Pacemaker Mediated Tachycardia

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67 yrs male, a known case of dilated cardiomyopathy, was implanted dual chamber pacemaker, two years back, for complete heart block. He presented with increased breathlessness and palpitation for two days. ECG showed wide QRS tachycardia (Figure 1, Figure 2, strip A). There was suggestion of pacing spike before the QRS complex. P waves were not identified. Since ventricular pacing was occurring at inappropriately higher rate, possibility of pacemaker mediated tachycardia (PMT)/endless loop tachycardia was kept. Magnet was kept over the generator, and the tachycardia was terminated (Figure 2, strip B). After this, post ventricular atrial refractory period (PVARP) was increased by programming the pacemaker and ECG showed atrial sensing with ventricular pacing (Figure 2, strip C).

Pacemaker-mediated tachycardia, also called endless-loop tachycardia, is used to refer to a form of a reentrant tachycardia and can occur in patients who have dual-chamber pacemakers. The pacemaker forms the anterograde (atrium to ventricle [A → V]) limb of the circuit and the atroventricular (AV) node is the retrograde limb (ventricle to atrium [V → A]) of the circuit.¹

The following is the most common scenario causing PMT. A dual-chamber pacemaker programmed DDD is implanted. The patient must have retrograde (V→A) conduction with an atrial activation time that is longer than the programmed PVARP. A ventricular-paced beat or a properly timed premature ventricular contraction (PVC) conducts retrograde via the AV node (or an accessory pathway, if present) to the atrium. If the atrial depolarization occurs after the set PVARP, but before the next timed atrial-paced beat, ventricular pacing will be triggered at the programmed AV interval. PMT tends to occur at or near the programmed upper rate limit and depend upon the programmed AV delay and the PVARP. This generates an incessant reentrant arrhythmia circuit that persists as long as there is continuous VA conduction with atrial activation outside the PVARP. Ventricular pacing at or near the upper rate limit of the pacemaker is evident on ECG. The presence of a paced rhythm exactly at the upper rate limit with atrial sensing and exact A-V association warrants evaluation for pacemaker-mediated tachycardia (PMT). A magnet placed on the pacemaker will stop the tachycardia.

Treatment, prevention, and termination of pacemaker-mediated tachycardia (PMT) typically involves altering the pacemaker programming to prevent sensing of the retrograde P wave. This is most easily done by prolonging the PVARP. During the PVARP, the atrial lead does not sense any atrial activity; hence, ventricular pacing is not triggered. Carotid sinus massage or AV nodal blocking drugs such as adenosine, verapamil, or beta-blockers can block VA conduction (ie, retrograde conduction) directly and can terminate PMT.

Reprogramming a dual-chamber, dual-mode, dual pacing, dual-sensing (DDD) pacemaker to AAI, VVI, or DVI (DDI) abolishes the PMT reentrant circuit, thereby prohibiting PMT from occurring. These other programming modalities can lead to serious problems as DDD pacing may be necessary (consider the difficulty of AAI pacing in a patient with complete heart block).

Atrial sensitivity may be programmed so that sinus P waves are detected but not retrograde P waves (which can be smaller).² The downside of this approach is that intrinsic P-wave amplitude can be lower at higher rates, which could potentially result in atrial undersensing.³ Making sure that atrial capture is adequate is also important. Attempting to adjust sensitivity is generally impractical. Most modern dual-chamber pacemakers are capable of detecting PMT and initiating PMT intervention by automatically prolonging the PVARP for the beat after a ventricular-sensed event that is not preceded by atrial pacing, ie, a PVC (PVARP extension).

References

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