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Mechanisms of torsade de pointes tachycardia in patients with spontaneous high-degree atrioventricular block: A modern look at old data☆

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ABSTRACT

Introduction: The mechanisms of torsade de pointes tachycardia (TdP) are incompletely understood. We aimed to investigate the mechanisms underlying TdP tachycardia in patients with spontaneous high-degree atrioventricular block (AVB).

Methods and results: This retrospective study reviewed old TdP recordings after ventricular temporary pacing interruption in 16 patients with spontaneous high-degree AVB. Five of them had also bipolar endocardial right ventricular (RV) apex recordings. The QT interval during AVB at a mean heart rate of 38.9 ± 7.5 bpm was 653.0 ± 67.2 ms. The critical coupling interval (CCI) between the last escape QRS during AVB and the first premature ventricular complex (PVC) was significantly shorter before the onset of TdP than before single PVCs and couplets. A morphologic crescendo of the escape T wave was observed before the onset of TdP, followed by a rhythmic and morphologic crescendo of PVCs. The escape RV apex electrograms (EGMs) showed the constant pattern of a rapid deflection similar to a Purkinje potential 40 to 80 ms after the onset of the QRS, superimposed on a smooth low amplitude signal in 4 out of 5 patients.

Conclusions: The major endocardial T wave prolongation and augmentation (morphologic crescendo) of the escape beat prior to the first PVC suggests a phase 2 reentry mechanism due to early afterdepolarization. The induced TdP can be due to the changing outputs from one or two simultaneous RV moving reentry circuits between depressed fibers and fast conducting ones, possibly located in the thin crista supraventricularis structure which has several connections with the septum and the RV free wall.

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Torsade de pointes tachycardia (TdP) is a rare but important cause of sudden death in young people with the genetically determined long QT syndrome (LQTS). In these patients, the length of the QT interval is directly related to the arrhythmic risk. TdP is also a major concern of the pharmaceutical industry for the development of cardiac and non-cardiac drugs. It is important to understand the basic mechanisms of this form of polymorphic ventricular tachycardia (VT) when it occurs in patients with high-degree atrioventricular block (AVB). In 1966, this arrhythmia received the descriptive term of "Torsade de pointes". This name was chosen to describe the QRS that twists around the isoelectric line by Desertenne [1]. Scientists reproduced this ECG pattern experimentally by pacing both ventricles in isolated pig hearts and confirmed the original hypothesis of two alternating activation sites [2]. Moreover, cellular electrophysiologists [3,4] determined that early after depolarization (EAD) should be the initial fundamental mechanism, as EAD are bradycardia-dependent and favored by QT prolongation, as are TdP. However, their perpetuation mechanism remains still discussed between automaticity and reentry. The role of M cells remains also discussed with diverging data from myocardium wedge preparations and from whole intact hearts [5].

The ECG recordings presented in this manuscript were performed between 1964 and 1970, at a time when most of these data was unknown. Some patients with coronary artery disease had "transient ventricular fibrillation (VF)", a term associated with poor prognosis, and others with complete AVB had what was called "tachyrhythmia" in English publications [6]. There was also a waiting period with temporary pacing leads in those early times of pacemaker (PM) implantation. These original

☆ We dedicate this work to our great mentor and teacher Guy H. Fontaine who is deeply missed.

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studies were undertaken to demonstrate that these arrhythmias could be prevented by pacing in some of these patients and that it reoccurred when pacing was interrupted. When this was confirmed, permanent pacemaker implantation became a routine procedure. In addition, not all patients with chronic AV block develop long QT and TdP, since there is genetic predisposition [7], but the exact mechanism of TdP and their relation to VT and VF was not fully elucidated.

In this study, we aimed to synthesize the actual knowledge with the data from these old observations. We also propose a potential site for the localization of this type of TdP.

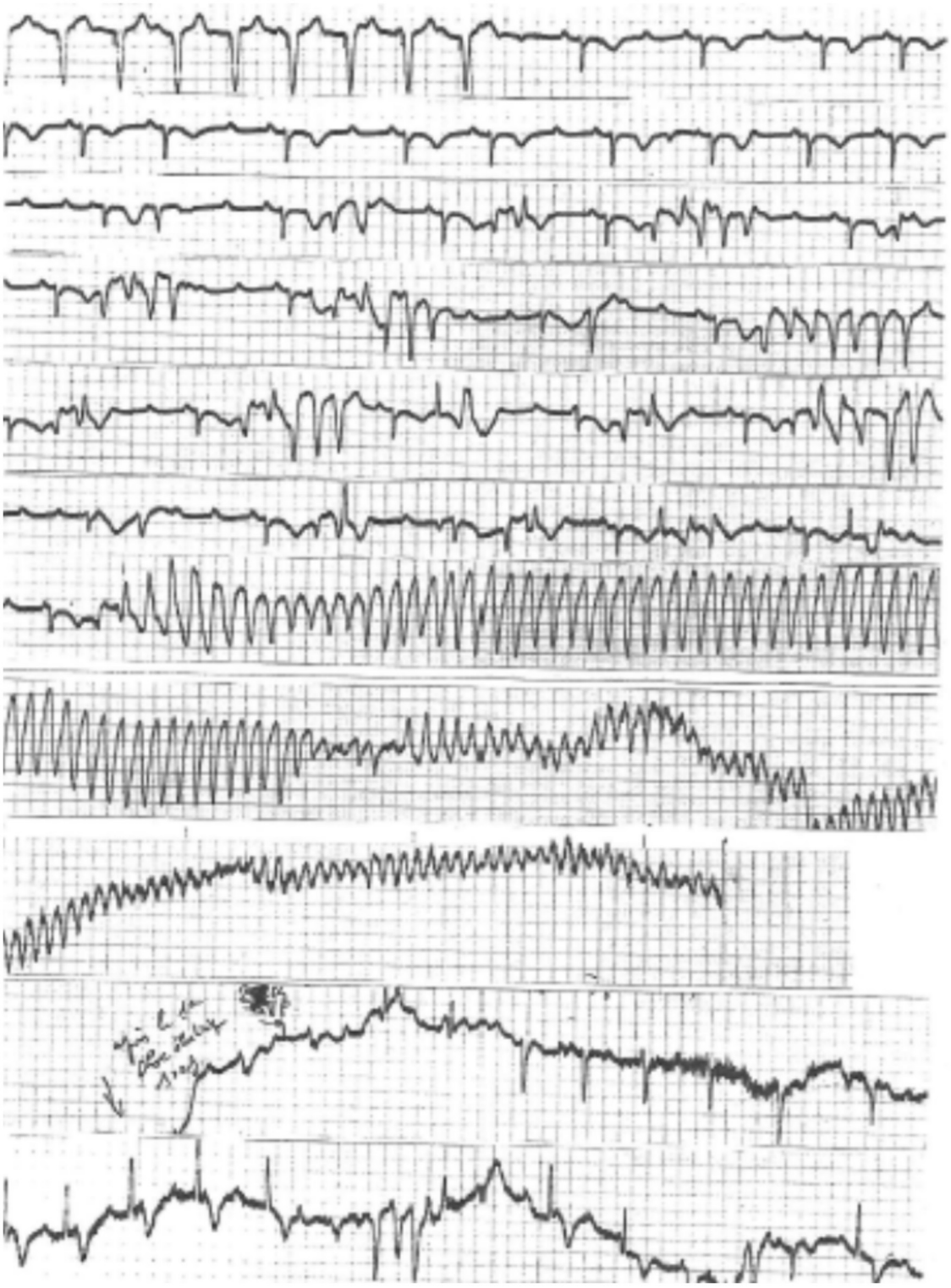


Fig. 1. Surface ECG (single lead derived from bipolar chest leads between V1 and V4): interruption of ventricular pacing (first line) shows long diastoles due to high degree AVB leading to a rhythmic crescendo and finally rapid VT pattern degenerating into VF converted by 100 Joules monophasic DC shock. Skeletal muscle artifacts (asterisk) are related to convulsions during 2 s at the recovery of consciousness (paper speed 25 mm/s).

Methods

Definitions

TdP has a complex definition, and what is specific is not really the torsades of QRS by itself, as there are other types of polymorphic VT, but its initiating complex, with a long short sequence on a long QT interval. In this series of AVB-induced LQTS, aborted sequences could be limited to the initial PVC or followed by one or several cycles of non-sustained VT with the same or different morphologies, with abrupt or progressive morphological changes, and sometimes degenerating into VF.

Study population

The study population consisted of 16 patients exhibiting 107 typical episodes of TdP during spontaneous high-degree AVB, and studied by the surface ECG recordings approximately 50 years ago. As explained above, these patients were waiting for definite PM implantation or replacement, and therefore had a temporary pacing bipolar electrode connected to an external pacemaker. They were >65 years old (range 66–80 years), 13 women and 3 men, and none was taking drugs known to prolong QT intervals. All had normal serum laboratory tests including serum potassium levels. There were no data on any familial disease.

All patients presented with type 2 second degree (Mobitz II) or third degree AVB. Most of them had well tolerated short episodes of TdP (<5 s) preceded by premature ventricular complexes (PVCs), couplets, triplets etc. After the recording of several episodes on a standard ECG recorder, it was noticed that some of them had the same QRS morphologies at the initiation of TdP. Therefore, to gain further insight, long-term bipolar ECGs were tape recorded in all, between a negative skin electrode placed in position V1 and a positive one placed in position V4, similar as lead II orientation, but in front of the right ventricle and the septum. Five of them had a simultaneous endocardial recording from the RV apex with the 1.2 cm bipolar pacing endocardial electrode. The precordial as the endocardial electrodes were connected to Tektronix 3A9 differential amplifiers with a band pass of DC to 60 Hz when specific electrophysiological equipment was not available in those times. The amplifier output was connected to a tape recorder (Ampex SP 300, Redwood City, CA, USA). The tapes were subsequently reviewed with an oscilloscope (Tektronix 561A-3A72) to select episodes of TdP to be printed on a channel ECG machine (Sanborn EH101371 Cambridge, MA, USA), later on a dual channel industrial ink pressure recorder (Clevite Brush 220 Chart Recorder Cleveland, OH, USA). All data were collected and reviewed by electrophysiologists before they were entered into a spreadsheet for statistical analysis (Dell Statisticav12, Tulsa, OK, USA). The critical coupling interval (CCI) between the last beat during AVB and the first abnormal ventricular events such as PVCs, couplets, triplets and TdPs (defined by >3 consecutive complexes) were identified, numbered and measured with a caliper with a precision <20 ms. Finally, the occurrence of TdP after pacing interruption was repeatedly assessed. The CCIs and the amplitude of the last TdP beats were also analyzed. Histograms of each numeric variable were drawn to identify outliers. The comparison of populations with continuous variables was performed by the Student's test. A P value<0.05 was considered significant.

Preliminary results of this study have been previously published [8–10]. This study is a more detailed and extended analysis of these previous reports.

Results

The QT interval during AVB at a mean heart rate of 38.9 ± 7.5 bpm was 653.0 ± 67.2 ms.

Ventricular event timings

All ventricular arrhythmic events were preceded by long diastole from 1000 to >1500 ms terminated by a junctional or ventricular escape (Fig. 1). After this escape beat, PVC occurred late, after the end of the prolonged T wave. Single PVCs were observed in 160 episodes with a CCI of 672 ± 44 ms (550–760), 121 couplets with a CCI of 676 ± 37 ms (560–760), 21 triplets with a CCI of 644 ± 39 ms (560–720), and TdP in 107 episodes with a CCI of 639 ± 52 ms (500–760) ($p < 0.05$ for TdP vs. single PVCs and couplets), suggesting a trend to shorter coupling intervals for the onset of sustained TdP. The case presented in Fig. 1 was the only one in which TdP did degenerate into fast regular VT and then VF. In the five patients with endocardial recordings (Fig. 2), the onset of endocardial activation on the RV apex diagnostic catheter was observed prior to or at the same time as the initiating PVC. TdP occurred separated by long periods without PVC in 11 patients.

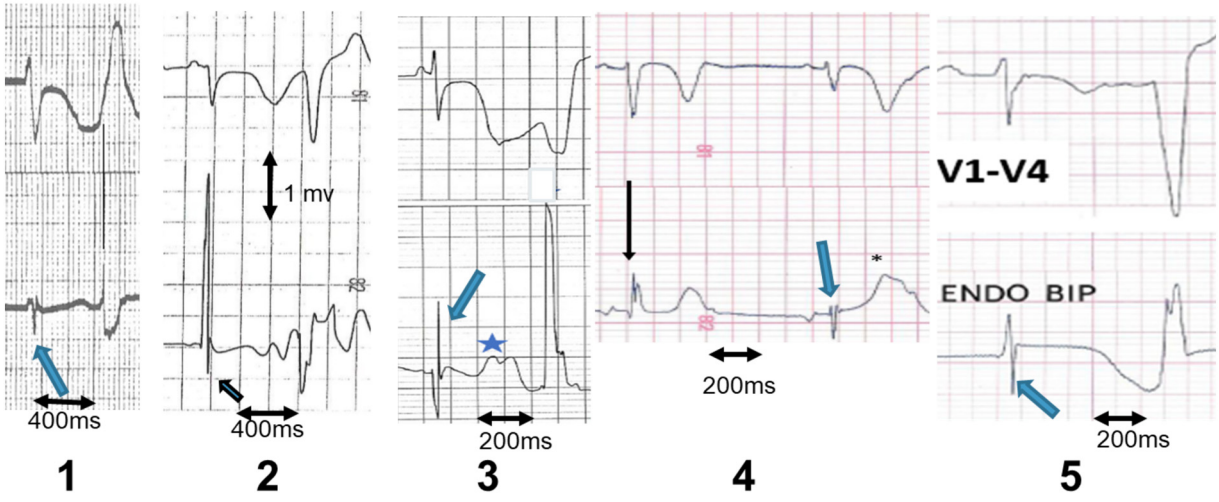


Fig. 2. ECG and apex EGM of last escape complex and initiating PVC in the 5 patients with both tape recordings. Top tracings: Surface ECG (single bipolar V1-V4 chest leads). Bottom tracings: Synchronous bipolar RV apex EGM replayed from the magnetic tape. The escape endocardial potential had in each patient the same pattern of a fast sharp signal (arrows) similar to a Purkinje potential superposed on a wider low amplitude signal in four of them, as a remote myocardial signal, or a depressed local myocardial signal. A similar sharp signal is visible after the initiating TdP PVC's onset as the transmission from a remote origin. It may be part of the Purkinje system (Asterisk indicates T wave augmentation (morphologic crescendo).

Repolarization crescendo

The severe bradycardia-induced progressive modifications of the T wave durations and shapes of the escape rhythms reflect an increasing dispersion of repolarization (Figs. 1, 3A and C) during several seconds before the first PVC. This “morphologic crescendo” (previously called "T wave augmentation" by our group) announcing the onset of TdP was observed in 12 patients (75%), sometimes associated with monstrous “bumps” better seen on the endocardial recordings, bumps which could also progressively regress (Fig. 3B), with PVC's disappearance.

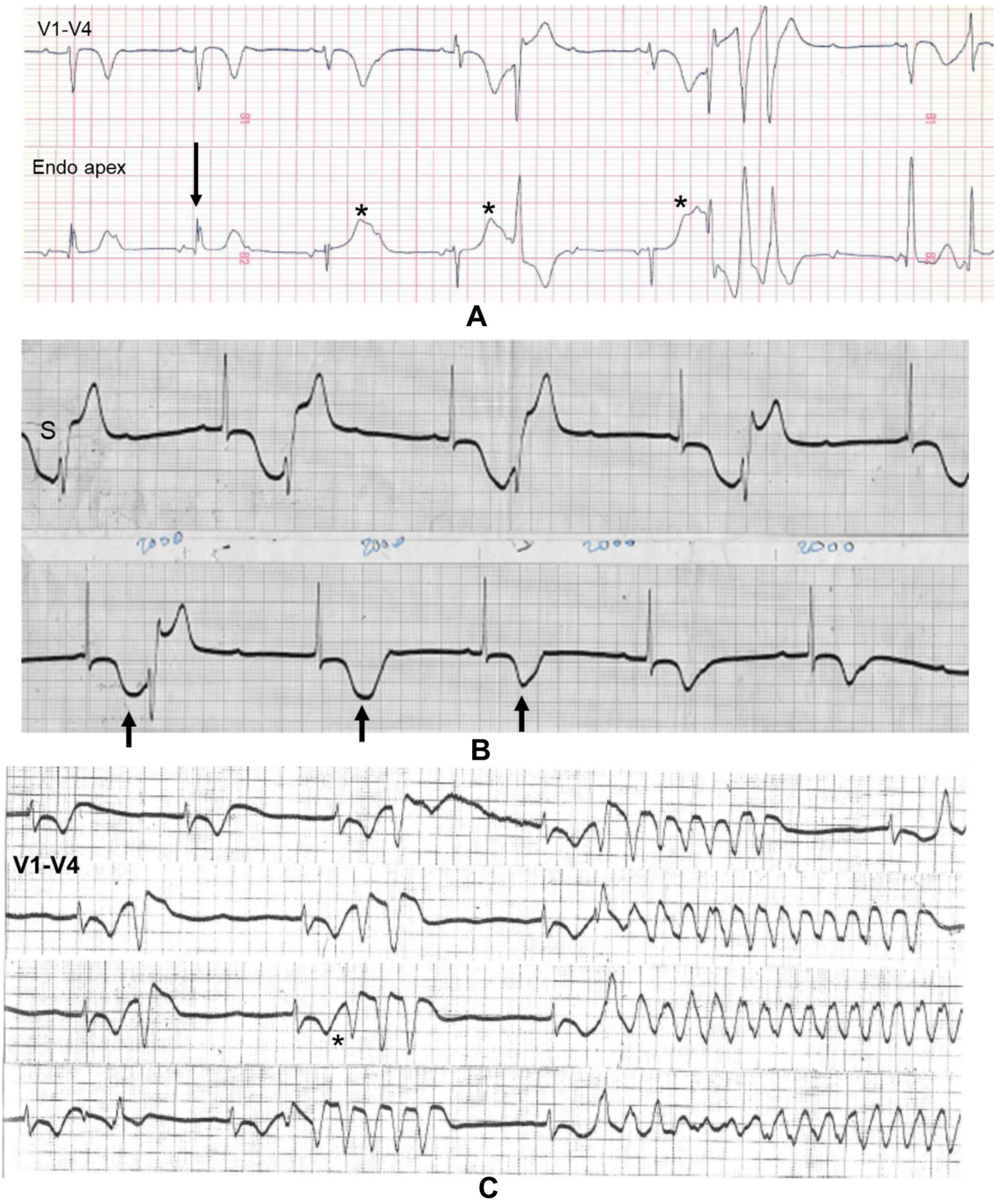


Fig. 3. A, ECG (bipolar precordial V1-V4, 25 mm/s) and below EGM repolarization crescendo (asterisk) (patient 4 in Fig. 2 recordings), before the PVC occurrence. B, ECG repolarization decrescendo with PVC disappearance (arrows). C, ECG (bipolar precordial V1-V4, 25 mm/s): ECG strips displaying the rhythmic crescendo with negative isolated PVC or salvos up to seven, suggesting a single circuit reentry. TdP are only induced by a positive one after a prolonged T wave. The progressive QRS changes in shape during the torsade on the three bottom strips suggest for each one a different site, and could be explained by a progressive fusion of two circuits with a small different phase or one with two exit points with different conduction times.

Rhythmic crescendo

TdP episodes were generally preceded by a "rhythmic crescendo" of isolated PVCs, then couplets, triplets, quadruplets etc., (Figs. 1 and 3C) inducing the classical long-short initiating cycle sequence. This was observed in all 16 cases. A rhythmic decrescendo of these arrhythmias was observed in 11 patients after the end of a TdP period.

Ventricular complex morphologies

Periods of non-sustained TdP (up to around 30 s) showed several repetitive, almost identical morphologies, which could be classified into four major categories: 4 patients had a single one, 5 patients had 2, 6 patients had 3, and one had 4 morphologies (Fig. 3C) with fusion beats. Of these, the initiating PVC morphology could be negative or positive in the bipolar precordial ECG recordings in the same patient (Figs. 3C and 4). An abrupt change of QRS orientation from upward to downward is illustrated in Fig. 5A, at the end of TdP and was observed in four cases, the others without major change from the preceding cycles. The single bipolar precordial derivation from the tape recordings did not allow a clearer identification of the site of PVC origin than a leftward and descending activation for a positive wave, which could be originating from the RV outflow tract (RVOT), and a negative one from the inferior part of the ventricles (Fig. 3C), and fusions complexes.

Endocardial data were available for 5 patients, all from the RV apex bipolar electrodes, and detailed elsewhere [9,10]. All had an escape EGM with a sharp Purkinje-like potential synchronous with the middle of the QRS, superimposed to a low voltage surrounding signal in 4 of them (Fig. 2). A similar potential could be observed at the onset of the initiating PVC in case 1 from Fig. 2, preceded by a smooth potential before the QRS onset in continuity with the T wave of the escape beat, interpreted as EAD's from the same region. This kind of thin potential was observed at other moments of the recording with a later timing, during PVC's of other morphologies (Fig. 4) in the same patient.

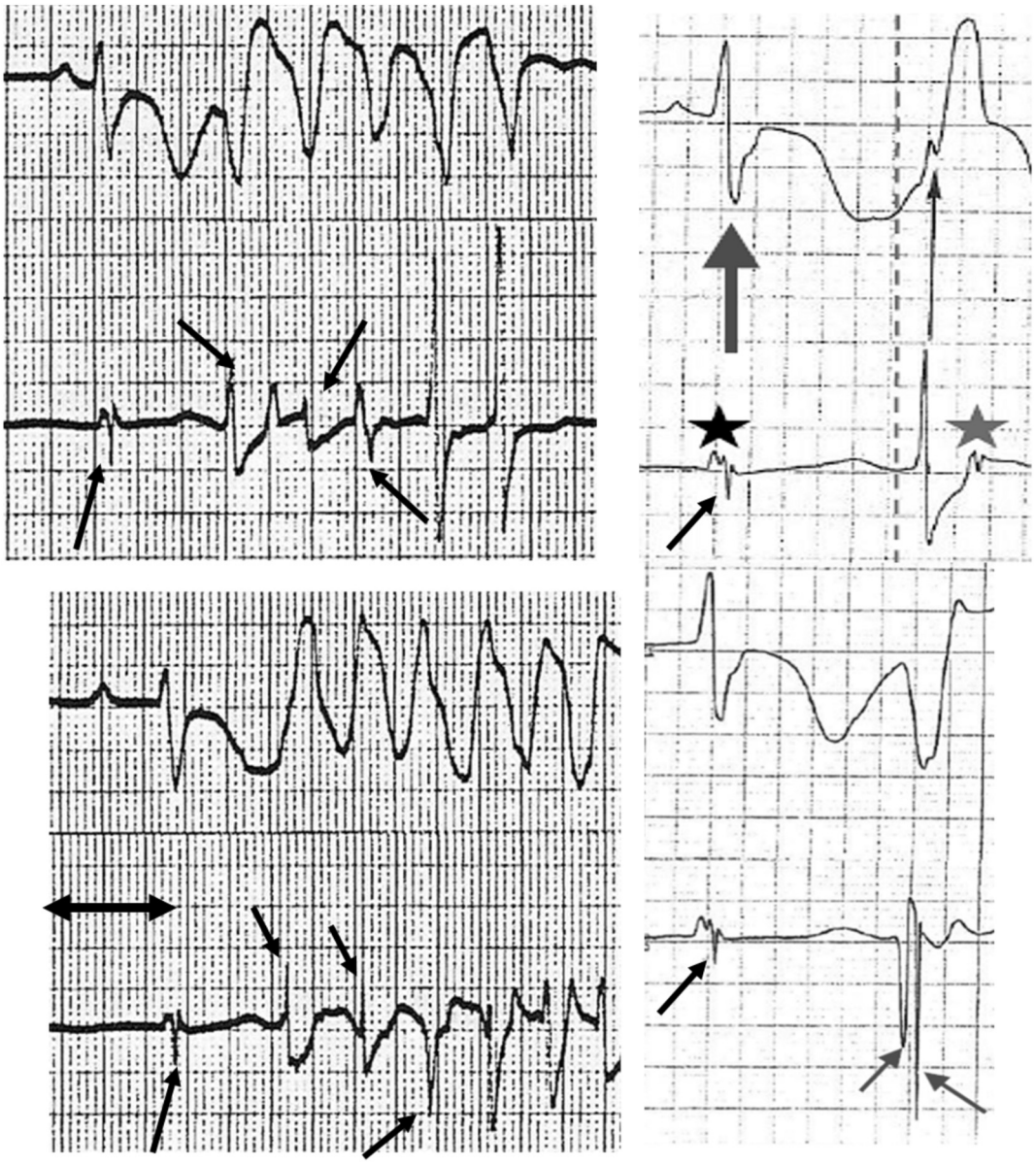
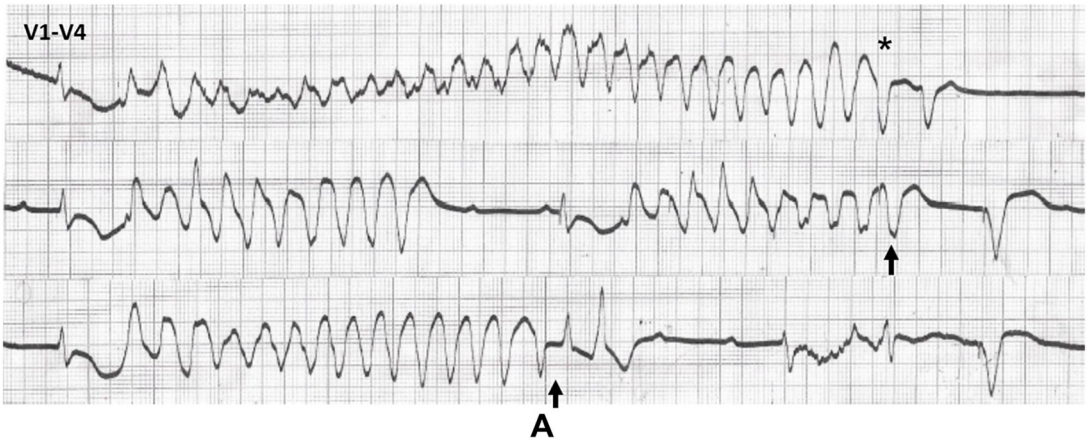


Fig. 4. Four different TdP onsets: ECG and EGM in patient 1 from Fig. 2. Note the different timings of the sharp Purkinje like signals within the different PVC's (the speed of the left column is 25 mm/s, the speed of the right column is 50 mm/s, arrows or stars).

Fragmentations were recorded in the next beats suggesting local inhomogeneity of the activation process (Figs. 2 and 4). On the nowadays retrieved tracings, complete endocardial recordings during TdP were only available for 1 patient (Fig. 5B).

Discussion

In this study, TdP were reproducibly observed in all patients with high-degree AVB. The CCI between the last escape QRS during AVB



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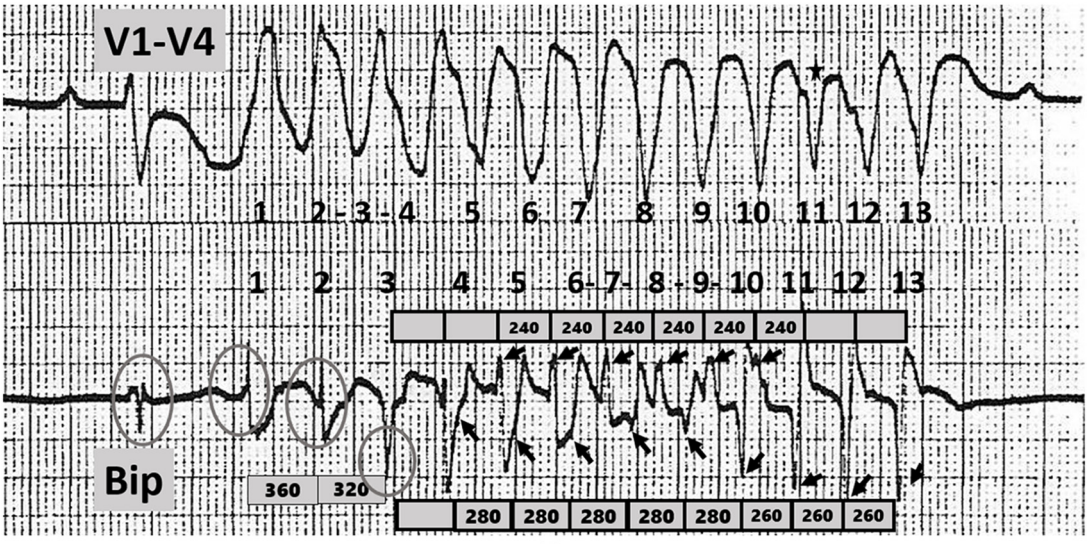

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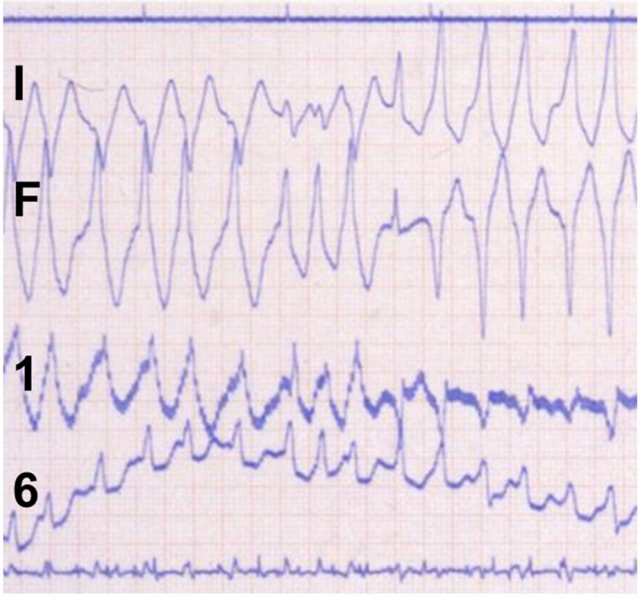

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and the first PVC was significantly shorter before the onset of TdP than before single PVCs and couplets. A morphologic crescendo of the escape T wave was observed before the onset of TdP, followed by a rhythmic and morphologic crescendo of PVCs. The escape RV apex EGMs showed the constant pattern of a rapid deflection similar to a Purkinje potential 40 to 80 ms after the onset of the QRS, superimposed on a smooth low amplitude signal.

The initiating complex

After interruption of ventricular pacing and the following occurrence of a junctional escape rhythm with severe bradycardia, there is progressive T wave prolongation and shape modification preceding the PVC or the first beat of TdP (Figs. 2, 3 and 4). This progressive modification reflects an increase in the heterogeneity of action potential durations and explains the arrhythmogenic substrate as well as the morphologic crescendo (T wave augmentation) with irregular huge T wave bumps (Figs. 1 and 3A), announcing the occurrence of TdP during AVB [11,12].

The following QRS complexes

There is still a controversial debate about the nature of those following PVC's, new phase 2 reentry from the previous PVC's, namely early afterdepolarizations (EAD), or a sizable reentry circuit. However, as action potentials progressively shorten with further shorter cardiac cycles, phase 2 reentry should stop after some of them. Perpetuation should be due to another mechanism. Conversely, the prematurity of the triggering PVC also enhances the repolarizations disparities, thus enhancing the anisotropic substrate allowing a functional reentry circuit to appear. Its moving location can explain the QRS changes, many times reproduced in experimental myocardial preparations as in full heart models, and interpreted as a rotor moving within the ventricles.

In our series some patients had sometimes only one morphology after the initiating QRS complex, but the others had at least two main upward and downward orientations (Figs. 3C, 4 and 5A) with fusion beats in between. For instance, in Fig. 5B, once the TdP initiated, our RV apex endocardial bipolar signal changed cycle after cycle, with small modifications on the ECG bipolar precordial lead. When compared with the surface QRS the EGMs appear sometimes early, sometimes late, due to fusion from at least two distinct activation zones, with early positive or negative components, according to the direction of the passing front through the bipolar apex electrode. We are therefore led to think that at least two different simultaneous ventricular activation sites are available. This was the explanation in the initial hypothesis by Dessertenne [1]. In our series the heart rate during TdP ranges up to 250–300 bpm. This speed is limited by the ventricular refractory periods, but as these progressively shorten with the cycle length the rate will accelerate. Moreover, at these rates, TdP can degenerate into VF as shown in Fig. 1 after 30 s of tachycardia.

Site of origin

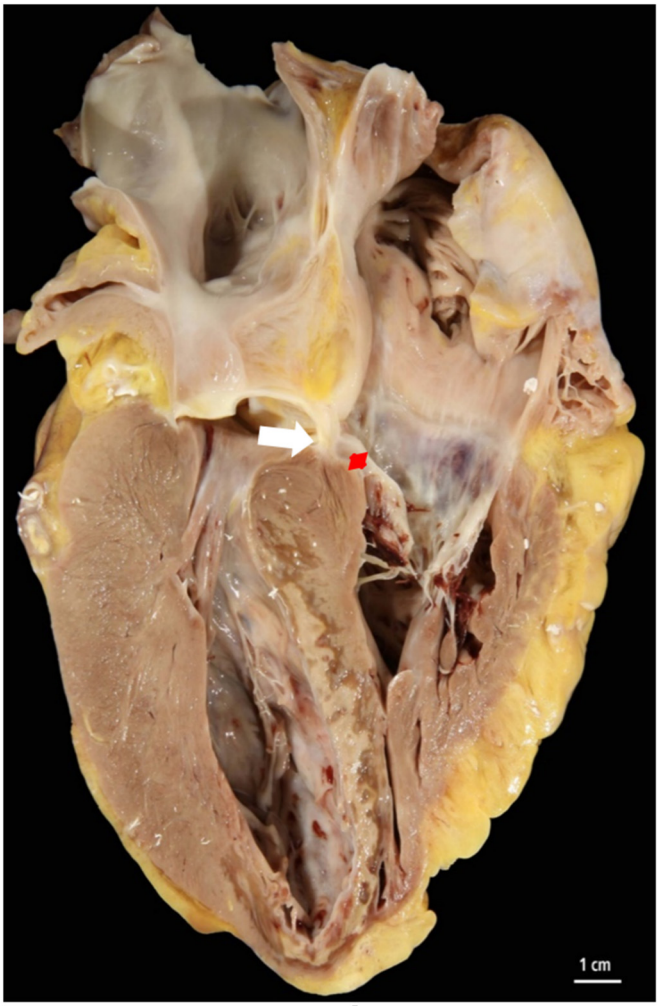
The site of origin of the initiating beat has been studied by Birati et al. [13] from surface ECG recordings in a population of 50 patients with various TdP etiologies, familial or drug-induced AVB. They found no correlation between etiology and QRS morphologies and most of the initiating complexes came from the RV. This fits with the early occurrence of the RV apical endocardial potentials in our 5 patients as shown in Fig. 2. To our knowledge, there has been no previous study investigating endocardial recordings from a catheter placed in the RV illustrating initiation of PVC-induced TdP in AVB. As the remaining 11 patients without EGM recordings had the same morphologies on their bipolar precordial ECG, we can assume that TdP also originated in the RV in those.

Proposed anatomical site for re-entry in TdP tachycardia

It is interesting to speculate that if a thin structure exists in the ventricles, apt to propagate a circular activation front as in Allessie's model [14], it should be in the right ventricle, which has a thickness of 2–3 mm as opposed to 5 to 12 mm for the left one. GHF speculated that the region of interest for TdP to propagate could be the RV crista supraventricularis. In the anatomic study of two hearts from two patients (Fig. 6A, B and C), the crista had a thickness of one to three millimeters, as Thomas James published in 1985 [15], and can get thinner during its course [16]. Therefore, to the best of our knowledge, we are the first group to propose that in the human heart, the crista supraventricularis, the thinnest part of the RV, could be the anatomical substrate for most of those TdP in high-degree AVB. The crista supraventricularis is described by T. James as a U-shaped folded structure connected to the high septal area, and with fibers diverging as a fan to the anterior RV. He stated that the crista "is crucially located to join the interventricular septum and left ventricle to much of the RV free wall" [15]. This structure can be linked to the interventricular septal Purkinje net and could transmit activation to the antero-superior and postero-inferior sulci. In that structure the genesis of the QRS changing from upward to downward activation morphologies with fusion could be (Fig. 6D) two simultaneous circuits with slightly different cycle lengths and entrance block from one another, as suggested in our Fig. 5A. Another mechanism could be one moving circuit with slight cycle length changes and dual changing exits, with Wenckebach type conduction block to the rest of the ventricles (Fig. 6D). This second hypothesis could explain the abrupt changes in two opposite directions observed at the beginning and at the end of a TdP (Fig. 5A), the two exit sites being then able to activate one after the other.

This was a retrospective study. All data in this study were obtained from patients treated in our hospital before 1970. The tape recordings and some individual patient data are no more available. Only a single precordial bipolar ECG lead, in front of the RV and the septal area, was recorded in most of the tracings. Therefore, some interesting

Fig. 5. A, Same legend as 3C: The end of TdP with longer intervals in the first line (asterisk) suggests a Wenckebach phenomenon on the last active circus movement (CM). An end of TdP without slowing is seen in the second line suggesting a Mobitz type 2 block on the last active CM (bold arrow). However, a small pacing spike before the last QRS could be subthreshold, but could also have interrupted the torsade as there is a paced ventriculogram after the end of the torsade. Abrupt change in the direction of QRS tips at the end of the third line (thin arrow) suggests a Mobitz type 2 block on only one of the two CMs. B, Simultaneous recordings (25 mm/s) of a TdP with the precordial bipolar V1-V4 ECG (upper trace and numbers) and its corresponding apex EGMs (lower trace and numbers) demonstrating two simultaneous ventricular activations moving cycle after cycle in the RV from the same patient. (Measures with a screen caliper calibrated with the ECG paper grid 1 mm = 40 ms). On the surface ECG, after an initiating positive QRS on a huge T wave, next QRS and its following one have a strange shape and seem biphasic. The next QRS's are negative, slightly irregular, with a progressive narrowing until beat 11. They then widen for the two last ones. These morphological changes suggest fusions from different activations waves, confirmed by the EGMs. The initiating apex EGM occurs at the onset of the surface QRS, and starts with a bipolar fast signal at the end of the prolonged T wave (circle), suggesting a local focal origin, followed by a remote, low voltage, 120 ms S wave. The next one starts with the early same fast signal, but the remote part of the signal changes, as the QRS shape, initiating the torsade for the next 11 beats. The fast signal is still seen within the third EGM, at the nadir of its S wave. A close analysis of next EGMs reveals two simultaneous cycle lengths, of 240 and 280 ms. It is obvious by looking at EGM 8, which has several components, as its surroundings. The first, marked by an ascending arrow under the tracing, consist of an early negative small potential synchronous to the onset of the surface QRS with an immediate positive wave. The next EGMs have also an early negative initial biphasic wave of increasing amplitude, with an identical cycle of 280 ms, and 260 ms for the last two QRS. When transposing the 280 ms intervals to prior EGMs, there are slurrings (arrows) on EGM S waves, still premature to the surface QRS, suggesting that this potential is fused with earlier EGMs end of other origin. The last component of EGM 8 is the second positive one, marked by descending arrows, and clearly seen on cycles 9 and 10, progressively fused with the first component, and may be deforming the R wave of the two last EGMs reflecting a last change of activation. This positive potential can be also recognized by reporting 240 ms on EGM 7 to 5, and may be 4 and 3. Both circuits seem to initiate simultaneously after EGM 3, as if there had been a bifurcation in the activation pathways. C, Example of dual ventricular activation by two different ventricular inputs in a case of two accessory pathways in atrial fibrillation resembling TdP.



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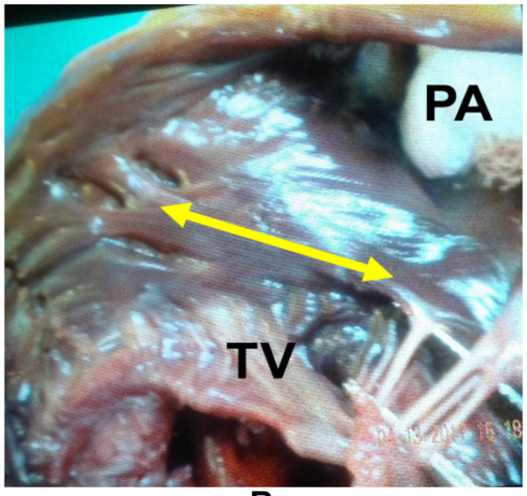

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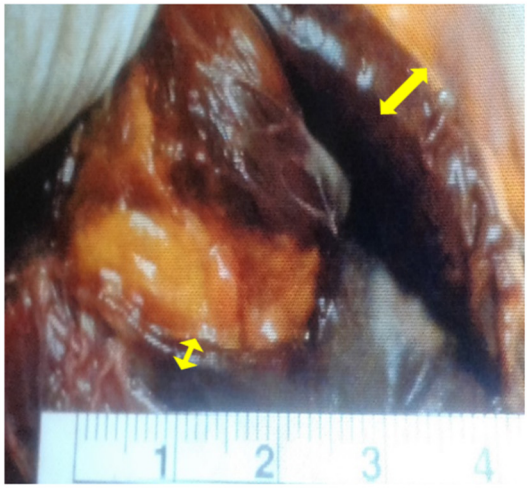

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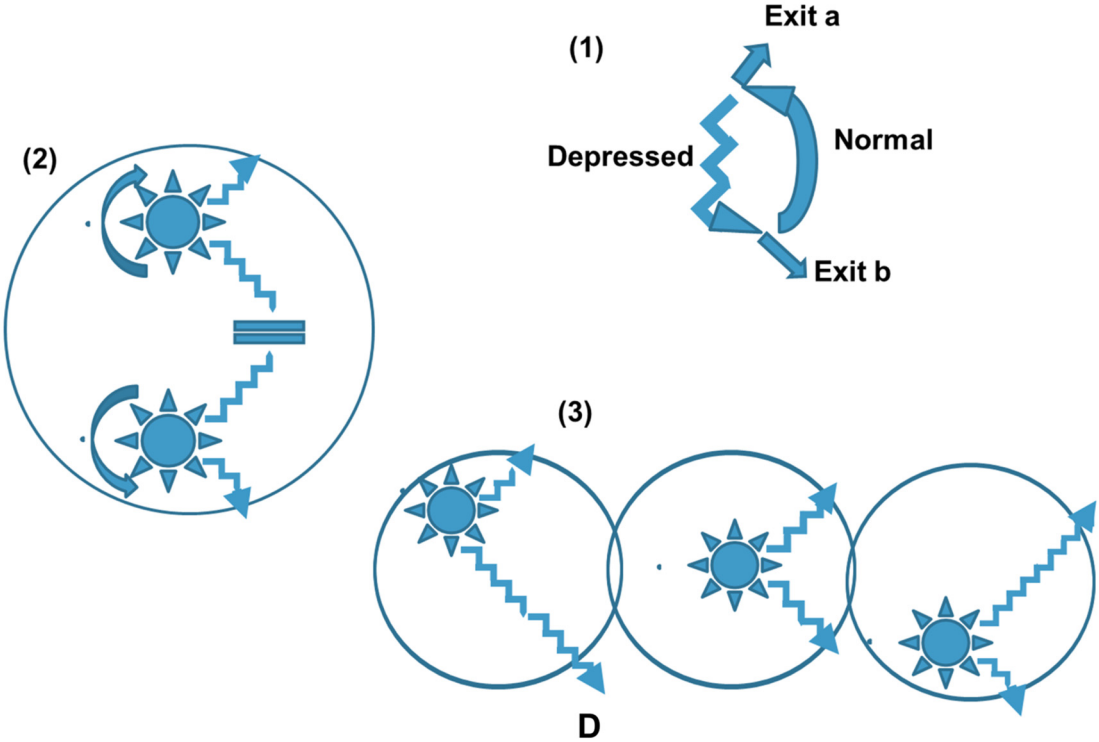

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information may have been missed and we could not show numerical data from each patient in this study. Endocardial recordings only explored the RV apex, and there is no data on other locations, as recordings from the crista supraventricularis and from the left ventricle. Therefore, new studies using 3D electroanatomic mapping with high resolution investigating the relationship of TdP initiation to Purkinje electrograms are warranted.

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Ethics

This is a retrospective study. When Guy H. Fontaine carried out these studies between 1965 and 1970, there were no ethical standards of a responsible committee on human experimentation (institutional or regional) as established 10 years later. This investigation was approved by the chief of cardiology as there was a clinical need for these patients to define in whom PM implant would be useful. An oral approved consent by the patient was obtained before the procedure.

Declaration of Competing Interest

Authors have no conflict of interest to disclose.

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Fig. 6. A, Human autopsy heart from a patient with coronary artery disease and chronic myocardial infarction showing the crista supraventricularis of the right ventricle (two-sided arrow), the thinnest structure in the human ventricles, in proximity to the His-Purkinje system. This crista may correspond to the region of adjacent fibers leading to a phase 2 reentry and initiation of TdP in patients with high degree AVB. Arrow: Crista supraventricularis. Note left septo-apical scar. B, Autopsy heart from a human without cardiac disease displaying the location of the crista supraventricularis (two-sided thin arrows). PA, white tissue in pulmonary artery; TV, tricuspid valve. Tweezers clamp under the cordae tendinae of the TV septal valve. C, The opening of the same heart along the crista displays the extreme thinness of this structure as compared to the thicker right ventricular free wall (two-sided bold arrow). Thick arrow: 3 mm for RV free wall. Thin arrow: 1 mm for Crista supraventricularis. D, Two hypotheses for the TdP inside a cardiac structure. The reentrant mechanisms occur between different fibers with different functional properties that could be defined as “Normal” or “Depressed” (1): One moving circuit and two exit points (2), or two different simultaneous circuits isolated one from the other (3). Since reentry can happen within a small area of myocardium two different circus movement tachycardia circuits can be simultaneously present, yielding different morphologies and cycle lengths as illustrated in 5B.