Retrograde Conduction to the Atria in Ventricular Tachycardia

By Albert D. Kistin, M.D.

Contrary to the prevalent view that retrograde conduction to the atria in ventricular tachycardia is rare, the author has recorded tracings consistent with such conduction relatively frequently with simultaneous standard and esophageal leads. The standard electrocardiographic tracings often fail to demonstrate atrial activity accurately, because the atrial deflections are small and lost in the deflections of the ectopic ventricular activity. This is apparent from a comparison of lead II with the simultaneously recorded esophageal lead in most of the illustrations of this paper, and probably explains why so few cases of ventriculo-atrial (V-A) conduction in ventricular tachycardia have heretofore been recognized.

Sir Thomas Lewis described the first clinical case of ventricular tachycardia in 1909 and observed that in dogs retrograde conduction to the atria was common. A review of the clinical literature by Foster and Thayer in 1950 yielded only three cases of 1:1 V-A conduction and six cases of V-A conduction with variable block. These authors concluded from the illustrations of 81 published cases of ventricular tachycardia that in 40 it was impossible to recognize the atrial activity. The interpretation of V-A conduction was made in standard electrocardiographic leads and recently in esophageal leads.

Material and Methods

Ventricular tachycardia for the purposes of this study is defined as five or more ectopic ventricular systoles in succession. Simultaneous esophageal and standard leads were recorded. During the period of study the interpretation of ventricular tachycardia was made in 21 cases, 14 in the course of clinical practice, five during cardiac catheterization, one during mitral valve surgery, and one during surgery for coarctation of the aorta.

Whenever the interpretation of ventricular tachycardia with 1:1 retrograde conduction to the atria was made, with one exception, the onset of one or more runs of tachycardia was recorded and consisted of a bizarre QRS different from the QRS of sinus origin. Parts of the tracing showing regular sinus rhythm were available for comparison. The esophageal leads ruled out the possibility of atrial tachycardia with aberrant conduction by demonstrating that the tachycardia did not start with a P wave. In the case in which the onset of the tachycardia was not observed, within a few beats after the cessation of the tachycardia isolated ventricular premature systoles with retrograde conduction to the atria were recorded identical in configuration with the complexes of the tachycardia.

In 10 earlier studies the esophageal electrode was paired with the Wilson V connection (VE lead). In 11 more recent studies a bipolar esophageal lead (BE lead) was recorded simultaneously with a standard lead, usually lead II, and two VE leads, one each from the electrodes of the BE lead as described by Copeland et al. Instead of the fluid-filled tubes recommended by these authors a simple Rehfuss tube was used with two German-silver rings, 3.0 mm. wide and 2.0 cm. apart. The lower of the rings was about 3 cm. from the tip of the tube, and each ring was connected to insulated wires passing up the inside of the tube. The holes in the tip of the Rehfuss tube were sealed with solder to avoid wetting the wires. The simpler BE electrode yields satisfactory tracings.

The BE lead is often superior to the VE lead for the study of V-A conduction. It is usually possible to select an esophageal position for the electrodes at which the BE lead records small QRS complexes and large P waves, the latter distinct even in complex arrhythmias when superimposed on QRS and T (figs. 1 and 3-6). Also it is often possible to select an esophageal level at which the BE lead records retrograde P waves more or less opposite in direction to the sinus P waves (figs. 1 and 3-5); such tracings are ob-
Figure 1

Case 1. Simultaneous lead II, bipolar esophageal lead, and V esophageal lead. Ventricular tachycardia with 1:1 V-A conduction. One sinus beat interrupts the tachycardia. The retrograde P waves in the BE lead are the large spiked downward deflections after each small rounded QRS; the sinus P wave is smaller, biphasic, with a larger upward component. The retrograde P waves are distinct in the VE lead, but there is less difference from the sinus P wave than in the BE lead. A similar consecutive run of 228 beats was observed with no increase of V-A conduction time.

Table 1 shows that cases noted for study varied in the number of V-A conduction. In the 21 cases studied, the 2:1 V-A conduction occurred in five. The 1:1 conduction often did not appear until the second beat of the tachycardia because of initial interference with the sinus beat, and then persisted to the end of the run. In five other cases one to many runs with 1:1 V-A conduction occurred in association with other runs in the same tracing with other atrial mechanisms. One of these cases was previously reported (case 7). The other mechanisms were either independent atrial rhythm or irregular V-A conduction including the Wenckebach phenomenon. In four cases V-A conduction occurred irregularly and there were no runs with 1:1 V-A conduction; in 3 cases there were also runs with independent atrial rhythm in the same tracing, and in 1 case runs that demonstrated the Wenckebach phenomenon. In only 7 of the 21 cases was the atrial mechanism always.

Observations and Discussion

Incidence of V-A Conduction

Table 1 is a summary of the cases with V-A conduction. Of 21 cases in which the interpretation of ventricular tachycardia was made 1:1 V-A conduction alone occurred in five. The 1:1 conduction often did not appear until the second beat of the tachycardia because of initial interference with the sinus beat, and then persisted to the end of the run. In five other cases one to many runs with 1:1 V-A conduction occurred in association with other runs in the same tracing with other atrial mechanisms. One of these cases was previously reported (case 7). The other mechanisms were either independent atrial rhythm or irregular V-A conduction including the Wenckebach phenomenon. In four cases V-A conduction occurred irregularly and there were no runs with 1:1 V-A conduction; in 3 cases there were also runs with independent atrial rhythm in the same tracing, and in 1 case runs that demonstrated the Wenckebach phenomenon. In only 7 of the 21 cases was the atrial mechanism always.

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<table>
<thead>
<tr>
<th>Patient no., sex, age (yr.) (fig.)</th>
<th>Diagnosis</th>
<th>Longest recorded uninterrupted run No. beats (min)</th>
<th>Sinus rate per min.</th>
<th>Average rate in ventricular tachycardia (per min)</th>
<th>P-R (sec.)</th>
<th>X-P' (sec.)</th>
<th>Atrial mechanism</th>
<th>Evidence for ventricular focus</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 M/69 (fig. 1) MI (old), PE</td>
<td>No</td>
<td>228</td>
<td>88</td>
<td>186</td>
<td>0.15</td>
<td>0.17</td>
<td>1:1 V-A</td>
<td>APS</td>
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<tr>
<td>2 M/68 (fig. 7) HAHD, PE, CWP</td>
<td>Yes</td>
<td>23</td>
<td>103</td>
<td>194</td>
<td>0.15</td>
<td>0.12</td>
<td>1:1 V-A</td>
<td>APS</td>
</tr>
<tr>
<td>3 M/16 CA (Surg)</td>
<td>No</td>
<td>5</td>
<td>76</td>
<td>94</td>
<td>0.14</td>
<td>0.18</td>
<td>1:1 V-A</td>
<td>——</td>
</tr>
<tr>
<td>4 M/64 (fig. 8) AHD, DM, PE</td>
<td>Yes</td>
<td>11</td>
<td>75</td>
<td>107*</td>
<td>0.15</td>
<td>0.14</td>
<td>1:1 V-A</td>
<td>QRS form</td>
</tr>
<tr>
<td>5 F/42 MS (Surg)</td>
<td>Yes</td>
<td>6</td>
<td>50</td>
<td>91</td>
<td>0.17</td>
<td>0.18</td>
<td>1:1 V-A</td>
<td>APS</td>
</tr>
<tr>
<td>6 F/43 AS, No HD</td>
<td>No</td>
<td>7</td>
<td>92</td>
<td>182</td>
<td>0.16</td>
<td>0.20</td>
<td>1:1 V-A + Irregular V-A (Wenckebach)</td>
<td>Reciprocal beats</td>
</tr>
<tr>
<td>7 M/38 (bibl. #27) AS, No HD</td>
<td>No</td>
<td>40</td>
<td>122</td>
<td>207</td>
<td>0.13</td>
<td>0.12</td>
<td>1:1 V-A + Independent</td>
<td>——</td>
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<tr>
<td>8 M/66 (fig. 3) HAHD, CT (old)</td>
<td>No</td>
<td>6</td>
<td>73</td>
<td>103</td>
<td>0.17</td>
<td>0.21</td>
<td>1:1 V-A + Irregular V-A</td>
<td>——</td>
</tr>
<tr>
<td>9 M/70 HAHD, PE, CWP, LS</td>
<td>No</td>
<td>7</td>
<td>98</td>
<td>151</td>
<td>0.19</td>
<td>0.13</td>
<td>1:1 V-A + Irregular V-A (Wenckebach)</td>
<td>APS</td>
</tr>
<tr>
<td>10 F/37 PHt, (R Cath)</td>
<td>No</td>
<td>7</td>
<td>82</td>
<td>130†</td>
<td>0.16</td>
<td>0.20</td>
<td>1:1 V-A + Irregular V-A</td>
<td>APS QRS form</td>
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<tr>
<td>11 F/68 HAHD, CT (recent)</td>
<td>No</td>
<td>8</td>
<td>51</td>
<td>96 to 154</td>
<td>0.14</td>
<td>0.12</td>
<td>Irregular V-A + Independent</td>
<td>APS</td>
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</table>
**Retrograde Conduction in Ventricular Tachycardia**

<table>
<thead>
<tr>
<th>Case</th>
<th>Rhythm</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>4-P J</td>
<td>1.6 0.16 APS Independent</td>
</tr>
<tr>
<td>13</td>
<td>4-P J</td>
<td>2.1 0.16 APS Independent</td>
</tr>
<tr>
<td>14</td>
<td>4-P J</td>
<td>&gt;2 min. 0.16 APS Independent</td>
</tr>
</tbody>
</table>

*During first few beats before terminal slowing.  
†During run with 1:1 V-A conduction and during first few beats of other run before terminal slowing. 

Where more than one atrial mechanism is given, this means that in the same tracing some runs of ventricular tachycardia were associated with one atrial mechanism, other runs with another. See text for discussion of column, Evidence for Ventricular Focus.

Abbreviations: AHD—coronary arteriosclerotic heart disease, APS—atrial premature systole, AS—anxiety state, CA—coarctation of the aorta, CT—cerebral thrombosis, CWP—coal workers' pneumoconiosis, DM—diabetes mellitus, HAHD—hypertensive and coronary arteriosclerotic heart disease, HD—heart disease, LS—latent syphilis, (L Cath)—tracing during left heart catheterization, MD—muscular dystrophy, MI—myocardial infarct, MS—mitral stenosis, PE—pulmonary emphysema, PHD—pulmonary heart disease, PHT—pulmonary hypertension, QRS form—maintenance of QRS form of ectopic beats in spite of pronounced variations in intervals between ectopic beats, (R Cath)—tracing during right heart catheterization, (Surg)—tracing during surgery, Tb—pulmonary tuberculosis, V-A—ventriculo-atrial, X-P—minimum ventriculo-atrial conduction time measured from onset of QRS in lead where it was earliest to onset of retrograde P in esophageal lead.

In a number of the cases of this study the duration of the runs was relatively brief. Is 1:1 conduction more persistent and prolonged ventricular tachycardias? This cannot be answered by the present study, but runs of 225 beats at 90 per minute (case 1, fig. 1) and 40 beats at 166 per minute (case 1, fig. 1) and 22 beats at 194 per minute (case 2, fig. 2) were observed with 1:1 V-A conduction, with 1:1 V-A conduction lasting only if the onset, or in one case the termination, of the tachycardia was noted. Persistent ventricular tachycardias with aberrant conduction have not been included in this study because they were not observed in the present study. Therefore, it is impossible to say whether the tachycardia is atrial or ventricular.

In some reported cases it is impossible to say whether the tachycardia is atrial or ventricular. Since the diagnosis of ventricular tachycardia with 1:1 V-A conduction was made a priori, it is impossible to say whether the 1:1 V-A conduction was made a priori. A P-QRS relation whose onset could not be observed are not included in this study. Since the study is weighted with intermittent ventricular tachycardias with bizarre QRS complexes and QRS complexes with bizarre QRS complexes, it follows that the study was weighted with intermittent ventricular tachycardias with bizarre QRS complexes. Persistent ventricular tachycardias with bizarre QRS complexes and QRS complexes with bizarre QRS complexes of possible variations in patient populations in view of the total found in the literature to date.
V-A conduction times of 0.12 to 0.17 second and no progressive increase of conduction time from the beginning to the end of the run.

The Rate of Tachycardia and V-A Conduction

One-to-one V-A conduction occurred with ventricular rates of 91, 94, 103, 107, 130, 151, 182, 186, 194, and 207. Independent atrial rhythm occurred with ventricular rates of 98, 120, 136, 140, 146, 150, 167, 171, 177, 207, and 273. Rate alone does not seem to be a factor determining 1:1 V-A conduction in this series, although it is possible that at ventricular rates greater than those observed in these patients, conduction might be interfered with because of rate alone. Lewis3 found
1:1 V-A conduction frequently in dogs at rates below approximately 220; at faster rates the mechanism was usually 2:1 V-A block and rarely 4:1 V-A block.

**Differentiation from A-V Nodal Tachycardia with Aberrant Conduction**

The question may be raised whether the ectopic complexes initiating the tachycardias originate not in the ventricle but rather in the A-V node and are conducted aberrantly because of occurrence during a partially refractory phase. Evidence that most of the tachycardias with retrograde conduction to the atria in this study were of ventricular origin consists of (1) the V-A conduction times, (2) normal forward conduction with atrial premature systoles, ventricular captures by sinus beats and reciprocal beats, (3) fusion between ectopic and sinus beats and fusion between ectopic and reciprocal beats, and (4) persistence of the bizarre form of the ectopic QRS with wide variations in the intervals between ectopic complexes.

**V-A Conduction Times**

The V-A conduction times were 0.12 to 0.52 second. Briefer intervals between QRS and P such as might be expected with A-V nodal rhythm did not occur. With the technic used it is possible to recognize P occurring simultaneously with QRS in A-V nodal rhythm and to measure small QRS-to-P intervals in such rhythm. Tachycardias in which the onset was with a P wave were not included in this study, so that so-called upper A-V nodal tachycardias are excluded.

In the five cases in which 1:1 V-A conduction alone occurred, the V-A conduction times were 0.12 to 0.20 second. In the five cases in which runs of 1:1 V-A conduction were associated with other runs with different atrial mechanisms, the V-A conduction times were 0.13 to 0.30 second. In the four cases with irregular V-A conduction and no runs with 1:1 conduction, the V-A conduction times were 0.12 to 0.52 second.

In 10 instances the V-A conduction times were about the same as P-R or longer (table 1), in four instances shorter (cases 2, 9, 11, and 12). Since the ventricular premature systole may occur during the refractory period produced by the previous systole, it is expected that the V-A conduction time will sometimes be longer than the A-V time. That the V-A time is often equal to or shorter
than A-V time requires comment, since it has been stated that V-A time in man is regularly longer and that there may normally be unidirectional block in the A-V node so that V-A conduction is blocked or delayed compared to A-V conduction.

Studies with esophageal leads show that V-A conduction occurs commonly, and while V-A intervals longer than A-V occur as expected, V-A intervals equal to or briefer than A-V occur also. In figure 7 there seems little doubt that the tachycardia originates from a ventricular focus (see below), and yet P-R is 0.15 second and the V-A conduction time is 0.12 second. There are a number of reasons why a V-A time equal to or shorter than P-R in the same tracing cannot be used as evidence against a ventricular origin of the ectopic focus. First, the experimental evidence was previously reviewed; the results varied, but in some studies V-A times were shorter than A-V times. Recent studies of conduction velocity in individual myocardial fibers show that conduction through the A-V node is about as rapid in one direction as in the other. There may possibly be a site of delay in retrograde conduction during the refractory period at the junction of the Purkinje fibers and myocardial fibers, but even here retrograde conduction during recovery is as rapid as in the forward direction. Second, the times as measured in the clinical electrocardiogram are a crude index of conduction velocity. There may be limitations in measurement; the onset of the ectopic ventricular systole may not be recorded in the leads used, or it may be unrecognizable, being superimposed on the preceding T wave. The fiber distance traveled in V-A conduction is not the same as that in A-V conduction, the exact paths not being known, and it could conceivably be shorter. For example, from a ventricular focus near the A-V node the electrocardiographically recorded V-A time is occupied by conduction from focus-to-AV node-to first part of atrium activated, a fiber distance which could conceivably be shorter than that from atrium near S-A node-to A-V node-to first part of ventricle activated, conduction along which is represented by P-R. Third, V-A conduction could possibly occur by a different pathway with faster conductivity. There is some clinical and experimental evidence for multiple
### Table 2

**Comparison of Ectopic Systoles Initiating Tachycardia and Atrial Premature Systoles in the Same Tracing**

<table>
<thead>
<tr>
<th>Patient number</th>
<th>Atrial premature systole followed by normal QRS</th>
<th>Ectopic systole initiating tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Duration of preceding cardiac cycle (sec.)</td>
<td>Coupling, preceding QRS to QRS of atrial premature systole (sec.)</td>
</tr>
<tr>
<td>1</td>
<td>0.68</td>
<td>0.41</td>
</tr>
<tr>
<td>2</td>
<td>0.56</td>
<td>0.37</td>
</tr>
<tr>
<td>3</td>
<td>0.58</td>
<td>0.39</td>
</tr>
<tr>
<td>5</td>
<td>1.12</td>
<td>0.63</td>
</tr>
<tr>
<td>9</td>
<td>0.58</td>
<td>0.43</td>
</tr>
<tr>
<td>10</td>
<td>0.60</td>
<td>0.38</td>
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<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>1.15</td>
<td>0.72</td>
</tr>
<tr>
<td>12</td>
<td>0.63</td>
<td>0.58</td>
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<td></td>
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</tr>
<tr>
<td>13</td>
<td>0.55</td>
<td>0.36</td>
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<tr>
<td>13</td>
<td>0.55</td>
<td>0.36</td>
</tr>
</tbody>
</table>

### Atrial Premature Systoles

Convincing evidence of ventricular origin of the ectopic beat initiating the tachycardia is the occurrence, in the same tracing under similar conditions, of forward conduction from above the bifurcation of the bundle of His, giving rise to normal QRS complexes. (By normal QRS is hereafter meant a QRS like one of sinus origin, although in two of the cases the QRS of sinus origin showed intraventricular block [fig. 8].) This was observed with atrial premature systoles in five of the cases with 1:1 V-A conduction (cases 1, 2, 5, 9, and 10) and in three of the cases with irregular V-A conduction (cases 11-13). The availability of atrial premature systoles for comparison was in part fortuitous, but also in part related to the long periods of recorded observation of some of the patients. For example, two atrial premature systoles occurred in 27 minutes of recorded tracings in case 1. During cardiac catheterization and cardiac surgery the occurrence of both atrial and ventricular premature systoles is usual.

Of the conditions that may influence refractoriness of myocardium and therefore aberrant conduction, there are two that can be measured in the electrocardiogram: (1) the duration of the cardiac cycle preceding the ectopic systole and (2) the coupling interval or the interval between the ectopic systole and the preceding ventricular systole. Aberrant conduction should occur more readily with longer preceding cardiac cycles and shorter coupling intervals. In table 2 some ectopic systoles initiating the tachycardias were selected for comparison with some atrial premature systoles in the same tracing giving rise to normal QRS complexes. The evidence is against an A-V nodal focus with aberrant...
conduction, except that this possibility is not ruled out in case 12 in which the coupling of the bizarre QRS is a little shorter than that of the QRS of the atrial premature systole.

Ventricular Captures, Reciprocal Beats, Fusion Beats

Forward conduction with normal QRS occurred also with ventricular captures from sinus beats during the tachycardia and fusion of sinus and ectopic beats (cases 13 and 14), and reciprocal beats (cases 6 and 13) and fusion of reciprocal and ectopic beats (cases 13). Reciprocal beats with normal QRS are illustrated in figures 2 and 4, and a ventricular fusion of reciprocal and ectopic beats is illustrated in figure 5. The
interval between the reciprocal beat and the preceding ectopic beat in figure 4 is close to the intervals between ectopic beats, suggesting a ventricular focus of the ectopic beats. The fusion beat in figure 5 practically excludes the possibility that the ectopic beats arise in an A-V nodal focus. In figure 2 the occurrence of the reciprocal beat with normal QRS is highly suggestive that the ectopic focus is ventricular, but it is not conclusive. The interval between reciprocal beat and previous ectopic beat is longer than the interval between ectopic beats during the tachycardia, and recovery from a refractory period for the reciprocal beat is possible.

The diagram of figure 5 illustrates the interpretation that reciprocal impulses from some ectopic beats fail to reach the ventricle because retrograde conduction from the immediately following ectopic beat produces refractoriness of the part of the conduction path common to retrograde and reciprocal impulses. Such a mechanism was postulated by Pick and Langendorf.46

Persistence of Form of QRS with Varying Intervals

In two cases of 1:1 V-A conduction (cases 4 and 10) the persistence of the form of the ectopic QRS, in spite of pronounced prolongation of the interval between ventricular complexes, is evidence of ventricular rather than A-V nodal origin of the ectopic focus. In figure 8 toward the end of the illustrated run, the interval between ectopic systoles is close to that between the sinus beats. There seems no reason why an A-V nodal focus should be conducted aberrantly at this time.

The Form of QRS

It is believed that the QRS more frequently assumes the pattern of right bundle-branch block in aberrant ventricular conduction,47 although the pattern of left bundle-branch block may occur also.48 There was no right bundle-branch block pattern in cases 1, 7, 8, and 14. There was a pattern possibly of atypical right bundle-branch block in cases 9 and 11. In the other patients the tachycardia was not observed in the leads necessary for the diagnosis; some patients with infrequent runs of tachycardia were observed as long as possible on lead II and esophageal leads, and the same procedure was used for observation during surgery and cardiac catheterization.

To complete the discussion of the differentiation of ventricular from A-V nodal systoles one must refer to the interesting observations of Rakita, Kennamer, Rothman, and Prinzmetal49 that experimental irritation of the A-V node may produce bizarre QRS complexes. According to the authors this occurs when the A-V node is injured, only part of the fibers from the node being activated, these fibers passing far out into the ventricle without anastomosis with adjacent fibers. Whether
Case 2. Simultaneous lead II and V esophageal lead. Comparison of preceding cardiac cycle and coupling interval of ectopic systole initiating tachycardia (left) and atrial premature systole (right) from same tracing. This illustrates the method of table 2. A-V conduction after the atrial premature systole giving rise to a QRS like the QRS after a sinus beat at intervals comparable to the ectopic systole that initiates the tachycardia is evidence that the ectopic focus is ventricular. Ventricular tachycardia with 1:1 V-A conduction starting with the second ectopic beat (left), retrograde P waves marked by arrows. No retrograde conduction after first ectopic beat because of interference with sinus beat. This tracing is exceptional in the series in that the retrograde P waves show clearly in lead II. A similar consecutive run of 23 beats was observed with no change in V-A conduction time.

Case 4. Simultaneous lead II and V esophageal lead. Lower strips continuous with upper. Ventricular tachycardia with 1:1 V-A conduction and pronounced slowing of ventricular rate. QRS form of the ectopic beats maintained even when interval between ectopic beats equals or exceeds that between sinus beats. This is evidence that the ectopic focus is ventricular, since at these longer intervals there is no reason for aberrant conduction from an A-V nodal focus. The peaks of the retrograde P waves in the VE lead are 2 to 5 mm. above the peaks of the ectopic QRS. First ectopic QRS probably superimposed on sinus P.

this is clinically significant, and whether anything like such a mechanism could be involved in cases without clinical evidence of A-V nodal injury, one cannot say at present. If an ectopic focus in part of the A-V node could produce bizarre QRS complexes by the suggested mechanism of Rakita et al. while an impulse from the atrium or a reciprocal route could pass through the A-V node to the ventricle to produce normal QRS complexes, then some of the evidence presented here for the ventricular origin of
the ectopic foci would not be conclusive. Nothing more can be said at present about such a possibility, which would question the origin of the common premature systoles conventionally considered ventricular.

Differential Diagnosis of Ventricular and Supraventricular Tachycardia

Should 1:1 V-A conduction in ventricular tachycardia occur with anything like the frequency suggested by this study, then the problem of differentiation between ventricular tachycardia and supraventricular tachycardia with aberrant conduction is more complicated than has been supposed. On the assumption that 1:1 V-A conduction in ventricular tachycardia is rare, the finding in esophageal tracings of a 1:1 relation of QRS and P has been used as evidence of supraventricular tachycardia. It has previously been emphasized that this is no absolute distinction, and the present study may indicate that it does not have even a probability value in differential diagnosis. The serious limitations of some of the criteria in use for the diagnosis of ventricular tachycardia have been thoroughly analyzed. This study casts additional doubt on one of the classical criteria, namely, the independent atrial rhythm, which may be absent in ventricular tachycardia more frequently than has been realized.

Summary and Conclusions

Ventriculo-atrial (V-A) conduction in ventricular tachycardia has been recognized relatively frequently in studies with simultaneous esophageal and standard leads. Of 21 cases interpreted as ventricular tachycardia there was 1:1 V-A conduction alone in five, 1:1 V-A conduction in some runs of tachycardia with other mechanisms in other runs in five, V-A conduction with variable block in four, and an independent atrial rhythm alone in seven.

Evidence that the ectopic foci in these cases are indeed ventricular rather than A-V nodal with aberrant conduction is based on (1) V-A conduction times, (2) normal forward conduction with atrial premature systoles, ventricular captures by sinus beats and reciprocal beats, (3) fusion between ectopic and sinus beats and fusion between ectopic and reciprocal beats, and (4) persistence of the bizarre form of the ectopic QRS in spite of varying intervals between ectopic beats.

The frequency of 1:1 V-A conduction in ventricular tachycardia complicates the differential diagnosis from supraventricular tachycardia with aberrant conduction.

A bipolar esophageal lead is often superior to a V esophageal lead for the study of complex arrhythmias and V-A conduction. It is more likely than a V esophageal lead to show retrograde P waves more or less opposite in direction to the sinus P waves.

Acknowledgment

The author gratefully acknowledges the contributions to the study of Dr. Richard Langendorf, who critically reviewed the manuscript, Drs. Roger E. Wilcoxon and John J. Marra, who helped obtain tracings during left heart catheterization and surgery, and Drs. Sam M. Fox III, and Joseph C. Greenfield, National Heart Institute, who obtained tracings during right heart catheterization.

References


RETROGRADE CONDUCTION IN VENTRICULAR TACHYCARDIA


Heart Failure

It is impossible thoughtfully to survey, in the light of daily experience, the field of medical work covering diseases of the heart, varied as the manifestations may be, without realising the central problem to be failure of the heart to accomplish its work in lesser or greater degree. This work consists in the propulsion of blood through the circle of vessels in adequate quantity to meet the needs of the body in the ordinary and varied circumstances of life. The very essence of cardiovascular practice is recognition and early heart failure and discrimination between different grades of failure. This simple truth is not stated here for the first time; in theory it receives occasional homage from many. It emerges into view for a fleeting moment, to retreat and lie concealed beneath a mass of technical, and by comparison trivial, detail; it does not dominate cardiac practice as it should. When a patient seeks advice and heart disease is suspected, or is known, to be present, two questions are of chief importance. Firstly, has the heart the capacity to do the work demanded of it when the body is at rest? Secondly, what is the condition of the heart's reserves? These questions can be answered, and correctly answered, in almost all cases by simple interrogations and by bedside signs; and the answers force all other considerations into the background in most cases of chronic heart disease; they are essentials to sound prognosis and treatment.—Sir Thomas Lewis. Diseases of the Heart. New York, The MacMillan Company, 1933, p. 1.
Retrograde Conduction to the Atria in Ventricular Tachycardia

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