Clinical Investigation

Transcatheter Electrical Ablation of Right Bundle Branch
A Method of Treating Macroreentrant Ventricular Tachycardia Attributed to Bundle Branch Reentry

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The present study describes the clinical and electrophysiological characteristics of sustained bundle branch reentrant ventricular tachycardia treated with electrical ablation of the right bundle branch. Seven patients presented with syncopal episodes, and six of the seven had documented episodes of ventricular tachycardia. All patients had depressed left ventricular ejection fraction with cardiomegaly. Six of the seven had dilated cardiomyopathy in the absence of significant coronary disease. Twelve-lead electrocardiograms in all seven patients during sinus rhythm were remarkably similar; six demonstrated intraventricular conduction defect resembling left bundle branch block, and one showed left anterior fascicular block. All patients showed prolonged His-to-ventricle intervals during sinus rhythm. Sustained ventricular tachycardia (with atrioventricular dissociation) because of bundle branch reentry was induced in all patients during baseline electrophysiological study. The His-to-ventricle intervals during tachycardia were similar to those seen during sinus rhythm. Electrical ablation of the right bundle branch was accomplished in each patient with delivery of two electrical shocks (170–310 J) through electrode catheters. Right bundle branch block developed on their surface electrocardiogram immediately after the ablation. Follow-up electrophysiological studies showed no inducible ventricular tachycardia. Clinical follow-up showed no recurrence of syncpe or ventricular tachycardia. From the data presented, the following can be concluded. First, right bundle branch ablation is a safe and promising means of treating ventricular tachycardia because of bundle branch reentry and can obviate the need for antiarrhythmic drug therapy and its frequent undesirable side effects. Second, there are common clinical and electrophysiological characteristics that are frequently seen in patients with this tachycardia, and the recognition of these common characteristics should alert the physician to a bundle branch reentrant mechanism of ventricular tachycardia. (Circulation 1988;78:246–257)

Macroreentrant ventricular beats with the His bundle and bundle branches in the reentrant pathway have been demonstrated to occur in the human His-Purkinje system.1–3 A single case of sustained macroreentrant ventricular tachycardia because of bundle branch reentry (Macro-HPS VT) was reported in 1980.4 Subsequently, six cases of such tachycardias were reported in 1982–1983.5–8 Although bundle branch reentry has not been a frequently observed mechanism of ventricular tachycardia, its recognition has important clinical implications regarding treatment. While control of this type of tachyarrhythmia may be difficult with medications, physical interruption of the macroreentrant circuit can be achieved relatively easily with catheter ablative techniques because of the well-defined and accessible electrical landmark of the right bundle branch potential. Preliminary studies8,10 describing catheter ablation of the right bundle branch in such patients indicated that this technique was quite effective. Touboul et al11 have described in greater detail the use of this technique in a patient who suffered from Macro-HPS VT.
In the present study, the clinical characteristics of seven patients who had sustained Macro-HPS VT that was reproducible in the electrophysiology laboratory and who underwent successful treatment with catheter ablation of the right bundle branch are reported.

**Patients and Methods**

**Electrophysiological Evaluation and Drug Testing**

Baseline electrophysiological studies were performed in six patients who were not taking any antiarrhythmic medications. A seventh patient had already been treated with amiodarone for 1 month before our evaluation. Each baseline study was performed with two or three multipolar electrode catheters inserted through antecubital or femoral veins. The catheter tips were positioned in the high right atrium for recording and pacing the right atrium, across the tricuspid valve for recording His bundle and right bundle electrograms, and in the right ventricle for recording and pacing that chamber. Right bundle (RB) or His (H) recordings were always obtained through quadrupolar electrode catheters with 1-cm interelectrode distance [US Catheter and Instruments (USCI) 6F or 7F, Billerica, Massachusetts]. Programmed stimulation was performed at the right ventricular apex, followed by the right ventricular outflow tract if no tachycardia could be induced at the first site. In one patient, pacing was also performed at the left ventricular apex in an attempt to reproduce a clinical tachycardia with a right bundle branch block configuration. Atrial catheters were omitted if a patient had atrial fibrillation.

The following factors were sought in each patient to support the mechanism of bundle branch reentry during an induced ventricular tachycardia: 1) the first beat of tachycardia, as well as all subsequent beats, was preceded by H or RB potentials; 2) during each induction of tachycardia, similar His-to-ventricle (HV) relations were seen, even if different programmed stimulations were used; 3) there was an absence of consistent H or RB deflections between QRS complexes during ventricular pacing at the cycle length of tachycardia; 4) surface QRS morphology, as well as activation sequence of intracardiac electrograms, must be consistent with depolarization of the ventricle through one of the bundle branches; and last and most important, 5) spontaneous variation in ventricle-to-ventricle (VV) intervals usually seen at initiation of tachycardia should be preceded, rather than followed, by similar changes in His-to-His (HH) or right bundle-to-right bundle (RB-RB) intervals.

Follow-up electrophysiological studies obtained to assess antiarrhythmic efficacy of oral medications or ablation were performed with one or more multipolar electrode catheters inserted through either antecubital or femoral veins and positioned in the right ventricle and His bundle region. Programmed electrical stimulation used for induction of ventricular tachyarrhythmias included pacing at three different cycle lengths at two right ventricular sites with up to three premature beats. Six patients underwent right bundle branch ablation because they were intolerant of conventional antiarrhythmic drug treatments or failed to respond to them as judged by clinical recurrence or follow-up testing. The seventh patient chose to undergo right bundle ablation without a trial with medications.

**Technique of Right Bundle Branch Ablation**

A 5F bipolar temporary pacing catheter was inserted through either an internal jugular or subclavian vein and positioned in the right ventricular apex to be used for ventricular pacing should the patient develop complete atrioventricular block after ablation. A new 6F or 7F quadripolar catheter (USCI) was then inserted from the femoral vein, positioned with the tip across the tricuspid valve, and manipulated until an RB recording could be obtained from the distal two poles of the catheter. Usual criteria for verification of RB recording\(^3\) (RB-V<30 msec) may not always apply in these patients because of the presence of His-Purkinje system disease causing prolongation of RB-V intervals. The following criteria were, therefore, used to determine proper catheter positioning for ablation. The RB recording from the distal two electrodes was at least 20 msec later than the H recording, and there was no large atrial recording on the RB tracing.\(^3\) Unipolar electrograms from the two distal electrodes of the ablation catheter were also examined to verify that the tip electrode indeed had the larger RB deflection. Intravenous pentothal was then administered to the patients until they were adequately anesthetized. A 230-J shock was delivered through the tip of the right bundle branch catheter to a back paddle positioned between the left scapula and the spine. The catheter tip served as the cathode. After verification of stable rhythm and blood pressure after the first shock, a second 170-J shock was delivered in the same manner. In the fifth, sixth, and seventh patients, the shock strengths were 310 and 230 J. The choices of shock energies were based on our experience with His bundle ablation. Recently, we have increased the energies used for His ablation to minimize the chances of conduction recovery. After each ablation, an attempt was made to record the RB potential again.

**Results**

**Clinical Characteristics**

Table 1 lists clinical characteristics of the seven patients. Ages varied from 50 to 81 years. Two patients had progressive, severe congestive heart failure at the time of ablation. Six of the seven patients had dilated cardiomyopathies that were unexplained by any significant coronary disease on arteriography. One patient had severe coronary artery disease that most likely represented the cause...
of underlying myopathy. Left ventricular ejection fractions as measured by radionuclide angiography were all depressed, ranging from 15% to 40%. Four of the seven patients had markedly enlarged cardiac shadows on their chest x-rays. The other three patients, who had ejection fractions of 35–40%, had mildly enlarged cardiac shadows. As depicted in Table 1, all seven patients presented with clinical episodes of syncope followed by complete spontaneous recovery. Six of the seven patients had wide QRS tachycardia documented clinically either in hospital or by emergency personnel. Of interest, patient 3 had numerous presyncopal and syncopal episodes during the 2 years before ablation. A final syncopal episode before ablation led to electrocardiogram documentation of two different ventricular tachycardias. One morphology resembled left bundle branch block, while the other was typical of right bundle branch block (Figure 1). Patient 4 did not have clinically documented tachycardia. He

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>SHD</th>
<th>Ejection fraction</th>
<th>Cardiac size*</th>
<th>Clinical symptoms</th>
<th>Documented clinical tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>73</td>
<td>M</td>
<td>IDC</td>
<td>35%</td>
<td>Mild</td>
<td>Syncope weakness and dyspnea</td>
<td>Sustained wide QRS tachycardia; CL&lt;300 msec</td>
</tr>
<tr>
<td>2</td>
<td>50</td>
<td>M</td>
<td>Severe three-vessel CAD</td>
<td>15%</td>
<td>Marked cardiomegaly</td>
<td>Syncope X 3 SCD X 1</td>
<td>Sustained VT; CL = 280 msec</td>
</tr>
<tr>
<td>3</td>
<td>62</td>
<td>M</td>
<td>IDC</td>
<td>40%</td>
<td>Mild cardiomegaly</td>
<td>Frequent presyncope and syncope</td>
<td>Sustained VT; CL = 290 and 250 msec</td>
</tr>
<tr>
<td>4</td>
<td>71</td>
<td>M</td>
<td>IDC</td>
<td>40%</td>
<td>Mild enlargement</td>
<td>Syncope X 4</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>62</td>
<td>M</td>
<td>IDC</td>
<td>26%</td>
<td>Marked cardiomegaly</td>
<td>Syncope X 3</td>
<td>Sustained VT; CL = 250 msec</td>
</tr>
<tr>
<td>6</td>
<td>50</td>
<td>M</td>
<td>IDC</td>
<td>15%</td>
<td>Marked cardiomegaly</td>
<td>Presyn. X 2 Syncope X 1 SCD X 1</td>
<td>Sustained VT; CL = 290 msec</td>
</tr>
<tr>
<td>7</td>
<td>81</td>
<td>M</td>
<td>IDC</td>
<td>18%</td>
<td>Moderate cardiomegaly</td>
<td>Syncope</td>
<td>Nonsustained VT; 12 sec CL = 300 msec</td>
</tr>
</tbody>
</table>

SHD, structural heart disease; IDC, idiopathic dilated cardiomyopathy, no significant coronary artery disease; CAD, coronary artery disease; VT, ventricular tachycardia.

*Cardiac size on chest x-ray.

FIGURE 1. Electrocardiographic documentation of two distinct ventricular tachycardias in patient 6. Note that the morphology shown in Panel A is left bundle branch block with a cycle length of 290 msec, while Panel B shows a tachycardia with right bundle branch block pattern and a cycle length of 260 msec.
was initially admitted to the hospital with three syncopal episodes occurring in 2 days. He had a fourth syncopal episode after admission but recovered consciousness spontaneously before cardiac monitoring. He was then referred for electrophysiological evaluation.

Electrocardiographical variables measured at baseline evaluations before drug therapy are listed in Table 2. Five patients were in sinus rhythm and two were in atrial fibrillation; PR intervals varied from the upper limits of normal to markedly prolonged. All patients had prolonged QRS duration ($\geq 104$ msec). The QRS morphology in lead $V_1$ was suggestive of left bundle branch block in six of the seven patients. The seventh patient’s electrocardiogram showed a typical left anterior hemiblock pattern.

**Electrophysiological Studies**

Pertinent data from control electrophysiological studies are shown in Table 3. Baseline HV intervals were prolonged and varied from 70 to 90 msec in six patients. In the seventh patient, only a distal HV interval was available, which measured 55 msec. Sustained monomorphic ventricular tachycardias were induced repeatedly in each patient with one to three ventricular premature stimulations. The cycle lengths of ventricular tachycardia ranged from 250 to 300 msec. Each induced tachycardia showed atrioventricular dissociation or persistence of atrial fibrillation throughout the tachycardia. Instances of spontaneous RR interval changes during tachycardia were seen in each patient, which were preceded by similar HH or RB-RB interval changes.

Figure 2 illustrates several pertinent observations taken from the electrophysiological study of patient 1. Sustained ventricular tachycardia was initiated with a single premature beat after a short-to-long cycle length change in the basic drive (Panel A). A right bundle branch deflection can be seen before each ventricular beat. Except for the first two beats of tachycardia where minor variation can be seen, spontaneous changes in VV intervals were preceded by identical changes in RB-RB intervals. Note that the His bundle electrograms were inconsistently visualized during tachycardia despite the fact that they were quite well seen with antegrade conduction during sinus rhythm (Panel B). This variability in visualization points out the importance of recording multiple electrograms from adjacent electrodes. Multiple recordings enhance the likelihood of obtaining an adequate H or RB deflection during tachycardia. Panel B shows the initiation of tachycardia with a single premature beat after a constant cycle length drive ($S_3$ and $S_4$ do not capture the ventricle). Despite a different method of

| Table 2. Twelve-Lead Electrocardiogram Before Right Bundle Ablation |
|-------------------------|------------------------|------------------------|------------------------|------------------------|------------------------|------------------------|------------------------|
|                         | Rhythm                 | 1                      | 2                      | 3                      | 4                      | 5                      | 6                      | 7                      |
|                         |                        | Sinus                  | Sinus                  | AF                     | Sinus                  | Sinus                  | AF                     | Sinus                  |
| PR interval (msec)      | 290                    | 210                    | ...                    | 210                    | 340                    | ...                    | 205                    |
| QRS duration (msec)     | 104                    | 160                    | 150                    | 125                    | 120                    | 116                    | 150                    |
| QRS morphology          | LAHB                   | IVCD                   | IVCD                   | IVCD                   | IVCD                   | IVCD                   | IVCD                   |

AF, atrial fibrillation; LAHB, left anterior hemiblock; IVCD, intraventricular conduction defect; LB, left bundle branch block type of IVCD; NA, normal axis; LA, left axis.

<table>
<thead>
<tr>
<th>Table 3. Baseline Electrophysiology Study</th>
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<tbody>
<tr>
<td>Patient</td>
</tr>
<tr>
<td>---------</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
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<tr>
<td></td>
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<td></td>
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<tr>
<td>4</td>
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<tr>
<td>5</td>
</tr>
<tr>
<td>6</td>
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<tr>
<td>7</td>
</tr>
</tbody>
</table>

*Distal His-to-ventricle interval = 55 msec; †pacing from left ventricle, all other stimulation from right ventricle; ‡Left bundle-to-ventricle = 30 msec.

HV, His-to-ventricle; VT, ventricular tachycardia; AV, atrioventricular; SR, sinus rhythm; LB, left bundle branch block; RB, right bundle branch block; AF, atrial fibrillation.
initiating the tachycardia, the RB and V relations remain the same as in Panel A. The tachycardia now terminated spontaneously in association with block within the His-Purkinje system as reflected by absence of a right bundle electrogram after the last beat of tachycardia. Such observations, when available, are additional evidence that the His-Purkinje system forms an integral part of the reentrant circuit. Panel C demonstrates resetting of the tachycardia with a single premature beat delivered to the right ventricle (arrow). Note that the ventricular electrogram on the RB recording showed no significant preexcitation from the premature stimulus. However, the RB-RB interval was shortened to 220 msec with identical shortening of the subsequent VV interval (VV intervals measured on the RB
recording). After this resetting, the tachycardia continued at the original cycle length. These observations are also consistent with His-Purkinje system participation in the reentrant circuit.

Figure 3 is an illustration of tachycardia initiation taken from patient 2 during programmed stimulation performed just before right bundle ablation. Again, tachycardia was triggered by a single premature beat after a short-to-long cycle length change. The patient’s underlying rhythm was atrial fibrillation. Note that spontaneous changes in VV intervals during tachycardia were preceded by similar HH and RB-RB interval changes, implicating participation of the His-Purkinje system in the tachycardia circuit.

In patient 3, both morphologies of clinical ventricular tachycardia were reproduced during electrophysiological study. As illustrated in Figure 4 (Panel A), the left bundle branch block pattern tachycardia was induced during right ventricular pacing with a single premature beat. During sinus rhythm, an H deflection could be seen on the proximal H recording, while a distal H (or RB) deflection was recorded on the distal recording. Once tachycardia was started, however, the proximal H recording could not be easily appreciated, while the distal H (or RB) recording can be clearly seen. Changes in cycle length of the first three beats of tachycardia were preceded by similar changes in HH intervals. The HV intervals of each tachycardia beat remained constant at 55 msec. In the same patient, left ventricular stimulation was also performed just before ablation. This revealed an inducible tachycardia with a typical right bundle branch block morphology with left axis deviation (Figure 4, Panel B) at a cycle length of 250 msec. A left bundle branch potential (LB) was recorded through the proximal electrodes of the left ventricular catheter and showed a deflection preceding each beat of tachycardia. The LB-V intervals during tachycardia were similar to those seen during sinus rhythm. The similarity of these intervals, as well as the early activation of the LB deflection (at least 30 msec earlier than ventricular activation on surface or intracardiac leads), would indicate that the His-Purkinje system was an active participant in the tachycardia circuit. Such timing of the LB deflection would be highly unusual as well as a remarkable coincidence for a myocardial source of the tachycardia with passive activation of the left bundle. The induction of two different tachycardias from opposing ventricles that corresponded to the two clinical tachycardias is consistent with the concept that one macroreentrant pathway could generate two radically different tachycardia morphologies, at least in some cases, by allowing conduction to occur in opposing directions.

Figure 5 illustrates examples of HH interval changes during tachycardia preceding VV interval changes in patients 4, 5, 6, and 7. Panel A shows a tracing taken from patient 4. A single premature ventricular beat after a short-to-long cycle length change initiated the tachycardia. Panel B shows data taken from a follow-up study of patient 5 while he was being treated with quinidine. Note that tachycardia cycle length had slowed somewhat from his baseline study (300 vs. 250 msec; Table 3). The QRS morphology of the tachycardia, however, was the same. Changes in VV intervals were preceded by identical HH interval changes. Panel C shows data taken from the baseline study of patient 6. The underlying rhythm in this patient was atrial fibrilla-
FIGURE 4. Electrocardiographic documentation of ventricular tachycardias induced with programmed stimulation in patient 3. Panel A: Induction of a tachycardia with left bundle branch block morphology. Note that HV intervals during tachycardia remain constant, while changes in VV intervals seen at initiation of tachycardia are preceded by similar HH interval changes. Also note that while a proximal His-bundle electrogram \( [\text{HB}(P)] \) is visible during sinus rhythm, it is difficult to appreciate during tachycardia. Only the distal His bundle \( [\text{HB}(D)] \), which could even be a right bundle recording, is clearly seen. Thus, the use of multiple recordings of the His-bundle electrogram (or right bundle) enhances the likelihood of obtaining adequate tracings from which the diagnosis of Macro-HPS VT can be made. Panel B: Right bundle branch block tachycardia induced in same patient during left ventricular stimulation. Electrograms recorded from a proximal pair of electrodes on the left ventricular catheter show a left bundle branch potential (LB) during sinus rhythm as well as during tachycardia with similar LB-V intervals. Two tachycardias shown here, which correspond to the two clinical tachycardias shown in Figure 1, demonstrate that a macroreentrant circuit involving the His-Purkinje system can potentially propagate in either direction, at least in some patients. (See text for further discussion.) 1, 2, V1, corresponding surface electrocardiographic leads; RV, right ventricular recording.

tion as seen on the proximal recording electrodes of the His bundle catheter (AV). The HV interval during atrial fibrillation was 90 msec. Sustained tachycardia was induced with triple extrastimuli delivered at the right ventricular apex. Large changes in VV intervals could be seen with the first four beats of tachycardia, which were preceded by similar HH interval changes. Panel D shows data taken from the electrophysiological study of patient 7. This episode of tachycardia was triggered by pre-
mature ventricular beats, probably mechanically induced, during catheter adjustment. The His bundle catheter was being advanced into the right ventricle in an attempt to record an RB branch potential. A right bundle deflection could be seen on the middle recording. The third, fourth, fifth, and sixth beats of the tracing were ventricular premature beats that occurred in a short-long-short pattern of cycle length change and initiated a macroreentrant tachycardia. Changes in VV intervals seen early in the tachycardia were preceded by similar changes in RB-RB intervals.

**Right Bundle Ablation**

After the first shock, all patients developed complete right bundle branch block that persisted after the second shock. One patient developed complete atrioventricular block after the second shock, which resolved spontaneously after 48 hours to sinus rhythm with a right bundle branch block and left axis deviation (left axis was present before ablation). Postablation bradycardias occurred in three patients (sinus in two, and atrial fibrillation with slow ventricular response in one) and required temporary ventricular pacing for up to 1 minute.

A typical example, taken from patient 7, of changes in surface and intracardiac electrograms that occurred after ablation is illustrated in Figure 6. Panel A shows the electrograms recorded through the quadrupolar ablation electrode just before ablation. The proximal intracardiac recording shows H deflection with a prolonged HV interval (70 msec). The distal recording shows RB recording with a prolonged RB-V interval and no visible atrial deflection. Panel B illustrates the corresponding electrograms obtained immediately after ablation. Ablation catheter was maintained in the same position. Note the development of a new right bundle branch block on the surface leads, prolongation of the HV interval to 90 msec, and absence of any RB deflection. On follow-up evaluation 2 days after ablation, HV interval had prolonged further to 120 msec, as listed in Table 4.

**After Ablation**

Repeat electrophysiological studies were performed 2–3 days after ablation. The measured HV
intervals are listed in Table 4. Postablation HV intervals were usually longer, increasing up to 40 msec over the corresponding value before ablation. None of the seven patients had inducible ventricular tachycardia or even a single macroreentrant beat at follow-up study.

At discharge, all patients still had right bundle branch block without complete atrioventricular block. However, because of prolonged HV intervals seen even before right bundle ablation in these patients, pacemakers were implanted after ablation in expectation of progressive His-Purkinje system disease that may culminate in complete heart block, especially because one bundle branch had been deliberately rendered nonconductive. None of the patients were discharged on antiarrhythmic medications. Patient 6, who had been on Amiodarone, underwent a repeat electrophysiological study 3 months after ablation. No tachycardia was inducible at this late repeat study.

Clinical Follow-up

Data obtained from the follow-up of these patients are listed in Table 4. Patient 2, who had a severe, dilated myopathy of ischemic origin before ablation, died in refractory congestive failure 3 months after ablation with no recurrence of his clinical ventricular tachycardia. Patient 5 continued to have progressive congestive heart failure. He died 6 months after ablation because of acute gastrointestinal bleeding, complicated by severe congestive heart failure. Patient 3 had a recurrence of "palpitations," and underwent a repeat electrophysiological study at another institution. No ventricular tachycardia could be induced at that time, not even a single macroreentrant beat, despite the use of triple premature extrastimuli.

All remaining patients contacted at follow-up were active and asymptomatic. Over the varying periods of follow-up, no patient developed second-or third-degree atrioventricular block.

Discussion

Efficacy of Right Bundle Ablation

The present study of seven patients with sustained Macro-HPS VT indicated that treatment of this arrhythmia can be effectively achieved with the procedure of right bundle branch catheter ablation, which was well tolerated by these patients. This procedure demonstrated few acute complications and revealed no chronic complications attributable to ablation during a follow-up of up to 55 months.

Despite the fact that these patients experienced multiple syncopal episodes before right bundle ablation, none of them had a recurrence after ablation. While one patient had an episode of "palpitations," the nature of this arrhythmia is unclear. Repeat electrophysiology study after those symptoms revealed no inducible tachycardia even though Macro-HPS VT was easily induced with programmed stimulation before ablation. Five of seven patients were active at the time of latest follow-up. The remaining two patients died at 3 and 6 months after ablation in refractory congestive heart failure. Even at the time of ablation, these two patients were suffering from progressive deterioration of their cardiac function as manifested by recurrent episodes of congestive heart failure that had become more difficult to treat with medications. It would, therefore, appear that prognosis of these patients after control of their ventricular tachycardia with right bundle ablation depended primarily on the nature and progression of their underlying cardiac disease.
TABLE 4.  After-Ablation Data

<table>
<thead>
<tr>
<th>Patient</th>
<th>HV interval (msec)</th>
<th>Program electrical stimulation</th>
<th>12-lead electrocardiogram</th>
<th>Duration of following (mo)</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>70</td>
<td>No inducible VT. Three extrastimuli. Two RV sites</td>
<td>SR, SRB, left axis</td>
<td>1</td>
<td>No recurrent symptoms. Active.</td>
</tr>
<tr>
<td>2</td>
<td>120</td>
<td>No inducible VT. Three extrastimuli. Two RV sites</td>
<td>Atrial fibrillation, RBBB, left axis</td>
<td>3</td>
<td>Died of severe congestive heart failure. No recurrence of VT.</td>
</tr>
<tr>
<td>3</td>
<td>90</td>
<td>No inducible VT. Three extrastimuli. Two RV sites</td>
<td>SR, RBBB, left axis</td>
<td>5</td>
<td>One episode of “palpitations.” Repeat EPS: no VT. Active.</td>
</tr>
<tr>
<td>4</td>
<td>90</td>
<td>No inducible VT. Two extrastimuli. Two RV sites</td>
<td>SR, 1° AVB, RBBB, left axis</td>
<td>55</td>
<td>No recurrent symptoms. Active.</td>
</tr>
<tr>
<td>5</td>
<td>140</td>
<td>No inducible VT. Three extrastimuli. Two RV sites</td>
<td>SR, 1° AVB, RBBB, left axis</td>
<td>6</td>
<td>Died with acute gastrointestinal bleeding. Had severe congestive heart failure.</td>
</tr>
<tr>
<td>6</td>
<td>90</td>
<td>No inducible VT. Three extrastimuli. Two RV sites</td>
<td>Atrial fibrillation, RBBB, left axis</td>
<td>7</td>
<td>No recurrent symptoms. Active.</td>
</tr>
<tr>
<td>7</td>
<td>120</td>
<td>No inducible VT. Three extrastimuli. Two RV sites</td>
<td>SR, 1° AVB, RBBB, left axis</td>
<td>16</td>
<td>No recurrent symptoms. Active.</td>
</tr>
</tbody>
</table>

HV, His-to-ventricle; VT, ventricular tachycardia; RV, right ventricle; SR, sinus rhythm; RBBB, right bundle branch block; AVB, atrioventricular block; EPS, electrophysiological study.

Clinical Characteristics of Patients With Macro-HPS VT

The rather similar clinical characteristics and presentation of these seven patients suggest that certain clinical features should alert a physician to the possible presence of Macro-HPS VT. All of these patients had a dilated myopathy, only one of which could be angiographically attributed to coronary disease. Four of the seven patients showed moderate to marked cardiomegaly on their chest x-rays, and they had ejection fractions of 15–26%. All patients had evidence of His-Purkinje system disease on 12-lead electrocardiogram. Six had left bundle branch block intraventricular conduction defects on their surface electrocardiogram during sinus rhythm, and one had left anterior hemiblock. The HV intervals during sinus rhythm were prolonged in all seven patients. While not seen in the present study, there has also been a study of Macro-HPS VT occurring in a patient with surface electrocardiogram showing right bundle branch block during sinus rhythm,11

The common features in the seven patients were strikingly similar to other studied cases of Macro-HPS VT. A review yielded 11 studied cases of patients with tachycardias suspected of being bundle branch reentry.4–11 Eight of the 11 patients had dilated left ventricles. Two of the remaining three patients had aortic valve disease, but their left ventricular sizes were not reported. No information regarding structural heart disease was available for the remaining patient. Of these 11 patients, two had “narrow” QRS on their surface 12-lead electrocardiograms. The remaining nine patients had QRS duration ranging from 120 to 190 msec (mean, 153 ± 24 msec). The HV interval during sinus rhythm was available in eight patients. One patient had a normal HV of 50 msec. The remaining seven patients had prolonged HV intervals with a mean of 87 ± 10 msec. It appears that the presence of dilated cardiomyopathy, widened QRS, and prolonged HV interval during sinus rhythm are a triad common to patients with Macro-HPS VT. Presence of these characteristics in a patient with syncope or documented ventricular tachycardia should alert physicians to the possible bundle branch reentrant mechanism of ventricular tachycardia, especially if the tachycardia has a typical bundle branch block morphology. Special attention can then be directed to proper recording techniques to verify bundle branch reentrant mechanism during electrophysiology study. Recognition of such a mechanism would have important consequences regarding the available means of treating this entity. While ablation of ventricular tachycardia not having a bundle branch reentrant mechanism may be more difficult to accomplish because of difficulties associated with identifying the source of tachycardia with catheter mapping, ablation of a Macro-HPS VT can be reliably accomplished because there exists a clearly identifiable electrophysiological landmark of the right bundle branch potential. Such a nonpharmacological approach to control of tachycardia could obviate the need for drug testing as well as chronic therapy with antiarrhythmic medications, which are often associated with a considerable amount of side effects.
Characteristics of Induced Macro-HPS VT

Macro-HPS VT was induced in each of the seven patients while pacing from the right ventricle. It was not surprising, therefore, that the induced tachycardias had left bundle branch block QRS patterns. Previous studies have elucidated mechanisms by which a left bundle branch block macroreentrant beat would more likely be induced than a corresponding right bundle branch block pattern when pacing was performed from the right ventricle. However, spontaneous Macro-HPS VT and those induced by pacing at sites other than the right ventricle may well have different morphologies. In the only patient in whom both left and right bundle branch block tachycardias were seen clinically (Figure 1), pacing from the right ventricle induced the left bundle branch block pattern tachycardia, while pacing from the left ventricle induced the other morphology (Figure 4, Panels A and B). Touboul et al.11 have reported the spontaneous initiation of a Macro-HPS VT during sinus rhythm with a right bundle branch morphology. In the same patient, programmed electrical stimulation from right ventricular apex revealed a Macro-HPS VT with a left bundle branch block morphology. Thus, the macroreentrant circuit can conduct in either direction in at least some patients. The morphology of a clinical Macro-HPS VT need not, therefore, be identical to that induced in the electrophysiology lab unless stimulation was attempted from both ventricles. Induction of a Macro-HPS VT with a right bundle branch morphology may require pacing from the left ventricle or, perhaps, from the atria. While Macro-HPS VT of left bundle branch block morphology may appear quite different from that with a right bundle branch block pattern, electrical ablation of the right bundle should be equally efficacious in interrupting the underlying reentrant circuit.

The rate of tachycardias induced in these seven patients were relatively rapid, with cycle lengths ranging from 250 to 300 msec. This range was quite similar to that seen with the nine of 11 studied tachycardias in which cycle lengths could be obtained (283 ± 52 msec). Given the cycle lengths of these tachycardias, it is not surprising that atrioventricular dissociation was seen in these nine cases. However, should a patient manifest 1:1 retrograde conduction during a Macro-HPS VT, it may be difficult to differentiate it from a supraventricular tachycardia with aberrant ventricular conduction. In such cases, it could be important to have both H as well as RB recordings to facilitate such a differentiation.

The HV intervals during Macro-HPS VT in the seven patients appeared to be similar to those seen during sinus rhythm, ranging from 10 msec shorter to 20 msec longer. Again, this range was not unlike that seen in the nine studied tachycardias with available HV intervals for comparison. HV intervals during those nine tachycardias ranged from 20 msec shorter to 90 msec longer than during sinus rhythm. This variation most likely represented interplay of two factors, one tending to prolong HV interval and the other tending to shorten it. The relatively rapid rate of tachycardia would contribute to longer HV times, especially in this group of patients who have underlying conduction defects within the His-Purkinje system. On the other hand, if above the branching point of the bundles, the site of H recording in a given patient need not actively participate in the reentrant circuit and, therefore, could be activated retrogradely in a bystander fashion. Such bystander activation can seemingly shorten the HV interval because of nearly simultaneous activation of the H recording site and upper portion of the bundle branch used antegradely during the induced tachycardia. These two factors in all likelihood interplayed to generate HV intervals during tachycardia, which approximated the HV intervals during sinus rhythm. While not seen in the present study, the possibility of markedly prolonged HV intervals during Macro-HPS VT should be considered. If the bundle branch used for antegrade propagation during reentry has greater delay marked by conduction than the other bundle, the HV interval during tachycardia may be dramatically longer than during sinus rhythm.

Other Tachycardia Mechanisms That May Mimic Macro-HPS VT

Because other unusual tachycardia mechanisms may also present similar electrophysiological pictures, they should be considered in this patient population. Atrioventricular nodal reentrant tachycardia showing retrograde nodoatrial block and antegrade aberrant conduction (a highly unusual combination) could manifest as a wide QRS tachycardia with typical bundle branch block morphology, atrioventricular dissociation, and a His bundle electrogram preceding each ventricular beat. Furthermore, spontaneous changes in RR intervals during such a tachycardia would be preceded by similar changes in HH intervals. In the seven patients, however, such a mechanism can be excluded by the events surrounding tachycardia initiation. To induce atrioventricular nodal reentry from ventricular pacing, the His bundle must be activated twice, once in each opposite direction, between the last premature ventricular beat and the first beat of tachycardia. That only a single H or RB deflection was seen within this interval is incompatible with the above-described mechanism.

Ventricular tachycardia originating from the myocardium with incidental "bystander" activation of the His-Purkinje system could also mimic a Macro-HPS VT. However, spontaneous VV interval changes in such instances would be expected to be followed by similar HH interval changes rather than preceded by them. The examples shown in Figures 2–5 would essentially exclude such possibilities.
Another hypothetical mechanism that may cause a tachycardia resembling Macro-HPS VT with atrioventricular dissociation is reentry involving retrograde conduction over a so-called "nodoventricular" fiber and antegrade conduction through the His bundle with concomitant unilaterial bundle branch block and retrograde nodoatrial block. Such an accessory pathway must also be "concealed" because ventricular preexitation would be readily detected during electrophysiological study. To date, however, there has not been any evidence to indicate that nodoventricular pathways are capable of retrograde conduction. To the contrary, evidence exists to suggest that such pathways are incapable of retrograde conduction. Furthermore, there is a growing suspicion that nodoventricular bypass tracts do not, in actuality, originate from the node but are more likely to be atrioventricular or atriofascicular accessory pathways with nodelike decremental properties. A wide QRS tachycardia with atrioventricular dissociation in such instances would be impossible. Therefore, a tachycardia that may be explained by this improbable and purely hypothetical mechanism is more likely Macro-HPS VT.

**Clinical Relevance of Laboratory-Induced Macro-HPS VT**

The same criteria used to judge "clinical significance" of any induced ventricular tachycardia should be equally applied to suspected Macro-HPS VT. These seven patients were experiencing frequent syncopal episodes before right bundle ablation and yet had no recurrence of these episodes after ablation. It is highly unlikely that the ablative procedure carried out at the high septum without mapping could have fortuitously eliminated focal sources of ventricular tachycardia in seven consecutive cases.

The seven patients in the present study and other patients cited in the literature strongly suggest that Macro-HPS VT can indeed be "clinical" arrhythmias. While the overall incidence of Macro-HPS VT is not high, it is probably higher than generally appreciated, especially in certain populations such as patients with dilated cardiomyopathies showing QRS conduction abnormalities. Therefore, the diagnosis of Macro-HPS VT should be strongly suspected in ventricular tachycardia showing HV interval approximating that of sinus beats and QRS morphology of a typical right or left bundle branch block pattern. In fact, when these electrophysiological criteria are met, the spontaneous or induced tachycardia should be suspected of being Macro-HPS VT unless proven otherwise. The recognition of the underlying mechanism of this arrhythmia has enormous value in expanding the availability of therapeutic measures. Data from the present study would indicate that catheter ablation of the right bundle is a relatively safe procedure that shows promise in eliminating the source of this tachyarrhythmia. Such a procedure could be preferable to long-term antiarrhythmic drug therapy with its associated undesirable side effects.

**References**


**Key Words** • ventricular tachycardia • macroreentry • His-Purkinje system
Transcatheter electrical ablation of right bundle branch. A method of treating macroreentrant ventricular tachycardia attributed to bundle branch reentry.
P Tchou, M Jazayeri, S Denker, J Dongas, J Caceres and M Akhtar

Circulation. 1988;78:246-257
doi: 10.1161/01.CIR.78.2.246

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1988 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

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