ECG Quiz 29

CASSA
CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
A SPECIAL INTEREST GROUP OF SA HEART ASSOCIATION

ANSWER

Let's consider the possible mechanisms of the intermittent wide QRS complexes.

a) AF with complete right bundle branch block (RBBB)

This is incorrect, because all except 2 of the complexes are narrow (about 90ms). There are, however, small terminal R-waves in V1 which are compatible with minor right ventricular conduction delay.

b) AF with premature ventricular complexes (PVCs)

The two wide QRS complexes are premature and could originate in the ventricles. However, the pattern in V1 is compatible with RBBB aberration, in that the initial R is small and narrow; the S is also rapid; all the delay resides in the latter part of the QRS; and the terminal R in V1. Unfortunately, the wide complexes are not seen in V5-6 to confirm normal left ventricular depolarisation.

Two additional features argue against PVCs. The coupling interval between the wide QRS complexes and the preceding narrow complexes is different (400 vs. 360ms). Usually, the coupling interval of similar PVCs is the same as they are dependent on the previous (normal) ventricular depolarisation. The mechanism for this is either triggered activity or localised re-entry. The other feature against PVCs is the lack of a compensatory pause following the wide beats. A compensatory pause usually follows a PVC even during atrial fibrillation. The mechanism is probably the same as during sinus rhythm, namely concealed retrograde penetration of the AV node. During sinus rhythm, this usually results in a fully compensatory pause (i.e. 2 R-R intervals between the normal complexes before and after the PVC), because the PVC conducts retrogradely into the AV node, without traversing it, but prolongs the AV node refractory period sufficiently to block the next sinus P-wave. In AF, this prolongation of AV nodal refractoriness also occurs, causing a longer R-R following a PVC, although this is not invariable.

c) AF with rate-related RBBB

This is a possibility, in that the wide complexes follow short R-R intervals. Rate-related bundle branch block occurs when the refractory period of the affected bundle is too long to conduct at faster ventricular rates. Against this, however, is the observation that the first wide QRS is followed by an even shorter R-R (368 vs. 400ms) and yet the resulting QRS is narrow, indicating that the right bundle branch had recovered excitability more quickly than before. This suggests a more dynamic process is taking place, with variability in the refractory period of the right bundle.

d) AF with Ashman phenomenon

This provides a better explanation than simple rate-related RBBB. Refractory periods in the tissues of the His-Purkinje system fluctuate with changes in rate, shortening at faster rates (shorter R-R intervals) and lengthening at slower rates. When R-R intervals vary widely, as in AF, a situation can arise in which a long R-R is followed by a much shorter interval (see Figure 1). As a result of the preceding long R-R, the refractory period of the right bundle (in this case) has prolonged. When the short R-R follows unexpectedly, the right bundle is still refractory, resulting in temporary RBBB. Despite further R-R shortening of the subsequent beat, the refractory period is reduced by the short R-R of the preceding beat. It therefore conducts normally.

This phenomenon was described by Gouaux and Ashman in 1947.[1] It can occur in any rhythm in which the R-R intervals vary widely, but is most common in atrial fibrillation. The physiological shortening of the refractory periods in the His-Purkinje system in response to tachycardia is probably responsible for the relative rarity of rate-related aberrant conduction during regular sustained supraventricular tachycardias. Right or left bundle branch block commonly occurs after induction of an SVT in the EP laboratory, but usually only lasts for less than a minute, during which the

refractory period of the affected bundle shortens in response to the tachycardia, with resumption of normal, narrow QRS conduction. By the time the patient with a spontaneous SVT reaches the doctor or Emergency Unit and an ECG is done, the QRS complexes have normalised, unless one of the bundles is intrinsically abnormal.

The Ashman phenomenon is of interest chiefly as a model of the physiological behaviour of the His-Purkinje system, rather than being of any great clinical significance. It is however, useful to be able to differentiate it from other mechanisms of intermittent QRS widening, such as premature ventricular complexes, which might be more sinister.

ANSWER: (d) Atrial fibrillation (AF) with Ashman phenomenon.

REFERENCE

1. Gouaux JM and Ashman R. Auricular fibrillation with aberration simulating ventricular paroxysmal tachycardia. Amer Heart J 1947;34;366-373.

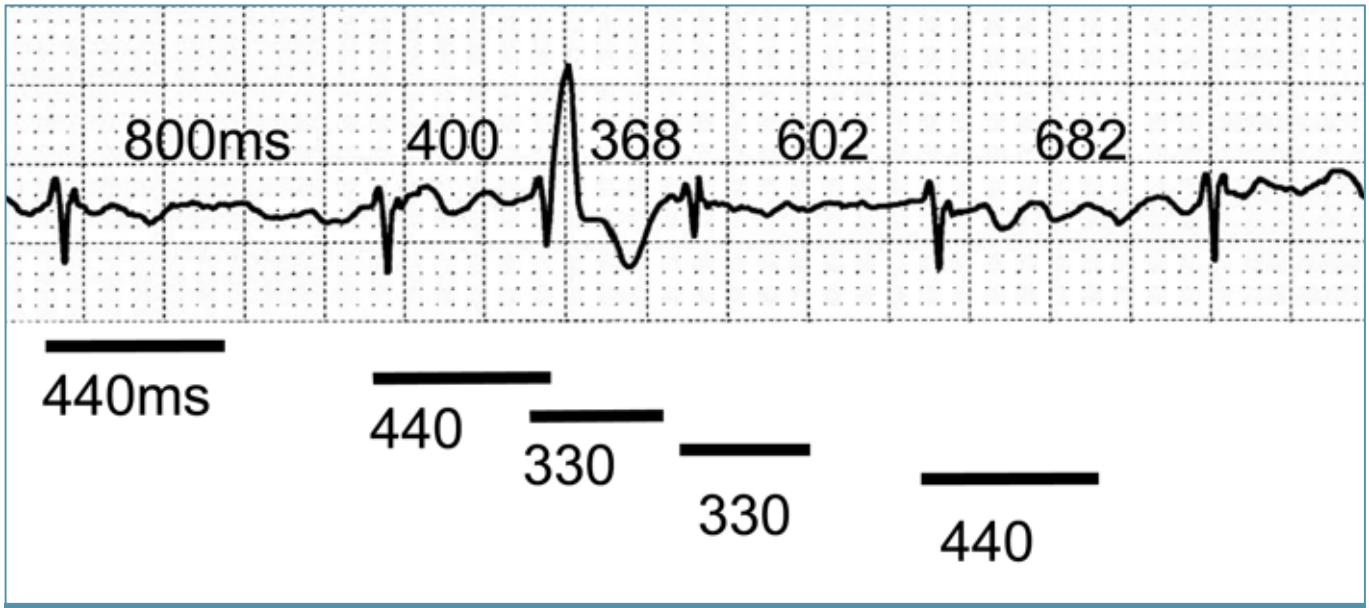

FIGURE 1: Widely varying R-R intervals could indicate AF with Ashman phenomenon.
The R-R intervals are marked above the ECG trace showing the first aberrant complex in V1. The postulated refractory periods (RP) of the right bundle branch appear below as horizontal bars. The RP of 440ms following the QRS before the long R-R interval allows full recovery and normal conduction. The following R-R is much shorter, resulting in activation before the right bundle had recovered. However, the short R-R causes the right bundle RP to shorten to 330ms, allowing the beat following an even shorter R-R to conduct normally.

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