to persistence of incessant VT during transfer to the operating room, with VT termination only during open-chest manipulation of
Location of Perforation

The exact location of perforation was confirmed in 6 patients at the time of cardiac surgery. Five were in the RV (4 outflow tract, 1 free wall) and only 1 was in the LV. Perforation at the RV apex by the diagnostic catheter was the most likely culprit in 2 subjects (cases 6 and 8). In both of these cases, mapping of the LV was performed in patients with structurally normal hearts, and in only 1 case was ablation performed at the mid LV septum for PVCs. In both cases, the fall in blood pressure was insidious, and the diagnostic catheter at the RV apex had to be repositioned during the case due to loss of capture. The site of perforation could not be determined in case 4 who had symptomatic hypotension 1 hour after undergoing ablation in the RV and LV outflow tracts for 3 different VTs. Transthoracic echocardiography demonstrated a 1.3-cm circumferential pericardial effusion with diastolic compression of the right atrium and ventricle. After fluid resuscitation, his blood pressure improved, and the pericardial effusion was managed successfully without pericardiocentesis.

At the time of surgery, the lacerations were estimated to be between 5 to 10 mm in length and at locations consistent with where catheters were positioned when steam pops were detected.

Outcome

All patients survived to hospital discharge. The average length of stay was significantly longer in patients who required surgical repair. (34.5±30.7 versus 4.0±2.6 days for those without surgical repair, \( P=0.03 \)) The ablation procedure was only completed in 3 of 11 patients. VT recurred in 5 patients (3 while in hospital and 2 after discharge) and was treated medically. No repeat ablation procedures were performed.

Discussion

The present study demonstrates an incidence of cardiac perforation and tamponade of 1.0% in a large cohort of patients undergoing more than 1100 endocardial catheter ablation procedures for ventricular arrhythmias. More than half of these patients required surgical repair; perforation after a steam pop was more likely to require surgery. Although most perforations occurred during ablation within the RV, in particular the outflow tract, LV perforation was seen but less commonly. Significant pericardial bleeds did not occur in any patients who had prior cardiac surgery. Cardiac tamponade complicating VT ablation has been sporadically reported. In a multicenter trial of 231 patients undergoing ablation of infract-related VT ablation using an open-irrigated ablation catheter, there was a single case of cardiac perforation and tamponade, with subsequent death, an incidence of 0.4%. 13 In a smaller trial of 146 patients with structural heart disease and VT undergoing ablation with an internally irrigated catheter, 4 cases (2.7%) of tamponade that resulted in a single death were reported. 2 Among reports from experienced high-volume centers, Della et al 4 and Sauer et al 7 have each described 4 cases of pericardial effusions without tamponade in 97 and 327 cases of VT ablation, respectively.

Several groups have reported perforation and tamponade during catheter ablation for atrial fibrillation. Of 622 patients who underwent atrial ablation at the Mayo Clinic, 15 procedures (2.4%) were complicated by perforation. 14 Of these, only 2 cases (13%) required open surgical repair, whereas the remaining 13 cases were treated with pericardiocenteses, intravenous fluids, and pressors. Similarly, Haissaguerre et al reported a 2.9% risk of cardiac tamponade during 348 AF ablation procedures. In 8 of 10 cases identified, an audible “pop” associated with an impedance rise was observed. 15 Only 1 (10%) of this group required surgical repair. Maximum power delivery in excess of 48 W was a risk factor of tamponade during atrial ablation, and limiting power delivery to <42 W successfully reduced the incidence of cardiac perforation. In contrast, patients with ventricular perforations, as seen in our series, were more likely to require surgical repair.

In the current study, the occurrence of a steam pop heralded the onset of tamponade in 6 of 11 cases. In 5 of 6 patients, the locations of lacerations repaired during cardiac surgery was consistent with the catheter location when the pop occurred, suggesting that ventricular perforation in these cases were to the ablation that resulted in a steam pop. Because of ventricular thickness, higher powers are often required for ventricular ablation. 16 Catheter irrigation is often used to cool the ablation electrode such that more power can be delivered without being limited by the formation of thrombus at the catheter-tissue interface. 17 Irrigation results in substantial disparity between the electrode and tissue temperature. Excessive intramyocardial heating can produce steam formation and abrupt volume expansion, which may be audible as steam pops. 18 Pops are capable of causing deep tissue tears, consistent with the findings in our patients at the time of surgery. In a swine model, Cooper et al reported that maintaining electrode temperature <40°C during application of radiofrequency energy with an internally irrigated catheter can reduce the risk of steam explosions. 19 However in the present study, the mean ablation electrode temperature before pop was 39.0±4.1°C. We have previously analyzed ablation parameters that are associated with steam pop during VT ablation with externally irrigated catheter in a report that included a subset of the more recently studied patients included in the present report. 20 Steam pops occurred in 1.5% of radiofrequency energy applications. Larger impedance falls and higher maximum energies were associated with pops. Eighty percent of pops occurred after impedance decreased by more than 18 Ω. This is consistent with the present study where the mean impedance fall in 6 applications that resulted in a pop was 22.5 Ω. Limiting radiofrequency power to prevent an impedance decrease of more than 18 Ω may reduce the risk of cardiac perforation. An important consideration that could not be quantified in our series is the contact force applied during ablation, which may affect the propensity for steam pops. Contact sensing catheters in development will hopefully, provide more insight.

It is not surprising to find that the majority of perforations occurred in the thinner-walled RV, which can occur during...
either radiofrequency ablation or even placement of diagnostic catheters. It is generally perceived that the thicker LV should be less prone to perforation because of its greater wall thickness. In addition, LV infarct scars, where ablation is often performed to target slow conducting channels, are usually firm and sometimes calcified. Our series shows that perforation can occur uncommonly during ablation in diseased LV as the result of prior myocardial infarctions. One LV perforation was caused by a steam pop, and therefore careful titration of radiofrequency current to avoid steam pops during LV ablation is warranted.

Study Limitations
This is a retrospective analysis of a large cohort of patients referred over 12 years to a tertiary center for VT ablation. During this time, ablation technologies and techniques have been continually modified. The exact location of ventricular perforation could be confirmed only in the patients who underwent surgical repair. In the rest of the patients, the site of perforation was based on circumstantial evidence and could be inaccurate. All our patients survived to discharge, in no small part because of the support and close collaboration provided by cardiac surgeons. Therefore, it is possible that the 1% risk may not necessarily reflect the actual risk associated with current practices including the use of new technologies. Despite the large cohort of patients analyzed, the number of perforations causing tamponade was small, limiting the strength of the associations reported in this series for steam pops in patients with prior cardiac surgery. However, these data represent the largest series of adult patients complicated by ventricular perforations. The number of cases in our study was too small to investigate the relationship between catheter design or ablation modality and the likelihood of perforations. ICE during VT ablation procedure and transthoracic echocardiography after the procedure is not routinely performed on all patients, and it is likely that some asymptomatic perforation without hemodynamic compromise were not diagnosed.

Conclusions
Ventricular perforation and tamponade occur in 1% of ventricular ablation procedures, most commonly at the RV outflow tract. In our cohort, this complication occurred exclusively in patients who have never had prior cardiac surgery. More than half of these cases required surgical repair, and perforation after a steam pop was more likely to require surgery. Perforation during ablation in diseased LV myocardium is rare but can occur.

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Disclosures
None.

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**CLINICAL PERSPECTIVE**

It is commonly perceived that the risk of ventricular perforation during catheter ablation is relatively small compared with atrial ablation because of the thicker ventricular walls. The present study provides the largest reported experience of cardiac perforation during catheter ablation for ventricular arrhythmias and describes its management and outcome. Of 1152 consecutive ventricular ablation procedures in 892 patients, 11 procedures (1.0%) were complicated by cardiac perforation. Cardiac tamponade was not encountered in the patients with prior cardiac surgery. In 6 patients, hemodynamic stability could not be restored despite pericardiocentesis and were emergently taken to the operating room for surgical repair. The exact location of perforation was confirmed at the time of cardiac surgery; 5 in the right ventricular (4 outflow tract, 1 free wall) and only 1 in the left ventricular. All patients survived to hospital discharge. Five of 6 patients who had steam pops during their last radiofrequency energy application required surgical repair; perforations occurring in relation to steam pops were more likely to require surgical repair ($P=0.07$). This series emphasizes the infrequent but life-threatening possibility of both right and left ventricular perforations during ventricular tachycardia ablations and the importance of timely cardiac surgical interventions for effective management.
Outcomes of Cardiac Perforation Complicating Catheter Ablation of Ventricular Arrhythmias
Michifumi Tokuda, Pipin Kojodjojo, Laurence M. Epstein, Bruce A. Koplan, Gregory F. Michaud, Usha B. Tedrow, William G. Stevenson and Roy M. John

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Clinical summaries of selected cases

Case 2

*Learning point: Thrombus can prevent evacuation of pericardial effusion by a percutaneous drain*

This 41 year old gentleman with non-ischemic cardiomyopathy, 15% EF awaiting cardiac transplantation was admitted with VT storm and worsening heart failure. Radiofrequency ablation at the RV outflow tract resulted in an audible pop which terminated radiofrequency energy delivery. Hypotension developed immediately, followed by cardiogenic shock and then cardiac arrest with ventricular fibrillation, refractory to external cardioversion. A pericardial drain was inserted but only 100mls of blood could be withdrawn without any improvement in hemodynamics. In the electrophysiology laboratory, the cardiac surgical team performed a subxiphoid pericardial window and evacuated large amounts of thrombus, which presumably prevented percutaneous pericardial drainage. Despite this effort, ventricular fibrillation remained refractory to external cardioversion. He was transferred to the operating room with ongoing CPR. After sternotomy and internal cardiac massage was instituted, he
spontaneously converted to a slow ventricular rhythm. Cardiopulmonary bypass was instituted. A tear on the anterior RV outflow tract was repaired. Despite the use of milrinone, dopamine and an intra-aortic balloon counterpulsation, it was not possible to wean him off bypass and therefore a biventricular assist device (Thoratec, USA) was implanted. His post-operative course was complicated by acute renal failure. He remained free from VT for the next 9 weeks and then underwent orthotopic cardiac transplantation.

Case 5

*Learning point: Tamponade can occur late after a steam pop*

67 year old female with non-ischemic cardiomyopathy and EF 15% was referred for VT ablation due to recurrent VT, refractory to amiodarone. In the electrophysiology lab, sustained VT at the cycle length of 370ms, LBBB morphology, right inferior axis and precordial transition at V3 was induced but had to be pace-terminated due to hemodynamic intolerance. Pace mapping at the free wall of the RV outflow tract yielded an excellent pace map match with the inducible VT. During radiofrequency application at this site, an audible pop occurred and ablation was promptly discontinued. However, hemodynamics remained stable and movement of the cardiac silhouette was
still evident on fluoroscopy. Further ablations were delivered slightly inferior to that location. Approximately 20 minutes after the pop, hypotension developed and there was absence of cardiac motion on fluoroscopy (Video S1). As protamine, fluids and inotropic agents were administered, transthoracic echocardiography confirmed the presence of a moderately sized pericardial effusion with collapse of the right heart. (Video S2) A pericardial drain was placed via the subxyphoid approach. Evacuation of pericardial blood restored blood pressure but bleeding continued. Hemodynamic stability was maintained by continued aspiration of pericardial blood and autotransfusion into the left femoral vein. The patient was transferred to the operating room where a further 500mls mixture of blood and thrombus was removed from the pericardial space. A perforation in the anterior RV outflow tract consistent with the site of the catheter at the time of the prior audible pop, was closed with pledgeted sutures. She remained free from VT and was subsequently discharged home, but required placement of a biventricular assist device two months later due to worsening heart failure status.

Case 10
Learning point: Adjunctive epicardial cryoablation can be employed at the time of cardiac surgery to prevent VT recurrence

An 87 year old male with prior anterior MI, 30% EF and VT storm despite mexiliteine and amiodarone was transferred to our institution. Incessant VT at 410ms with a LBBB, superior axis configuration and predominantly S waves in the precordial leads was induced and left ventricular endocardial mapping identified earliest endocardial activation at the apical left ventricular septum. Irrigated radiofrequency application at this site slowed and transiently terminated VT but it remained easily inducible. Suspecting that the critical isthmus was deep within the interventricular septum, the ablation catheter was brought onto the apical RV septum where pre-systolic electrical activity was detected. Radiofrequency application was followed by hypotension due to cardiac tamponade, necessitating emergent pericardiocentesis and administration of protamine. Pericardial bleeding persisted and he was transferred to the operating room where a laceration at the RV apex consistent with the last site of ablation was found and sutured. As VT had remained incessant and was only terminated during open-chested manipulation of the heart, a ring of cryoablation lesions was placed around a large anterio-apical calcified aneurysm which was in close proximity to the identified endocardial exit sites for VT. He remained free from VT. However, his hospital stay
was prolonged due to bleeding from the left saphenous vein graft site requiring transfusions and fever of unknown origin which required broad spectrum antibiotics. He was eventually discharged to a local rehabilitation unit 20 days after his ablation procedure.

Case 11

Learning point: Left ventricular perforation can rarely occur during ablation of diseased LV myocardium

A 67 year old gentleman with non-ischemic cardiomyopathy, moderate aortic stenosis and recurrent VT necessitating ICD shocks 7 months after a prior catheter ablation procedure was admitted for VT ablation. Three different VTs with cycle lengths between 350 to 540ms were induced. After transeptal access was achieved, a left ventricular anatomical shell was created by CartoSound system and a voltage map was superimposed showed a low voltage region in the inferoposterior basal LV near the mitral annulus, where the presence of late potentials, long stim-QRS delays and pace-mapping matched QRS morphologies of induced VTs. The location of the scar was consistent with the presence of a posterior-basal aneurysm visible on intracardiac echocardiography. (Figure 3) Using a substrate modification strategy, radiofrequency
ablation was applied to the area. During the last ablation close to the septal side of the scar, a steam pop was heard and delivery of radiofrequency energy promptly stopped. A large intramural hematoma in the posterior-lateral left ventricle, together with an enlarging sized pericardial effusion, was visible on intracardiac echocardiography. (Figure 2) ST segment elevation was seen in the lateral leads and coronary angiography was promptly performed which showed no obstruction or spasm of the epicardial vessels. Approximately 25 minutes after the pop, systolic blood pressure fell to less than 70mmHg and a pericardial drain was inserted. One litre of pericardial blood was drained over the next 40 minutes which restored hemodynamic stability, but frank bleeding continued and the patient was transferred to the operating room for emergent surgical repair. He was placed on cardiopulmonary bypass. A large dissecting, intramural hematoma with 500-600mls of clot was observed within the posterior-lateral left ventricle, in the territory of the obtuse marginal branches. Dissection of the hematoma through the ventricular wall and swelling resulted in visible separation of obtuse marginal branches from some myocardial perforating vessels. Given the extent of the injury, a left ventricular assist device (TandemHeart, USA) was implanted and the stenotic aortic valve was replaced. (Mitroflow, CarboMedics Inc., USA) Due to significant ongoing oozing from the repaired posterior-lateral left ventricle,
sternal closure was not initially performed. His post-operative course was further complicated by thrombus formation in the left ventricular outflow tract which required re-sternotomy and thrombectomy. The left ventricular assist device was removed 23 days following his first operation. He was successfully discharged for rehabilitation after 45 days and has since remained free from VT after 4 months of follow-up.

Figure legend

Figure 3.

Left panel – voltage map of left ventricle superimposed on a registered intracardiacechocardiogram (ICE) image showing low-voltage myocardium (<1.5mV in red) and normal myocardium (in purple). Location of ablation lesions marked in red dots and site of ablation which resulted in a pop.(white arrow) Right panel – the registered ICE image showing the presence of a basal posterior aneurysm (white arrow) around which putative isthmus sites were targeted
Additional Videos for Online Data Supplement

Video 1. Fluoroscopy showing the absence of movement of the cardiac silhouette and the injection of contrast via a drain into the pericardium

Video 2. Subxiphoid view showing the presence of a pericardial effusion measuring at least 2 cm associated with collapse of the right sided chambers, consistent with tamponade physiology.
Figure 3.