Reevaluation of Electrocardiographic and Bedside Criteria for Diagnosis of Ventricular Tachycardia

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With the technical assistance of Mr. Howard Jackson

SUMMARY

Electrically induced ventricular and atrial tachycardias were observed in 12 cases, and ventriculoatrial (V-A) and atrioventricular (A-V) relationships were studied. It was noted that retrograde V-A conduction is a common phenomenon and that A-V dissociation is not a prerequisite for diagnosis of ventricular tachycardia. Conduction through the A-V node occurred readily in both antegrade and retrograde directions; and, all grades of block from first to complete and including the Wenckebach phenomenon were observed in both directions. The intensity of the first heart sound and the amplitude of the jugular a wave were governed directly by the temporal relationship between the P and the QRS regardless of whether conduction was antegrade, atrioventricular, or retrograde, ventriculoatrial.

It is concluded that at present no electrocardiographic or bedside criteria are available for an unequivocal diagnosis of ventricular tachycardia and that further studies are needed.

Additional Indexing Words:
Electrical stimulation of ventricles
Phonocardiograms

The statement that "the large variety of manifestations of rapid ectopic rhythms and specially the numerous features common to supraventricular and ventricular forms render their distinction on clinical grounds very difficult and uncertain," made by Pick and Langendorf,1 serves to emphasize the difficulties in differentiating the two types of tachycardia. The electrocardiographic diagnosis of the origin of tachycardia is fraught with considerable uncertainty because, on the one hand, the bizarre configuration and long duration of the QRS complexes secondary to aberrancy of intraventricular conduction are common in supraventricular tachycardias.2

Further, the atrioventricular (A-V) dissociation considered the hallmark of ventricular tachycardia can no longer be regarded as essential in view of the recent demonstrations of frequent retrograde ventriculoatrial (V-A) conduction2–8 and the frequent occurrence of A-V dissociation with A-V junctional rhythms.9

Clinicians have sought assistance from bedside examination of the patient with special attention to the intensity of the first heart sound (S1) and the constancy of the jugular venous pulse waves (JVP).10–13 It has been reasoned that, in the presence of ventricular tachycardia, the dissociation of the ventricles from the atria would lead to a constantly changing relationship between atrial and ventricular contractions and this, in turn, would...
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alter the intensity of the first heart sound and 
the amplitude of the a waves in the jugular 
veins. The value of these criteria could be seri-
sously hampered, however, in view of the fre-
quency occurrence of retrograde V-A conduc-
tion.2–8

A graphic study of these points is difficult in 
most clinical settings, for patients with tachy-
cardias often are in distress and cannot toler-
ate delays necessitated by recording of sounds 
and pulses. It was felt, therefore, that study of 
electrically induced atrial and ventricular tachycardias in a rigidly controlled laboratory 
environment in patients undergoing diagnostic cardiac catheterization would be of value 
in the reexamination of these criteria. It is the 
purpose of this communication to present the 
results of such a study and to point out the diagnostic difficulties and uncertainties which 
may stem from the frequent occurrence of 
retrograde V-A conduction.

Methods

Electrical stimulation of the right and left ventricles was carried out under constant surveillance in the cardiac laboratory in 11 patients in normal sinus rhythm, undergoing the diagnostic procedures of right and left heart catheterization (table 1). In addition, a patient with atrial fibrillation with electrically induced ventricular tachycardia (case 12) was included for the purpose of pointing out the differences brought about by the absence of regular atrial activity. Consents were secured from all patients prior to the study. The right ventricle was stimulated electrically at varying rates in 10 cases and the left ventricle in one. For comparison of heart sounds and JVP in ventricular and supraventricular tachycardias in the same subjects, the right atrium was stimulated in eight of the cases. The stimulating apparatus was an R-wave coupled pulse generator (Medtronic, Model 5873-A) permitting delivery of the stimulus to the ventricle during the safe interval after the vulnerable period. The right ventricle was stimulated at rates between 125 and 170/min and usually under 140/min. The frequency of retrograde V-A

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>RV stimulation, orthograde A-V conduction</th>
<th>RA stimulation, orthograde A-V conduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>D.B.</td>
<td>69</td>
<td>F</td>
<td>Suspect, aneurysm of abdominal aorta</td>
<td>None</td>
<td>Not done</td>
</tr>
<tr>
<td>M.B.</td>
<td>68</td>
<td>F</td>
<td>Aneurysm, thoracic aorta</td>
<td>None</td>
<td>1:1 with changing PR</td>
</tr>
<tr>
<td>A.W.</td>
<td>40</td>
<td>M</td>
<td>Pulmonary embolism</td>
<td>1:1 with changing RP'</td>
<td>1:1 with changing PR</td>
</tr>
<tr>
<td>C.G.</td>
<td>29</td>
<td>M</td>
<td>Cardiomyopathy</td>
<td>1:1 with constant RP'</td>
<td>Not done</td>
</tr>
<tr>
<td>C.B.</td>
<td>34</td>
<td>M</td>
<td>Hypertensive heart disease</td>
<td>1:1 with constant RP'</td>
<td>1:1 with constant PR</td>
</tr>
<tr>
<td>M.T.</td>
<td>31</td>
<td>F</td>
<td>Pulmonary embolism</td>
<td>None</td>
<td>1:1 with constant PR</td>
</tr>
<tr>
<td>W.P.</td>
<td>46</td>
<td>M</td>
<td>Cardiomyopathy</td>
<td>1:1 with constant RP'</td>
<td>1:1 with constant PR</td>
</tr>
<tr>
<td>C.Y.</td>
<td>56</td>
<td>M</td>
<td>Aortic regurgitation</td>
<td>Variable</td>
<td>Variable</td>
</tr>
<tr>
<td>J.K.</td>
<td>47</td>
<td>F</td>
<td>Mitral stenosis</td>
<td>None</td>
<td>Variable</td>
</tr>
<tr>
<td>C.J.</td>
<td>57</td>
<td>M</td>
<td>Hypertensive heart disease</td>
<td>Variable</td>
<td>1:1 with constant PR</td>
</tr>
<tr>
<td>V.H.</td>
<td>76</td>
<td>F</td>
<td>Renal artery stenosis; suspect</td>
<td>1:1 with changing RP'</td>
<td>Not done</td>
</tr>
<tr>
<td>E.D.</td>
<td>69</td>
<td>F</td>
<td>Ischemic heart disease</td>
<td>Atrial fibrillation</td>
<td>Not done</td>
</tr>
</tbody>
</table>

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Conduction was probably not increased by the relatively slow rates of ventricular stimulation because, in the present as well as in previous studies, \(^7\) stimuli delivered at rates higher than 200/min were conducted retrograde just as readily as those delivered at slower rates.

Atrioventricular dissociation was investigated by simultaneous recording of a bipolar esophageal lead with lead II, lead V\(_1\), or both. External phonocardiograms were recorded simultaneously from either the tricuspid or the mitral area using a Sanborn microphone. Venous pulses were recorded either indirectly over the right jugular vein using a Sanborn pulse pick-up (Model 374) or directly from the cavity of the right atrium or the jugular vein through a catheter. Recordings were made on a research recorder (Electronics for Medicine, DR-8) at a paper speed of 50 mm/sec with time lines at 0.1 or 0.2-sec intervals. The P waves on the bipolar esophageal lead and their relationship to the QRS complexes were studied and compared in the two types of tachycardia. Similarly, the intensity of the first heart sound and the amplitude of the venous waves were analyzed and compared. Six of the 12 cases have been selected for detailed presentation.

Results

Retrograde V-A conduction occurred in seven of 11 cases with normal sinus rhythm. Conduction was predominantly 1:1 in five and variable in two. Atrioventricular conduction in cases of atrial tachycardia was 1:1 in six and variable in two. In cases of 1:1 V-A conduction, the RP' intervals were constant, but changed in the manner of the Wenckebach phenomenon in the remaining two cases. In cases of atrial tachycardia with 1:1 A-V conduction, the PR relationship was constant in four and variable in the other two.

The behavior of S\(_1\) and JVP was intimately governed by the relationship between the P and the QRS or the QRS and the ensuing retrograde P'. When these relationships were constant or changed very little, the S\(_1\) and the JVP did not vary. This situation was encountered when there was 1:1 or 2:1 A-V conduction, 1:1 V-A conduction, and the interesting, fortuitous association of a ventricular rate exactly (or nearly exactly) double the atrial rate. On the other hand, a changing PR or RP' relationship brought about profound variations of S\(_1\) and JVP regardless of whether the origin of the tachycardia was supraventricular or ventricular. Generally, when the P waves preceded the QRS complexes and the PR intervals varied from beat to beat, the first heart sound was found to be inconstant and quite variable. On the other hand, the appearance of the P wave immediately beyond or some time after the QRS complexes at varying RP' intervals did not seem to alter the first heart sound very significantly but caused profound changes in the size of the JVP, and specifically, of the a waves.

Data on Cases and Description of Figures

Case 1, D.B. (Fig. 1)

In this patient with no documented heart disease, the right ventricle was stimulated electrically at varying rates, and no V-A conduction

![Figure 1](http://circ.ahajournals.org/Downloaded from http://circ.ahajournals.org/)

Case 1, D.B. See text for explanation of figures.
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Figure 2
Case 2, M.B.

Figure 3
Case 3, A.W.

could be provoked. The top tracing in figure 1 is lead II; the tracing immediately below it and the third tracing are bipolar esophageal leads taken at two different levels; the fourth tracing is a phonocardiogram recorded from the tricuspid area, and the bottom tracing is a direct jugular pressure tracing. Beat number 1 of the left hand panel is the only normal beat displayed. Beats 2 to 23 are those of electrically induced ventricular tachycardia. It may be noted that normal P waves persist throughout the three episodes of tachycardia with no evidence of retrograde conduction. Downward arrows point to the stimulus artifacts. Beats 4 to 9 show ventricular tachycardia at the rate of 144/min. The normal P waves occur just before every other electrically induced QRS with a constantly changing PR interval. This is associated with gross changes in the intensity of the first heart sounds marked S₁ but with relatively unimpressive variations in the jugular a waves. In the middle panel in beats 10 through 15, the normal P waves occur predominantly after the electrically induced QRS complexes and for this reason the constantly changing RP intervals are translated into marked variations in the size of the jugular a.
Figure 4
Case 4, C.G.

Figure 5
Case 11, V.H.
waves. In this figure the first heart sounds show unimpressive variations. In beat 16, however, the P precedes the QRS and is associated with a loud S1. In the right panel, the ventricular rate is almost exactly twice the normally occurring sino-atrial rate so that the normal P waves fall equidistant between the artificially induced QRS complexes. The result is a relatively constant first heart sound and constant a waves separated by smaller cv waves.

Case 2, M.B. (Fig. 2)

The patient had dilatation of the ascending aorta and aortic regurgitation. The ECG and phonocardiogram were recorded simultaneously with the right atrial pressure. In figure 2 beats 1 and 2 are normal sinus beats; beats 3 to 8 are those of right ventricular stimulation at a rate of 148/min, and beats 9 through 13 are the result of right atrial stimulation at a rate of 152/min. As in case 1, ventricular stimulation fails to provoke retrograde V-A conduction, and as would be expected, the intensity of S1 and the amplitude of the right atrial pulsations are variable. In the right panel, there is a gradually increasing PR interval, and as a result, the intensity of the first heart sound varies markedly. Simultaneously, the height of the right atrial a waves increases gradually as the interval between the QRS and the next P diminishes. It can be seen that differentiation between the two types of tachycardia on the basis of S1 and JVP would have been difficult in this case.

Case 3, A.W. (Fig. 3)

This patient had pulmonary embolism. The left panel of figure 3 shows ventricular stimulation at a rate of 130/min, and the right panel depicts right atrial stimulation at a rate of 136/min. Beats 1 and 2 and 11 and 12 are normal. Beats 3 through 10 show retrograde V-A conduction with the resultant retrograde P waves marked P'. V-A conduction begins as 1:1 with a gradual increase in RP' intervals culminating in 2:1 V-A block in beats 7 to 10. These marked variations in RP' relationship account for changing intensity of S1 and also gross variations in the JVP. In the right panel, the antegrade conduction after right atrial stimulation begins as 1:1 with increasingly longer PR intervals culminating in a blocked P wave which is hidden in the QRS of beat 17. The nearness of the P to the preceding QRS may give the erroneous impression of an A-V junctional rhythm with retrograde conduction. Here, too, as in the left panel, the first heart sound and the JVP vary considerably. Comparison of the two panels makes it abundantly clear that clinical distinction between these two phenomena would be impossible.

Case 4, C.G. (Fig. 4)

The patient had cardiomyopathy of undetermined origin. This was the only case in which the left ventricle was electrically stimulated. In figure 4 the first four beats are normal, and the remaining beats are those of left ventricular stimulation. Due to constant 1:1 retrograde V-A conduction, each electrically stimulated QRS is followed by retrograde atrial activation marked P' at a constant RP' interval. For this reason the first heart sound and the JVP are constant and therefore indistinguishable clinically from that of sinus tachycardia or supraventricular tachycardia with 1:1 conduction.

Figure 6

Case 12, E.D.
Case 11, V.H. (Fig. 5)

This patient was suspected of having stenosis of the left renal artery. The top tracing in figure 5 is a bipolar esophageal lead, the middle tracing is a direct intracavitary right atrial lead, and the bottom tracing is lead V1. The left panel shows two normal beats; the middle panel depicts right ventricular stimulation with retrograde 1:1 V-A conduction. The greatly prolonged, but almost fixed RP' of 0.25 sec causes the P' to be nearer the subsequent QRS than the preceding one and to appear to be responsible for the former. The right panel shows 1:1 V-A conduction with gradually increasing RP' intervals (retrograde Wenckebach's phenomenon) culminating in a dropped P' after beat 10. In a spontaneously occurring ventricular tachycardia of this type in which esophageal or intracavitary electrograms are taken for diagnosis, the P' waves may be easily misinterpreted as P waves responsible for the ensuring QRS complexes. Alternatively, the arrhythmia may be viewed as a combination of a ventricular or A-V junctional tachycardia with an independent atrial tachycardia.

Case 12, E.D. (Fig. 6)

This patient had arteriosclerotic heart disease and atrial fibrillation. The left panel in figure 6 is the control tracing showing rather marked variations in the intensity of the first heart sound but relatively constant c wave waves in the JVP. In the middle panel, the right ventricle is stimulated at a rate of 120/min. The change in the intensity of the first heart sound becomes less striking, and the JVP remains indistinguishable from that in the left panel. Recognition of the ventricular origin of the tachycardia would have been impossible in this case in which the preexisting atrial fibrillation, that is, the absence of regular atrial activity, prevented any predictable variations in S1 or jugular a waves. The right panel was recorded after conversion of atrial fibrillation by electric countershock and the spontaneous occurrence of an A-V junctional rhythm at a rate of 72/min. In this the retrograde P' waves occur regularly after QRS complexes at constant R-P' intervals. The intensity of the first heart sound varies relatively little. The most impressive feature, however, is the constancy of the appearance of the JVP tracing showing spiking c waves followed by small a waves related to the retrograde P' waves. At the bedside, these giant c waves were mistaken for normal or large a waves.

Discussion

Electrocardiographic characterization of the nature of paroxysmal tachycardia is often difficult and uncertain because of the frequent overlapping of the features of supraventricular and ventricular tachycardias. The dissociation between the atria and the ventricles, that is, between the P and the QRS complex, has been considered a key feature of ventricular tachycardia, whereas the demonstration of A-V conduction has been accepted as proof of supraventricular origin of the ectopic rhythm. The demonstration by a number of authors2-8 of retrograde V-A conduction in a high percentage of cases of ectopic beats originating in the ventricles and also documented in the present study indicates that A-V dissociation can no longer be considered a prerequisite for diagnosis of ventricular origin of an ectopic rhythm. Similarly, the bizarre configuration and long duration of the QRS complexes accepted so readily as features of ventricular tachycardia are found commonly in beats of supraventricular origin associated with aberration of intraventricular conduction. At least four possible mechanisms may explain this abnormality of the QRS complexes in beats of supraventricular origin.2 This, therefore, can no longer be held as a reliable criterion of ventricular tachycardia. An A-V junctional tachycardia with wide QRS complexes due either to preexisting bundle-branch block or the association of aberrant intraventricular conduction, in which retrograde conduction to the atria is absent, would be indistinguishable from a classical case of ventricular tachycardia.

The behavior of the first heart sound and JVP has been used as a supplement to the electrocardiographic diagnosis and their variability has been accepted as an indication of A-V dissociation and, therefore, a diagnostic feature of ventricular tachycardia. Here, too, the intensity of S1 and the configuration of JVP are governed entirely by the relationship between the P wave and the QRS complex. This study, while serving to emphasize the accuracy of this thesis, makes it abundantly clear that differentiation between the ventricular and supraventricular tachycardias on the basis of S1 and JVP is impossible. It
was demonstrated in case 4 that constancy of PR or RP' relationship irrespective of the origin of the QRS complexes is sufficient to create a constant S₁ and an unchanging JVP. Conversely, changing PR or RP' relationship due either to changing antegrade A-V conduction in a supraventricular tachycardia or variable retrograde V-A conduction in ventricular tachycardia is sufficient to give rise to marked beat-to-beat variations of S₁ and JVP (cases 1, 2, and 3). Since in most clinical situations, even with the benefit of an electrocardiogram, the differentiation between antegrade and retrograde conduction may be impossible, it is clear that observation of S₁ and JVP cannot solve the problem in difficult situations. In a spontaneous attack of tachycardia, even an esophageal or intracavitary electrogram may fail to clarify the origin of the QRS complexes as long as the atrial and ventricular rates are equal. The nearness of the P to the next QRS cannot be taken as proof of antegrade atrioventricular conduction, for this may be a reflection of prolonged V-A conduction time such as occurred in case 11 of this study (fig. 5). Conversely, the nearness of the P to the preceding QRS need not indicate retrograde V-A conduction, because this situation may be observed in supraventricular tachycardia with first degree A-V block (case 3, fig. 3). Only in cases in which the ventricular rate is more rapid than the atrial can one say with certainty that the origin of the tachycardia is below the atrium, that is, within the ventricles or in the A-V junctional tissue.

In view of these difficulties in differentiating ventricular from supraventricular tachycardias, Kistin² and Langendorf,20 and Marriott (personal communication from H. L. Marriott) have observed that in the usual clinical circumstances the definitive diagnosis of ventricular origin of a tachycardia is impossible unless the ectopic rhythm is artificially induced with the stimulating electrode lying on or within the ventricles.

Review of the published reports of ventricular tachycardia and of descriptions of criteria for differential diagnosis of tachycardia makes it clear that most writers depend to a great extent on the demonstration of A-V dissociation and changing S₁ and JVP. It is hoped that the present study will serve to emphasize the difficulties that surround a definitive diagnosis and to stimulate further studies with the objective of establishing firmer criteria.

References

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50 Years Ago:
Growth and Form

In the case of the heart we have, within each of its cavities, a pressure which, at any given moment, is constant over the whole wall-area, but the thickness of the wall varies very considerably. For instance, in the left ventricle, the apex is by much the thinnest portion, as it is also that with the greatest curvature. We may assume, therefore (or at least suspect, that the formula, \( t(1/r + 1/r') = C \), holds good; that is to say, that the thickness \((t)\) of the wall varies inversely as the mean curvature. This may be tested experimentally, by dilating a heart with alcohol under a known pressure, and then measuring the thickness of the walls in various parts after the whole organ has become hardened. By this means it is found that, for each of the cavities, the law holds good with great accuracy. Moreover, if we begin by dilating the right ventricle and then dilate the left in like manner, until the whole heart is equally and symmetrically dilated, we find (1) that we have had to use a pressure in the left ventricle from six to seven times as great as in the right ventricle, and (2) that the thickness of the walls is just in the same proportion.—D'ARCY WENTWORTH THOMPSON: On Growth and Form. Cambridge, University Press, 1917, p. 666.
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