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Cycle length oscillation during ventricular tachycardia: What is the mechanism?

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Case presentation

A 55 year-old man with a history of inferoapical myocardial infarction presented with symptomatic tolerated ventricular tachycardia (VT) and was referred for catheter ablation. A linear tridecapolar catheter (1-1-1 mm, T3, Access Point Technologies, Rogers, MN) was positioned along the region of endocardial isochronal crowding on the isochronal late activation map. Stable monomorphic VT with right bundle branch superior axis with V_4 transition was induced and exhibited spontaneous variability in tachycardia cycle length (TCL; 360–415 ms). What is the mechanism of oscillation in TCL? (Figure 1)

Commentary

The TCL oscillates between 360 and 415 ms with the same QRS morphology. The endocardial activation map showed an exit breakout in the inferolateral apex with early diastolic activation (LV 1,2–LV 7,8) observed more basally. A significant activation gap was present during late diastole, suggesting that the distal isthmus propagated on either the midmyocardium and/or epicardium (Online Supplemental Videos 1 and 2). Changes in the activation pattern of early to mid-diastolic potentials corresponded to changes in the V-V intervals of 360 and 365 ms with longer V-V intervals of 410 and 415 ms. During shorter V-V intervals, the higher amplitude diastolic potentials on the bipole LV 10,11 were earlier than the fragmented lower amplitude diastolic potentials on bipole pairs LV 5,6 and LV 7,8. In contrast, when VT slowed, diastolic potentials on LV 10,11 preceded changes on LV 5,6 and LV 7,8 during longer V-V intervals. This finding indicates that the diastolic potential on LV 11,12 was recorded from a critical circuit isthmus component with constant prediction of the TCL, with secondary activation of LV 5,6. Akin to bundle branch reentrant tachycardia, where the His-His interval that precedes and predicts the V-V interval, the isthmus can be confirmed without entrainment if the "isthmus-isthmus" interval predicts V-V in scar-related VT.[1]

Entrainment was attempted from the high-frequency diastolic potential on bipole LV 11,12 (Figure 2). During overdrive pacing at 330 ms from electrodes LV 11,12, initial capture was observed before termination of VT. For the first 2 captured beats, concealed fusion with orthodromic exit capture and S-QRS interval of 198 ms was observed. The low amplitude fractionated potential on electrodes LV 5,6 appeared to be accelerated to the paced cycle length. The third paced beat showed local capture with prolongation of the S-QRS, suggesting loss of capture or decremental conduction. The fourth paced beat showed local capture with block to the exit, without a resultant QRS complex, indicating termination without global capture.[2] This pacing response provided additional evidence that the local potential on LV 11,12 was recorded from the isthmus.

In summary, a novel criterion that can mechanistically prove an isthmus site is demonstrated with variable isthmus conduction interval that predicts TCL oscillations, when present. In this case, overdrive pacing with a nonpropagated stimulus further confirmed this observation. This illustration provides insights into the underlying mechanisms of TCL oscillation, which are attributable to spontaneous changes in intra-isthmus conduction.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2023.01.026.

References

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KEYWORDS Tachycardia cycle length; Ventricular tachycardia; Diastolic potential; Catheter ablation; Electrogram; Mapping; Entrainment (Heart Rhythm 2023;■:1)

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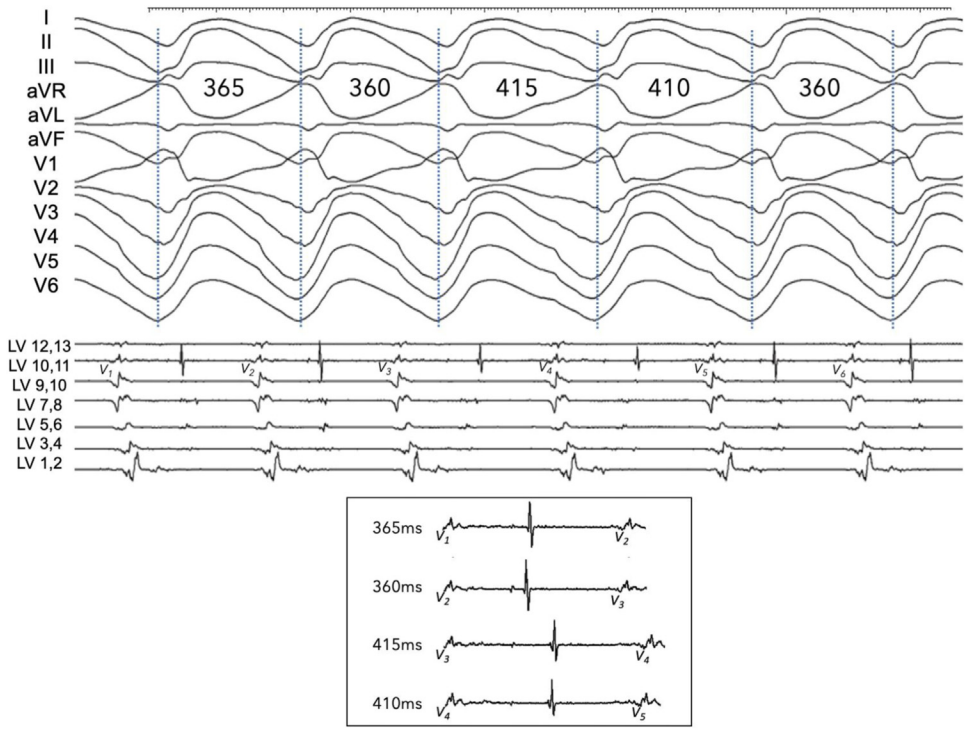

Figure 1 Surface and intracardiac electrograms of selected electrodes with sweep speed 200 m/s during monomorphic ventricular tachycardia. The labeled RR intervals show spontaneous tachycardia cycle length variation. The local ventricular activation coincident with QRS breakout (V1–V6) on the LV 11-12 electrodes is numbered in sequence. The lower panel shows all local electrograms recorded from the LV 11-12 electrodes aligned vertically and scaled up for each respective cycle length.

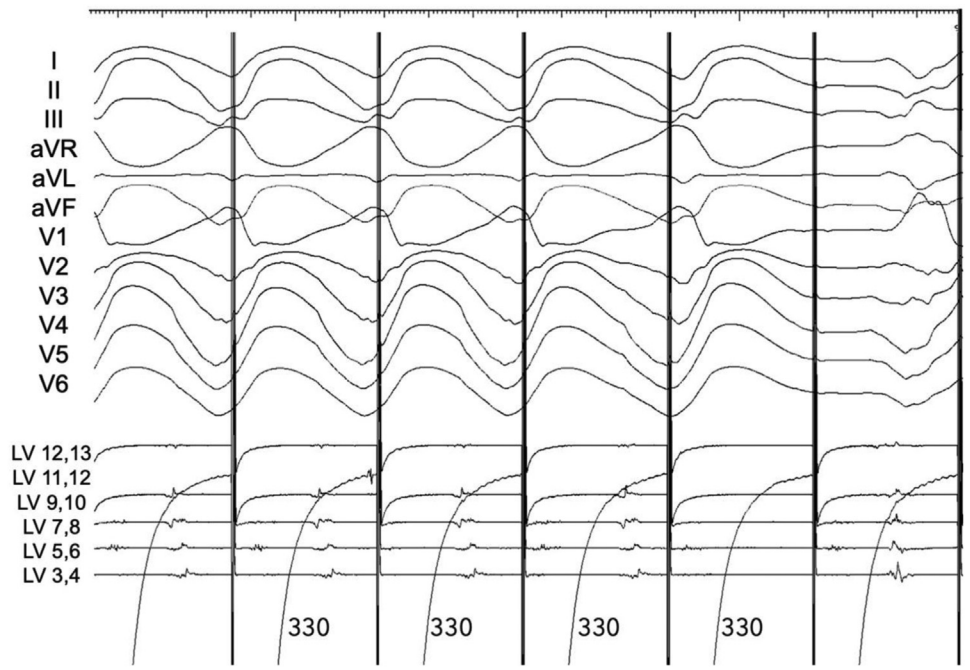

Figure 2 Overdrive pacing at 330 ms with capture of the first 2 beats. The third pacing stimulus has a longer S-QRS, which is consistent with loss of capture or decremental conduction. The fourth paced beat does not yield a QRS despite local capture (LV 5,6), which is consistent with nonpropagated stimulus and an isthmus site critical for reentry maintenance.