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Unexpectedly short postpacing interval in a left free wall accessory pathway mediated tachycardia: A pitfall of the corrected postpacing interval algorithm?

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1. Introduction

One of the maneuvers that help in the differential diagnosis of supraventricular tachycardias is the analysis of the response to transient entrainment or resetting of the tachycardia by right ventricular pacing. The basis of the analysis is the subtraction of the tachycardia cycle length (TCL) from the postpacing interval (PPI) measured at the pacing site, so called "PPI-TCL", as first described for the differential diagnosis between AV nodal reentrant tachycardias (AVNRT) and tachycardias using a septal accessory pathways (AP) [1]. Further refinements of the maneuver included a correction for the rate-related increase in AV node delay (subtraction of the AH interval during tachycardia from the AH interval resulting from the last paced beat, the "corrected PPI-TCL") [2], the use of single premature extrastimuli resulting in resetting [3] or a simplification of the maneuver by just subtracting the ventriculoatrial interval during tachycardia (VA interval) from stimulus to atrial interval during entrainment (SA interval), leading to the "SA-VA" value [1,4]. Shorter intervals of all these measurements are found in tachycardias mediated by AP as compared to AVNRT because the ventricles are part of the circuit in the former [1–4]. However, within AP mediated tachycardias, those using septal pathways have shorter intervals as compared to tachycardias mediated by left free-wall AP [2–4]. We present the case of a left free-wall AP mediated tachycardia and an unexpectedly short "corrected PPI-TCL".

2. Case presentation

48 year-old man, without structural heart disease with history of previously treated Hodgkin's lymphoma, was admitted to the Hospital with pneumonia; during his stay he had an episode of wide QRS complex tachycardia, that was poorly tolerated and required electrical cardioversion. After the pneumonia was resolved he was referred for electrophysiologic evaluation.

In the baseline condition, during ventricular stimulation, a wide QRS complex tachycardia (morphologically identical to the spontaneous tachycardia) with left bundle branch block (LBBB) was induced. The tachycardia cycle length was 344 ms, the HV interval 60 ms and the VA interval 150 ms (Fig. 1). Atrial activation sequence was eccentric, earliest in the proximal coronary sinus (CS catheter advanced to a clock type 3 position). An extrastimulus from the right ventricle, delivered when the His bundle was refractory (depolarized 30 ms before the extrastimulus), advanced the following atrial depolarization (Fig. 1). Both findings were consistent with a left free-wall AP mediated tachycardia. Rapid right ventricular mid septal pacing at a cycle length of 330 ms entrained the tachycardia (Fig. 2).

When measuring the corrected PPI-TCL, the value was 28 ms after resetting (Fig. 1) and 30 ms after entrainment (Fig. 2), totally unexpected for a circus movement tachycardia mediated by a left free-wall AP. Moreover, the SA-VA interval was 12 ms in response to resetting (Fig. 1) and 18 ms in response to entrainment (Fig. 2), again extremely short and totally unexpected values for a tachycardia mediated by a left free-wall AP.

2.1. Comentary

The explanation for the different corrected PPI-TCL (and SA-VA) in relation to location of accessory pathway during right ventricular stimulation relays in the principles of the interpretation of the

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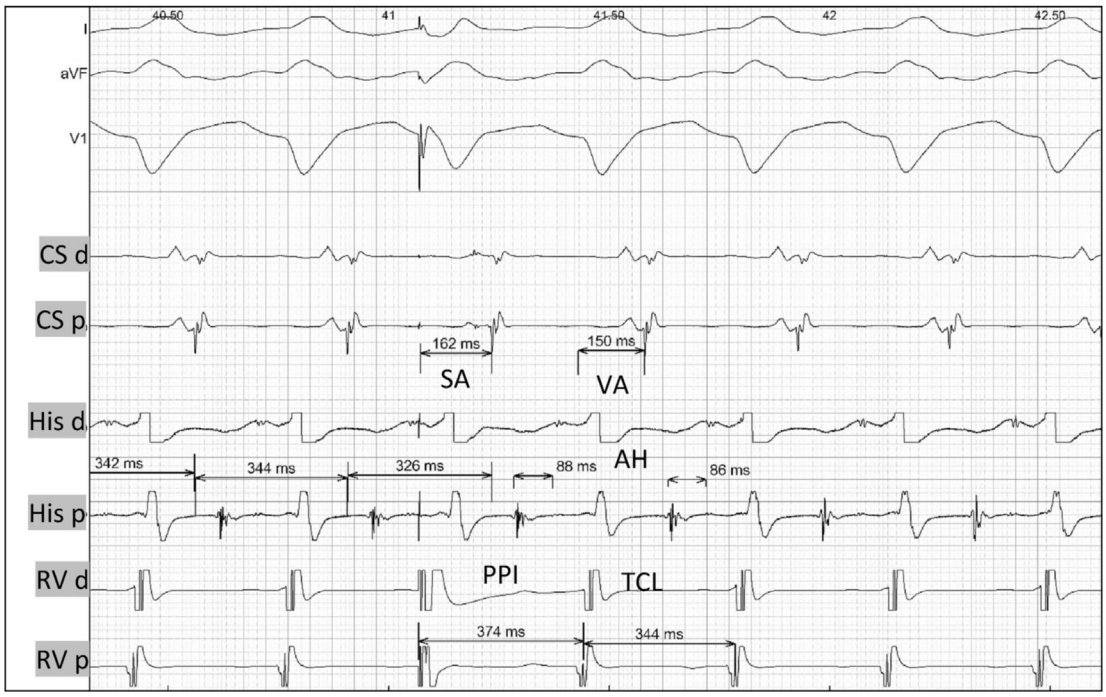

Fig. 1. ECG and intracardiac recordings from the coronary sinus (CS), His bundle area (HIS) and right ventricle (RV) during wide QRS complex (LBBB) tachycardia. Both eccentric atrial activation sequence (earliest in CS) and tachycardia reset by a single right ventricular extrastimulus when the His bundle is refractory are consistent with a left free-wall AP mediated tachycardia. Note unexpectedly short corrected PPI-TCL (30 ms) and SA-VA (12 ms).

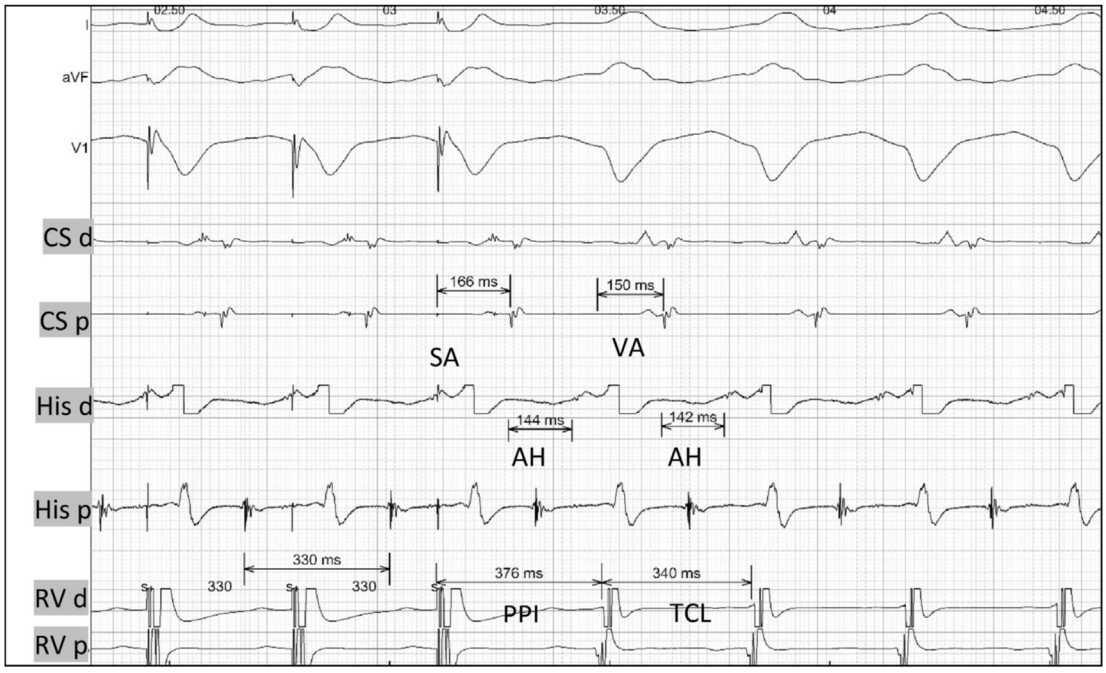

Fig. 2. ECG and intracardiac recordings as in Fig. 1. The tracing shows the end of a pacing train delivered during tachycardia (the same tachycardia recorded in Fig. 1) that produces transient entrainment. Note unexpectedly short corrected PPI-TCL (32 ms) and SA-VA (30 ms).

resetting and entrainment responses, as recently reviewed [5]: PPI exceeds the TCL by the amount of time that the paced wavefront takes to reach the reentrant circuit from the stimulated site plus the amount of time it takes from the exit of the circuit to the pacing site. During circus movement tachycardias mediated by left free-wall accessory pathways the actual ventricular wavefront that travels across the ventricles to engage the ventricular insertion of the AP is expected to be generated by the left bundle branch and Purkinje fibers connected to it, travelling a relatively short distance to access the ventricular insertion of the AP at the mitral annulus (Fig. 3). In such situations, the circuit is “far away” from the right ventricular pacing area (in terms of conduction time), and corrected PPI-TCL

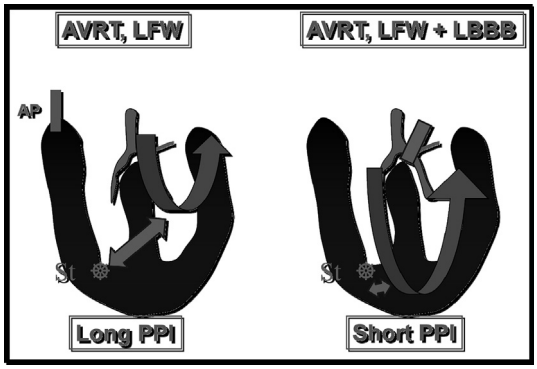

Fig. 3. The figures show how circuit becomes nearly to stimulation place during wide QRS complex (LBBB) tachycardia explaining PPI-TCL shorter compared to QRS complex narrow tachycardia.

(and SA-VA) are expected to be longer than in circus movement tachycardias mediated by septal (or right free-wall) AP since the circuit is closer to the pacing site in the latter. The mean value for corrected PPI-TCL was found to be 81 ± 21 ms and the mean SA-VA 73 ± 26 ms during entrainment (77 ± 20 ms and 71 ± 19 during resetting) in free-wall accessory pathways mediated tachycardias, as opposed to 44 ± 20 ms and 32 ± 24 ms during entrainment (37 ± 18 ms and 33 ± 16 during resetting) in septal accessory pathways mediated tachycardias [2,3]. So numbers in Figs. 1 and 2 fall well into the septal accessory pathway category.

The above will account for corrected PPI-TCL (and SA-VA) in narrow QRS tachycardias. However, when left bundle branch block (LBBB) develops during circus movement tachycardias mediated by left free-wall AP, the actual ventricular wavefront that engages the ventricular insertion of the AP has to emanate from the right bundle branch, traversing the septum to reach the left ventricle (Fig. 3). Thus, the circuit is enlarged and located in close proximity to the right ventricular apical area, so the pacing site is close to the circuit and corrected PPI approximates TCL (and SA approximates VA). This is the situation in Figs. 1 and 2.

Further support for this explanation is presented in Fig. 4. A narrow QRS tachycardia with identical atrial activation sequence appeared in this same patient in response to a single premature ventricular extrastimulus (not shown). Fig. 4 shows that a single ventricular extrastimulus reset the tachycardia but now with a longer corrected PPI-TCL interval. Thus, for the same tachycardia mechanism but with narrow QRS, the values for the corrected PPI-TCL and SA-VA are within the limits expected for a left free-wall accessory pathway mediated tachycardia (94 and 74 ms respectively).

The accessory pathway was successfully ablated and tachycardia was no longer inducible.

Can this be considered a pitfall of the corrected PPI-TCL (and SA-VA) algorithms? 1) For the differential diagnosis between AVNRT and AP mediated tachycardia it is not a pitfall and actually makes even clearer the differential diagnosis since LBBB shortens more the values for some AP mediated tachycardias (and LBBB is not expected to influence the response during AVNRT). 2) For AP localization it certainly is a pitfall, since left free-wall AP mediated tachycardias will behave, in the presence of LBBB, as septal or right free-wall AP mediated tachycardias. This means that, in the presence of LBBB, the corrected PPI-TCL (and SA-VA) algorithms cannot be used for AP location approximation. 3) It is of note that in the series of Gonzalez-Torrecilla et al. [2,4], cases with wide QRS complex were excluded. The present case suggests that the corrected PPI-TCL (and SA-VA) algorithms can be extended to

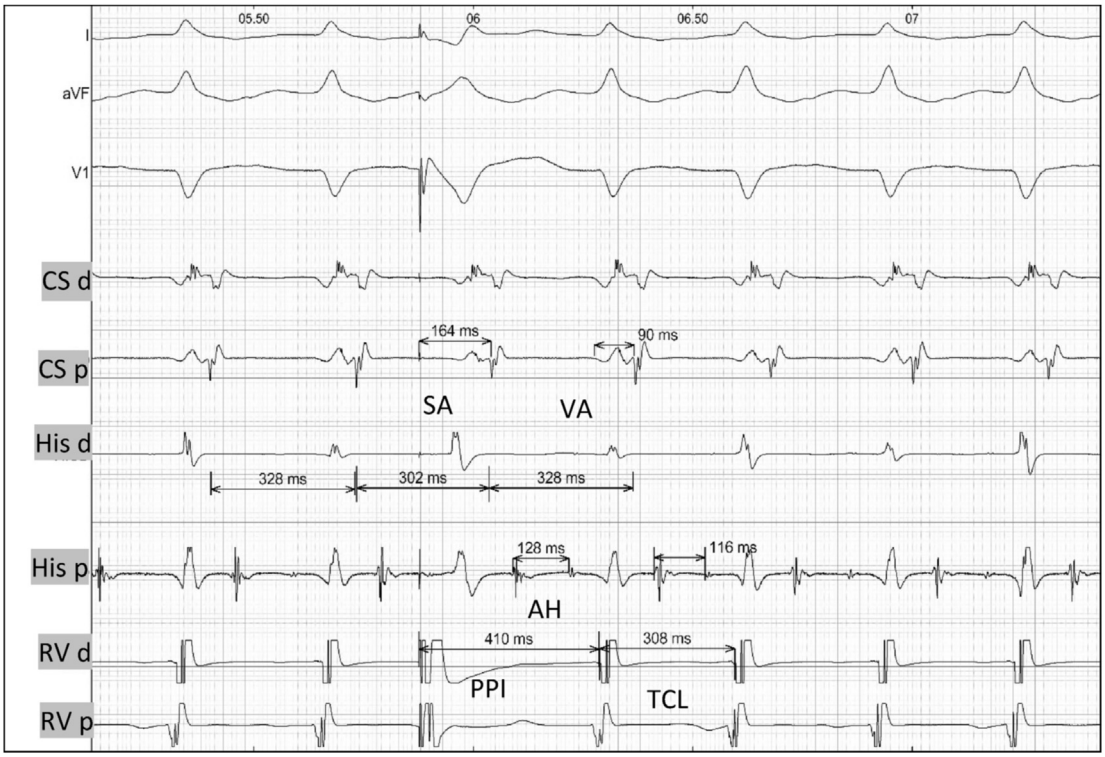

Fig. 4. ECG and intracardiac recordings during narrow QRS complex tachycardia with identical mechanism as the tachycardia in Fig. 1 (atrial activation sequence is identical). Intracardiac recordings as in Fig. 1. A single right ventricular extrastimulus produces tachycardia reset when the His bundle is refractory. Note that both corrected PPI-TCL (92 ms) and SA-VA (74 ms) are in the range that is expected for a left-free wall AP mediated tachycardia.

tachycardias with LBBB (and probably right BBB too) for the differential diagnosis between AVNRT and AP mediated tachycardia 4) This example remembers that the PPI-TCL maneuver is based on the resetting-entrainment principles, and thus relates to the whole tachycardia circuit and not just to the AP. 5) From a practical standpoint, the good news is that the cases in which the corrected PPI-TCL (and SA-VA) algorithms can be misleading for AP localization are easily identified by the presence of LBBB, and if it is functional (as in the present case), the algorithms can help in the same patient during narrow QRS tachycardia.

3. Conclusion

This case shows that, in the presence of LBBB, the corrected PPI-TCL (and SA-VA) response to resetting and/or entrainment with right ventricular stimulation during left free-wall AP mediated tachycardias can resemble that observed during septal or right free-wall AP mediated tachycardias. However, LBBB does not obscure the contribution of resetting and/or entrainment to the differential diagnosis between AVNRT and AP mediated tachycardia.

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