Atrial Premature Beats During Decrementally Conducting Antidromic Tachycardia

Running title: Sternick et al.: Role of atrial premature beats during Mahaim tachycardia

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Abstract:

Background - Advancement of ventricular activation by an atrial premature beat (APB) given during His bundle refractoriness, followed by resetting of an antidromic tachycardia (AT) in patients (pts) with decrementally conducting accessory pathway (DAP) is a helpful maneuver to prove pathway existence and participation in the circuit. We aim to assess in a large cohort the role of APB during AT in pts with a DAP.

Methods and Results - 33 pts with a DAP having 34 AT were included in the study: 29 pts had an atriofascicular pathway, 1 had a long atrioventricular (A-V) DAP and 4 had a short A-V fiber. APBs were delivered initially from the lateral RA, scanning diastole with a 10 ms decrement until AT termination or refractoriness. We observed 4 patterns of response following APB during AT: advancement of activation (29 cases), delay (2), advancement followed by delay (3), and termination (7). Eight pts required an earlier APB in order to advance or delay ventricular activation. These 8 pts had a shorter AT cycle length (median of 273 vs. 315 ms, p=0.003), and had a shorter resetting zone (median coupling interval of 30 vs. 50 ms, p<0.01).

Conclusions - APB delivered during AT in pts with a DAP advanced and/or delayed ventricular activation in all pts. In 1/5 of cases the AT was terminated by a single APB. In approximately a quarter of the patients an earlier coupled APB was needed to reset AT. The high RA was an adequate stimulation site in all right-sided DAP.

Key Words: atriofascicular pathway, decremental conduction, antidromic tachycardia, arrhythmia (mechanisms)
Introduction

Presence of a decrementally conducting accessory pathway (AP) during a pre-excited tachycardia can be suspected by measuring the A-V interval at the His bundle recording during tachycardia (1). Proof of AP participation in the tachycardia circuit can be obtained by advancing ventricular activation following a critically timed atrial premature beat delivered during AV node or His bundle refractoriness (2). The aim of our study was to appraise the role, and the appropriate stimulation site, of atrial premature beats during pre-excited tachycardia in a large cohort of patients with anterogradely decrementally conducting A-V fibers.

Population and Methods

Definitions:

Antidromic decrementally conducting circus movement tachycardia (AT): a tachycardia incorporating a slow and decrementally conducting accessory A-V pathway as antegrade limb of the tachycardia circuit.

Atriofascicular pathway: a decrementally conducting accessory A-V pathway connecting the right atrium with the distal part of the RBB. Characterized by a QRS complex width during tachycardia ≤ 140 ms, and a short V-H interval (≤30 ms), unless retrograde conduction occurs over the left bundle branch because of retrograde right bundle branch block (case 3).

Long atrioventricular pathway: a decrementally conducting accessory A-V pathway between the atrium with the ventricle with the ventricular end closer to the ventricular apex than to the annulus. It is characterized by a QRS width during tachycardia > 140 ms, and a longer V-H interval (> 30 ms).
Short decrementally conducting AV fibers: decrementally conducting accessory A-V pathways with their ventricular end close to the tricuspid or mitral ring.

Latent AP: presence of a decrementally conducting right-sided accessory A-V pathway only capable of conduction during antidromic tachycardia. Absence of “Mahaim physiology”: no ventricular preexcitation during sinus rhythm or atrial pacing at increasing rates.

Decremental conduction: a minimal increase of 30 ms in accessory pathway conduction during atrial pacing at increasing rates.

Study Population: The population consisted of 33 consecutive patients (2 with a mild asymptomatic form of Ebstein’s disease, cases 5 and 22) from 4 Institutions (Biocor Instituto, Nova Lima, Brazil, University Hospital, Maastricht, Netherlands, Herzzentrum, University of Leipzig, Germany, and Holy Family Hospital, Mumbai, India) with preexcited tachycardias associated with an antegrade decrementally conducting accessory pathways (AP).

All patients underwent electrophysiologic assessment and successful radiofrequency catheter ablation as reported elsewhere (3). Data were collected retrospectively in 10 patients and prospectively in 23 patients. The study population consisted of 33 patients (19 females) with a mean age of 25 ± 11 years, with 34 antidromic tachycardias caused by an atriofascicular pathway in 29 patients, short decrementally conducting atrioventricular pathways (4) in 4 patients (1 with a “latent” conduction) (5), and a long atrioventricular decrementally conducting AP (6) in 1 patient.

Additional arrhythmia substrates: Five patients also had inducible sustained AVNRT. No patient had bystander preexcitation during AVNRT. Case 2 also had a manifest anterogradely conducting right postero-lateral AP. After ablation of this rapidly conducting AP, the atriofascicular pathway was diagnosed.
Measurements: All measurements were done by two independent observers (EBS, LG) during pre-excited tachycardia at a paper-speed of 100 and 200 mm/sec. We used the high lateral right atrium and the proximal His bundle recording as the main leads for making interval measurements. We measured in a beat-to-beat fashion, the preceding tachycardia cycle length, the HH, and the AA interval, the coupling interval (A pacing-spike), the H1H2, and the V1V2 interval (V2, and H2 were right apical ventricular and His bundle activation following the atrial extrastimulus), V2V3 interval (the return cycle after the captured ventricular beat), the AHR-AHIS interval. The delta A-V decrement following an APB delivered during AT was the maximum atrial stimulus spike-V following an APB – baseline A-V interval. These values are shown in the Table.

Statistical Analysis: Values are given as mean ± standard deviation, and as median. The significance of differences (p < 0.05) between groups of clinical, and electrophysiologic parameters was assessed by the Kruskal Wallis test. Interobserver agreement of antidromic tachycardia cycle length was quantified (kappa statistic) using STATA statistical software package (STATA 11 Software, NC, USA). Overall interobserver agreement was defined as good if k > 0.6. We used analysis of average values between the two observers (EBS, and LG) for data analysis.

Results
In our cohort of 33 patients (57.5 % female) with a mean age of 26 ± 10 years, 34 antidromic tachycardias were assessed. Twenty-eight patients had an atriofascicular pathway, 4 a short decrementally conducting fiber, and 1 a long decrementally conducting fiber.

The cycle length of antidromic tachycardia ranged from 222 to 410 ms (mean of 304 ± 39 ms). The tachycardia reset zone coupling interval ranged from 10 to 90 ms (46 ± 18 ms). During
antidromic tachycardia the V-H interval ranged from 0 to 150 ms (20 ± 25 ms). The delta A-V interval [(A2-V2) - (A1-V1)] ranged from 30 to 150 ms (76 ± 36 ms).

In all 34 cases a critically timed atrial premature beat (APB) delivered at a time of His bundle refractoriness affected subsequent ventricular activation. We observed 4 patterns of response following an APB (Figures 1, 2, and 3): 29 of the 34 cases (85,3%) had advancement of the ventricular activation (Type I), 2 cases (5,9%) delayed ventricular activation (Type II), and 3 cases (8,8%) both advancement and delay of ventricular activation (Type III). The maximal VV delay in the 5 patients showing delay of ventricular activation was 60, 60, 80, 16, and 12 ms, respectively (Table I). In 3 of 5 patients the A-V decrement had a “jump-like” pattern (Table I, cases 9, 24, and 29). Termination (Type IV) of antidromic tachycardia by a single APB was seen in 7 cases.

Earlier atrial premature beats:

Eight patients needed a shorter coupling interval to affect ventricular activation during antidromic tachycardia (cases 27 thru 34, Table I). The atrial electrogram in those “earlier” APBs was recorded 10 to 35 ms before the His bundle electrogram (21 ± 9 ms). Those 8 patients, as compared to the remaining 25 patients who did not require “earlier” atrial premature beats, did not differ regarding gender, mean age, presence of additional arrhythmia substrates, Mahaim fiber location, maximum A-V decrement, type of response (I, II, III, and IV) to an atrial premature beat delivered during AT (p= ns). However, patients who needed an earlier atrial premature beat had a shorter AT cycle length (mean cycle length of 269 ± 28 vs. 315 ± 26 ms, p= 0,001) (median of 273 vs. 321 ms, KW= 8,33, p= 0,003), and a smaller resetting window (mean coupling interval of 35 ± 23 vs. 50 ± 15 ms, p= 0,04) (median coupling interval of 30 vs. 50 ms, KW= 5,97, p= 0,01).
Interobserver variability: Kappa = 0.628 (Std error= 0.0373, z= 16.84, Prob>|z|= 0.0000) for antidromic tachycardia cycle length.

Discussion

The major findings of this study were: 1. There are four types of response to atrial premature beats delivered during antidromic tachycardia in patients with a decrementally conducting AP (atriofascicular, long, and short atroventricular pathways); 2. Reset of tachycardia can be obtained either with advancement and delay ventricular activation; 3. Right atrial lateral wall is an adequate stimulation site in most patients.

What is the diagnostic value of delaying ventricular activation?

Advancing or delaying ventricular activation by a premature beat followed by resetting of the tachycardia cycle length proves participation of the decremental AP in the tachycardia circuit. Delaying activation in a decrementally conducting AP after a critically timed premature beat was initially reported in patients with the permanent junctional reciprocating tachycardia (9). The authors called it a “paradoxical delaying of activation”. Broadhurst et al (10) were the first to report delayed ventricular activation in a patient with a latent atriofascicular pathway. In their patient they got advancement with a longer coupling interval and delay (type III) with a shorter one. In the present study we report for the first time delayed activation as a single response (type II) after premature atrial beats during antidromic tachycardia in a patient with an atriofascicular pathway.

Does it matter where to deliver APBs?

In 1988, when Tchou et al (2) reported the role of a late atrial premature beat in differentiating atriofascicular from nodofascicular fiber in a single patient, the prevailing idea was that Mahaim fibers were nodofascicular or nodoventricular pathways, with the proximal end at the AV node,
so it made more sense to apply atrial premature beats close to the AV node, or the right atrial septum. However, we now know that most of the decrementally conducting fibers have their atrial on the lateral part of the tricuspid annulus, suggesting that is better to apply premature beats on the right atrial lateral wall, because of the proximity to the accessory pathway atrial end. Of course, if the decrementally conducting AP is para-septal or left-sided, right atrial septal or left atrial premature beats may be required to affect timing of ventricular activation during antidromic tachycardia. This we observed in a single case of a left-sided decrementally conducting AP (case 24, figure 3).

**Failure to advance ventricular activation as an argument for nodofascicular fiber**

Failure of an APB to advance ventricular activation is usually regarded as an argument favoring the presence of a nodofascicular fiber (11, 12).

In one of the largest series with atriofascicular pathways (13), ventricular activation was advanced with late right atrial premature beats in all 23 patients with antidromic tachycardia. Failure to advance ventricular activation while stimulating close to the accessory pathway proximal end was reported once (14). The patient had an atriofascicular pathway and was successfully ablated guided by an AP potential. The author’s explanation for the inability to advance ventricular activation was related to a delay in antegrade conduction over the atriofascicular pathway. An alternate possibility was that only long-coupled premature beats were delivered. In our cohort, 8 patients needed a shorter coupling interval.

**Earlier-coupled premature beats may have the same diagnostic value as late-coupled atrial premature beats?**

Considering that normal intraatrial conduction time takes 20 to 50 ms, atrial stimulation from the lateral wall affords a wider window of stimulation than stimulating from the septum, due to the
time interval that the excitation wave front takes to depolarize the atrial septal region. The fact that in all patients in whom a shorter coupling interval was needed to reset the tachycardia, the AA (at the His bundle electrogram) interval remained unaffected, stresses the diagnostic value of earlier-coupled premature beats.

Why is it that only a few patients showed types II and III response (delay of ventricular activation)?

Surprisingly only 5 patients (14.7%) showed delay of ventricular activation following critically timed APBs during antidromic CMT. As shown in figure 4, delay of activation may be explained by longitudinal dissociation in the decrementally conducting AP. Spontaneous longitudinal dissociation may cause cycle length alternans during antidromic CMT, and an incessant tachycardia due to 1:2 A-V conduction over the decrementally conducting AP (15, 16). Duality of conduction over Mahaim-type fibers because of longitudinal dissociation may also be validated by demonstration of a “jump-like” response following critically timed APBs during ACMT. Three of the 5 patients (cases 9, 24, and 28) had a “jump” like response to a 10 ms decrement in APB coupling interval (Table), an observation which is consistent with this hypothesis. Probably the low incidence of types II and III in our cohort is related to the incidence of longitudinal dissociation in this setting.

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Conflict of Interest Disclosures: None.
References:


Table 1: Clinical and Electrophysiological data

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Decremental physiology refers to the decrement in the A-V conduction that occurs with each subsequent impulse, which is a measure of the A-V decrement. The antenna V-interval following APB (AV-APB) is compared to the A-V interval (AV) measured before the APB. The difference between these two measures is referred to as the delta AV. A positive delta AV indicates that the A-V decrement is present, while a negative delta AV indicates that the A-V decrement is absent.

Abbreviations: delta AV= net A-V decrement, which is the pacing spike-V interval following APB minus the A-V interval of the preceding beat during CMT; w/= with. At the bottom of the Table, means + standard deviation and medians are displayed, respectively.
Figure Legends:

**Figure 1**: Type I response (case 29). A His bundle refractory atrial premature beat (S) with a coupling interval of 200 ms advances ventricular activation by 25 ms, and resets the short VH antidromic tachycardia. Black arrows points to atrial potentials from the HRA (A₂), and proximal His bundle electrogram (lower arrow). Note that in spite of an A_{HRA}-A_{HIS} interval of 30 ms, the A_{A_HIS} interval remains unaltered (the septal atrium was not captured by the earlier-coupled APB). Abbreviations: CS p, m, d- proximal, middle, and distal coronary sinus, HRA- high right atrium, HIS- His bundle, RB- right bundle branch. Paper speed at 150 mm/sec.

**Figure 2**: Type II response (case 1). A His bundle refractory atrial premature beat (S) with a coupling interval of 300 ms delays ventricular activation by 40 ms, and resets the short VH antidromic tachycardia. This proves participation of the decremental AP in the tachycardia circuit. Abbreviations: same as figure 1. Paper speed at 100 mm/sec.

**Figure 3**: Left panel: Type III response (case 24) in a patient with a decrementally conducting left posteroseptal A-V pathway. Note that in this patient a right atrial midseptal APB first shortens (left panel) the arrival of ventricular activation during tachycardia (coupling interval 280 ms), followed by delay when the coupling interval of the APB is shortened (right panel). Abbreviations: same as figure 1. RVA- right ventricular apex. Paper speed at 100 mm/sec.

**Figure 4**: Effect of atrial premature beats (coupling intervals) on tachycardia cycle length (TCL). V₁-V₂ interval measures the effect of the APB on the following ventricular activation, while V₂-
V₃ interval assesses the return cycle and the resetting zone. **Upper panel:** Type I response (case 6) - Baseline TCL was 330 ms. An APB with a coupling interval of 270 ms advances ventricular activation by 24 ms and resets tachycardia. Ventricular activation advancement and resetting were still observed decreasing coupling interval prematurity until 190 ms (resetting window of 80 ms). **Middle panel:** Type II response- (case 1) – Baseline TCL was 350 ms. An APB with a coupling interval of 340 ms delays ventricular activation by 10 ms and resets tachycardia (the return cycle is always shorter than the CMT cycle length). **Lower panel:** Type III response- (case 9) – Baseline TCL was 330 ms. An APB with a coupling interval of 280 ms advances ventricular activation by 20 ms and resets tachycardia. The same pattern of response was seen until a coupling interval of 240 ms. An APB with a coupling interval of 230 ms delays the following ventricular activation by 24 ms (total resetting window of 60 ms).
Atrial Premature Beats During Decrementally Conducting Antidromic Tachycardia
Eduardo Back Sternick, Yash Lokhandwala, Carl Timmermanns, Luiz Gerken, Frederico Soares, Liana Dias, Yan Huo, Gerhard Hindricks and Hein J.J. Wellens

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