## **RHYTHM PUZZLE - ANSWER**

## Saturday night broad QRS complex tachycardia in a young male

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## Answer to the rhythm puzzle

The ECG in the question (Fig. 1) shows a regular wide complex tachycardia with a rate of 237 beats/min with changing QRS width. The QRS axis is negative in II, III, aVF and positive in aVR suggesting an extreme right axis deviation. The QRS complexes show a right bundle branch block (RBBB)-like morphology. The QRS complexes in V6 show an RS pattern. There is no monomorphic R wave in aVR and the QRS duration varies. This is clearly seen in leads II, III, aVF and V1 when one compares the width of the first six with the last three QRS complexes. There is no AV dissociation and there are no fusion or escape beats. In lead III there is a notch visible in the initial part of the ST segments of the first six QRS complexes. In lead II there is a notch visible in the terminal part of the ST segments of the last five QRS complexes.

According to the classical Wellens criteria this tachycardia shows a right bundle branch block morphology. It is of supraventricular origin because there is no left axis deviation, a relatively narrow QRS width of 140 ms and the absence of a monomorphic R wave or left rabbit ear sign in the first eight beats of lead V1 [1].

According to the criteria of Brugada, there are RS complexes in the precordial leads, the longest R-S interval is less than 100 ms, there is no AV dissociation and the classical criteria for ventricular tachycardia (VT) in the precordial leads are missing [2].

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The criteria for VT presented by Verreckei et al. are by far the easiest to apply [3]. These investigators came up with the idea to look purely at the QRS morphology in lead aVR. According to their criteria there is no monomorphic R wave in aVR, there is no initial q wave greater than 40 ms and there is no initial delay or notch in the initial part of the QRS complex in lead aVR. However, the vertical excursion of the QRS in the last 40 ms is larger than in the first 40 ms and therefore suggests VT.

Spontaneously the ECG changes (Fig. 2). It demonstrates group beating at a rate of 167 beats/min with two types of QRS complexes, i.e. narrow QRS complexes with left axis deviation and a slightly wider QRS complex with incomplete RBBB morphology. The RR-time differs between the two types as is shown in lead 2.

Lead V1 demonstrates an atrial flutter waves with a FF interval of 240 ms with 3:2 AV Wenckebach conduction to the ventricles. Every third flutter wave is not conducted to the ventricles.

What is interestingly to note is that the first conducted flutter wave to the ventricles produces a narrow QRS complex. Paradoxically, the second wave with a more prolonged AV nodal conduction time produces an incomplete RBBB and the third flutter wave is blocked in the AV node.

In conclusion, the initial ECG showed an atrial flutter at a rate of approximately 240 bpm with 1:1 AV conduction to the ventricles with RBBB aberrancy.

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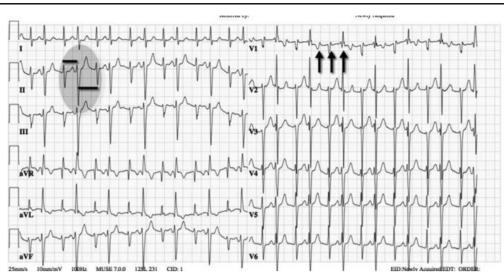


Fig. 2 ECG a few minutes after admission

## References

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