Pacemaker therapy for ictal asystole: potentially hazardous programming?

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A 75-year-old woman experienced transient loss of consciousness (T-LOC) five times over 15 months; each episode was associated with prodromal symptoms including dyspepsia and chest discomfort, but without convulsions, tongue biting, or urinary incontinence. Consciousness returned spontaneously without sequelae. Consequently, syncope rather than seizure was suspected during initial evaluation.

Physical and laboratory findings were normal; her baseline 12-lead electrocardiogram showed normal sinus rhythm with a heart rate of 89 b.p.m. Holter monitoring did not show sinus node dysfunction, paroxysmal atrioventricular block, or ventricular tachyarrhythmias. She had no history of vasovagal syncope or orthostatic hypotension. A conventional electroencephalogram (EEG) was without epileptic waveforms. Since the basis for T-LOC was unidentified, an implantable cardiac monitor (ICM) was implanted.

Two months later, the patient experienced an episode of T-LOC. The ICM recording showed cardiac asystole for 19 s (Figure 1). Similar cardiac pauses were recorded during both sleeping and waking hours, 11 times over the recording period. Consequently, a permanent pacemaker (Accolade®; Boston Scientific Inc., Marlborough, MA, USA) was implanted and programmed to dual-chamber pacing mode with sensing and pacing in both atria and ventricles (DDD) mode at a low rate (35 ppm) with a 200 ms-AV delay, with the sudden brady response function (SBR) switched ‘ON’.

For 10 months, she experienced no syncope but had frequent, intermittent light-headedness and episodes of clouded consciousness. The cumulative percentage of atrial and ventricular pacing was <1% and SBR occurred 23 times with these symptoms. Accordingly, although she had not collapsed, based on the assumption that pacing at 35 ppm was too slow during ictal asystole (IA) spells, the pacemaker was reprogrammed to DDD mode at a lower rate of 60 ppm with a 200 ms-AV delay; SBR was turned ‘OFF’.

Figure 1 ECG during development of T-LOC recorded by ICM prior to pacemaker implantation. The recording was obtained when the patient collapsed 2 months after implantation of the ICM. In this ECG, the heart rate was sustained at 75 beats/min, but temporarily rose to 90 beats/min and then gradually dropped to 30 beats/min, resulting in a 19-s cardiac arrest. ECG, electrocardiogram; ICM, implantable cardiac monitor; T-LOC, transient loss of consciousness.
Unexpectedly, episodes of T-LOC, associated with urinary incontinence and automatism resumed. An EEG revealed left temporal lobe epilepsy; treatment with oral levetiracetam was initiated, and her pacemaker was reset to 35 ppm in DDD mode without SBR. During follow-up, pacing was confirmed several times before levetiracetam therapy ultimately proved to be effective for eliminating the recurrences of T-LOC. Subsequent follow-up indicated that she no longer needed atrial or ventricular pacing.

The main observations from this case are: (i) SBR (pacemaker backup rate, 35 ppm) successfully prevented syncope, but not prodromal symptoms, (ii) DDD mode pacing at a lower rate of 60 ppm was accompanied by worsening of epilepsy-related symptoms presumably by preventing the protective mechanism of spontaneous seizure termination, that is, the diminution of cerebral blood flow at low heart rates.

Currently, it is accepted that pacemaker therapy may be effective for preventing syncope associated with IA.1,2 Occasionally, this asystole transiently reduces cerebral perfusion to force ischemia-induced termination of epileptic activity.2,3 Consequently, while pacemakers may avert asystole, the characteristic T-LOC after epileptic seizures may be aggravated by maintenance of cerebral perfusion.

In conclusion, although pacing is an accepted treatment for prevention of syncope associated with IA, excessive pacing may inadvertently exacerbate epileptic symptoms. Thus, pacemaker programming must be carefully considered in IA, and antiepileptic medication remains important for effective treatment.

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References