Recurrent Sustained Ventricular Tachycardia

4. Pleomorphism

MARK E. JOSEPHSON, M.D., LEONARD N. HOROWITZ, M.D., ARDESHIR FARSHIDI, M.D., SCOTT R. SPIELMAN, M.D., ERIC L. MICHELSO, M.D., AND ALLAN M. GREENSPAN, M.D.

SUMMARY Two or more morphologically distinct ventricular tachycardias were observed during electrophysiologic study in 14 patients with chronic sustained ventricular tachycardia. Nine of these patients had clinical ventricular tachycardia with multiple morphologies. During the study 13 patients manifested both right bundle branch block (RBBB) and left bundle branch block (LBBB) morphologies. The remaining patient had RBBB with both right and left axis deviation. Changing morphologies were observed spontaneously in four patients and could be produced in all 14 by ventricular stimulation. In 12 patients both RBBB and LBBB originated in the left ventricle, and in 11 of these patients, from within a left ventricular aneurysm. Diastolic fragmented activity representing reentry was unchanged during both morphologies in four patients and during one morphology in five patients. Epicardial mapping confirmed the aneurysm as the site of origin of multiform ventricular tachycardias in two patients. Our data suggest that 1) ventricular tachycardia is frequently pleomorphic; 2) multiple morphologies usually represent variable exit sites and/or ventricular activation during the same tachycardia; and 3) there is a frequent association of pleomorphic ventricular tachycardia with left ventricular aneurysm.

THE SITE OF ORIGIN of ventricular arrhythmias has usually been determined by electrocardiographic criteria. It has been assumed that a left ventricular site of origin produces right bundle branch block (RBBB) morphology, and right ventricular origin left bundle branch block (LBBB) morphology.1,4-6 Hence, multiple morphologies have been considered to represent multifocal origins of ventricular arrhythmias. However, recent experimental data using endocardial and epicardial mapping of ventricular tachycardias in the postinfarction canine model suggest that the ECG, and occasionally epicardial mapping, may not be adequate to localize a site of origin of ventricular tachycardia.6-9 We recently described a technique for endocardial mapping of ventricular tachycardias; the results confirmed these animal studies.9 The demonstration that ventricular tachycardia with LBBB morphology may arise in the left ventricle was particularly important. Recent experimental studies have shown that fragmented diastolic activity during such arrhythmias represents reentrant activity.10-14 This study evaluates multiple distinct morphologies of ventricular tachycardia in patients to determine the relationship of morphological variations, site of origin and endocardial activation. The therapeutic implications (regarding surgery) of the electrophysiologic evaluation of these patients are discussed.

Methods and Materials

After giving informed consent, 26 patients with sustained ventricular tachycardia underwent electrophysiologic evaluation in the non-sedated, postabsorptive state. Although each patient had minor spontaneous or stimulation-induced changes in ventricular tachycardia morphology during the study, 14 patients had two or more morphologically distinct tachycardias characterized by a greater than 90° shift in frontal plane axis and/or a change of bundle branch block pattern. They included 11 of 13 patients with left ventricular aneurysms and three of 13 patients without aneurysms. Nine of these 14 patients had polymorphic ventricular tachycardia clinically. These 14 patients form the basis of this report (table 1). The patient population included 12 men and two women ranging in age from 21–63 years. Eleven had coronary artery disease with left ventricular aneurysms.

Three to six bi-, tri-, and/or quadripolar electrode catheters with a 1-cm interelectrode distance were inserted percutaneously or by cutdown and positioned under fluoroscopic control at sites within the heart. These sites usually included the atrioventricular junction at the His bundle recording position, right ventricular apex, coronary sinus, right atrium and left ventricle. The right and left ventricular catheters were also used for endocardial mapping.8 The intracardiac electrograms were filtered at 40–500 Hz and were simultaneously displayed with one to three surface
1. premature depolarization; 460
CIRCULATION
1-4-cm deflections. Signals ranged from 1-15 mV in amplitude and gains were adjusted to maintain 1-4-cm deflections.

In each case the tachycardias were initiated by ventricular, and in one case, atrial stimulation, which included incremental pacing and/or the introduction of progressively premature single and/or double extrastimuli according to previously described methods. Stimulation was accomplished using a specially designed programmable stimulator and a constant current source (Bloom Associates Ltd, Narberth, Pennsylvania). The site of origin of the tachycardia or region of myocardium in which reentrant electrical activity was presumably located was defined by the earliest discrete local ventricular electrogram or fragmented diastolic activity, as previously described. Such fragmented activity has been considered to represent recording of reentrant activity, and should be distinguished from discrete diastolic potentials recorded by epicardial mapping during surgery. A reentrant ventricular tachycardia should be preceded by prolonged diastolic fragmentation before the first complex and between subsequent complexes of the tachycardia.

In each case of diastolic fragmented activity, artifact was excluded by the demonstration of a temporal relationship of this activity to the tachycardia. A flat diastolic baseline was observed immediately before initiating and immediately after terminating the tachycardia. Furthermore, such activity was not noted during pacing unless the tachycardia was induced, suggesting catheter movement was not responsible for these potentials. Since the morphology of the initiated ventricular tachycardia was usually constant, it was only with this form that diastolic activity preceded the onset of the tachycardia. When the

**Table 1. Clinical Data**

<table>
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<tr>
<th>Case</th>
<th>Age</th>
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<td>RBBB</td>
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<td>RBBB</td>
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<td>LBBB</td>
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Abbreviations: ASHD = atherosclerotic heart disease; ASMI = anteroscpetal myocardial infarction; AVB = atrioventricular block; HCVD = hypertensive cardiovascular disease; IMI = inferior myocardial infarction; IVCD = intraventricular conduction defect; LAD = left axis duration; LBBB = left bundle branch block; LV-An = left ventricular aneurysm; MR = mitral regurgitation; NHD = no heart disease; RBBB = right bundle branch block; RHD = rheumatic heart disease; VPD = ventricular premature depolarization; VT = ventricular tachycardia; CL = cycle length.
morphology changed either spontaneously or in response to stimulation, the relationship of the fragmented diastolic activity with the QRS varied. However, a presystolic component could be obtained during the second morphology with slight catheter manipulation.

The data were displayed on a multichannel oscilloscope (Electronics for Medicine, DR 16, White Plains, New York), stored on an eight-channel tape (Honeywell 5600), and later retrieved on photographic paper at speeds of 150–200 mm/sec. In two cases intraoperative activation mapping was performed using a hand-held bipolar probe with a 1-mm interelectrode distance according to previously reported methods. The data obtained during intraoperative mapping were compared with that obtained during endocardial catheter mapping before surgery.

**Results**

Thirteen of the 14 patients had both RBBB and LBBB morphologies during ventricular tachycardia (table 1). In the remaining patient (case 5), the tachycardia had a RBBB morphology with both left and right axis deviation (−75 and +135°, respectively).

**Morphological Changes and Relationship to Site of Origin**

Four patients had spontaneous alterations during the electrophysiologic study. Such changes were either gradual (fig. 1) or abrupt (fig. 2). In these four patients ventricular stimulation produced changes similar (RBBB to LBBB) to those occurring spontaneously (fig. 3). Morphologic changes could be induced by rapid ventricular pacing (375–200 msec) in all 14 patients and by the introduction of one or two ventricular extrastimuli in nine patients (fig. 4). Changes in types of right (R, qR, rsR') or LBBB (QS, rS) configurations resulted from different ventricular activation patterns despite an unchanged site of origin. However the relationship between the electrical activity at the site of origin and the onset of the QRS of different morphologies varied.

In all cases of ventricular tachycardia associated with left ventricular aneurysms (11 of 14) endocardial mapping revealed that both RBBB and LBBB morphologies originated within the aneurysm as documented by earliest discrete electrogram or by the demonstration of diastolic and systolic activity. In cases 3 and 7, the different morphologies appeared to have different sites of origin, i.e., both left and right ventricles (fig. 5). We could not perform extensive mapping of both ventricles, however, during both morphological forms. However, no fragmented diastolic activity was observed during either morphology in either patient. Neither of these patients had coronary artery disease. In the remaining patient without an aneurysm the site of origin of both morphologies was in the left ventricle.

Electrical activity at the site of origin associated with one tachycardia morphology was analyzed during changes in morphology. In nine patients with ventricular aneurysms the site of origin was recognized by fragmented diastolic activity recorded in the aneurysm. The source of this activity is unclear. It may come from Purkinje fibers or small islands of viable myocardium which may be observed in human aneurysms (Spear et al., unpublished observations) and/or from areas of viable myocardium around the

![Figure 1](https://example.com/figure1.png)  
*Figure 1*. Gradual change in morphology during ventricular tachycardia (case 11). From top to bottom are ECG lead V1, and electrograms from the atrioventricular junction (AVJ), right ventricular apex (RVA), coronary sinus (CS), left ventricular aneurysm (LV-An), and time lines (T). Ventricular tachycardia with a right bundle branch block morphology appears in the first three complexes. The site of origin is recorded in the LV-An which shows low amplitude fragmented diastolic activity 50 msec before the onset of the QRS (left vertical dashed line). The QRS then gradually changes to a left bundle branch block morphology in the last three complexes. Activity in the LV-An is earliest, beginning before the QRS (right vertical dashed line) and its configuration and cycle length are unaffected by the morphologic changes. The slight presystolic activity in the AVJ, RVA, and CS probably represents the oncoming wavefront from the LV-An. Time lines are 10 and 100 msec.
Figure 2. Abrupt change in QRS morphology during ventricular tachycardia (case 11). The figure is arranged as in figure 1. The ventricular tachycardia morphology changed abruptly from a right to left bundle branch block pattern following the fifth complex. The change in morphology occurs with little or no change in timing of electrograms from either the atrioventricular junction (AVJ), right ventricular apex (RVA), or the left ventricular aneurysm (LV-An). The major change is a sudden delay in activation of the posterior left ventricle as recorded on the coronary sinus (CS). Thus, the change in QRS morphology primarily represents a change in activation pattern within the left ventricle with the site of origin at the LV-An unchanged. During both morphologies low amplitude presystolic potentials appear (left of vertical dashed lines).

Figure 3. Morphologic changes induced by rapid ventricular pacing (case 11). The figure is organized identically to figures 1 and 2. Ventricular tachycardia with left bundle branch morphology is shifted to a right bundle branch block morphology by a burst of rapid ventricular pacing (arrows) initiated from the right ventricular apex (RVA) after the third complex. More obvious low amplitude diastolic fragmented activity appears during the transition and then the right bundle branch block pattern develops. AVJ = atrioventricular junction; CS = coronary sinus; LV-An = left ventricular aneurysm.
Ventricular aneurysm was demonstrated as the site of origin by preoperative endocardial mapping, we performed open heart surgery with aneurysmectomy in an attempt to ablate the tachycardia. In both patients, extensive epicardial mapping revealed early breakthrough of all morphologically distinct forms along the border of the aneurysm (fig. 7). In both cases the LBBB form of the tachycardia originated from the area of the aneurysm which incorporated the intraventricular septum, while the RBBB morphology originated either along the free wall or septal edge of the aneurysm.

Discussion

Ventricular depolarizations or sustained ventricular tachycardia originating in the left ventricle have been considered of RBBB morphology and those in the right ventricle have been considered of LBBB morphology. A corollary of this assumption is that in a patient with both right and left bundle branch block morphologic types of ventricular tachycardia, the tachycardias originate from two foci. Observations in experimental ventricular tachycardia suggest, however, that the QRS morphology is merely a reflection of the epicardial activation pattern and cannot be assumed to define accurately the site of origin of the tachycardia. In addition, using endocardial catheter mapping we have demonstrated that in seven of 11 patients with sustained ventricular tachycardia with LBBB morphology, the site of origin was either in the left ventricle or septum.9

Assessment of Site of Origin

The site of origin of a tachycardia as determined by catheter endocardial recording is primarily limited by reproducibility and degree of resolution. The catheter recording technique can only yield reproducible results if segmental areas of myocardium are analyzed; thus, the site of origin actually represents a segment of origin. These segments are small enough (approximately 10–12 cm²) to allow localization to an aneurysm or an area supplied by a major coronary artery. Therefore, such data can identify areas which can be surgically ablated — the ultimate goal for localizing the tachycardia. Thus, while pinpoint localization is not possible with standard catheter techniques, the accuracy is sufficient to guide appropriate surgical intervention.

Confusion may arise from the changing relationships and duration of fragmented systolic and diastolic activity to the onset of the QRS during multiple morphologies. Recent experimental data suggest that such fragmented activity represents recording of reentrant activity. This fragmented activity should be compared with discrete diastolic potentials recorded at surgery at aneurysm borders, which has been felt to represent slowed conduction out of the aneurysm and a marker for reentry. Whether continuous or non-holodiastolic fragmentation is recorded depends on the catheter position and size of the reentrant circuit. The smaller the circuit and closer the catheter to that circuit, the more likely continuous fragmented activity will be recorded. If only part of
the reentrant circuit is recorded due to catheter position relative to the spatial and geometric arrangement of the reentrant circuit, this activity may be recorded early or late in the cardiac cycle, depending on changes in ventricular activation resulting from varying exit sites. This is schematically shown in figure 8. If part of the reentrant circuit is recorded, the constancy of the interelectrogram intervals during changes in morphology suggests that the same reentrant circuit is responsible for both morphological forms. In each

**Figure 5.** Two morphologically distinct ventricular tachycardias with different sites of origin (case 3). Both panels are organized from top to bottom as ECG leads 2 and V\(_1\) and electrograms from the atrioventricular junction (AVJ), mid-septum of the right ventricle (RV), and high right atrium (HRA). In the top panel ventricular tachycardia with a left bundle branch block morphology is present. The site of origin was the RV septum. Low amplitude potentials (arrow) at the onset of the QRS probably represent the oncoming wavefront from the septum. In the bottom panel ventricular tachycardia with right bundle branch block morphology is shown. The site of origin (vertical dotted line) was the left ventricular anterior wall (LV-ant). No fragmented diastolic activity was observed during either morphology. T = time lines.
Mechanisms of Pleomorphic Forms

Our study demonstrates that recurrent sustained ventricular tachycardia is frequently pleomorphic — in 14 of 26 of our patients. A left ventricular aneurysm was the source of the pleomorphic tachycardias in 11 of these 14 patients. The observation that spontaneous alterations in the QRS could occur without change in the underlying activity within the aneurysm suggests that changes in QRS morphology reflect changing exit sites from the reentrant circuit, rather than changes in the site of origin, and that ventricular activation from each of these exit sites produces different morphological patterns. This hypothesis is also supported by observations that 1) multiple morphologies can be produced by extrastimuli in the presence of unchanged continuous electrical activity, 2) during epicardial mapping different epicardial exit sites produce different morphologies, and 3) exit block at the border of the aneurysm at the left ventricular free wall has previously been demonstrated to be a mechanism for shift from right-to-left bundle branch block morphology. Since all aneurysms were left ventricular and involved the septum, the development of a LBBB pattern was probably the result of preferential left-to-right transseptal activation due to early exit along the septal border of the aneurysm. The fact that left ventricular aneurysmectomy has cured ventricular tachycardia with a LBBB pattern supports this concept.16-22 If the impulse can exit the aneurysm from multiple sites, ventricular activation and the morphology of the resulting QRS will not only depend on the exit site but will additionally be influenced by the pathological state of the myocardium.

We and others10, 15, 22-25 have suggested that the mechanism of sustained ventricular tachycardia is reentry. The ability to change QRS morphologies appears to be unrelated to the underlying mechanism but is related to spontaneous or electrically induced changes in the patterns of ventricular activation. Therefore, changing morphologies cannot be diagnostic for any mechanism.

Clinical Significance of Pleomorphic Ventricular Tachycardia

Some investigators have stated that ventricular tachyarrhythmias with a LBBB morphology occur most commonly in the absence of organic heart disease, while those with RBBB morphology are more often associated with significant heart disease.3-5, 18, 26, 27 This opinion is not, however, shared by others.19-22 Since all prior investigations used ECG morphology to identify the site of origin, the ambiguities of clinical correlates can be explained by the inability of the surface ECG to identify accurately the site of origin of ventricular tachycardia.
No previous study has analyzed the clinical substrate of spontaneous or stimulation-induced pleomorphic ventricular tachycardia. Of two patients reported by Spurrell et al., with morphologically distinct ventricular tachycardias induced by stimulation, one had a left ventricular aneurysm secondary to myocardial infarction and one had alcoholic cardiomyopathy. In our 14 patients, all but one had cardiac disease, including 11 with coronary artery disease and left ventricular aneurysms. The single patient with pleomorphic ventricular tachycardia in the absence of heart disease was one of the two patients in whom two foci may have been responsible. The unusually high incidence of left ventricular aneurysms in our series suggests that the presence of pleomorphic ventricular tachycardia should imply myocardial dysfunction, particularly coronary artery disease with aneurysm formation, as the cause of the arrhythmia. This is supported by the fact that identical stimulation techniques produced pleomorphic ventricular tachycardia in 11 of 13 patients with left ventricular aneurysms and in three of 13 patients without aneurysms. In fact, two of the three patients without aneurysms who demonstrated pleomorphism appeared to have two distinct tachycardias.

**Therapeutic Implications**

Therapy of ventricular tachycardia is difficult. While pharmacologic or pacemaker therapy is often successful, surgery may be considered in some patients. Aneurysmectomy is currently the only acceptable surgical modality for ablation of ventricular tachycardia. However, if the presence of two morphologies implies two foci, one in each ventricle, surgery is often considered contraindicated. Our study suggests that pleomorphic ventricular tachycardia is not a contraindication of surgery since in most patients, particularly those with aneurysms, both forms represent the same ventricular tachycardia. In such patients electrophysiologic study should be performed to evaluate polymorphic forms of ventricular tachycardia as a means to an effective therapeutic program.
FIGURE 8. Schema of catheter recording of local reentrant activity during ventricular tachycardia with two morphologies. A bipolar electrode catheter is schematically positioned over part of the reentrant circuit and records local fragmented (reentrant) activity during different parts of the cardiac cycle, depending on the relationship of the exiting wavefront to the catheter recording site. If the ventricles are depolarized by a wavefront that exits after passing the electrode (tachycardia on right) fragmented activity will be recorded before the QRS. If ventricular activation occurs before reaching the area of catheter recording, then the fragmented activity will appear during and after the QRS (tachycardia on left). Thus, the right and left bundle branch block morphologies shown here arise from the same reentrant circuit, despite a changing relationship of the fragmented electrogram to the onset of the QRS.

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References

22. Spurrell RAJ, Sowton E, Deuchar DC: Ventricular tachycard-
Cardiac Arrhythmias in the Conscious Dog
After Excision of the Sinoatrial Node and Crista Terminalis

D. E. Euler, B.A., S. B. Jones, Ph.D., W. P. Gunnar,
J. M. Loeb, Ph.D., D. K. Murdock, M.D., and W. C. Randall, Ph.D.

SUMMARY A stable junctional rhythm was produced in 16 dogs after surgical excision of the sinoatrial node and a large section of the crista terminalis. An unstable ectopic atrial rhythm appeared within the first week after surgery in 94% of the dogs. The pacemaker instability was characterized by spontaneous pacemaker shifts and periodic episodes of asystole which were prominent for several months after surgery in most of the dogs. In contrast to sinus arrhythmia observed before surgery, the ectopic atrial arrhythmias were not related to the respiratory cycle. The prompt disappearance of the asystoles after atropine or during treadmill exercise indicated the essential role of the vagus in producing the unstable rhythms. Atropine increased the average rate of the ectopic rhythms from \(63 \pm 3\) beats/min to \(107 \pm 9\) beats/min \((p < 0.001)\) and shortened the corrected recovery time \((CRT)\) following overdrive pacing from \(3.8 \pm 0.3\) seconds to \(1.9 \pm 0.6\) seconds \((p < 0.001)\). Propranolol, in the absence of atropine, decreased the spontaneous heart rate from \(56 \pm 5\) beats/min to \(39 \pm 6\) beats/min \((p < 0.01)\) and increased the \(CRT\) to \(6.5 \pm 2\) seconds \((p < 0.001)\) when administered after atropine. The data suggest that unstable ectopic atrial pacemakers could be responsible for some of the arrhythmias associated with the sick sinus syndrome in man.

THE PRODUCTION OF A STABLE junctional rhythm (JR) or ectopic atrial rhythm (EAR) after the removal or suppression of the sinoatrial node (SAN) in acute animal experiments has long been recognized. However, little is known about the stability of supraventricular subsidiary pacemakers in chronic SAN dysfunction. Depression of automaticity in escape pacemakers has been implicated in the production of arrhythmias associated with the sick sinus syndrome in man. Early experimental studies by Borman and Meek showed that atrial pauses were common for several days after experimental destruction of the SAN by implantation of radon seeds. In more recent experiments, surgical exclusion of the SAN from the remainder of the right atrium resulted in a transitory period of pacemaker instability after the animals recovered from anesthesia. The instability was characterized by shifting pacemaker sites, asystole, and tachycardias with a gradual return to a stable EAR within the first 3 weeks after surgery.

The SAN in the dog has been shown histologically to extend for a distance of 8–20 mm along the sulcus terminalis starting at the cavo-auricular junction. Surgical excision of just the SAN, with little damage to the crista terminalis, produces an immediate, stable EAR with little change in P-wave morphology or P-R interval in both the anesthetized and conscious dog. Epicardial mapping of these ectopic rhythms in the anesthetized dog locates the earliest point of negativity on the sulcus terminalis just distal to the site of the excised SAN. Since the crista terminalis underlying the sulcus terminalis contains specialized fibers capable of spontaneous diastolic depolarization, it is possible that stable ectopic atrial rhythms might arise from these fibers in the chronic absence of the SAN.

Our study chronically examines the ectopic rhythms produced by removal of the SAN and a large section of the crista terminalis. Since the autonomic nervous system plays an important role in modulating pacemaker function, we tested the response of the subsidiary pacemakers to atropine, propranolol, and treadmill exercise. We also evaluated the susceptibility of the subsidiary pacemakers to overdrive suppression.

From the Department of Physiology, Loyola University of Chicago, Stritch School of Medicine, 2160 South First Avenue, Maywood, Illinois 60153.
Supported by NIH Grant HL08682.
Address for reprints: David Euler, Department of Physiology, Loyola University Medical Center, 2160 South First Avenue, Maywood, Illinois 60153.
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