A dangerous spike: cause or coincidence?

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Case presentation

A 62-year-old male was admitted to hospital because of cardiac arrest. He had a history of chronic bradycardic atrial fibrillation for which a single chamber pacemaker was implanted 15 years ago (programmed VVIR 50–140). He also had chronic moderate to severe functional mitral regurgitation with normal left ventricular systolic function. His therapy consisted of oral anticoagulation, beta-blocker, ACE-inhibitor and diuretic. ECG during resuscitation manoeuvers is showed in figure 1.

Question

What is the cause of the cardiac arrest in this patient?

Commentary

The ECG shows an idioventricular rhythm at 45 bpm with QTc within the normal range. There are regular spikes at 50 bpm (red arrows) (lower programmed rate) with loss of capture, evident for the first spike, and undersensing of the R waves. The third spike arises on the terminal upstroke part of the T-wave (the beginning of the vulnerable period) with the subsequent onset of a ventricular complex (R on T) (black arrow) initiating a polymorphic ventricular tachycardia degenerating into ventricular fibrillation.

This case therefore illustrates the development of a ventricular fibrillation on the basis of an R on T phenomenon, when the ventricular depolarisation falls in the vulnerable period. The question is if the R on T is due to a paced ventricular complex or is spontaneous with the pacemaker spike on the T-wave being only coincidental. The key problem in addressing this issue is to understand if the spike on the T-wave captures the ventricle or not.

There are arguments for the two possibilities. After successful defibrillation, the interrogation of the pacemaker confirmed a lead dysfunction with high threshold (3 V at 0.4 ms) causing loss of capture at the programmed value of 2.5 V, so that the spike on the T-wave should probably not produce the ventricular response. The first beat of the ventricular arrhythmia begins about 180 ms after the spike and this latency renders a capture rather unlikely. Moreover, the morphology of the first QRS of the ventricular arrhythmia available in the limb leads is slightly different from the morphology of the paced ventricular complexes seen in the limbs leads immediately after reprogramming the pacemaker to ensure capture (fig. 2). These arguments render rather unlikely that the pacing stimulus captures the ventricle so that the spike could be coincidental.

Considering that the threshold was only slightly higher than the programmed value (see above), the possibility of intermittent capture is reasonable. Moreover, it is known [1] that a subthreshold impulse, as could be the case in our patient with a dysfunctional pacemaker lead, can produce a full ventricular response (albeit generally at the end of the T-wave). A paced ventricular complex on the T-wave may intensify the ventricular electrical instability leading to the onset of the ventricular arrhythmia especially in the presence of the structural heart disease as in our case [2]. Additionally, the above mentioned 180 ms latency between spike and ventricular capture could be explained by slowed conduction which could also slightly change the QRS axis of the captured beat. These arguments would be in favour of a direct link between the pacemaker stimulus on the T-wave and the beginning of the ventricular fibrillation, a phenomenon on the other hand rarely reported [3].

After successful resuscitation, the pacemaker was reprogrammed reassuring the correct sensing and capture with the lower rate of 90 bpm (fig. 2). Laboratory analysis did not show significant electrolyte abnormalities and the patient did not take any potentially proarrhythmic medications. An echocardiogram showed severe biventricular dysfunction and severe mitral regurgitation, a coronary angiography did not documented significant coronary arteriosclerosis. The patient was treated for decompensated heart failure, the cardiac function normalised 5 days after admission and the pacemaker was replaced with a defibrillator.

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Figure 1
12-lead ECG during resuscitation (ECG registration in limb leads is not simultaneous with precordial leads). At the beginning of the registration there is an idioventricular rhythm at 45 bpm. Spikes at 50 bpm (red arrows) are noted with loss of capture (evident for the first spike) and undersensing of the R waves. The third spike arise at the terminal part of the ascending limb of the T wave and is followed by a ventricular complex (R on T) (black arrow) initiating a polymorphic ventricular tachycardia rapidly degenerating into ventricular fibrillation.

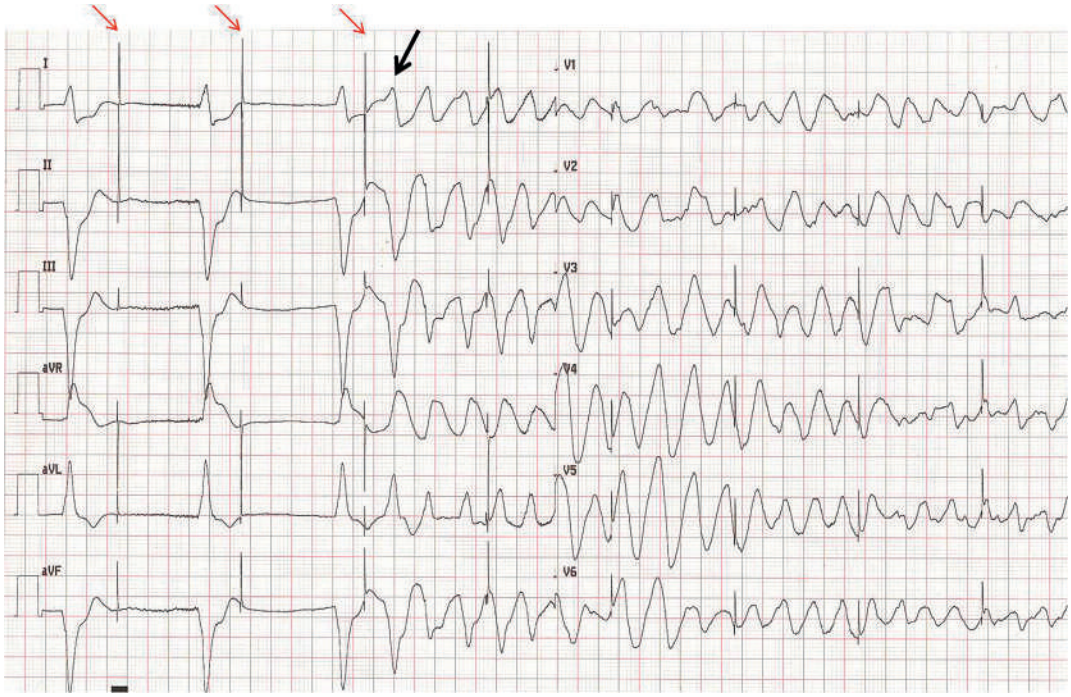
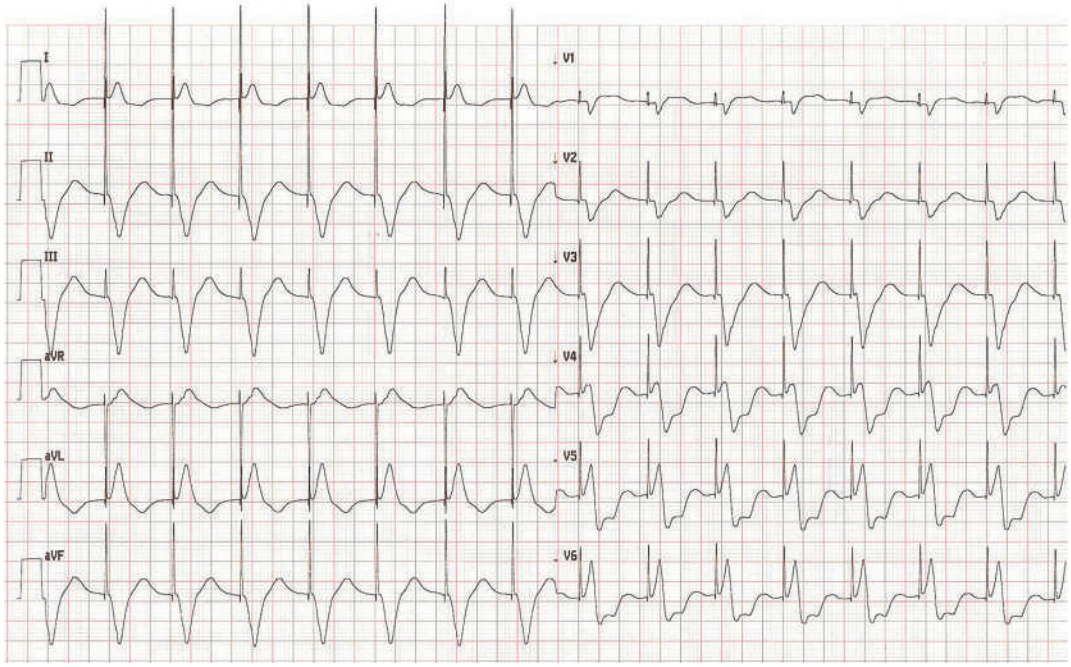


Figure 2
12-lead ECG after reprogramming the pacemaker (increase of output, increase of sensing). Normal ventricular capture at 90 bpm.



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