Preventive algorithm ending up being the 'Cause'!

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Abstract

N/A

1— Case Presentation

A 75 year-old male, who is a known case of chronic renal insufficiency and on twice weekly renal replacement therapy, presented with sudden onset and rapidly progressive dyspnoea of 3 days duration associated with palpitations. He had previously undergone a dual chamber automatic implantable cardioverter defibrillator (AICD) implantation (Abbott, Ellipse^{TM} DR MRI) for degenerative intermittent complete heart block (CHB) and non-ischemic cardiomyopathy with a left ventricular ejection fraction of 35%. An electrocardiogram (ECG) in the emergency department demonstrated a regular wide QRS tachycardia at 110 beats per minute (bpm) with left bundle branch block (LBBB) morphology a pacing spike preceding each QRS complex. His device was programmed to a lower rate of 40 bpm and a maximum track rate of 110 bpm. The pacemaker mediated tachycardia (PMT) response was turned 'ON'. The device was interrogated and the ongoing electrogram (EGM) is shown in Figure 1. It shows a tachycardia annotated by the device as PMT and the tachycardia continues despite PMT intervention by the device. What is the probable mechanism of initiation and perpetuation of the tachycardia?

2—Discussion

The differential diagnoses of a regular wide QRS tachycardia with morphology identical to the paced QRS morphology at the maximal tracking rate in a patient with a dual chamber pacemaker includes PMT, sinus tachycardia/ atrial tachycardia tracked by the ventricular lead and ventricular tachycardia (VT) with exit

site close to the ventricular lead implant site. Ventricular tachycardia is ruled out here as the ventricular complexes follow pacing stimulus throughout the trace. An onset of tachycardia which is sudden and heralded by a premature ventricular contraction argues against sinus tachycardia or atrial tachycardia.

The device EGM in Figure 1 shows three channels- atrial near-field EGM (AEGM), ventricular near-field EGM (VEGM) and the device marker annotations. The trace begins with a sinus atrial signal being tracked by the ventricular lead at the programmed AV delay. Next comes a premature ventricular complex (PVC) with retrograde ventriculoatrial (VA) conduction (VA- 120 ms) to the atrium (note the difference in atrial EGM near-field characteristics from the sinus node origin atrial EGM). This is followed by atrial pacing approximately 330 ms after the atrial event which fails to capture the atrium (Note the atrial EGM corresponding to Ap doesn't have an evoked potential).

This Ap at 330 ms after an atrial event following a PVC is the characteristic 'PVC response' seen in Abbott's newer generation of pacemakers. Ventricular pacing then follows at the programmed AV delay. Loss of atrial capture allows the paced ventricular complex to conduct retrogradely to the atrium (note the near- field atrial EGM morphology matches that of retrograde atrial EGM) which is in turn is tracked by the ventricular lead and starts a cycle of PMT at the upper tracking rate. After confirming the stability of the Vp- As intervals on the 2nd attempt (each attempt includes a count of 8 Vp-As intervals) which includes an extension of the AV delay by 50 ms, the device extends the post- ventricular atrial refractory period (PVARP) followed by an atrial pacing event at 330 ms similar to the PVC response. However, as seen earlier in the trace, this Ap fails to capture again and allows the subsequently ventricular paced QRS complex to conduct retrogradely to the atrium and start another cycle of PMT. Thus, a vicious cycle is perpetuated leading to heart failure symptoms.

The current generation of Abbott pacemakers have PMT prevention (*PVC response*) and termination (*Auto-detect*) algorithms with operation settings similar to other device manufacturers, that is, extension of the post ventricular atrial refractory period (PVARP). Their algorithms, in addition, involve atrial pacing after the retrograde atrial event followed by ventricular pacing at the programmed paced AVD.[1]

The PVC response algorithm detects and responds to PVCs when the device is in DDD(R) mode. It detects a PVC if: 1) an R-wave is not preceded by an atrial event; or 2) a P-wave is detected in the relative refractory portion of the PVARP period but is not followed by an R-wave within 280 ms of the atrial event.

The Atrial Pace setting is a response to a PVC confirmation. The response consists of a continuous extension of the PVARP setting to 475 ms, followed by an atrial alert period of 330 ms until a P-wave is tracked outside the extended PVARP period, followed by an atrial pace.

The PMT termination algorithm (Auto- detect) also behaves in a similar fashion confirming Vp- As interval stability and ultimately manifesting itself as PVARP extension followed by an atrial pace.

The probable reason for loss of capture with the Atrial Pace setting of the PVC response algorithm (even 330ms after the last atrial depolarisation) in this was the long refractory period of atrial myocardial tissue due to the metabolic milieu of chronic renal insufficiency.

Also noted was a VA interval which was longer for paced QRS morphology than for the PVC which could be explained by the closer location of the PVC origin to the His- Purkinje conduction system. This long intrinsic VA was responsible for the perpetuation of the PMT, the retrograde A falling outside the PVARP each time.

Although any event causing AV dissociation can start a PMT, PVCs are the most common triggers and hence the need of a specific algorithm directed towards it. But the same proved to be arrhythmogenic as has also been reported previously in literature but with respect to ventricular and atrial tachyarrhythmias.[2]

This case represents a unique hitherto unheard-of clinical scenario, where an algorithm (PVC response) meant for a specific purpose (prevent PMT initiation) not only fails but starts the very phenomenon it was meant to prevent. In fact, the PMT would not have been initiated if it were not for the algorithm. The PVC

response was eventually switched off, as was Atrial Pace setting after PVARP extension resulting resolution of symptoms.

References

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Figure Legends

Figure 1- Three channel Device EGM showing initiation of tachycardia with Vp-As sequences and device label of Pacemaker Mediated Tachycardia (PMT)

Figure 2- Explanation-

(A) The first intrinsic atrial event is tracked by the ventricle (Vp) at the programmed atrioventricular delay (AVD). This is followed by a premature ventricular contraction (PVC) with retrograde atrial activation with ventriculoatrial interval (VA) of 120 ms. The PVC triggers the 'PVC response' which include a Post Ventricular Atrial Refractory Period (PVARP) extension followed by atrial pacing (Ap) at 330ms from the retrograde atrial event. However, the Ap fails to capture atrial myocardium because of longer refractory period which allows the next Vp to have retrograde conduction to the atrium. This starts the Pacemaker Mediated tachycardia (PMT) which is detected by the device (B) after one failed attempt (as 8 consecutive Vp-As interval average was more than 16 ms longer than the first Vp-As). However, the PMT response from the device fails to terminate the tachycardia in the same way as the PVC response and again starts the PMT (C).