Inhibition of ventricular output in a dual chamber pacemaker with normal pacing and sensing parameters: What is the mechanism?

Debabrata Bera a, *, Radhey Shyam Joshi a, Suchit Majumder b, Sanjeev S. Mukherjee c

a Dept of Cardiology, RTIICS, Kolkata, India
b Dept of Cardiology, Apollo Gleneagles Hospital, Kolkata, India
c Dept of Cardiology, Medica Superspeciality, Kolkata, India

Article history:
Received 12 May 2021
Received in revised form 19 July 2021
Accepted 1 September 2021
Available online 4 September 2021

Keywords:
Far field P wave Oversensing
Inhibited ventricular output
Early insulation failure

A 65-year-old gentleman with dual chamber pacemaker presented with presyncope. The ECG raised concerns of oversensing which was confirmed by magnet response. The device interrogation revealed noise in ventricular channel temporally associated with P wave. The pacing thresholds were normal. Although the ventricular lead impedance was within normal limit the impedance trend suggested 100 Ohm decline over last 1 year. RV lead insulation failure was speculated and supported by the bipolar and unipolar intracardiac electrogram. Device was programmed to DOO temporarily. He underwent RV lead replacement uneventfully.

© 2022 Indian Heart Rhythm Society. Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

1. Case

A 65-year-old gentleman underwent permanent pacemaker implantation 11 years ago for infra-hisian complete heart block with normal LV function (dual chamber DDDR, St Jude, St Paul, MN; Verity ADx device, Isoflex leads). Right atrial (RA) lead was in RA appendage and right ventricular (RV) lead at the apex. He was on regular follow-up and was asymptomatic. Recently he presented with recurrent episodes of dizziness and presyncope. Pacemaker setting: DDDR, Lower rate 60, Upper rate limit 110, AV delay(s/p) 150/200 ms, RA- Amplitude 3.5 V @0.4 PW [Pulse width], RV amplitude (Uni/Bi) - Cap confirm @0.4 ms PW, Atrial sensitivity: 0.75 mV, Ventricular sensitivity: 2 mV(Uni/Bi). His 12 lead ECG revealed intermittent non-tracking of P waves (Fig. 1A). A loss of output was noted and over-sensing was suspected. Magnet application rectified the problem confirming the diagnosis of oversensing (Fig. 1B). Fluoroscopy revealed no abnormality (Fig. 2ABC). The device interrogation revealed that there was intermittent but frequent oversensing of noise exclusively after properly sensed P waves (Fig. 3AB). What could be the possible mechanism and how to troubleshoot?

2. Commentary

The oversensing was inhibiting the RV output to be delivered. During a prolonged interrogation of real time EGM, it was noted that there was oversensing in marker channel which was timed consistently around P waves. The noise was cyclical. The temporal correlation with atrial activity also suggests the noise to be intracardiac. However, the over-sensing took place at variable As-Vs interval from the sensed P waves (Fig. 3A and B, 80 ms-240 ms). The sensing polarity was changed to unipolar from Uni/Bi but the result remained same (Fig. 3B). His RV lead impedance remained consistently between 360 and 381 Ohm. The pacing threshold was also normal. The arm movement or posture change didn’t have any effect on the frequency of oversensing which kept on coming at random intervals but in quick succession. Fortunately, the over-sensing was seen in maximum of 2 consecutive beats, hence prolonged asystole was not experienced, at least during the prolonged interrogation over 2 hours. A battery longevity of 1.75 to 2 years was noted (battery voltage 2.7 V). The fluoroscopy did not reveal any macroscopic lead fracture or insulation defect (Fig. 2ABC). Though his RV lead impedance was still within the normal range, a noticeable 100 Ohm decline over last 1 year was observed. Until now, the RV lead impedance stayed between 450 and 510 Ohm consistently for last 10 years. An early insulation defect was the provisional diagnosis. In view of pacing dependence, it was decided to change the RV lead at the earliest (temporarily it was made DOO @85/min
until lead change). On operative table, the lead impedances and thresholds were tested, both in unipolar and bipolar mode, which were same (360–385 Ω). After placing the new RV lead, the old pulse generator was connected and a normal pacing was observed ruling out a rare possibility of device circuit failure. As less than two years of battery longevity was remaining, battery was replaced as well. After the procedure, the patient is well and relieved of previous complaints.

Fig. 1. 1A: As-Vp rhythm with intermittent non-tracking of P waves. 1B: Magnet application solved the problem with consistent Ap-Vp, confirming the diagnosis of oversensing in the previous ECG in Fig. 1A.

Fig. 2. ABC: Fig: Fluoroscopy showing appropriate RA and RV lead position with no evident loss of lead integrity.
The differential diagnosis of apparent lack of atrial tracking (like Fig. 1A) in the presence of an adequate atrial sensing includes: 1) P wave in the post-ventricular refractory period (PVARP) (without ventricular oversensing), 2) upper rate behaviour during a sinus or atrial tachycardia, 3) ventricular oversensing of a variety of signals in the AV delay (as in our case) or beyond the AV delay [1]. In the latter case, oversensing generates a new post-ventricular atrial period that causes false atrial undersensing. The first two options were unlikely in our case and ventricular oversensing was evidently the culprit here.

The unique finding of the oversensing here was the consistent relation with P wave. The possibilities were: (1) Oversensing of P waves itself, (2) Noise generated due to friction between two leads during atrial contraction when there is lead integrity issue. Far-field sensing of the P wave by the ventricular channel with intact ventricular capture, can occur usually with dislodgement of a ventricular lead toward the RV inflow tract or into the coronary sinus [2–5]. This could also happen in integrated bipolar ICD leads in a small RV. In the present case it was a chronic lead at RV apex [Fig. 2ABC].

In very rare instances when atrial output (during ‘Ap’) is high with low impedance, the ventricular circuit in close proximity to atrial circuitry (within the pulse generator) can wrongly sense (i.e, over-sense) the atrial output as ‘Vs’ and might inhibit RV output [6,7]. However, in those cases the time interval between A→V in marker channel will be fixed in every over-sensed beats, unlike our case. Moreover, this can only happen in Ap beats, hence not a differential in our case.

Intriguingly, the amplitude of oversensed noises were paradoxically larger in bipolar setting [Fig. 3A and B]. An insulation break in atrial portion of a ventricular lead can mimic far-field P wave sensing whenever atrial and ventricular leads rub against

---

**Fig. 3.** A: Evidence of ventricular channel oversensing in 4th and 7th beat the Vs was associated with P wave (red arrows). This was seen at frequent intervals. In the 8th beat the oversensed noise came little later after QRS onset and Vp, although this was a rare occurrence. The fifth and 6th beats also shows similar but smaller signals (black arrows) that the device did not register and delivered a Vp. It was possibly related to the amplitude of the signal.

3B: In the 4th and 6th beat there was evidence of oversensing. Although the Vs were in relation with P waves, the time interval between As and Vs in those beats were variable (80 ms in 4th, 110 ms in 7th and 240 ms in 8th beat in Figs. 2A and 80 ms in 6th and 120 ms in 6th beat in Fig. 2B). The smaller amplitude of the noise during unipolar sensing despite larger gain (as compared to bipolar in Fig. 2A) possibly pointed towards outer insulation failure.
each other in atrial systole. In this situation, movement can create disturbance that generates critically timed false signals [8]. This possibility should always be considered with apparent far-field P wave sensing by a well-positioned ventricular lead at the right ventricular apex especially in unipolar sensing setting [8]. Absence of ‘make and break’ noise or oversensing with arm movement dissuaded against a micro-fracture which can also rarely present with oversensing/loss of output and apparently normal lead impedance. This case highlights the importance of looking at the intracardiac electrogram and impedance trend in cases having oversensing and reemphasizes that parameters within normal range does not rule out lead malfunction.

Funding

None.

Consent

Consent has been taken from patient.

Data availability statement

All raw data and recording during the case are available for review.

Declaration of competing interest

None.

References

[1] Barold SS, Garrigue S, Clémenty J. Far-field P-wave sensing by the right ventricular lead of conventional dual chamber pacemakers. J Intervent Card Electrophysiol 2002;6(1):77–80. https://doi.org/10.1023/a:1014132408411.
[2] Ben-Zur UM, Gross JN, Goldberger MB, et al. Oversensing of pacemakers in the bipolar pacing configuration: paradoxical resolution with programming to unipolar sensing. Am Heart J 1994;128:817–9.
[3] Weinstock M, DeGuia R, Daniell M, et al. Inhibition of transvenous pacing through the coronary sinus by the atrial P wave: diagnosis with the aid of isoproterenol. Chest 1971;59:563–6.
[4] van Gelder LM, el Gamal MI, Tielen CH. P-wave sensing in VVI pacemakers: useful or a problem? PACE (Pacing Clin Electrophysiol) 1988;11:1413–8.
[5] Barold SS, Falkoff MD, Ong LS, et al. Oversensing by single-chamber pacemakers: mechanisms, diagnosis and treatment. Cardiol Clin 1985;3:565–85.
[6] Santucci PA, Mitra RL. Ventricular output failure in a DDD permanent pacemaker associated with increased atrial output. Pacing Clin Electrophysiol 1997;20(11):2860–3.
[7] Ben-Zur UM, Kahn S, Gross JN, Platt SB, Goodfriend MA, Furman S. Suppression of ventricular output by noise in the atrial channel in a dual chamber pacemaker. Pacing Clin Electrophysiol 1995;18(8):1586–8.
[8] van Gelder BM, Bracke FA, el Gamal MI. P wave oversensing in a unipolar VVI pacemaker. PACE (Pacing Clin Electrophysiol) 1995;18:370.