Ventricular tachycardia with QRS configuration similar to that in sinus rhythm and a myocardial origin: differential diagnosis with bundle branch reentry

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Introduction Tachycardia with a QRS configuration which resembles that in sinus rhythm is usually thought to be supraventricular. Ventricular tachycardia, with a similar QRS configuration to that in sinus rhythm on the 12-lead ECG, can occur. The mechanisms of this form of ventricular tachycardia have not been previously reported.

Methods and Results The mechanism of ventricular tachycardia was defined during electrophysiological study in five patients. During sinus rhythm, all patients had a wide QRS complex (>0.12 s) on the 12-lead ECG. The morphology remained grossly unchanged during spontaneous, symptomatic tachycardia. Four of the five patients had coronary artery disease and left ventricular dysfunction. The remaining patient had idiopathic dilated cardiomyopathy. The relationship between the His bundle, deflection, the right bundle branch and the QRS complex was evaluated during tachycardia. Atrial and ventricular pacing, and ventricular activation mapping were performed during tachycardia to define the tachycardia mechanism. The tachycardia induced at electrophysiological testing, which was similar to the clinical tachycardia, was proven to be ventricular tachycardia in each patient. The morphology of ventricular tachycardia was right bundle branch block in two patients and left bundle branch block in three patients. The median tachycardia cycle length was 300 ms (range: 260–480 ms). His bundle activation occurred in a 1:1 relationship with ventricular activation during tachycardia in all patients at least intermittently. The tachycardias were thought initially to be bundle branch reentry tachycardia. With further intervention and continued observation, it became clear that His bundle activation was passive and was not required for the tachycardia to sustain. During tachycardia, His bundle activation appeared to precede the local ventricular activation. Instead, the His bundle was activated slowly from the previous ventricular beat causing a long ventricular-His (VH) interval. This was shown by: (1) activation patterns, (2) response to pacing, (3) intermittent VH dissociation, and (4) termination of ventricular tachycardia.

Conclusion A unique form of ventricular tachycardia is described. The QRS complex morphology on the 12-lead ECG during tachycardia was grossly similar to that during sinus rhythm. The His bundle activation was passive and occurred with a long activation time from the ventricle to the His bundle. Although it mimics usual bundle branch reentry, this form of ventricular tachycardia appears to be due to a different mechanism in which the His bundle is not obligatory for the continuation of the reentrant phenomenon.

Key Words: Arrhythmia, bundle branch block, electrophysiology, reentry, tachycardia.
complexes, the exact diagnosis requires electrophysiological testing provided the tachycardia is inducible. When the QRS complex morphology on the 12-lead ECG during tachycardia resembles that in sinus rhythm, the tachycardia is, as a rule, supraventricular in origin. Less well recognized is ventricular tachycardia with QRS complexes similar to those in sinus rhythm. When the QRS complex morphology during ventricular tachycardia resembles that in sinus rhythm, the ventricular activation pattern is likely to involve the His-Purkinje system. This is the case for ventricular tachycardia related to bundle branch reentry. An exit resulting in septal activation similar to that in sinus rhythm is another possibility. Invasive electrophysiological studies, using His bundle electrogram recordings and programmed electrical stimulation, can help determine the mechanism of these tachycardias and can help assess the role of the His-Purkinje system. In this report, electrophysiological testing was used to evaluate the relationship between His-Purkinje and ventricular activation during ventricular tachycardia in patients with a grossly similar QRS complex morphology to that in sinus rhythm.

When the QRS complex morphology during ventricular tachycardia resembles that in sinus rhythm, the ventricular activation pattern is likely to involve the His-Purkinje system. This is the case for ventricular tachycardia related to bundle branch reentry. An exit resulting in septal activation similar to that in sinus rhythm is another possibility. Invasive electrophysiological studies, using His bundle electrogram recordings and programmed electrical stimulation, can help determine the mechanism of these tachycardias and can help assess the role of the His-Purkinje system. In this report, electrophysiological testing was used to evaluate the relationship between His-Purkinje and ventricular activation during ventricular tachycardia in patients with a grossly similar QRS complex morphology to that in sinus rhythm, and to show that the origin was myocardial. Identification of the exact mechanism is essential as ablation of the right branch would not be the appropriate therapy.

Methods

Patient characteristics

Five patients with symptomatic, wide QRS complex tachycardia (QRS width > 0.12 s on the 12-lead ECG) were referred for electrophysiological testing. The clinical tachycardias were initially suspected to be supraventricular tachycardias and treated with either a calcium channel blocker or digoxin. On this therapy, all patients had recurrent tachycardia episodes. The clinical characteristics are presented in Table 1. All patients had a baseline bundle branch pattern during sinus rhythm. The tachycardia and the sinus rhythm were considered similar when they resembled each other in QRS morphology on 12-lead ECG, including bundle branch block pattern and axis orientation. Each patient had at least one episode of a spontaneous wide QRS complex tachycardia with a similar 12-lead morphology as in sinus rhythm (Fig. 1).

Cardiac catheterization, including coronary angiography and left ventriculography, was performed in each patient. Four patients had coronary artery disease and had a history of myocardial infarction before evaluation. No patient was in the acute phase of myocardial infarction. The remaining patient had idiopathic dilated cardiomyopathy.

Electrophysiological study

After written, informed consent, electrophysiological testing was performed in all patients using a standard protocol of antiarrhythmic medication for five or more half-lives, and all patients had a repeat electrophysiological test after receiving an antiarrhythmic drug. Four or more electrocardiographic leads, filtered between 0.5 and 100 Hz, were recorded simultaneously with bipolar endocardial electrograms from His bundle and right ventricular sites, filtered between 30 and 500 Hz. All data were monitored on a multichannel oscilloscope (Siemens-Elema, Inc, Solna, Sweden or Electronics for Medicine, White Plains, NY, U.S.A.) recorded simultaneously on magnetic tape (Teac model XR-510 Montebello, CA, U.S.A.) and printed on an ink-jet recorder (Mingograph, Siemens-Elema) or on photographic paper (Electronics for Medicine, White Plains, NY, U.S.A.) for subsequent review. Programmed electrical stimulation was performed using an optically isolated constant current source stimulator (Bloom Associates, Ltd, Narberth, PA, U.S.A. or Medtronic 1349A, Minneapolis, MN, U.S.A.) at a pulse duration of 2 ms and at twice diastolic threshold.

The protocol used to induce tachycardia included single premature atrial extrastimuli after an eight-beat drive train and rapid atrial pacing to assess atrioventricular conduction and the presence of inducible supraventricular tachycardia. Ventricular tachycardia induction was attempted by delivery of up to three premature extrastimuli after an eight-beat drive train at

Table 1  Clinical characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/gender</th>
<th>Diagnosis</th>
<th>Ejection fraction (%)</th>
<th>QRS characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>78/M</td>
<td>CAD</td>
<td>&lt;= 20 (LV)</td>
<td>LAD, LBBB</td>
</tr>
<tr>
<td>2</td>
<td>58/M</td>
<td>CAD, LV resection</td>
<td>&lt;= 20 (LV)</td>
<td>LAD, LBBB</td>
</tr>
<tr>
<td>3</td>
<td>75/F</td>
<td>CAD</td>
<td>&lt;= 20 (LV)</td>
<td>LAD, LBBB</td>
</tr>
<tr>
<td>4</td>
<td>46/M</td>
<td>CAD, LV aneurysm</td>
<td>15 (LV)</td>
<td>LAD, RBBB</td>
</tr>
<tr>
<td>5</td>
<td>31/M</td>
<td>DCM</td>
<td>49 (LV)</td>
<td>LAD, RBBB</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ejection fraction (%)</th>
<th>QRS characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axis</td>
<td>Configuration</td>
</tr>
</tbody>
</table>

CAD, coronary artery disease; DCM, idiopathic dilated cardiomyopathy; LV, left ventricle; RV, right ventricle; RBBB, right bundle branch block; LBBB, left bundle branch block; LAD, left axis deviation.
two paced cycle lengths from the right atrium and right ventricle (two sites: right ventricular apex and infundibulum if necessary). His bundle activation was recorded during ventricular tachycardia induction. The endpoint of the protocol was the induction of the clinical morphology, sustained, monomorphic tachycardia or completion of the entire protocol. A 12-lead ECG was obtained during each induced tachycardia.

**Assessment of the origin of the tachycardia**

During tachycardia, intra-cardiac recordings allowed the evaluation of the relationships between the His bundle, the right bundle (when recorded) and the QRS complex. Attempts were made to influence the activation relationship without disrupting the tachycardia and to dissociate the His bundle from the tachycardia by rapid atrial and ventricular pacing, or by timed premature extrastimuli. Special attention was given to the His-ventricular (HV) and ventricular-His (VH) relationships. The association and timing of the VV and HH intervals were assessed during spontaneous or induced variation in tachycardia cycle length.

**Criteria to diagnose ventricular tachycardia**

In the electrophysiology laboratory, the following criteria were used to diagnose ventricular tachycardia: (1) dissociation of the His bundle from the QRS complex during tachycardia, (2) local ventricular activation

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**Figure 1.** Morphological comparison of sinus rhythm and ventricular tachycardia. 12-lead ECGs during both sinus rhythm and the ventricular tachycardia are illustrated. (a) ECGs from Patient 4 with a right bundle branch block morphology. (b) ECGs from Patient 1 with a left bundle branch block morphology.
during tachycardia preceding the QRS complex and His bundle recordings, (3) HV activation (HV interval) in tachycardia shorter than that in sinus rhythm, and (4) reversal of the His bundle/right bundle activation inconsistent with the bundle branch morphology and different from sinus rhythm (i.e. if the morphology of the tachycardia is similar to that in sinus rhythm, a supraventricular rhythm would be inconsistent with a shift in the activation pattern of the right bundle and the His bundle except if a concealed Mahaim fibre is present).

In all cases, antegrade and retrograde pre-excitation was excluded. Atrifascicular, nodovenous and nodofascicular accessory pathways were considered and were eliminated as the cause for tachycardia. Ventricular tachycardia was diagnosed in all patients as the cause for the tachycardia.

### Intra-operative mapping, catheter mapping and attempted ablation

Two patients (Patients 4 and 5) underwent intra-operative mapping of the ventricular tachycardia for purposes of myocardial resection and/or cryoablation. In one patient (Patient 5), a multipolar sock electrode containing 56 bipolar pairs was positioned on the epicardial surface of the heart (BARD Electrophysiology, MA, U.S.A.). The epicardial activation map during tachycardia recorded the earliest site of activation in Patient 5 for purposes of cryoablation. Patient 4 underwent sequential endocardial bipolar mapping to record the earliest site of endocardial activation during ventricular tachycardia.

Two patients (Patients 1 and 2) underwent sequential site catheter mapping procedures during tachycardia. Transcatheter ablation was attempted at the earliest endocardial activation sites in these patients.

### Results

In all patients, electrophysiological testing induced ventricular tachycardia episodes that were similar in morphology and rate to the clinical tachycardia. All tachycardias were confirmed to be ventricular in origin using the above mentioned criteria.

In all patients, there was an apparent association between the His bundle activation and ventricular activation [Fig. 2(b)]. Dissociation of the His bundle activation from ventricular activation was detected during continuous observation of the tachycardia.

### Morphological and electrophysiological characteristics of the tachycardias

In sinus rhythm, all patients had bundle branch block, and the spontaneous and induced tachycardias had similar 12-lead ECG morphology. Two patients had right bundle branch block and three had left bundle branch block (Table 1). The cycle length of the tachycardias varied from 260 to 480 ms (Table 2). The tachycardias in Patient 4 had similar QRS morphologies, but two distinct cycle lengths (245 ms and 420 ms). Representative cases are illustrated in Fig. 1.

In all patients, tachycardias were induced reproducibly from the right ventricular apex with double premature extrastimuli. The tachycardias were all terminated by rapid ventricular pacing. Tachycardia induction was not suppressed by intravenous procainamide. Other electrophysiological characteristics are listed in Table 2.

Atrioventricular dissociation or block was present intermittently during tachycardia, as documented by the electrophysiological study in all five patients.

Initially, a concordant relationship was seen between the His bundle and ventricular activation during all induced tachycardias [Fig. 2(a)]. The ‘apparent’ HV activation times varied from 30 to 170 ms with a median HV interval of 62.5 ms in these five patients (Table 2). In Patient 4, the local ventricular electrogram during tachycardia preceded the QRS complex at one of the two rates of the same morphology tachycardia [Fig. 2(b)]. In this patient, HV activation in the faster tachycardia induced, was apparently shorter (HV=30 ms) than that in sinus rhythm. The His bundle activation was not necessary for continuation of ventricular tachycardia, as 2:1 VH conduction was noted with prolonged observation [Fig. 2(b)]. The slower tachycardia induced in this patient had an apparently prolonged HV interval (Table 2).

Three of five patients had ‘apparent’ HV intervals during tachycardia that were prolonged, as seen in bundle branch reentry. In four patients, the HV interval recorded during tachycardia was longer or equal to the HV interval in sinus rhythm (Table 2). In Patient 5, as well as in one episode of ventricular tachycardia of Patient 4, the HV was slightly shorter but the His bundle electrogram still preceded the onset of the QRS complex.

HV dissociation was found in all patients during tachycardia with prolonged observation. In two patients (Patients 3 and 4), atrial pacing captured the His bundle antegrade without resetting, or otherwise influencing, the tachycardia rate or morphology (Fig. 3). In two other patients (Patients 2 and 5), HV dissociation occurred spontaneously during the tachycardia without change in cycle length. In the remaining patient (Patient 1), analysis of the His-right bundle activation sequence showed a varying activation pattern during tachycardia. The His bundle activation either preceded the right bundle recording (Fig. 4, Panel A) or changed to be activated nearly simultaneously or slightly after the right bundle recording (Fig. 4, Panel B), despite the presence of left bundle branch block morphology. Special attention was given to assure that there was no catheter displacement during intra-cardiac recordings. In addition, HV dissociation was observed in Patient 1 during ventricular tachycardia with accompanying atrial flutter. AH interval was fixed in contrast with the HV relationship.
Premature ventricular extrastimuli and rapid ventricular pacing were capable of activating the His bundle retrogradely. The His bundle was activated with a long activation time (range: 200–400 ms). The ‘apparent’ $HV$ interval actually represented an extremely long retrograde or $VH$ interval, present only with short coupling intervals. Curiously, these long $VH$ intervals were associated with normal $HV$ intervals in sinus rhythm, and with no $HV$ prolongation with premature atrial extrastimuli in sinus rhythm. In another patient (Patient 4), the retrograde His bundle activation was variable during tachycardia.

Activation mapping was performed in four of the five patients during the induced clinical morphology, tachycardia to determine the earliest site of ventricular activation. In four patients, left ventricle and right ventricle endocardial activation mapping was performed in the electrophysiological laboratory (Table 3). In two

Figure 2. Surface ECG and endocardial electrogram recordings from Patient 4. During one episode of tachycardia in this patient, an apparent 1:1 $HV$ relationship resembling that seen in usual bundle branch reentry was noted (a). With further observation, an intermittent 2:1 $VH$ conduction, then 2:1 $VA$ conduction, was registered. The tachycardia continued at almost the same rate, despite the lack of 1:1 His bundle activation (b). Also shown in (b) is that local activation from the apex of left ventricle preceded the QRS complex (last electrogram recording).
patients (Patients 4 and 5), endocardial and epicardial activation mapping was performed in the operating room. In these two patients, the earliest site of ventricular activation was at the left ventricular apex [Fig. 2(b)].

Patient 5 had an unsuccessful attempt at endocardial catheter ablation and underwent surgery. In the operating room, the earliest site activation appeared to be at the epicardial surface of the left ventricular apex and was successfully ablated. This patient had normal coronary arteries and slightly impaired left ventricular function. Seven years after surgery, he remains free of tachycardia. Patient 4, with a severe ischaemic cardiomyopathy and a left ventricular aneurysm, had an early ventricular activation site during catheter mapping (of both fast and slow ventricular tachycardias) at the anterolateral left ventricular wall. Pace mapping showed nearly identical 12-lead ECG morphologies, similar to that in sinus rhythm and to the clinical ventricular tachycardias at multiple disparate pacing sites in the left ventricle. Both attempted catheter and surgical ablation were unsuccessful in this patient.

**Discussion**

The cause for tachycardia with wide QRS complexes includes: (1) ventricular tachycardia, (2) supraventricular tachycardia with pre-existing or rate-dependent bundle branch block, or (3) supraventricular tachycardia with antegrade activation via an accessory pathway. Based on intracardiac recordings, the wide QRS complex tachycardia was proven to be ventricular tachycardia in the five patients reported in the present study.

A wide QRS complex tachycardia can have similar ECG morphology to that in sinus rhythm and still be ventricular in origin. While uncommon, the close similarity in morphology invites careful assessment of the tachycardia mechanism. Since the morphology is that of a bundle block similar to that seen in sinus rhythm, it is generally thought that the tachycardia originates in the His-Purkinje system and may be due to bundle branch reentry. Careful observation of the ECG may depict some subtle differences in morphology.

**Table 2** Characteristics of sinus rhythm and tachycardia

<table>
<thead>
<tr>
<th>Patient</th>
<th>Rhythm</th>
<th>Cycle length</th>
<th>QRS</th>
<th>AH</th>
<th>HV</th>
<th>VH</th>
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<tbody>
<tr>
<td>1</td>
<td>SR</td>
<td>820</td>
<td>180</td>
<td>100</td>
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</tr>
<tr>
<td></td>
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<td>180</td>
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<td>70</td>
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<tr>
<td></td>
<td>VT</td>
<td>358</td>
<td>150</td>
<td>170</td>
<td>188</td>
<td></td>
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<tr>
<td>3</td>
<td>SR</td>
<td>970</td>
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<td>120</td>
<td>70</td>
<td>na</td>
</tr>
<tr>
<td></td>
<td>VT</td>
<td>480</td>
<td>120</td>
<td>na</td>
<td>70</td>
<td>410</td>
</tr>
<tr>
<td>4</td>
<td>SR</td>
<td>670</td>
<td>150</td>
<td>70</td>
<td>40</td>
<td>na</td>
</tr>
<tr>
<td></td>
<td>VT-1</td>
<td>245</td>
<td>150</td>
<td>na</td>
<td>55</td>
<td>190</td>
</tr>
<tr>
<td></td>
<td>VT-2</td>
<td>420</td>
<td>150</td>
<td>na</td>
<td>30</td>
<td>390</td>
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<tr>
<td>5</td>
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<td></td>
<td>VT</td>
<td>300</td>
<td>160</td>
<td>na</td>
<td>35</td>
<td>265</td>
</tr>
</tbody>
</table>

VT, ventricular tachycardia; SR, sinus rhythm; na, not applicable. Numbers for cycle length and intervals are in ms.

**Figure 3.** Atrial pacing alters the HV relationship. During one episode of ventricular tachycardia in Patient 4, atrial pacing was applied at high right atrium, at a cycle length different from that of ventricular tachycardia (260 ms vs 420 ms). These pulses were able to conduct to and capture the His bundle, therefore were able to alter the VH relationship without interrupting the ventricular tachycardia.
between the QRS complexes of the tachycardia and those in sinus rhythm, which could suggest that the mechanism of the tachycardia may not be bundle branch reentry. This has therapeutic implications. However, electrophysiological testing is particularly indicated in order to determine the exact mechanism. During intra-cardiac recordings in these patients, the His bundle was activated passively as a bystander during tachycardia, and an 'apparent' short HV interval was actually a prolonged VH activation with passive His bundle activation causing an apparent association of the His to the following QRS complex, as shown in Fig. 5. The tachycardias did not depend on His bundle or atrial activation to perpetuate themselves. This is supported by the continuation of ventricular tachycardia, even in the absence of a preceding His activation usually seen in typical His-Purkinje reentry tachycardia.

The study patients had a reentrant mechanism for their tachycardia, as it could be induced and terminated with premature ventricular extrastimuli. Entrainment criteria, including entrainment from the atrium in one patient (Patient 4), were seen in three patients confirming a reentrant mechanism\[^3,4\]. The ventricular tachycardias were related to reentry, but reentry may be due to a number of different mechanisms. These include: (1) macroreentry involving the His-Purkinje system (bundle branch reentry tachycardia), (2) reentry solely requiring the ventricular myocardium and part of the Purkinje system (fasciculo-ventricular reentry), or (3) reentry in the fascicles (interfascicular reentry).

Bundle branch reentry was the mechanism reported by Touboul et al.\[^5\] in their three patients with tachycardias with QRS complexes similar in morphology to those seen in sinus rhythm. This was also the case for the seven patients described by Cohen et al.\[^6\].

Bundle branch reentry tachycardia is reported to be responsible for 6% of ventricular tachycardias, and has been studied extensively by the team of Akhtar et al.\[^7-12\]. Unlike other types of ventricular tachycardia, it can be treated by ablation of the right bundle branch.\[^8,9\]. The diagnosis of bundle branch reentry tachycardia is based on His-Purkinje/ventricular activation relationships\[^10\]. In the patients from the present study, the tachycardias were inconsistent with the conventional bundle branch reentry criteria described by previous studies\[^5-12\], since His bundle activation was not needed for tachycardia.

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**Table 3 Earliest site of ventricular activation during tachycardia**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Catheter mapping</th>
<th>Surgical mapping</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>LV infero-apical wall</td>
<td>Not done</td>
</tr>
<tr>
<td>2</td>
<td>RVOT</td>
<td>Not done</td>
</tr>
<tr>
<td>4</td>
<td>LV anterolateral wall</td>
<td>LV apex</td>
</tr>
<tr>
<td>5</td>
<td>LP apex</td>
<td>LV apical epicardium</td>
</tr>
</tbody>
</table>

LV, left ventricle; RVOT, right ventricular outflow tract.
perpetuation and because of the changing relationships between His bundle and right bundle activation (Fig. 4).

The implications of this study suggest a circumspect approach to ablation in patients with HV activation concordance without further careful evaluation of the tachycardia mechanism. Since these patients had a His bundle activation apparently preceding each QRS complex during tachycardia, a diagnosis of the usual bundle branch reentry tachycardia seemed likely. On further evaluation, VH conduction was found to be the cause for His bundle activation. The long VH conduction time was misleading. Although VH conduction during ventricular tachycardias occurs frequently, peculiarly long VH conduction times, as shown here (Fig. 5), have not been described.

Intra-cardiac, including His bundle, recording has been viewed as an essential way to clarify the origin and mechanism of a wide complex tachycardia. A very short HV interval (shorter than HV in sinus rhythm) indicates that the His bundle is activated retrogradely rather than anterogradely. By the same argument, an abnormally long HV interval may indicate antegrade activation by way of the His Purkinje system. This has been confirmed in studies by Akhtar et al.[11,12]. Patients exhibiting bundle branch reentry tachycardia have a functional conduction delay or block that results in prolongation of the HV interval. This prolongation is the driving force behind the macroreentry circuit.

Lack of ability to capture the His bundle may be a criterion of bundle branch reentry since it may remain tonically activated and refractory[13]. The excitable gap may be small. However, the same may be true with passive His bundle activation. The lack of antegrade capture of the His bundle may be amplified by marked delays in conduction to the His bundle.

Downar[14] has described endocardial and epicardial activation patterns in a single patient with similar QRS complex configuration in both sinus rhythm and ventricular tachycardia. Using detailed epicardial and endocardial mapping techniques, he suspected the mechanism to be reentrant and involving part, but not all, of the His-Purkinje system and the ventricular myocardium[15]. In addition, his data suggested that the origin of the tachycardia was at a distance from the His bundle, similar to that in the present patients[14]. Gilmour et al.[16] found similar results in an experimental study.

It is possible that in the study patients, reentry may involve part of the Purkinje system and some ventricular myocardium but not the His bundle (Fig. 6(c)). Alternatively, it is possible that reentry was in the fascicles as has been described[13,17].

Implied mechanisms for the similar ECG morphology in tachycardia to sinus rhythm include a site of origin of the tachycardia near to the site of exit from the Purkinje system, and a tachycardia that includes the Purkinje system as part of the reentry circuit. Ventricular activation may proceed through the His-Purkinje system without participation of the His-Purkinje system in the reentry circuit. It is possible for conduction to proceed through the His-Purkinje system during tachycardia with the same activation sequence as that in sinus rhythm, but without the need for the His-Purkinje system to participate in the reentrant circuit of the tachycardia.

Figure 6. Diagrams illustrate possible mechanisms that can explain His bundle activation during ventricular tachycardia. (a) The His bundle is represented as a necessary limb of the reentry circuit. This exists in typical bundle branch reentry. (b) Special type of bundle branch reentry, in which the His bundle is activated passively and is not part of the obligate reentry circuit. Slow retrograde activation is shown with the stippled line. In this case, it would be highly unusual to find block in the common bundle before His activation, if such an anatomical-electrophysiological possibility can even exist. (c) The most likely possibility that explains the present findings. In this case, fascicular activation occurs via retrograde passive activation and then proceeds to the His bundle. The authors suspect that this fascicular/ventricular reentry with passive activation of the His bundle explains these findings.
Conclusion

This paper describes a unique form of ventricular tachycardia with a similar QRS morphology on the 12-lead ECG to that in sinus rhythm, but with a mechanism that does not appear to be due to bundle branch reentry. The mechanism appears to involve ventricular myocardium, although part of the Purkinje system with slow retrograde activation of the His bundle remains possible.

References


