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Sustained Ventricular Tachycardia Associated With Corrective Valve Surgery

Robert E. Eckart, DO; Tomasz W. Hruczkowski, MD; Usha B. Tedrow, MD; Bruce A. Koplan, MD; Laurence M. Epstein, MD; William G. Stevenson, MD

Background—The causes of sustained monomorphic ventricular tachycardia (VT) after cardiac valve surgeries have not been studied extensively, although bundle-branch reentry has been reported.

Methods and Results—Records of 496 patients referred for electrophysiology study and catheter ablation of recurrent VT were reviewed. Twenty patients (4%) had VT after aortic or mitral valve surgery in the absence of known myocardial infarction. The median age was 53 years, and the median ejection fraction was 45%. In 4 patients, VT occurred early after surgery, and electrophysiology study was performed 3 to 10 days later. In the remaining patients, electrophysiology study was performed a median of 12 years (interquartile range 5 to 15 years) after surgery. Sustained VT was inducible in 17 patients. VT was attributed to scar-related reentry in 14 patients (70%) and to bundle-branch reentry in 2 (10%). Multiple VTs were present in 9 of 14 patients with scar-related reentry. A total of 42 induced VTs were targeted for ablation. Of the 14 patients with scar-related reentry, 9 (64%) had periannular scar, and 10 (71%) had an identifiable endocardial circuit isthmus. Ablation abolished 41 (98%) of the 42 targeted VTs. At a median follow-up of 2.1 years, 3 deaths occurred 8 to 14 months after ablation. One patient with incessant VT early after valve surgery suffered a stroke with residual hemianopsia. Of the 20 patients, 3 required repeat ablation after recurrence, and 2 of these who were not inducible during electrophysiology study had clinical recurrence that necessitated ablation.

Conclusions—Sustained VT after valve surgery appears to be bimodal in presentation, occurring either early after surgery or years later. In this referral population, reentry in a region of scar is more common than bundle-branch reentry. Catheter ablation can be successful. (Circulation. 2007;116:2005-2011.)

Key Words: ablation ▪ arrhythmia ▪ catheter ablation ▪ valves ▪ tachycardia, ventricular

Sustained monomorphic ventricular tachycardia (VT) after valve surgery is both uncommon and not well-studied. Cases of focal VT1 and small series of bundle-branch reentry have been reported.2,3 Information is limited as to the cause and interventional treatment of other types of VT in these patients. We report a cohort of patients with monomorphic VT after valve surgery who underwent detailed electrophysiology study with mapping that elucidates the likely substrate and mechanisms of VT in this population.

Methods

Clinical records of the 496 patients referred for electrophysiological study of recurrent VT from January 2000 to December 2005 were reviewed to determine the presence of valvular heart disease that required surgery before catheter ablation. The 20 patients with valve surgery before the occurrence of VT and no known history of prior myocardial infarction form the basis for the present report. Coronary artery disease was defined either angiographically as a >50% stenosis or as a history of coronary revascularization before testing. Mortality was determined with the use of both hospital records and the Social Security Death Index.

Electrophysiology Study

Electrophysiological studies with catheter mapping and ablation were performed as described previously.4 Access to the left ventricle was achieved via a transseptal approach in patients with mechanical aortic valves (11 patients) or by a retrograde aortic approach in the remaining patients. Patients were anticoagulated with heparin. Surface ECG leads and intracardiac electrograms were stored with the Prucka CardioLab EP System (GE Healthcare, Wis). Nonfluoroscopic electroanatomic mapping was performed with CARTO (Biosense-Webster, Inc, Diamond Bar, Calif). An anatomic low-voltage scar was defined as 2 adjacent points within a structural territory with a bipolar voltage <1.5 mV.5–6 The mechanism of VT was defined as scar-related reentry when VT was inducible with programmed stimulation, could be entrained, and originated from a low-voltage area consistent with scar. Reentry circuit sites were...
### Table. Characteristics of 20 Patients Referred for Ablation of Documented VT After Aortic or Mitral Valve Surgery

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, y</th>
<th>Heart Disease*</th>
<th>Mechanism</th>
<th>Anatomic Source of Clinical VT</th>
<th>Cycle Length, ms</th>
<th>Morphology</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>34</td>
<td>MV repair, EF 55%</td>
<td>Noninducible</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>2</td>
<td>67</td>
<td>AV prosthesis, (+) CAD, EF 50%</td>
<td>Noninducible</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>3</td>
<td>41</td>
<td>MV repair, EF 50%</td>
<td>Noninducible</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>4</td>
<td>74</td>
<td>MV repair, EF 10%</td>
<td>Automatic</td>
<td>Distal left bundle, no ablation; preexisting RBBB</td>
<td>430</td>
<td>RBBB, left superior axis</td>
</tr>
<tr>
<td>5</td>
<td>65</td>
<td>AV prosthesis, EF 30%</td>
<td>Bundle-branch reentry</td>
<td>Right bundle ablated</td>
<td>426</td>
<td>LBBB, inferior axis</td>
</tr>
<tr>
<td>6</td>
<td>36</td>
<td>AV prosthesis, EF 12%</td>
<td>Bundle-branch reentry and scar-related reentry</td>
<td>Right bundle ablated</td>
<td>337</td>
<td>RBBB, left superior axis</td>
</tr>
<tr>
<td>7</td>
<td>44</td>
<td>AV prosthesis, (+) CAD, EF 60%</td>
<td>Scar-related reentry</td>
<td>Anteroseptal and inferior base of LV, apex of RV</td>
<td>390</td>
<td>LBBB, left superior axis</td>
</tr>
<tr>
<td>8</td>
<td>38</td>
<td>AV prosthesis, EF 50%</td>
<td>Scar-related reentry</td>
<td>Anteroseptal MV annulus</td>
<td>296</td>
<td>RBBB, right inferior axis</td>
</tr>
<tr>
<td>9</td>
<td>41</td>
<td>AV prosthesis, EF 30%</td>
<td>Scar-related reentry</td>
<td>Anteroseptal mid to apical LV</td>
<td>280</td>
<td>RBBB, right inferior axis</td>
</tr>
<tr>
<td>10</td>
<td>39</td>
<td>AV prosthesis, EF 65%</td>
<td>Scar-related reentry</td>
<td>Midanterolateral LV</td>
<td>335</td>
<td>RBBB, right inferior axis</td>
</tr>
<tr>
<td>11</td>
<td>76</td>
<td>AV prosthesis, EF 30%</td>
<td>Scar-related reentry</td>
<td>Posterolateral AV annulus</td>
<td>463</td>
<td>LBBB, right inferior axis</td>
</tr>
<tr>
<td>12</td>
<td>53</td>
<td>AV prosthesis, EF 60%</td>
<td>Scar-related reentry</td>
<td>Inferoseptal AV annulus</td>
<td>240</td>
<td>RBBB, right inferior axis</td>
</tr>
<tr>
<td>13</td>
<td>53</td>
<td>AV prosthesis, EF 15%</td>
<td>Scar-related reentry</td>
<td>Inferolateral RV</td>
<td>433</td>
<td>RBBB, left superior axis</td>
</tr>
<tr>
<td>14</td>
<td>49</td>
<td>AV prosthesis, (+) CAD, EF 40%</td>
<td>Scar-related reentry</td>
<td>MV and AV trigone</td>
<td>490</td>
<td>RBBB, right superior axis</td>
</tr>
<tr>
<td>15</td>
<td>76</td>
<td>AV prosthesis, (+) CAD, EF 55%</td>
<td>Scar-related reentry</td>
<td>Anteroseptal MV annulus</td>
<td>327</td>
<td>RBBB, left inferior axis</td>
</tr>
</tbody>
</table>

(Continued)
defined by entrainment mapping and pace mapping as reported previously.7 Reentrant circuit isthmus sites were those at which entrainment occurred with concealed fusion with a postspacing interval within 30 ms of the tachycardia cycle length and a stimulus-to-QRS interval within 30 ms of the tachycardia cycle length. When VT was unstable for mapping, exit sites were targeted on the basis of pace entrainment occurred with concealed fusion with a postpacing interval within 70% of the tachycardia cycle length. When VT was unstable for mapping, exit sites were targeted on the basis of pace entrainment occurred with concealed fusion with a postpacing interval within 30 ms of the tachycardia cycle length and a stimulus-to-QRS interval within 30 ms of the tachycardia cycle length. When VT was unstable for mapping, exit sites were targeted on the basis of pace entrainment occurred with concealed fusion with a postpacing interval within 30 ms of the tachycardia cycle length and a stimulus-to-QRS interval within 30 ms of the tachycardia cycle length. When VT was unstable for mapping, exit sites were targeted on the basis of pace entrainment occurred with concealed fusion with a postpacing interval within 30 ms of the tachycardia cycle length and a stimulus-to-QRS interval within 30 ms of the tachycardia cycle length. When VT was unstable for mapping, exit sites were targeted on the basis of pace

### Results

The 20 patients were all men, with a median age of 53 years (IQR 41, 71 years; range 34 to 76 years; Table). No patient was known to have congenital heart disease. The surgical procedure was aortic valve replacement in 12 patients, mitral valve replacement in 1, mitral valve repair in 5, and double-valve surgery in 2. Median left ventricular ejection fraction was 45% (IQR 18%, 59%), with an end-diastolic internal diameter of 6.3 cm (IQR 5.4, 7.4 cm) and interventricular septal thickness of 1.2 cm (IQR 1.0, 1.3 cm). Sustained monomorphic VT occurred during the index hospitalization associated with valve surgery in 4 patients, and an electrophysiology study was performed 3, 6, 8, and 10 days after surgery, respectively, in these patients. In the remaining 16 patients, VT occurred late after surgery (range 1 to 16 years). Although the exact time at which the arrhythmias first began is uncertain, the time to initiation of treatment for VT was a median of 12 years (IQR 5, 15 years) after the most recent surgical procedure.

Three patients underwent implantation of an implantable cardioverter defibrillator (ICD) after electrophysiology study; 12 of the 20 patients had a preexisting ICD implanted a median of 4.5 years (IQR 1.9, 6.1 years) before referral. Only 3 of the patients had coronary artery disease, and all of these patients had undergone prior revascularization. Sixteen of the patients were receiving beta-blockers. Antiarrhythmic drug therapy immediately before ablation consisted of mexiletine in 6 patients (30%), amiodarone in 5 (25%), sotalol in 2 (10%), moricizine in 1 (5%), procaainamide in 1 (5%), and quinidine in 1 (5%); 5 patients were taking combinations of antiarrhythmic drugs. The reason for referral was repetitive ICD therapies in 10 patients (50%), symptomatic self-terminating VT in 4 (20%) with near-syncope in 2, hemodynamically stable sustained VT in 5 (25%), and cardiac arrest with documented sustained VT in 1 (5%).
VT was incessant in 1 patient (5.0%). No VT was inducible despite documented spontaneous VT in 3 patients (15.0%). In the remaining 16 patients (80%), sustained monomorphic VT was induced by ventricular stimulation with single (3 patients, 15.0%), double (6 patients, 30.0%), or triple (3 patients, 15.0%) extrastimuli or burst pacing (4 patients, 20.0%). Two patients required isoproterenol before tachycardia could be induced with burst pacing. VT was attributed to scar-related reentry in 14 patients (70.0%), bundle-branch reentry in 2 (10.0%), and focal origin (presumed automatic) in 1 (5.0%).

**Scar-Related Reentry**

Electroanatomic mapping with creation of voltage maps during sinus rhythm was performed in 15 patients. Voltage mapping was not performed on the 3 patients with no inducible arrhythmia, on 1 patient with an automatic focus, and on 1 patient with bundle-branch reentry VT. A low-voltage area consistent with scar was noted in 14 patients (93.3%), all of whom had scar-related reentry VT (Figure 1). The scar extended adjacent to the aortic or mitral annulus in 9 (64.3%) of the 14 patients with scar. Of the 14 patients with scar-related reentry, 9 had prior isolated aortic valve surgery, 3 had isolated mitral valve surgery, and 2 had both aortic and mitral valve surgery. Voltage maps revealed scar adjacent to the valve that had been operated on in 8 of 14 patients. Patients with scar-related reentry had a total of 40 sustained monomorphic VTs induced. The majority of VTs had right bundle-branch block morphology, with an inferiorly directed axis. Endocardial isthmus sites were identified in 12 patients (Figure 2). Of those with scar-related reentry, middiastolic potentials were identified in 6 cases (42.8%), and “fractionated” signals consistent with slowed conduction in the scar region were identified in 7 (50.0%).

Endocardial ablation (median 18 lesions per patient) abolished, at least in the short term, all inducible monomorphic VTs in all 14 patients with scar-related reentry. In those cases in which the isthmus approximated the valve annulus, a line of lesions was placed from the isthmus to the electrically unexcitable region of the annulus. At the conclusion of the procedure, no patient with an identified endocardial isthmus had VT inducible with up to 3 extrastimuli to a coupling interval of 180 ms or refractoriness.

In 1 patient with 4 induced VTs, no endocardial isthmus sites were identified. Pace mapping was used to guide endocardial ablation, after which only nonsustained VT was inducible, which suggests that the circuit was sufficiently close to the endocardium for ablation. The patient was started on sotalol therapy and brought back to the laboratory 4 days later, at which time no VT could be induced.

During a median follow-up of 2.1 years, 11 patients remained free of spontaneous VT. Three patients had repeat ablation due to recurrent VT. A patient with a history of aortic valve replacement had recurrent VT after ablation and underwent a second procedure 5 weeks after the first. During the second procedure, a single VT with the same QRS morphology but a slower cycle length than previously observed (410 ms compared with 335 ms) was found arising from a region of scar in the midanterolateral left ventricular wall, where ablation again abolished inducible VT. He had recurrence 7 months after his second procedure and was found to have the same VT QRS morphology, albeit slower (tachycardia cycle length 450 ms), and ultimately, he had a successful outcome with ablation at the same site as the first 2 procedures. The second patient, with a history of mitral valve repair, had recurrent sustained VT at 5 years later. Repeat study revealed an isthmus location near the mitral valve annulus, similar to the original study, and ablation again achieved short-term success. The third patient, with a history of aortic valve replacement, who had 4 induced VTs, had recurrence of a
slower right-bundle/right-superior-axis VT (tachycardia cycle length 460 ms) 21 months after first presentation, and ablation was again successful.

**Bundle-Branch Reentry and Focal VT**

Three patients had VTs that were not due to scar-related reentry. One patient with prior mitral valve repair and a nonischemic cardiomyopathy (ejection fraction 10%, end-diastolic internal diameter 7.6 cm) had a VT with right bundle-branch block, left superior axis, with precordial transition at V5 that appeared to have a focal origin originating from the left bundle branch. Because the patient had right bundle-branch block, ablation was not attempted to avoid the risk of a complete heart block. Amiodarone therapy was initiated. The patient died of uncertain cause 8 months after the electrophysiology study. Two patients with depressed left ventricular function (left ventricular ejection fraction 12% and 30%, respectively) who had undergone aortic valve replacement demonstrated bundle-branch reentry, which was abolished with ablation of the right bundle branch. One of these patients had a second VT identified as scar-related reentry that was successfully ablated. One underwent orthotopic heart transplantation 11 days later for chronic heart failure.

In the remaining 3 patients, no VT was inducible. One had a cardiac arrest 6 days after aortic valve replacement and coronary revascularization. An ICD was implanted. He presented 26 months later to an outside facility with recurrent VT. Mapping revealed a large anterior scar and 3 VTs that were rendered noninducible after ablation. The second patient had sustained monomorphic VT 10 days after mitral valve repair and had an electrophysiology study after initiation of amiodarone, with no inducible VT during the electrophysiology study; no ablation was performed. A third patient, who developed symptomatic VT 8 years after mitral valve repair that was not suppressed by sotalol, did not have inducible VT at electrophysiology study; during 45 months of follow-up, rare pace-terminable VT without need for defibrillation was recorded on ICD interrogations.

**Complications and Recurrences**

The median procedure time was 3.1 hours (IQR 2.2, 4.4 hours), with a median fluoroscopy time of 48 minutes (IQR 26, 61 minutes; 114 cGy · cm$^{-2}$ [IQR 32, 156 cGy · cm$^{-2}$]). One patient who required ablation for incessant VT 3 days after mitral valve repair had a periprocedural stroke with residual hemianopsia. No other significant complications occurred. The median follow-up was 1.9 years (IQR 0.8, 3.9 years). Two patients underwent postprocedural orthotopic heart transplantation at 2 weeks or 3 months, respectively, for chronic heart failure that had been present before ablation. Two additional patients died of unknown causes at 7 and 12 months, respectively, after the procedure. Of the 4 patients who died or received a heart transplant, all had depressed left ventricular function (mean left ventricular ejection fraction 17 ± 9%, left ventricular end-diastolic internal dimension 7.4 ± 1.5 cm) without coronary artery disease.

**Discussion**

We found that most clinically significant VTs after valve surgery were due to scar-related reentry in the present referral population. Previous reports have shown bundle-branch reentry and focal VT after valve surgery. The importance of scar-related VT in this population has not been emphasized.
previously. The scars are often, but not always, located in proximity to a valve annulus. Catheter ablation approaches developed largely in patients with VT from infarct scars are often successful for controlling these VTs. Second, VT after valve surgery appears to be bimodal in presentation, with some cases presenting soon after surgery but the majority presenting years later. Because those with early postoperative VT may have been recognized more quickly, treatment (including ablation) may have been performed sooner than those in whom VT presented late. Although the precise time of presentation for late VTs was not known, the median time to initiation of therapy for VT was 12 years. It appears unlikely that these VTs were present for long durations before detection, given their rates and associated symptoms.

Sustained monomorphic VT early after valve surgery is not common. In 1 of the largest series reporting postoperative outcomes, VT of >30 seconds was identified in only 6 of 813 patients with isolated valve-correcting surgery. Narasimhan and coworkers studied 29 patients with sustained monomorphic VT after valve surgery and found bundle-branch reentry in one third of patients. Bundle-branch reentry was particularly prevalent when VT occurred early after surgery. Of the 11 patients with VT within 30 days of surgery, 8 (72%) had bundle-branch reentry compared with only 1 of the 18 patients presenting >30 days after surgery. VT in the later-presenting patients was stated to be “myocardial VT,” although the mechanisms were not determined and mapping and ablation were not reported. Many of these VTs were likely due to prior infarction, because 75% of patients had documented coronary artery disease and depressed ventricular function. The present observations are in agreement with this. Of the 4 patients we studied within 30 days postoperatively, 2 were noninducible, 1 had bundle-branch reentry, and 1 had scar-related reentry, whereas of those 16 presenting >30 days postoperatively, 13 (81.3%) had scar-related reentry. Although we found a smaller proportion of patients with bundle-branch reentry, it should be recognized that referral bias favoring VT that is more difficult to control is likely to have influenced the present patient population.

Patients with scar-related reentry had low-voltage regions, often with low-amplitude, multicomponent, “fractionated” electrograms consistent with the types of scars that cause VT after infarction and in some cardiomyopathies. However, we do not know whether these findings are specific to patients with VT after valve surgery or whether they may occur after valve surgery in the absence of VT. Although the cause of the presumed scar is not clear, several possibilities exist. In contrast to prior reports, we excluded patients with a history of known myocardial infarction. It is possible, however, that a coronary embolism related to the valvular abnormality or at the time of surgery caused an infarct. This appears less likely to be the cause of the scars that were periartotic in location. It is tempting to speculate that the disease process affecting the valve or surgery in the region is somehow related to scarring adjacent to the aortic annulus. Although largely abandoned, anteroaortic scars might be the result of a left ventricular vent placed during surgery, causing incisional reentry. This is speculative, however, and we are further limited by access to operative reports when surgery occurred at another institution many years before our evaluation. It is also possible that some scars are related to replacement fibrosis in the setting of ventricular dysfunction before surgery. The frequent location of scars adjacent to the valve annulus raises concern for possible entrapment of the mapping catheter in a mechanical valve during mapping. We were concerned about and aware of this possibility and fortunately did not encounter it, although it likely remains a risk that warrants attentive monitoring of catheter position.

The vast majority of VTs in the present series had an endocardial origin. The pericardial adhesions from surgery would have made percutaneous epicardial catheterization challenging, and this strategy was not pursued.

Conclusions

Recurrent monomorphic VT after aortic or mitral valve surgery is often due to reentry in a region of ventricular scar, although bundle-branch reentry and focal mechanisms also occur. Catheter ablation is often effective.

Disclosures

Dr Epstein has received speaking honoraria from and/or served in an advisory capacity for Boston Scientific, Inc and Biosense-Webster, Inc. Dr Stevenson has received speaking honoraria from Biosense-Webster, Inc and Boston Scientific, Inc and is a consultant to Biosense-Webster, Inc. The remaining authors report no conflicts.

References


**CLINICAL PERSPECTIVE**

Sustained monomorphic ventricular tachycardia (VT) after aortic and mitral valve surgery is uncommon. The causes are not well-defined, although bundle-branch reentry has been reported. We examined 20 patients without prior infarction referred for ablation of clinically significant monomorphic VT after mitral or aortic valve surgery. Sustained monomorphic VT occurred during index hospitalization associated with valve surgery in 4 patients but was not documented until a median of 12 years postoperatively in 16 patients. Electrophysiology study revealed no inducible VT in 3 patients. VT was due to scar-related reentry in 14 patients, bundle-branch reentry in 2 patients, and focal origin (presumed automatic) in 1 patient. After ablation, 11 patients remained free of spontaneous VT during a median follow-up of 2.1 years. Three patients had repeat ablation for recurrent VT. One patient with incessant VT early after mitral valve repair had a periprocedural stroke with residual hemianopsia. In this selected population, scar-related reentry was the major cause of monomorphic VT late after valve surgery. The cause of the arrhythmogenic scar is not clear. Catheter ablation by methods that target scar-related VTs can be effective.