Ventricular tachycardia as default diagnosis in broad complex tachycardia

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Summary

In the differential diagnosis of broad-complex tachycardia, the most important decision is whether or not the tachycardia is ventricular, since this type carries the worst prognosis. However, the rules for a diagnosis of ventricular tachycardia are so complex that they are not satisfied in many cases, and the default diagnosis, supraventricular tachycardia, is erroneously accepted. We sought to reverse this strategy; unless simple rules for a positive diagnosis of supraventricular tachycardia were satisfied, ventricular tachycardia was diagnosed by default.

The criterion for a diagnosis of supraventricular tachycardia was electrocardiographic (ECG) findings typical of bundle branch block (left = rS or QS wave in leads V1 and V2, delay to S wave nadir < 70 ms, and R wave and no Q wave in lead V6; right = rSR' wave in lead V1 and an RS wave in lead V6, with R wave height greater than S wave depth). Twelve-lead ECGs were done for 102 consecutive patients with broad-complex tachycardia (QRS width > 110 ms). Two observers, who were unaware of definitive diagnoses validated by electrophysiology, by our diagnostic rules made correct diagnosis of ventricular tachycardia in 63 and 62 of 69 patients, respectively, and correct diagnoses of supraventricular tachycardia in 28 and 22 of 33 patients (sensitivity for ventricular tachycardia 90% and 91%, specificity 67% and 85%). One observer then sought independent P waves in cases diagnosed as supraventricular tachycardia; sensitivity for the diagnosis of ventricular tachycardia rose to 96%, with a specificity of 64%.

These criteria, which require only knowledge of typical bundle branch block patterns, were highly sensitive for the important diagnosis of ventricular tachycardia.

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Introduction

Because the outlook is much worse for a patient with ventricular than with supraventricular tachycardia, the most important decision in the differential diagnosis of broad-complex tachycardia is whether or not the origin is ventricular. However, the rules required for this diagnosis are so complex that they are not satisfied in many cases[1,2] and the default diagnosis, supraventricular tachycardia, is made.[3–10] We sought to reverse this strategy, by use of simple rules, which would be widely applicable in general electrocardiographic diagnosis, for a positive diagnosis of supraventricular tachycardia. If these simple rules were not satisfied, ventricular tachycardia would be diagnosed by default.

Patients and methods

Data were collected prospectively from 102 consecutive patients referred to St George's Hospital, London, between November, 1986 and November, 1988. On twelve-lead electrocardiogram (ECG), each patient had broad-complex monomorphic tachycardia (QRS width greater than 110 ms). If a patient had more than one type of tachycardia, that recorded at first presentation was analysed. Patients who had had a myocardial infarct in the previous seven days were exluded. ECGs were recorded with a paper speed of 25 mm/s.

The definitive diagnosis was made by invasive electrophysiological study in all patients. Diagnosis of ventricular tachycardia was confirmed by QRS complexes during tachycardia without a preceding His deflection; or QRS complexes with a shorter HV interval than that recorded in sinus rhythm. In patients with a normal or long HV interval during tachycardia, atrial or His deflection dissociation (or both) from the tachycardia confirmed that it was ventricular in origin. Pre-excitation of the ventricles was excluded by atrial pacing in sinus rhythm.

Two observers, who were unaware of the electrophysiological diagnoses, reported on ECGs using our criteria for a diagnosis of supraventricular tachycardia (table 1). One of the observers was then asked to look for independent P waves, or a less than one to one V-A relation, after he had applied the above rules and diagnosed supraventricular tachycardia.

Results

The two observers produced very similar results (table 2) with high sensitivity for the diagnosis of ventricular tachycardia (90–91%), but with lower specificity (67–85%).

	BBB pattern
Right	
V1	rSR' pattern with R' > r RS (RS) pattern with R > S; Q wave
V6	< 40 ms and < 2mm (0 2 mV) were allowed
Left	
V1	rS or QS pattern with time to 5 wave nadir < 70 ms
V6	R wave with no Q wave

BBB = bundle branch block.

Table 1: **Electrocardiographic criteria for diagnosis of supraventricular tachycardia**

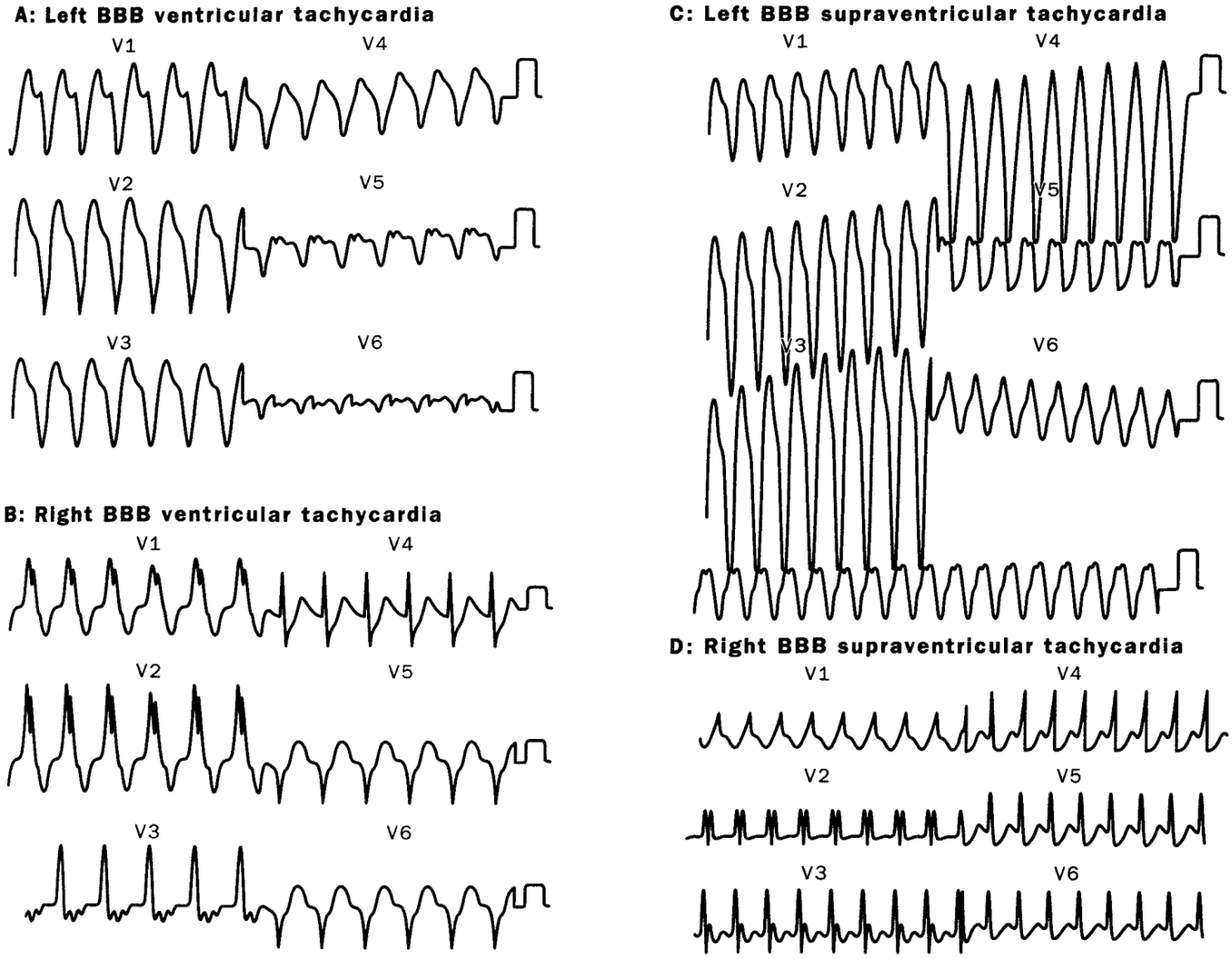

Figure: **Typical bundle branch block pattern tachycardias**

A: lead V1 shows 100 ms to S wave nadir; lead V6 has a Q wave (>40 ms long and >2 mm deep). B: lead V1 has RSR' pattern with R >R'; lead V6 has a Q wave (>40 ms long and > 2 mm deep). C: lead V1 has QS wave with 50 ms delay to S wave nadir; lead V6 has RS wave. D: lead V1 has rSR' pattern with R' >r; lead V6 has RS pattern with R >S.

Five out of 6 patients wrongly identified as having supraventricular tachycardia by observer 1 (5 of 7 patients for observer 2) had a left bundle branch block pattern with right axis deviation; these patients had right ventricular outflow tract tachycardia.

Observer 2 identified independent P waves in 4 of 7 patients who were wrongly diagnosed with supraventricular tachycardia, compared with only 1 of 22 who had a correct diagnosis of supraventricular tachycardia. Diagnosis on the basis of independent P waves alone had a low sensitivity (51%) but high specificity (94%) for the diagnosis of ventricular tachycardia. The criteria have been applied successfully to right and left bundle branch block pattern tachycardias (figure).

	Sensitivity	Specificity
Observer 1	91% (63/69)	85% (28/33)
Observer 2	90% (62/69)	67% (22/33)
Observer 2 and independent P waves	96% (66/69)	64% (21/33)
Independent P waves alone	51% (35/69)	94% (31/33)
Brugada criterion	83% (57/69)	67% (22/33)

Sensitivity: predicted positives/true positives.
Specificity: predicted negatives/true negatives.

Table 2: **Predictive value of criteria for diagnosis of ventricular tachycardia**

Discussion

Many patients presenting with broad-complex tachycardia are misdiagnosed as having supraventricular tachycardia with abberrant conduction.[1,2] This mistake can have immediate disastrous consequences because the patient may be given verapamil or other inappropriate therapy. If the error is not quickly recognised, subsequent management may also be inadequate and even lethal.

Misdiagnosis of supraventricular as ventricular tachycardia does not have the same dangers and will probably be rapidly recognised since most patients with ventricular tachycardia undergo electrophysiological testing. Despite many reports on this subject, misdiagnoses continue to be made. The main reason may be the central philosophy of the diagnostic process,[3-10] through which ventricular tachycardia must be positively diagnosed and supraventricular tachycardia is diagnosed by default. Rules for the positive diagnosis of ventricular tachycardia are multiple and complex, and the usual approach by inexperienced doctors is a search for independent P waves. If they are not identified (as in nearly half the patients in this study), the diagnosis of supraventricular tachycardia is made. If this philosophy were reversed, overdiagnosis of ventricular tachycardia would occur; this result would be much safer. Simple criteria such as typical left or right bundle branch block, which are part of basic

electrocardiographic training, can be easily memorised and used. A secondary search for independent P waves in the presence of typical bundle branch block increases the diagnostic yield for ventricular tachycardia.

When tested on our study population, these simple criteria were very sensitive for diagnosing ventricular tachycardia. The sensitivity was improved by the search for independent P waves when the initial diagnosis was supraventricular tachycardia. The sensitivity (96%) of this method compares very well with all other methods that have been described and evaluated.[3,5,8,10,12] The low specificity is less important, because the misdiagnosis of supraventricular as ventricular tachycardia carries little risk to the patient in the short term.

Most of the patients with ventricular tachycardia misdiagnosed as supraventricular tachycardia in this study had right ventricular outflow tachycardia. This type of ventricular tachycardia is difficult to recognise on morphological electrocardiographic criteria alone since the tachycardias have the typical morphology of left bundle branch block and the tachycardia is terminated by adenosine, a suggested diagnostic agent for supraventricular tachycardia. However it is usually possible to make the diagnosis of ventricular tachycardia by identifying independent P waves on the ECG in patients with right ventricular outflow tachycardia (415 patients in the series).

Combined with the change in diagnostic philosophy to make ventricular tachycardia the default diagnosis for broad-complex tachycardia, these criteria should lessen the likelihood of the serious and frequent error of diagnosing ventricular as supraventricular tachycardia with aberration.

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Short reports

Hepatitis C virus in multiple episodes of acute hepatitis in polytransfused thalassaemic children

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We investigated the course of distinct episodes of acute non-A, non-B (NANB) hepatitis in three polytransfused thalassaemic children. In each case, the first episode was associated with the appearance of serum hepatitis C virus (HCV) RNA and anti-HCV seroconversion. The second episode was accompanied by the reappearance of HCV viraemia, which in two patients was due to reinfection with a different HCV strain and in the third could be the result of either reactivation of primary infection or reinfection with a new but closely related strain. Thus HCV infection may not induce protective immunity, which has implications for vaccine development.

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With the development of serological assays to detect antibodies against hepatitis C virus (HCV),[1] we now know that HCV causes more than 90% of cases of post-transfusion hepatitis. Thus, other agents may account for only a few cases. Before the introduction of universal screening of blood donors for anti-HCV, thalassaemic patients on long-term transfusion therapy were at high risk of exposure to HCV. We documented that 61% of thalassaemic children experienced non-A, non-B (NANB) hepatitis.[2] As seen in drug addicts,[3] haemophiliacs,[4] and haemodialysed patients,[5] we observed multiple distinct episodes of acute NANB hepatitis in individual polytransfused thalassaemics.[2] Cross-challenge studies in chimpanzees provide evidence that reinfection with either homologous or heterologous strains of HCV may occur, suggesting a lack of protective immunity against HCV.[6] These findings prompted us to investigate the course of the multiple episodes of acute NANB hepatitis seen in thalassaemic children.

Markers of HCV viraemia and humoral immune response were analysed in three children, selected among those who had more than one episode of acute NANB hepatitis.[2] The three entered a long-term transfusion programme at age 8, 24, and 10 months, respectively. Before starting transfusion, all children were anti-HCV negative, as measured by first-generation and second-generation enzyme immunoassay (EIA-1 and EIA-2, Ortho), and had normal alanine aminotransferase (ALT) values. The design of the study and the storage conditions have been described.[2] The first episode of acute HCV hepatitis developed after transfusion of 19, 32, and 24 units of blood, respectively. The diagnosis of acute HCV hepatitis was based on increased ALT (peaks of 1870, 1050, and 740 IU/L, respectively), on the appearance of serum HCV RNA (polymerase chain reaction [PCR] amplification with