Case reports

British Heart Journal, 1974, 36, 512-515.

Ventricular tachycardia with retrograde conduction *Simplified diagnostic approach*

Gerald L. Evans¹, M. Arthur Charles, and Charles T. Thornsward

From the Cardiology Service, Walter Reed General Hospital, Washington D.C., U.S.A.

The diagnosis of ventricular tachycardia is primarily dependent upon the demonstration of dissociated atrial and ventricular activity. When retrograde conduction remains intact in patients with ventricular tachycardia, preserving a 1:1 relation between the ventricles and the atria, the diagnosis cannot be established in the usual fashion.

This report demonstrates a simplified diagnostic approach to this problem.

The diagnosis of ventricular tachycardia is primarily dependent upon the demonstration of dissociated atrial and ventricular activity (Mackenzie and Pascual, 1964). This may be visualized as asynchronous pulsation of the neck veins or may be evident on auscultation by a varying intensity of the first heart sound. With rapid ventricular rates these observations are not easily made. In addition, more and more patients with ventricular tachycardia are being recognized with intact retrograde conduction, preserving a 1:1 relation between the ventricles and atria (Massumi, Tawakkol, and Kistin, 1967; Goldreyer and Bigger, 1970). In such cases, the usual diagnostic tools are not applicable, and the distinction between a supraventricular tachycardia with aberrant conduction and ventricular tachycardia with retrograde conduction has not been an easy one. The following case illustrates both the problem and a simple bedside solution.

Case report

A 60-year-old man had an acute illness at 29 years of age characterized by severe substernal pain, profuse sweating, nausea, and vomiting. He received no definitive medical care and recovered after several days of extreme prostration.

Six years later, at age 35, a routine chest x-ray showed an abnormal bulge in the area of the left ventricle and an electrocardiogram was consistent with an anterior wall myocardial infarction. He was entirely well for the next 21 years until, at the age of 56, he began experiencing palpitations of one to two minutes' duration. At the age

of 58 a persistent episode of palpitations occurred and he was seen for the first time at Walter Reed General Hospital. An electrocardiogram (Fig. 1a) showed a regular rhythm at a rate of 150 a minute with a QRS of 0.13 sec duration. There were no discernible P waves. Carotid sinus massage produced no changes. The presumptive diagnosis was ventricular tachycardia and he was promptly cardioverted without difficulty. After cardioversion his electrocardiogram (Fig. 1b) showed a normal sinus rhythm at a rate of 60 a minute. The R wave was absent in leads V1 to V5 with persistent ST segment elevation in the same leads. Posteroanterior x-ray of the chest (Fig. 2) showed a densely calcified left ventricular aneurysm.

The arrhythmia recurred frequently over the next 18 months, despite therapy with digoxin, quinidine, procainamide, propranolol, and phenytoin, singly and in various combinations. On each occasion cardioversion was necessary to terminate the tachyarrhythmia. At no time was atrial activity evident by venous pulse waves, auscultatory variation of the first heart sound, or standard electrocardiography.

On one occasion, in order to determine definitively the nature of the rhythm disturbance, a unipolar, 'teflon'-coated, platinum-tipped electrode wire was inserted in the right antecubital vein and guided fluoroscopically to the right atrium. Proper grounding of all equipment was ensured to protect the patient against any electrical hazard. Using a Sanborn dual channel recorder, a simultaneous lead III and an intra-atrial electrocardiogram were recorded at a paper speed of 50 mm/sec (Fig. 3a). It can readily be seen in the intra-atrial recording (lower tracing) that there is one P wave for every QRS complex. This tracing is consistent with either an atrial tachycardia with aberrant ventricular conduction, or a ventricular tachycardia with 1:1 retrograde conduction. In an effort to differentiate between these two possi-

¹ Present address: Department of Cardiology, Framingham Union Hospital, Framingham, Massachusetts, U.S.A.

FIG. 1 (a) 12-lead electrocardiogram showing a ventricular rate of 150 a minute with a QRS of 0.13 sec. Atrial activity is not evident. (b) 12-lead electrocardiogram after cardioversion, showing a sinus rhythm at a rate of 60 a minute. PR interval = 0.20 sec; QRS = 0.08 sec. Absent R wave in leads V1 to V5 with persistent ST segment elevation indicates an antecedent anterior wall myocardial infarction and possible aneurysm formation.

FIG. 2 Posteroanterior chest x-ray showing a densely calcified left ventricular aneurysm.

FIG. 3 (a) Simultaneous lead III (above) and intra-atrial lead (below). Recorded at 50 mm/second. Ventricular rate = 150/minute. There is one P wave for each QRS. The distinction between atrial tachycardia with aberrant ventricular conduction and ventricular tachycardia with 1:1 retrograde conduction cannot be made. (The AV ladder diagram (given to help the reader) indicates that the diagnosis is ventricular tachycardia.) (b) With application of carotid sinus massage, the QRS complexes remain unaltered, but the P waves disappear. Retrograde block has been produced, establishing the diagnosis of ventricular tachycardia.

bilities, carotid sinus massage was again attempted (Fig. 3b). The QRS complexes remained unaltered, but the P waves disappeared. Carotid sinus massage produced retrograde block and established the diagnosis of ventricular tachycardia. This effect was not apparent during earlier attempts at carotid sinus massage because P waves were not demonstrable on the standard electrocardiogram. The combination of an intra-atrial electrocardiogram and carotid sinus massage was necessary to establish this diagnosis.

Discussion

The distinction between supraventricular tachy-

cardia with aberrant ventricular conduction and ventricular tachycardia is usually made without great difficulty. The presence of a right bundle-branch block pattern, particularly when triphasic, supports a diagnosis of supraventricular tachycardia with aberrant ventricular conduction. Intermittent conducted beats from the atrium producing capture or fusion beats confirm a diagnosis of ventricular tachycardia. Demonstration of dissociated atrial and ventricular activity, though difficult on physical examination, can frequently be made by standard electrocardiography. Variation of

the QRS configuration produced by independent atrial activity is usually evident. When the routine 12-lead electrocardiogram does not show atrial activity, bipolar chest leads, an oesophageal lead, or intra-atrial recordings will invariably demonstrate atrial activity. However, when ventricular tachycardia occurs with intact retrograde conduction, the distinction between supraventricular tachycardia with aberrant conduction and ventricular tachycardia with retrograde conduction is considered almost impossible to make (Massumi *et al.*, 1967; Kistin, 1961).

That retrograde conduction occurs frequently has only recently been recognized (Kistin, 1961). Since this phenomenon is not readily seen on the standard electrocardiogram, most observations have been made in the catheterization laboratory during the recording of intracavitary electrocardiograms (Massumi *et al.*, 1967). Retrograde conduction is fairly common in the presence of premature ventricular contractions, particularly in normal patients (Goldreyer and Bigger, 1970). While less common in patients with significant heart disease, it is certainly seen and has been observed in patients with acute myocardial infarction (Scheinman, 1971) and even in patients with bilateral bundle-branch block (Schuilenburg and Durrer, 1970). The conduction pattern may be 1:1, as in our patient, or show varying degrees of second-degree retrograde block, either of the Mobitz I (Wenckebach) or Mobitz II type (Massumi *et al.*, 1967; Kistin, Tawakkol, and Massumi, 1969).

It has been suggested that comparison of antero-grade and retrograde conduction times might be helpful in distinguishing these two arrhythmias (Damato, Lau, and Bobb, 1970). These authors found in dogs that antero-grade conduction time is consistently shorter than retrograde conduction time at comparable ventricular rates. Others dispute the value of this finding and show examples in man where retrograde conduction time is actually less than antero-grade conduction time (Kistin *et al.*, 1969). When antero-grade and retrograde conduction times were analysed in our patient, the findings did not support the proven diagnosis of ventricular tachycardia. Since the antero-grade conduction time in normal sinus rhythm at a rate of 60 a minute was 0.20 sec, the retrograde conduction time when he was in ventricular tachycardia at a rate of 140 a

minute should have been greater than 0.20 sec. It was, however, exactly 0.20 sec. Analysis of antero-grade and retrograde conduction time was, therefore, not useful.

The key diagnostic feature in solving this problem was the production of junctional block while recording the intra-atrial electrocardiogram. This can be accomplished pharmacologically by drugs such as propranolol (Maytin, Castillo, and Castellanos, 1971) or more simply, as we have done, by carotid sinus massage. Carotid sinus massage influences conduction through the junctional tissue in both an antero-grade and retrograde direction. This effect was not visualized on the standard electrocardiogram because the atrial activity was not visible but was clearly demonstrated on the intra-atrial electrocardiogram.

Thus, the simultaneous application of two frequently used diagnostic tools enabled us to make the diagnosis of ventricular tachycardia, when either one alone was unsatisfactory.

References

- Damato, A. N., Lau, S. H., and Bobb, G. A. (1970). Studies on ventriculo-atrial conduction and the reentry phenomenon. *Circulation*, **41**, 423.
- Goldreyer, B. N., and Bigger, J. T., Jr. (1970). Ventriculo-atrial conduction in man. *Circulation*, **41**, 935.
- Kistin, A. D. (1961). Retrograde conduction to the atria in ventricular tachycardia. *Circulation*, **24**, 236.
- Kistin, A. D., Tawakkol, A., and Massumi, R. A. (1969). Atrial rhythm in ventricular tachycardia occurring during cardiac catheterization. *Circulation*, **35**, 10.
- Mackenzie, G. J., and Pascual, S. (1964). Paroxysmal ventricular tachycardia. *British Heart Journal*, **26**, 441.
- Massumi, R. A., Tawakkol, A. A., and Kistin, A. D. (1967). Reevaluation of electrocardiographic and bedside criteria for diagnosis of ventricular tachycardia. *Circulation*, **36**, 628.
- Maytin, O., Castillo, C. A., and Castellanos, A., Jr. (1971). Complete V-A block (without impairment of forward conduction) produced by propranolol. *American Journal of Cardiology*, **27**, 687.
- Scheinman, M. M. (1971). Retrograde ventriculoatrial conduction in patients with acute myocardial infarction. *Chest*, **59**, 165.
- Schuilenburg, R. M., and Durrer, D. (1970). Observations on atrioventricular conduction in patients with bilateral bundle-branch block. *Circulation*, **41**, 967.

Requests for reprints to Dr. Charles T. Thornsward, Cardiology Service, Walter Reed General Hospital, Washington, D.C. 20012, U.S.A.