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Transient loss of capture after pulse field ablation due to pacing threshold elevation

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A 58-year-old man was referred for ablation of paroxysmal atrial fibrillation. He underwent surgical closure of an atrial septal defect at age twenty and ablations for atrial flutter and incisional atrial reentry. A single-chamber pacemaker had been inserted shortly after surgery for the occurrence of sick sinus syndrome. A screw-in-atrial lead had been inserted 7 years ago at the right appendage (original lead abandoned at the same position), with a chronic stable pacing threshold around 1 Volt.

Pulsed field ablation (PFA) of the four pulmonary veins was performed under general anaesthesia. Because of fractionated potentials just below the superior vena cava and repeated induction of Afib after mechanical contact in this area, an application of PFA energy was decided at this spot. The 35 mm Farawave ™ catheter (Boston Sc) was positioned just below the opening of the SVC in a basket-like conformation.

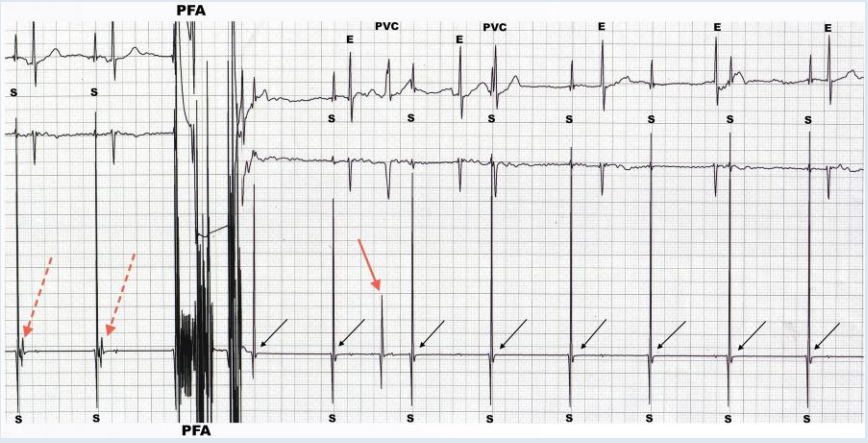

Figure 1 Surface ECG (upper tracings, leads II and V1) and intracardiac recordings (lower tracing, from farawave ™ catheter) immediately before, during, and after PFA, showing the loss of atrial capture: red dashed arrows are showing the consistent atrial capture before PFA, while there is no more capture after PFA (black arrows). Red arrow indicated the only atrial spontaneous escape event after PFA (S = spike artifact, E = junctional escape beats, PVC = premature ventricular beats).

Immediately after the first application (1.8 KV), loss of atrial capture happened (*Figure 1*). This application was automatically interrupted after 1 s by the generator, probably because of the restricted opening of the catheter and too close position of some splines as seen on fluoroscopy although direct contact to the pacing lead with a shortcut cannot be ruled out (but not visually evident and without artifact on intracardiac recordings).

Immediate control revealed an elevated threshold (2.5 V) which slowly recovered (1.8 V at 10 min in uni and bipolar configurations, and normalized < 1 V at 4 h and the day after). Impedance was normal (380 Ohms in uni and bipolar configurations). No further ablation was performed. The pacemaker was programmed with high output amplitude (4 V) and the patient was discharged without any relevant event over the following weeks.

This case highlights the risk of transient loss of capture, possibly due to impressive local sideration (reversible electroporation) or transient lesion, when PFA is delivered too close to pacing electrodes, even with reduced amplitude and very short duration (aborted application). It is likely that more 'regular' PFA application may have induced even more severe effects. The role of the pacing lead and device in concentrating energy by some antenna phenomenon is also plausible. Electrical cardioversion may also increase the pacing threshold, possibly because of some electroporation effect of DC shocks,[1] but also by direct injury to the pacing lead interface to the tissues. Even if no device damage has been published so far with PFA,[2] this case will preclude the use of PFA nearby pacing electrodes in the future. The Farawave™ catheter instructions for use should be reviewed and discussed with regards to PFA delivery in the proximity of pacing leads or other metallic structures.

Conflict of interest: None declared.

References

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