ECG Quiz 58

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

ANSWER

OVERVIEW OF THE ECG

This patient has a cardiac resynchronisation therapy (CRT) or biventricular (BiV) pacemaker with atrial, right ventricular (RV) and left ventricular (LV)/coronary sinus leads. The CRT pacemaker was programmed in the DDD mode with a lower base rate of 60bpm. Although the settings of the pacemaker are not given, general ECG observations can still be made.

This ECG shows a wide complex rhythm with variable paced QRS morphologies with a ventricular rate of 78bpm. The rhythm is regular for the first 5 beats (around 70bpm), followed by an irregular rhythm which is faster. No P waves are visible. Atrial fibrillatory waves are seen (best seen in V1), confirming the patient is in atrial fibrillation (AF). At least 3 different QRS morphologies are visible (best seen and compared in the V1 rhythm strip). See Figure 1.

QRS morphology type 1

Complexes 2, 3, 4 and 7 are identical. This morphology has a dominant R in V1 with a Qr complex in lead I with a duration of 150 - 160ms, with an indeterminate axis. A ventricular pacing spike is seen at the beginning of each complex. This morphology is compatible with CRT (simultaneous RV and LV) pacing. CRT-paced morphology can be highly variable between patients because of different positions of the RV and LV leads. A qR or Qr complex in lead I is usually indicative of CRT pacing. This complex matches a CRT-paced complex from an ECG recorded 1 year ago (see Figure 2).

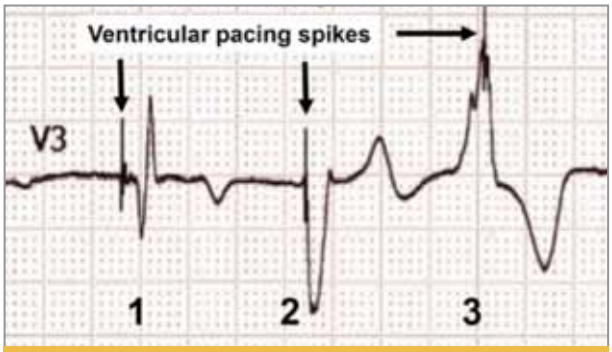

FIGURE 1: Three different QRS morphologies in V3. QRS morphology 1 is a true CRT paced complex with a pacing spike at the onset of the complex. QRS morphology 2 is a RV-sensed/trigger-paced fused complex (fusion between intrinsic conduction and simultaneous right ventricular and left ventricular pacing), with a pacing spike occurring immediately after the onset of the complex. QRS morphology 3 is a premature ventricular complex with triggered pacing spike 120ms after onset of the complex. See text for explanation.

QRS morphology type 2

Complexes 1, 5, 6, 8, 10, 11, 12 and 13 are identical. This complex has a duration of 120ms and resembles a left bundle branch block (LBBB) pattern (V1 - V3). The beginning of the complex is rapid, suggestive of initial His-Purkinje conduction. A ventricular pacing spike can be seen immediately after the onset of every complex, including after the irregular complexes – this is best seen in complexes 11, 12 and 13 (V4 - V6). This suggests that the ventricular pacing spike is not initiating the complex, but is triggered by the complex (triggered pacing can occur with some pacing algorithms – see later for explanation). This complex may be a fused (intrinsic conduction and pacing) or pseudo-fused (intrinsic conduction without pacing) complex. Comparison with an ECG prior to CRT pacing with LBBB (Figure 3) confirms that QRS morphology type 2 is a fused complex, which is much narrower than baseline LBBB (180ms).

QRS morphology type 3

Complex 9 is a very wide QRS complex, not preceded by a pacing spike, and is a premature ventricular complex (PVC). A pacing spike is seen in the middle of the complex.

EXPLANATION

In patients with a CRT pacemaker (with an atrial lead), AF causes a mode switch from DDD to DDI mode (when the atrial rate is >170bpm) to prevent inappropriate tracking of rapid atrial fibrillatory waves. In DDI mode, no tracking of fibrillatory waves can occur, and the device will only pace the ventricles if the conducted ventricular rate in AF is slower than the mode switch base rate.

It is important to know what type of pacemaker is inserted and how the pacemaker is programmed. This patient has a Boston Scientific CRT pacemaker with a mode switch base rate of DDI 70bpm. When the conducted AF rate is slower than 70bpm, the patient is paced at 70bpm with QRS

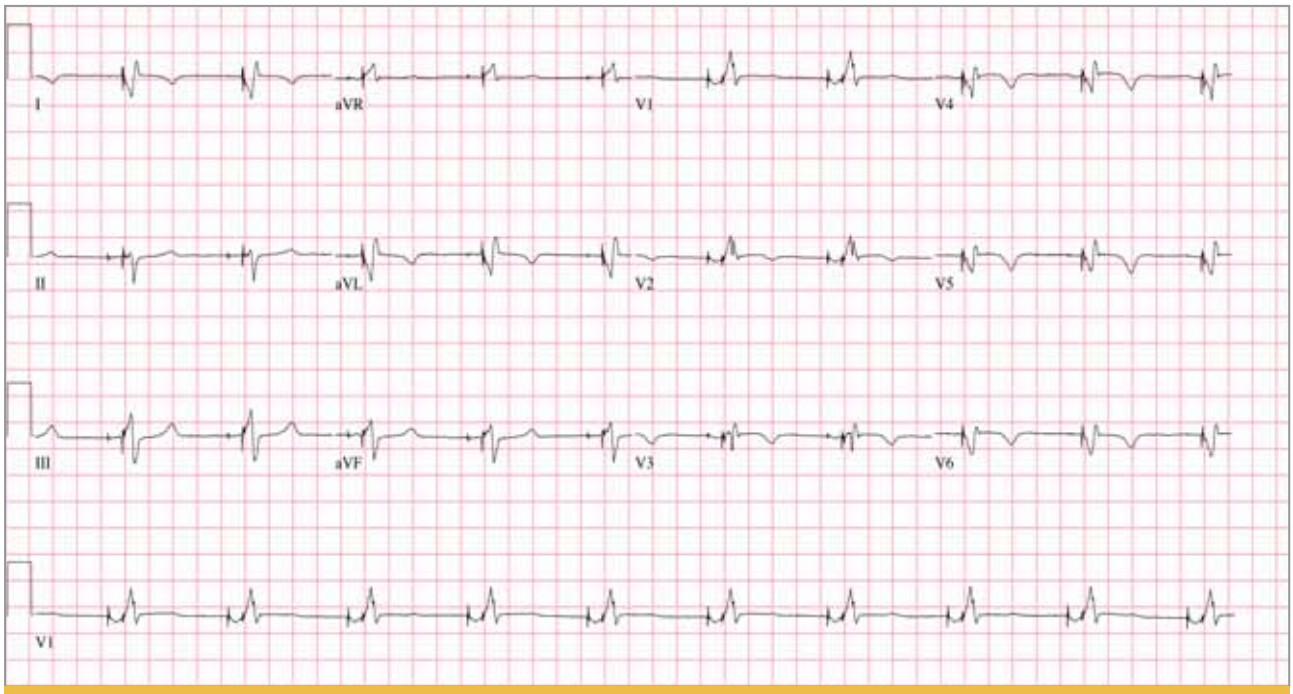

FIGURE 2: ECG of the same patient 1 year ago. Note the paced morphology is similar to QRS complex type 1 = CRT paced complex. The patient is paced in the atrium (DDD 60bpm).

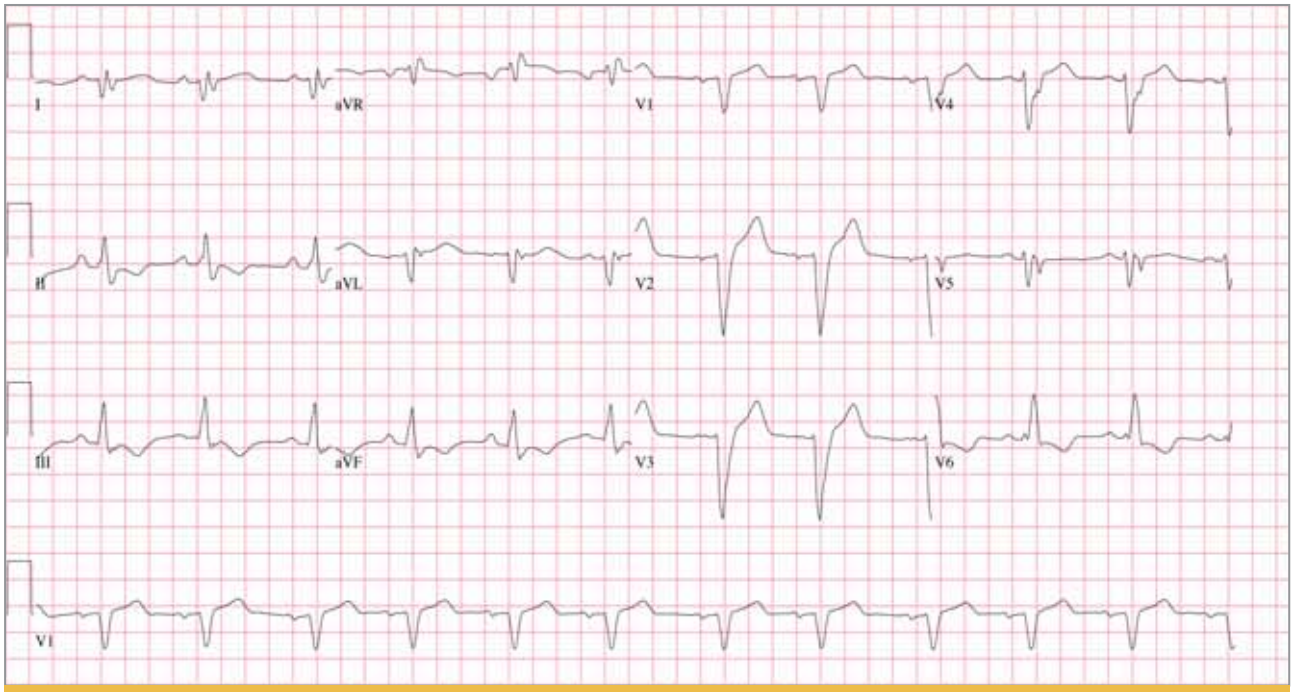

FIGURE 3: Basline ECG without CRT pacing. Note the wide left bundle branch pattern with a QRS duration of 180ms.

morphology type 1 – i.e. CRT pacing (complexes 2, 3 and 4). Complex 7 is also CRT-paced, but occurs slightly faster than 70bpm because of a "rate smoothing" algorithm that allows pacing above the base rate of 70bpm in an attempt to prevent sudden changes in heart rate.

In many CRT patients who develop AF, pacing is inhibited as the conducted AF rate is faster than the mode switch base rate, as AV node conduction is frequently intact. In this ECG, when the conducted AF rate increases above 70bpm, true CRT-pacing is inhibited and triggered pacing occurs.

This form of triggered pacing can be found in almost all CRT devices and in most algorithms is turned "on" as default. This form of triggered pacing was designed to provide ongoing RV and LV pacing, even when the conducted AF rate is faster than the lower mode switch pacing rate. These algorithms sense a RV beat and trigger an immediate simultaneous RV and LV pacing pulse after a RV sensed beat. This results in a fused beat (intrinsic conduction followed immediately by RV and LV pacing) rather than a true CRT paced beat (RV and LV pacing only) – since the RV septum has already started depolarising. In this patient with a Boston Scientific CRT device, the algorithm is called "BiV Trigger™", which delivers a RV and LV pacing spike 8 - 10ms after an intrinsic RV sensed beat. This paced beat creates QRS morphology type 2, which explains the initial LBBB morphology, spike occurring 8 - 10ms after the onset of the QRS complex, and the QRS width of 120ms (much narrower than baseline LBBB of 180ms), because of fusion. Interestingly, QRS morphology type 2 is narrower than the QRS morphology type 1 (true CRT paced) beat, suggesting better electrical resynchronisation with BiV fusion pacing than true CRT pacing. While these algorithms make theoretical and practical sense, there are no randomised trials evaluating the efficacy of these algorithms.[1]

Atrial pacing spikes without atrial capture are visible before QRS complexes 4, 5 and 6, which require further explanation. The atrial fibrillatory waves are very low in amplitude and are likely intermittently undersensed – i.e. atrial undersensing is present. When 8 atrial beats are <170bpm, the mode switch algorithm stops and the patient reverts back to DDD mode with atrial pacing. When this occurs, inappropriate atrial pacing occurs, which does not capture the atrium because the patient is in AF. When 8 atrial beats are sensed at >170bpm, mode switch occurs back to DDI. This can be confirmed by interrogating the ECG rhythm strips of the patient around the time of the ECG. Atrial undersensing is another cause of a lack of CRT pacing in sinus rhythm. A CRT paced beat can only occur after a sensed or paced P wave after a programmed sensed or paced AV interval.

Frequent PVCs can also result in a lack of CRT pacing, as PVCs cannot trigger CRT pacing (similar to atrial undersensing). However, in this case, the "BiV trigger" algorithm delivers a spike in the QRS complex (best seen in V3). In this case there is a 120ms delay between the onset of the QRS and pacing spike, probably because the PVC is left sided in origin with a delay to the RV sensing electrode.

The best answer is therefore e) A, B and C (atrial fibrillation, premature ventricular complexes and atrial undersensing).

DISCUSSION

It is important to perform and carefully analyse ECGs in all patients with CRT devices at follow-up. Comparison should be made to prior ECGs (paced and non-paced ECGs), as this can help interpret ECGs' findings and alert the physician about possible device malfunction.

Numerous studies have shown that a high percentage (>98%) of BIV pacing is required for a clinical response. This ECG shows 3 potential causes of lack of response to CRT pacing: atrial fibrillation, frequent PVCs, and atrial undersensing. Other causes include inappropriately programmed sensed or paced atrioventricular intervals, sinus tachycardia or supraventricular arrhythmias (above the upper track rate) or device programmed in a non-tracking mode (e.g. DDI).

To overcome some of these potential causes like AF or frequent PVCs, devices have algorithms which aim to maximise LV pacing with AF. Many of these algorithms are nominally turned "on" at implant and clinicians and technologists need to be aware of these algorithms. Awareness of these algorithms can prevent the incorrect labelling of device malfunction, which can lead to inappropriate device revision or programming.

Although studies have yet to prove the effectiveness of these algorithms, it is reasonable to turn them "on" until a definitive therapy for the AF or PVCs can be instituted – e.g. antiarrhythmic drug therapy, catheter ablation or AV node ablation can be considered in patients with AF and suboptimal BiV pacing. In this case, we opted to perform an AV node ablation.

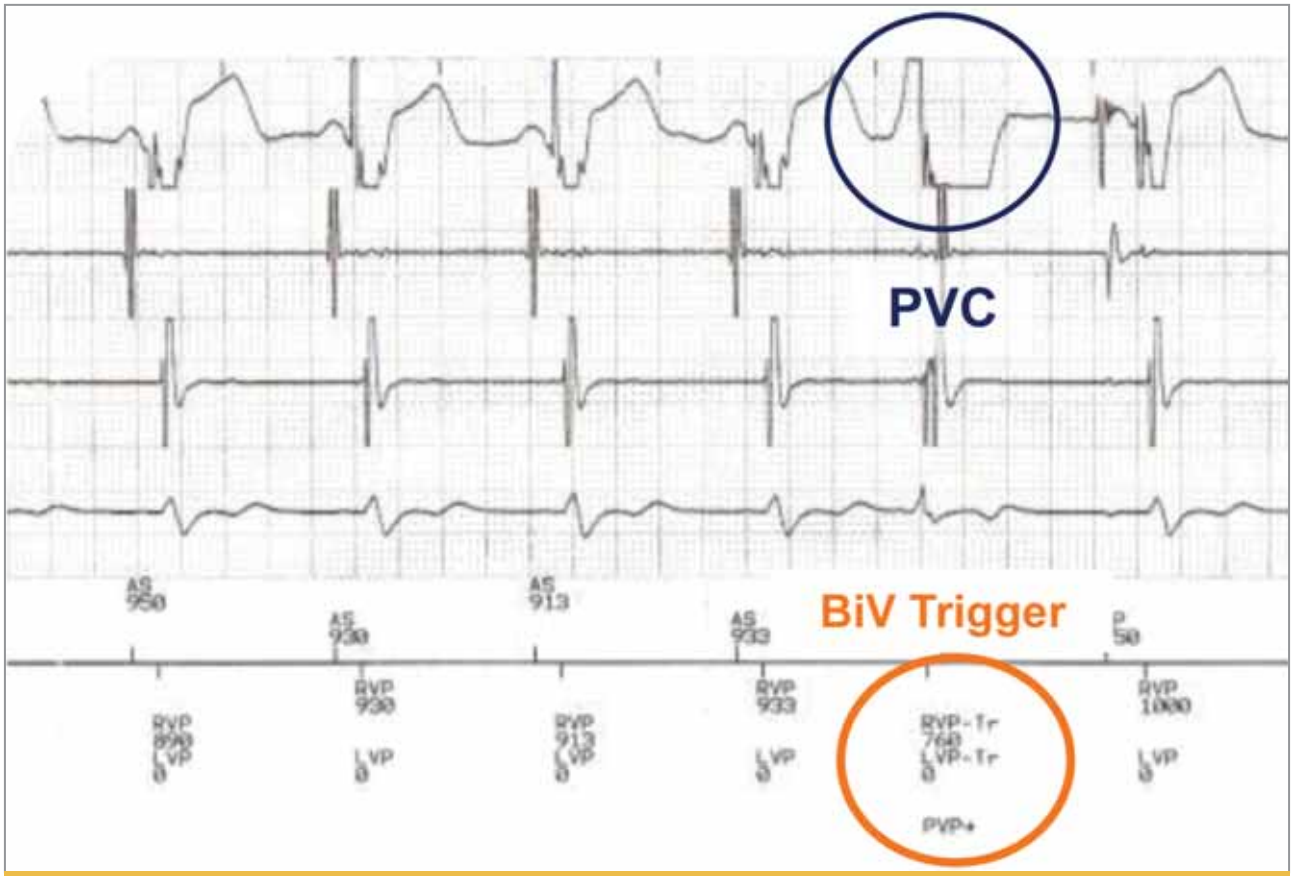

FIGURE 4: EGM of another patient with a Boston Scientific CRT device. The premature ventricular complex (PVC) is sensed in the right ventricle, which initiates a right ventricular and left ventricular pacing impulse. The biventricular (BiV) triggered beat is annotated RVP-Tr/LVP-Tr (EGM courtesy of Boston Scientific).

Additional helpful information is to obtain device EGM interrogation strips of the paced rhythm and device programming parameters. This information is extremely valuable, as this can often give insights into what the device is "seeing and doing". Marker annotations should be carefully inspected, which then can be correlated to the ECG. For example, "BiV Trigger" complexes (Boston Scientific) are annotated "RVP-Tr/LV-Tr" on EGM strips. In Figure 4, "BiV Trigger" algorithm allows a pacing spike after a PVC is sensed.

CONCLUSION

Atrial fibrillation, atrial undersensing and frequent PVCs are important causes for a lack of CRT pacing. Treatment of these conditions will allow close to 100% CRT pacing, which is needed for an optimal clinical response.

CRT devices have algorithms to promote ongoing RV and LV pacing even with PVCs or conducted AF rates faster than the mode switch base rate, by sensing RV conduction and then triggering simultaneous RV and/or LV pacing. This is not true CRT pacing and is a form of triggered fusion pacing.

Although electrical resynchronisation can occur with triggered fusion pacing, hard clinical benefits of this form of triggered pacing remain unclear.

Conflict of interest: none declared.

REFERENCES

1. Lo M, Obel O. Maximising cardiac resynchronisation therapy in patients with atrial fibrillation. J Innov Card Rhythm Management 2011;2:584-593.

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