A new algorithm in the differential diagnosis of wide complex tachycardia

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This editorial refers to ‘Application of a new algorithm in the differential diagnosis of wide QRS complex tachycardia’† by A. Vereckei et al., on page 589.

A correct electrocardiographic diagnosis of the mechanism of a wide complex tachycardia (WCT) is important when instituting emergent therapy and for long-term prognostic and therapeutic considerations. While any algorithm has the risk of oversimplifying a complex problem, it is absolutely essential to have an initial strategy for the acute evaluation of an arrhythmia.

Causes of wide QRS tachycardia include (1) supraventricular tachycardia (SVT) with pre-existing or functional bundle branch block, including sinus tachycardia, atrial tachycardia, atrial flutter, atrial fibrillation (AF) and AV nodal re-entry tachycardia, (2) orthodromic circus movement tachycardia with pre-existing or functional bundle branch block, (3) SVT with conduction over an accessory pathway, (4) Antidromic circus movement tachycardia using an accessory pathway in the anterograde direction and AV node in the retrograde direction, (5) ventricular tachycardia, and (6) ventricular paced rhythm. Ventricular tachycardia is by far the commonest underlying mechanism of WCT.†

The overall goal of any algorithm is to make things simpler yet effective.

Most criteria are based on QRS complex duration, QRS axis, concordant pattern, presence of Q-waves, AV dissociation, fusion beats, absence of preordial RS complex, QRS alternans, and presence of multiple wide complex morphologies.

Starting in 1978, several criteria have been developed to aid in the diagnosis of WCT. For evaluation of RBBB morphology tachycardias, Wellens et al.‡ proposed (1) duration of QRS > 140 ms, (2) left-axis deviation, (3) certain configurational characteristics of QRS, and (4) atrioventricular (A-V) dissociation as the criteria for diagnosis of VT vs. SVT. In 1988, specific criteria were proposed to differentiate WCT in the presence of LBBB morphology.§ These included (1) an R-wave in V1 or V2 of greater than 30-ms (>40 ms) duration; (2) any Q-wave in V6; (3) a duration of greater than 60 ms (>70 ms) from the onset of the QRS to the nadir of the S-wave in V1 or V2; and (4) notching on the down stroke of the S-wave in V1 or V2.

The criteria for width of QRS complex has been later modified to suit for RBBB and LBBB (140 vs. 160 ms).‡ Subsequently in 1991, Griffith et al. (simpler version)¶ and Brugada et al. (more complex version)¶ separately published algorithms to diagnose WCT. The algorithm proposed by Brugada et al. included (1) absence of RS complexes in all precordial leads, (2) R to S interval > 100 ms in one precordial lead, (3) AV dissociation, and (4) morphological criteria in V1–2 and V6. Many other authors proposed a number of other EKG criteria to further qualify or differentiate wide complex morphologies with various permutations and combinations. In spite of a simplistic algorithm-like approach, the Brugada criteria are not widely accepted due to inapplicability of complex morphological criteria and lower than expected sensitivity and specificity. Alberca et al.¶ suggested a limited applicability of most of the existing morphological criteria favouring ventricular tachycardia in patients with IVCD since they were also present in a substantial percent of patients during sinus rhythm. Despite all this effort, the diagnosis of WCT remains quite error-prone, suggesting the intrinsic complexities and subtleties in the analysis and also suggesting the limitations of a single objective algorithm.

Vereckei et al.¶ propose a new algorithm with four elements for differentiating wide-complex tachycardia from SVT. They analysed 453 monomorphic WCTs recorded from 287 patients based on (1) presence of AV dissociation; (2) presence of an initial R-wave in lead aVR; (3) did the morphology of the WCT correspond to bundle branch or fascicular block? (4) estimation of initial (Vi) and terminal (Vt) ventricular activation velocity ratio (Vi/Vt) by measuring the voltage change on the ECG tracing during the initial 40 ms (Vi) and the terminal 40 ms (Vt) of the same bi- or multiphasic QRS complex. The new algorithm correctly classified 409 of 453 WCTs (90.3% overall test accuracy (TA)) and was superior (P = 0.006) to that of Brugada algorithm [384/453 (84.8% overall TA)]. The TA of the fourth Brugada criterion was significantly lower [68 vs. 82.2%, P = 0.004] than the TA of the Vi/Vt criterion in the fourth step accounting for the majority of the difference in
outcome between the two methods. The rationale proposed for this criterion is that during WCT due to SVT, the initial activation of the septum should be invariably rapid and the intraventricular conduction delay causing the wide QRS complex occurs in the mid- to terminal part of the QRS. In contrast, in WCT due to VT, there is initial slower muscle-to-muscle spread of activation until the impulse reaches the His-Purkinje system, after which the rest of the ventricular muscle is more rapidly activated.8

Two elements of the algorithm, the ‘Vi/Vt’ ratio and the presence of an initial R-wave in aVR, are original contributions to this topic. In addition, the subgroup analyses evaluating this algorithm in patients with pre-existing bundle branch blocks and on antiarrhythmic medications provide useful insight. While the ‘Vi/Vt’ ratio is original and reflects the electrophysiology of many VT’s, there are a number of exceptions to these criteria, particularly patients with a history of infarct. Similarly, an initial R-wave in aVR can be seen in patients with left posterior fascicular block and prior inferior infarction. These exceptions do not invalidate the criteria, but need to be considered as part of the whole picture. There are other specific criteria that may apply to specific sub-category of patients. A Q-wave in V6 and notching on the downstroke of the S-wave in V1 or V2 were more common in VT patients with anterior myocardial infarction, whereas an R-wave > 30 ms in V1 or V2 was more common in patients with inferior myocardial infarction.

The effect of history of prior infarction, pre-excited tachycardias, antiarrhythmic medication usage, precordial lead placement, transplant status and the presence of congenital heart disease on these morphology criteria should be taken into account while applying these elements. Pre-excited tachycardias may not be differentiated consistently with the proposed criteria, especially those using epicardial left-sided para-septal or left-sided inferoposterior bypass tracts. These tachycardias can produce initial R-wave in aVR. In addition, some cardiomyopathies can have a baseline QRS during sinus rhythm that meet the proposed criteria. One has to be careful not to get carried away by criteria and replace clinical judgement by algorithms alone. It is important to recognize and balance the potential effects of an algorithm on proposed treatment and need for rapid action in the presence of clinical instability. It is also important to reaffirm the well-known fact that haemodynamically stable WCTs are not automatically SVT. Physical findings that suggest AV dissociation such as cannon A-waves in the jugular venous waveform, variable intensity in S1, and variable intensity in BP unrelated to respiration are quite useful in distinguishing the cause of WCT.

Despite the limitations, the data presented here indicate that a systematic analysis of the 12-lead electrocardiograms alone can be used to accurately diagnose the origin of WCTs, especially in presence of a normal ECG at baseline. Attention to these criteria combined with clinical judgement may lead to more rapid and effective therapy.

Conflict of interest: none declared.

References