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Sudden QRS morphology change during ventricular pacing in a scar-related isthmus What is the mechanism?☆

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Abstract We present an example of sudden modification in QRS morphology during ventricular pacing inside a scar-related isthmus. This is explained by a "concealed" sudden block in both the orthodromic and antidromic wavefront directions, allowing then the activation to proceed through a now overt antidromic conduction.

Keywords: Isthmus; Reentry circuit; Ventricular tachycardia; Pace-mapping; Entrainment

A 71-year-old woman with non ischemic dilated cardiomypathy (left ventricular ejection fraction 45%) was investigated during a procedure of percutaneous radiofrequency (RF) ablation for incessant monomorphic ventricular tachycardia (VT) with a right bundle branch block pattern and right inferior axis, and a cycle length of 400 milliseconds (Fig. 1). She had been implanted 16 years before with an ICD after a first VT episode and was treated by beta-blockers.

Pace-mapping was used to delineate the potential VT isthmus [1] (280 ms cycle length, 10 mA, 2 ms bipolar pacing between the two distal poles of the ablation catheter). At a scar area at the basal left ventricular free wall close to the mitral annulus where pace-mapping during sinus rhythm perfectly reproduced the VT morphology, QRS morphology suddenly changed to a right bundle branch pattern and right inferior axis clearly different from the VT morphology (Fig. 1). This happened without any dislodgment of the catheter both on fluoroscopy or 3D navigation system with local good contact (basal left ventricular free wall with bending of the catheter). Which mechanism would explain this sudden modification in QRS morphology during otherwise stable ventricular pacing?

In this patient, pacing was performed within a protected scar-related isthmus possibly included in the VT circuit, at a site displaying a long conduction time and paced-QRS exactly matching the VT morphology. The exclusive orthodromic conduction along the VT isthmus is explained by the collision, in a more proximal part of the isthmus, of the retrogradely propagating antidromic wavefront with the orthodromic wavefront generated by the previous paced beat (Figs. 2 and 3). Before the sudden change in QRS morphology occurs, the S-to-QRS interval slightly increased (Fig. 2). One stimulus was then suddenly not followed by any QRS complex, but all subsequent pacing stimuli again led to ventricular capture but with a completely different QRS morphology and a shorter and fixed stimulus-to-QRS interval (Fig. 1).

Similarly to what happens for the third criterion of transient entrainment, as described during postoperative typical atrial flutter [2,3], but also during VT [4], the absence of QRS for one paced beat explains the sudden change in QRS morphology for the next paced beat. It is unlikely that this simply occurred because of a sudden lack of local capture since it has not been observed either before or after this event and, in addition, such a phenomenon would have possibly promoted VT induction. It is also unlikely that capture of far-field structures happened after block in the orthodromic conduction into the VT circuit, because of the still long delay between the spike artifacts and the QRS after QRS morphology changes. Another more likely explanation is that purely local capture happened together with bidirectional conduction block within the VT isthmus – i.e. both in the orthodromic (after progressive increase in conduction time) and antidromic directions (above mentioned collision with the previous orthodromic wavefront) – thus without any detectable ventricular activation on surface ECG (Figs. 2 and 3).

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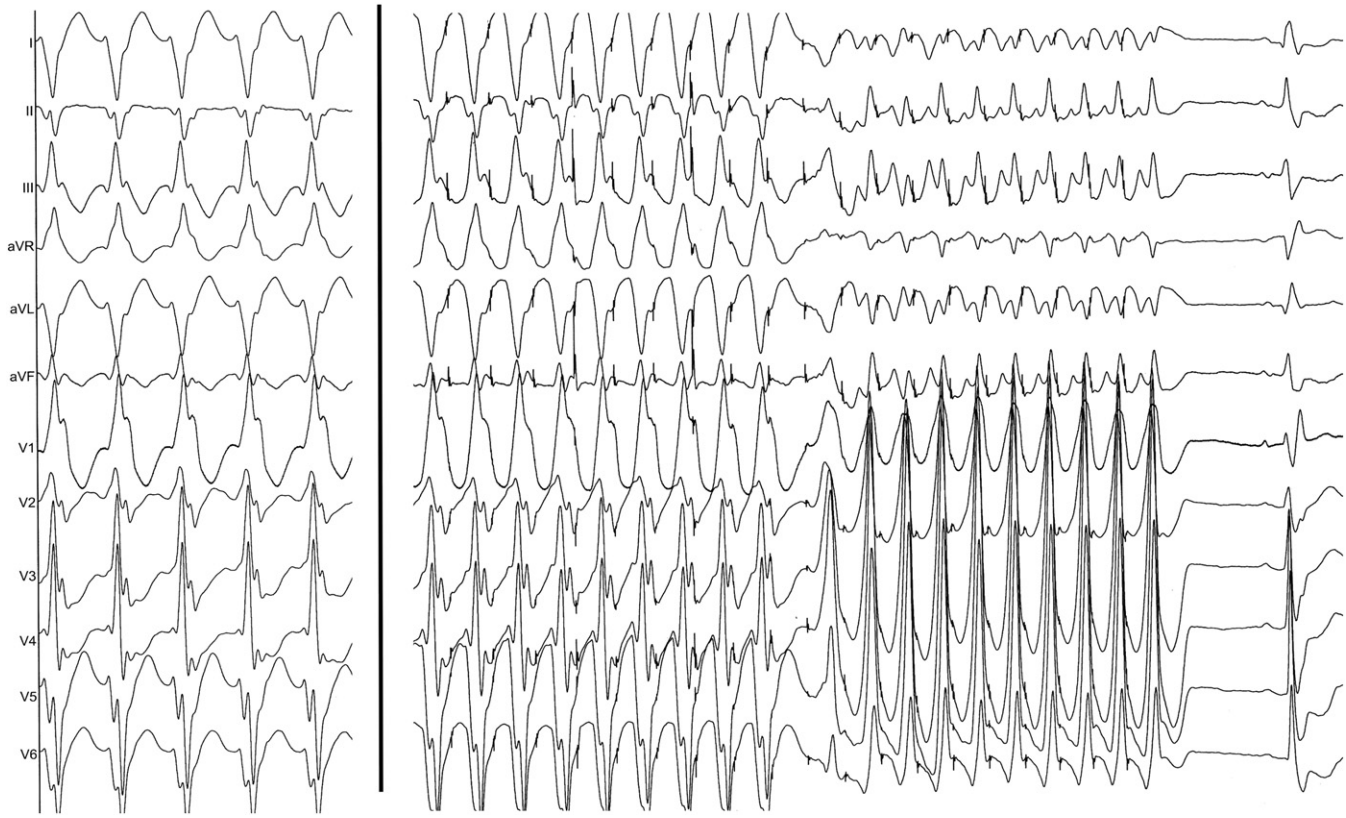

Fig. 1. 12-lead ECG of the spontaneous VT (left) and of the pace-mapping at 320 ms cycle length during sinus rhythm (right). At the initial part of the tracing, pace-mapping perfectly matches the VT morphology in any lead with a long delay between the pacing stimulus and the entrained QRS. Suddenly, QRS morphology changes from one beat to the next after a non conducted pacing event. Thereafter, QRS are still entrained at the pacing rate but with a different morphology and with shorter spike-QRS interval. (see text for explanation).

Such a sudden bidirectional conduction block for one beat allowed then the next pacing stimulus to generate an antidromic wavefront able to propagate freely retrogradely over the isthmus (since it did no more collide with any orthodromic activation) and to depolarize either the entrance site of the VT circuit or other bystander isthmus entrances/exit and with a shorter conduction time (Figs. 2 and 3). Thus, QRS morphology suddenly changed because a significant part of the ventricular muscle was now activated from the antidromic conduction, possibly with some degrees of fusions with the activation coming from the orthodromic wavefront, less participating to the ventricular activation due to its longer conduction time.

Similarly to what can be seen during the third entrainment criterion [2–4], activation at some sites – i.e. the right ventricular apex in Fig. 2 – was different after the non-conducted paced beat and this activation occurred with a shorter activation time. This was due to the modification of the preferential activation exit from pacing site inside the isthmus, which leads to changes in activation patterns and in activation time at some remote sites.

Changes in paced QRS morphology while pacing within a scar area have been already described [5] but the authors did not give the same explanations for these changes despite the fact that some exit block were noted in their examples. They rather mention the possibility of multiple exits from the circuit instead of reversal of activation through overt antidromic conduction after local concealed block inside the circuit as explained here.

Delineation of VT isthmuses during sinus rythm is an important tool for VT ablation. Recently, pace-mapping coupled to automated analysis of QRS morphologies has been shown to reliably characterize post-infarction VT isthmuses [1]. This technique is based on the greatest QRS changes occurring for the closest pacing sites, reflecting the fact that ventricular activation proceeds preferentially along the «exit» or «entrance» parts of the isthmus according to slight changes in the pacing site. In this case, the QRS changes did not happen by the change of pacing site but after the occurrence of a sudden transient block in both orthodromic and antidromic directions, but the conclusions made are the same: the pacing was possibly performed inside the VT critical isthmus (even if definitive proof for that would have needed classical parameters of concealed entrainment or activation mapping during VT). Moreover, the long stimulus-to-QRS delay in both directions proves that pacing site was located deep inside the protected isthmus, which is usually considered as the best target for ablation. The fortuitous fast rate of pacing, allowing block in the orthodromic direction, is probably the cause of this