Value of Physical Signs in the Diagnosis of Ventricular Tachycardia

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Background Although the use of physical signs for the diagnosis of ventricular tachycardia (VT) was described in the early 1900s, their value in this role has never been systematically assessed.

Methods and Results Using a blinded, randomized protocol, we examined the ability of 26 clinicians to detect ventriculoatrial (VA) dissociation during cardiac pacing in 21 patients with both atrial and ventricular pacing wires in situ after successful ablation of accessory pathways. In protocol 1 (10 patients), pacing was randomized to either ventricular pacing alone (simulating VT) or to atrioventricular sequential pacing (simulating supraventricular tachycardia or VT with intact VA conduction) at rates of 150 or 180 beats per minute. Each patient was examined by four clinicians blinded to the pacing mode. Clinicians were asked to make a diagnosis of “VA association” or “VA dissociation” after examining the patient for variability of the arterial pulse, jugular venous pulse (JVP), and first heart sound. In protocol 2 (11 patients), randomization of pacing mode was performed between examination of each of the three physical signs so that the value of each sign was assessed individually. In protocol 1, a diagnosis of VA dissociation (VT) was made in 21 of 40 observations, with a specificity of 75%, sensitivity of 70%, and a positive predictive value (PPV) of 71%. In protocol 2, from a total of 132 observations (44 for each sign), the sensitivity, specificity, and PPV for a diagnosis of VT were as follows: arterial pulse, 61%, 71%, 70%; JVP, 96%, 75%, 82%; and first heart sound, 58%, 100%, 100%.

Conclusions It is concluded that, in patients with a regular tachycardia of uncertain origin, clinically detectable variations in the first heart sound and JVP are highly specific and sensitive indicators, respectively, of a diagnosis of VT. Assessment of the arterial pulse is of little value in this role.

Methods

Patient and Clinician Selection

All patients on the cardiology wards with temporary atrial and ventricular pacing wires inserted for clinical reasons were asked if they would participate in the study. Approval for the study was given by the local ethics committee, and informed consent was obtained from all patients. Twenty-one patients were included in the study (10 in protocol 1, 11 in protocol 2). Nineteen had temporary pacing wires inserted for repeat electrophysiological testing after catheter ablation of accessory pathways. None of these patients had clinical or echocardiographic evidence of structural heart disease, and mean age was 37 years. One 62-year-old patient had dilated cardiomyopathy, and one 65-year-old patient had complete heart block and an otherwise normal heart. Temporary pacing electrodes (6F bipolar right ventricular lead, 5F bipolar “J” right atrial lead) had been inserted via the right or left subclavian venous route, as is our usual clinical practice. Patients who had given consent to the study underwent ventricular pacing at three rates (120, 150, and 180 beats per minute [bpm]) for 5 minutes at each rate while an electrogram was recorded from the atrial lead (Fig 1). Five patients were excluded from further study because intact VA conduction was evident (from the atrial electrogram) at these rates. Three patients were excluded because of discomfort during pacing at the higher rates.

Clinicians were recruited for the study if they had regular contact with medical patients in an acute setting (emergency room or hospital ward) and were in the vicinity of the cardiology ward at the time that a patient was being studied. No clinicians refused to take part in the study.

Protocol 1

Patients were randomized to ventricular pacing or AV sequential pacing and to one of two rates (150 or 180 bpm).

Key Words: pacing • tachycardia • diagnosis
Analysis of Results

The sensitivity of the physical signs to detect VA dissociation was defined as true positives (correct diagnoses of VT) divided by the sum of true positives and false negatives (incorrect diagnoses of VA association). Specificity was defined as true negatives (correct diagnoses of VA association) divided by the sum of true negatives and false positives (incorrect diagnoses of VT). Positive predictive value refers to the study population (50% VT, 50% AV association) and was defined as true positives divided by the sum of true positives and false positives.

Results

Clinician Characteristics

Twenty-six clinicians took part in the study: one attending physician, 2 senior cardiology fellows, 7 junior cardiology fellows, 8 senior residents, and 8 junior residents. All clinicians had some experience of assessment of physical signs in the setting of clinical cardiology (range, 3 months to 8 years), but only 2 claimed to have any experience of the use of physical signs as a means of arrhythmia diagnosis. Eleven clinicians examined only 1 patient, 3 examined 2 patients, 4 examined 5 patients, and 6 examined 6 patients. The great majority of clinicians took less than 5 minutes for the examination, and none took longer than 8 minutes.

Value of Physical Signs to Diagnose VT

A total of 40 observations were made in protocol 1 (10 patients each examined by 4 clinicians). A diagnosis of “VT” was made in 21 of the 40 observations, with a sensitivity of 75% (with 15 true positives from 20 observations during VA dissociation), specificity of 70% (with 6 false positives from 20 observations duringVA association), and positive predictive value of 71% for this diagnosis in the study population. A total of 132 observations were made in protocol 2. Eleven patients were each examined by 4 clinicians, resulting in 44 observations for each of the three physical signs. The sensitivity, specificity, and positive predictive value of the individual signs for a diagnosis of VT were as follows: arterial pulse, 61%, 71%, 70%; jugular venous pressure, 96%, 75%, 82%; and first heart sound, 58%, 100%, 100%. A clinically detectable variation in the first heart sound was absolutely specific for a diagnosis of VT, with no false positives (from a total of 20 observations during VA association) and 14 true positives (from 24 observations during VA dissociation). The relatively poor predictive value of the clinical signs when assessed together (protocol 1) compared with examination of the first heart sound alone (protocol 2) may have been due to relative neglect of the findings of auscultation in favor of those of the arterial and venous pulse when all the signs are considered together (Table 1). The arterial pulse was found to be of little value in the diagnosis of VT both in terms of specificity and sensitivity. Although variability of the venous pulse was highly sensitive for a diagnosis of VT (with 23 true positives from 24 observations during VA dissociation), this sign was only 75% specific (5 false positives from a total of 20 observations during VA association).
1. Ventricular Tachycardia Protocol

2. Protocol Effect

VT Diagnosis

3. TABLE 1. Value of Physical Signs in the Diagnosis of Ventricular Tachycardia

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<th>Sensitivity, %</th>
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<th>PPV, %</th>
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<td>JVP</td>
<td>96</td>
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<td>82</td>
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<tr>
<td>First heart sound</td>
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PPV indicates positive predictive value; JVP, jugular venous pulse.

Effect of Tachycardia Rate on Ability to Diagnose VT

The effect of tachycardia rate on the value of clinical signs to diagnose VT is described in Table 2. In protocol 1, 6 patients were examined at 150 bpm (total of 24 observations) and 4 were examined at 180 bpm (total of 16 observations). In protocol 2, 4 patients were examined at 120 bpm (16 observations for each sign), 4 at 150 bpm (16 observations for each sign), and 3 at 180 bpm (12 observations for each sign). When all the signs were assessed together (protocol 1), tachycardia rate had no detectable effect on the usefulness of the signs. Similarly, there was no clear relation between heart rate and the value of the arterial pulse when this sign was assessed separately (protocol 2). The absolute specificity of a variable first heart sound for a diagnosis of VT was, of course, independent of heart rate. The sensitivity of this latter sign, however, showed a direct relation with heart rate, so that at a rate of 180 bpm the sensitivity was 75%, compared with 50% for 150 bpm and 27% for 120 bpm. The specificity and sensitivity of the venous pulse also increased at the higher heart rate (Fig 2), with both values reaching 100% at 180 bpm, although only 12 observations were made for each sign at this rate (8 observations of VA dissociation and 4 of VA association, all correctly diagnosed).

Effect of ‘Learning’ on the Value of Clinical Signs

Twenty-one clinicians took part in protocol 1. Eleven of these examined only 1 patient. Five clinicians saw 2 patients, 3 saw 3 patients, and 2 saw 5 patients. When assessment of the value of the physical signs is based only on the first patient examined by any clinician in protocol 1, the specificity and positive predictive value of all the signs together for a diagnosis of VT fall to 54% and 58%, respectively (Table 3). This suggests that the

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<tr>
<td>First heart sound</td>
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PPV indicates positive predictive value; JVP, jugular venous pulse.

*First patient examined by any particular clinician only.
ability to detect VA dissociation during tachycardia from the three physical signs together may be particularly poor in the absence of previous experience of assessment of these signs during tachycardia, possibly because of inappropriate attention to the arterial pulse (see below). Because the true pacing mode was revealed to the clinicians after the study of each patient, this may have led to a degree of “learning” in terms of the relative usefulness of the individual signs for making a diagnosis of VT.

Twenty-one clinicians took part in protocol 2. Ten of these examined only 1 patient. Six clinicians saw 2 patients, 2 saw 3 patients, 1 saw 5 patients, and 2 saw 6 patients. When assessment of the value of the physical signs was based only on the first patient examined by any clinician in protocol 2, it can be seen that there was a small fall in the sensitivity of the first heart sound and venous pressure as a test for VT, but specificity and positive predictive value were well maintained for both signs. These results suggest that, even in the absence of previous specific experience, these particular physical signs are very useful in the diagnosis of VA dissociation during tachycardia.

Discussion

Sir James MacKensie recorded the occurrence of slow independent jugular venous pulsations during presumed VT as long ago as 1908. Gallavardin recognized the clinical value of this observation and stated that if inspection of the jugular veins showed a recurring impulse occurring at a rate at about half that of the arterial rate, a diagnosis could be made of VT with conservation of the normal atrial rhythm. Other physical signs of mechanical AV dissociation were described by Levine (variability of intensity of the first heart sound during tachycardia) and by Wilson and coworkers (variability of arterial blood pressure). More recently, Schrire and Vogelpol examined the value of combined ECG, phonocardiographic, and jugular venous pulse recordings in 9 cases of proven VT, the definitive diagnosis having been made usophagial recordings of atrial activity. Eight of the 9 cases demonstrated both changing intensity of the first heart sound and independent slow jugular venous pulsations ("Cannon" A waves). In the remaining patient, coexistent atrial fibrillation was demonstrated. Although not tested in any controlled manner, the authors of this report were clearly of the opinion that these features of VT were detectable clinically, that is, without the use of phonocardiographic and jugular venous pulse recordings. Interest in the clinical signs of mechanical AV dissociation waned, however, with the recognition of cases of VT with 1:1 retrograde conduction to the atria and the development of ECG “rules” for the diagnosis of VT.

The use of pacing techniques to simulate tachycardias rather than the use of spontaneously occurring arrhythmias in the study allowed a blinded, randomized design that otherwise would not have been possible. As a direct consequence of the study design, the great majority of patients enrolled had structurally normal hearts, in contrast to patients presenting with spontaneous wide-complex regular tachycardias, who may have a number of structural myocardial abnormalities. The signs of mechanical AV dissociation during VT are dependent primarily on the presence of atrial contraction and will not be seen if the atrium is fibrillating or if there is another cause of atrial inactivity (electrical or mechanical). The coexistence of atrial fibrillation with VT in some patients is likely to result in a reduction of the sensitivity of physical signs for the diagnosis of VT in clinical practice. The presence of significant tricuspid incompetence (due to primary valve disease or secondary to right ventricular dilation) and the occurrence of VT with intact retrograde conduction are other potential causes of a decrease in sensitivity of the physical signs in clinical practice. In contrast, it is very unlikely that the specificity of the physical signs of mechanical VA dissociation would be reduced by the presence of structural heart disease. Although it is possible that variability of arterial pulse amplitude may be simulated by pulsus alternans associated with abnormal left ventricular function, it is difficult to conceive of a structural or functional anomaly that could mimic the slow, independent jugular venous pulsations or variability of the first heart sound associated with mechanical VA dissociation.

VT is not the only cause of VA dissociation during wide-complex tachycardia. AV nodal reentrant tachycardia may be conducted with aberration to the ventricles (producing a regular wide-complex tachycardia) and with retrograde block to the atria. This causes of VA dissociation is so rare, however, that it continues to be the subject of case reports 20 years after its initial recognition. As a consequence, its relevance to the specificity of physical signs for identifying VT is likely to be minimal.

By design, none of the patients included in this study suffered hemodynamic compromise during pacing-simulated tachycardia. The relevance of the findings of the study to very fast and hemodynamically unstable arrhythmias is not known. However, as the value of a clinical means of tachycardia diagnosis applies principally to hemodynamically stable arrhythmias that do not require immediate cardioversion, we do not consider this a major limitation.

Most clinicians currently rely on patient history and specific ECG features to make a diagnosis of VT. Among patients referred for electrophysiological evaluation of broad-complex tachycardia, a history of previous myocardial infarction is known to have a very high predictive value for a diagnosis of VT. However, as pointed out by Tchou and coworkers, this patient group is already highly selected and is likely to differ from that presenting acutely to the emergency room. Patients with nonschismic cardiomyopathy (who may have VT in the absence of previous infarction) or atrial flutter in the setting of coronary disease (who may have supraventricular tachycardia after infarction) are often not referred for electrophysiological investigation. As a consequence, the true predictive value of the patient
history in the setting of the emergency room may be considerably less. Similarly, the sensitivity and specificity of the standard ECG criteria for the diagnosis of VT are suboptimal, with leads V₁ and V₆ frequently showing discordant morphology patterns that suggest different diagnoses.¹⁸ The most recent “rules” are those of Brugada and coworkers,¹⁸ who have devised a stepwise ECG approach based on the presence and width of an RS complex in the precordial leads during tachycardia, VA dissociation inferred from P-wave activity, and specific QRS patterns in V₁ and V₆. Although VA dissociation inferred from the ECG was 100% specific for VT, it could only be detected in 21% of VT episodes. Nevertheless, using their stepwise approach, these workers achieved very high sensitivity (99%) and specificity (96%) for a diagnosis of VT. It should be noted, however, that these results refer to ECG of patients not taking antiarrhythmic drug therapy. Specificity is very likely to fail if the patients are taking agents of antiarrhythmic class 1 or 3 at the time of presentation.¹⁹,²⁰ In addition, these figures refer to ECG interpretation (including specific patterns in V₁ and V₆) by arrhythmia specialists at a time remote from patient presentation. Numerous publications have established the much poorer performance of nonspecialists at the time of first presentation of the arrhythmia.⁶⁻⁸ The current study was not designed to compare the value of physical signs with that of the patient history or ECG (or any other means of diagnosis) for the identification of VT. Nevertheless, the very high positive predictive value of a variable first heart sound (and of Cannon A waves during tachycardias with rates of 180 bpm) is likely to compare favorably with the patient history, the ECG, or the combination of the two in the diagnosis of VT.

The findings of this study support those of Gallavardin⁹ (1920) and Levine¹⁰ (1927) and suggest that examination of the venous pressure waveform and first heart sound during tachycardia is of considerable value in the differential diagnosis of a broad-complex regular tachycardia. Direct comparison with other diagnostic methods in the clinical setting is warranted.

References

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Circulation. 1994;90:3103-3107
doi: 10.1161/01.CIR.90.6.3103

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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