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Correlation between the sudden jump-like increases of the atrio-Hisian interval induced during burst atrial pacing and during programmed atrial stimulation in patients with atrioventricular nodal reentrant tachycardia

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ABSTRACT

Purpose: To study the correlation between the sudden prolongations of the atrio-Hisian (AH) interval with ≥50 ms during burst and programmed atrial stimulation, and to define whether the AH jump during burst atrial pacing is a reliable diagnostic criterion for dual AV nodal physiology.
Methods: Retrospective data on 304 patients with preliminary ECG diagnosis of AV nodal reentrant tachycardia (AVNRT), confirmed during electrophysiological study, was analyzed for the presence of AH jump during burst and programmed atrial stimulation, and for correlation between the pacing modes for inducing the jump. Wilcoxon signed-ranks test and Spearman's bivariate correlation coefficient were applied, significant was P-value <0.05.
Results: The population was aged 48.5 ± 15.7 (12-85) years; males were 38.5%. AH jump occurred during burst atrial pacing in 81% of the patients, and during programmed stimulation – in 78%, P = 0.366. In 63.2% AH jump was induced by both pacing modes; in 17.8% – only by burst pacing; in 14.8% – only by programmed pacing; in 4.2% there was no inducible jump. There was negative correlation between both pacing modes, ρ = −0.204, P<0.001.
Conclusion: Burst and programmed atrial stimulation separately prove the presence of dual AV nodal physiology in 81 and 78% of the patients with AVNRT, respectively. There is negative correlation between the two pacing modes, allowing the combination of the two methods to prove diagnostic in 95.8% of the patients.

1. Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common form of paroxysmal supraventricular tachycardia in the adult person. The presence of dual AV nodal physiology remains at the centre of diagnostic and treatment approaches to AVNRT [1–3]. Even though two of the most commonly used methods of dual AV nodal physiology exposure and AVNRT induction – burst atrial pacing and programmed atrial stimulation, have emerged simultaneously [1,4,5], no express comparison of the two has been performed regarding their diagnostic value in proving dual AV nodal physiology. The traditional criterion for the presence of a dual AV nodal physiology is the sudden increase (jump) by ≥ 50 ms of the atrio-Hisian (AH) interval during programmed atrial stimulation with a gradual shortening of the coupling interval by 10-ms steps. Sudden increase of the AH interval by ≥ 50 ms during burst atrial pacing has not been studied extensively as a diagnostic criterion for the presence of dual AV nodal physiology and the information about the added benefit of the method is scarce and derived from small case series [4–6]. We aimed to study the correlation between the sudden prolongations of the AH interval by ≥ 50 ms during burst atrial pacing and during programmed atrial stimulation in patients with AVNRT, and to define whether the AH jump during burst atrial pacing can serve as a reliable diagnostic criterion for the presence of dual AV nodal physiology, as well as to correlate the induction of AVNRT to a pacing mode.

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2. Material and methods

2.1. Study population

The data records of 528 consecutive patients admitted between March 2012 and April 2016 with previous ECG recordings pointing to the diagnosis of AVNRT and with electrophysiological (EP) study and radiofrequency ablation performed were retrospectively collected. Of those, 224 patients had either some other type of tachycardia induced, or the pacing protocol was not completed as described below, thus prohibiting further analysis, or no tachycardia was induced at all. Included in the current study is the data on the first procedure of the remaining 304 patients in whom the preliminary diagnosis was confirmed during the EP study by induction of AVNRT with burst and/or programmed atrial pacing. Redo ablations of the patients with recurrences after the first procedure were not included in the analysis due to supposed change in the electrophysiological properties of the slow AV nodal pathway during the first ablation.

2.2. Electrophysiological study

Electrophysiological study was done in fasting non-sedated state. In 256 patients the antiarrhythmic drugs were stopped at least 48 h before the procedure. In the remaining 48 patients they were stopped less than 48 h before the study or not stopped at all. The EP study setup and the pacing protocol described below are uniformly used for study and induction of supraventricular tachycardias in our institution and were used in all study patients. Two 4-polar diagnostic catheters were inserted in the right ventricular apex and over the His bundle area and one 6-polar catheter was inserted in the coronary sinus. The signals were filtered at 30-500 Hz and recorded at a speed of at least 100 mm/s. Atrial pacing was performed using the proximal electrode pair of the coronary sinus catheter. The pacing protocol included rapid atrial pacing with bursts of 13 stimuli at a constant rate starting at a cycle length of 600 ms and with a 3-s pause introduced between the bursts. The cycle length of the subsequent bursts was repeatedly shortened by 20 ms down to the point of Wenckebach conduction block or to 240 ms. The second step was programmed atrial pacing at a drive cycle length above the sinus rate, usually 600 ms or less, and a single extrastimulus. The coupling interval of the extrastimulus was shortened at a step of 10 ms until tachycardia was induced or the effective refractory period of the AV node was reached. If tachycardia was not induced with single extrastimuli, the programmed atrial pacing was repeated with the introduction of 2 or 3 extrastimuli or with a faster drive cycle length. If tachycardia was still not inducible, hexoprenaline (isoprenaline is not available in our country) was infused intravenously and burst and programmed pacing was repeated. Then rapid and programmed ventricular pacing with the same pacing protocols was performed, except that programmed pacing was done with single extrastimuli only. After induction and confirmation of AVNRT was achieved, ablation was done by delivery of radiofrequency current by a 4-mm tipped ablation catheter positioned in the inferior to midseptal area of the tricuspid annulus in front of the coronary sinus ostium where an A to V potential amplitude ratio <1 was recorded.

The prolongation of the AH interval during the programmed atrial stimulation was measured at every pacing cycle and compared to the previous cycle for the presence of a jump of

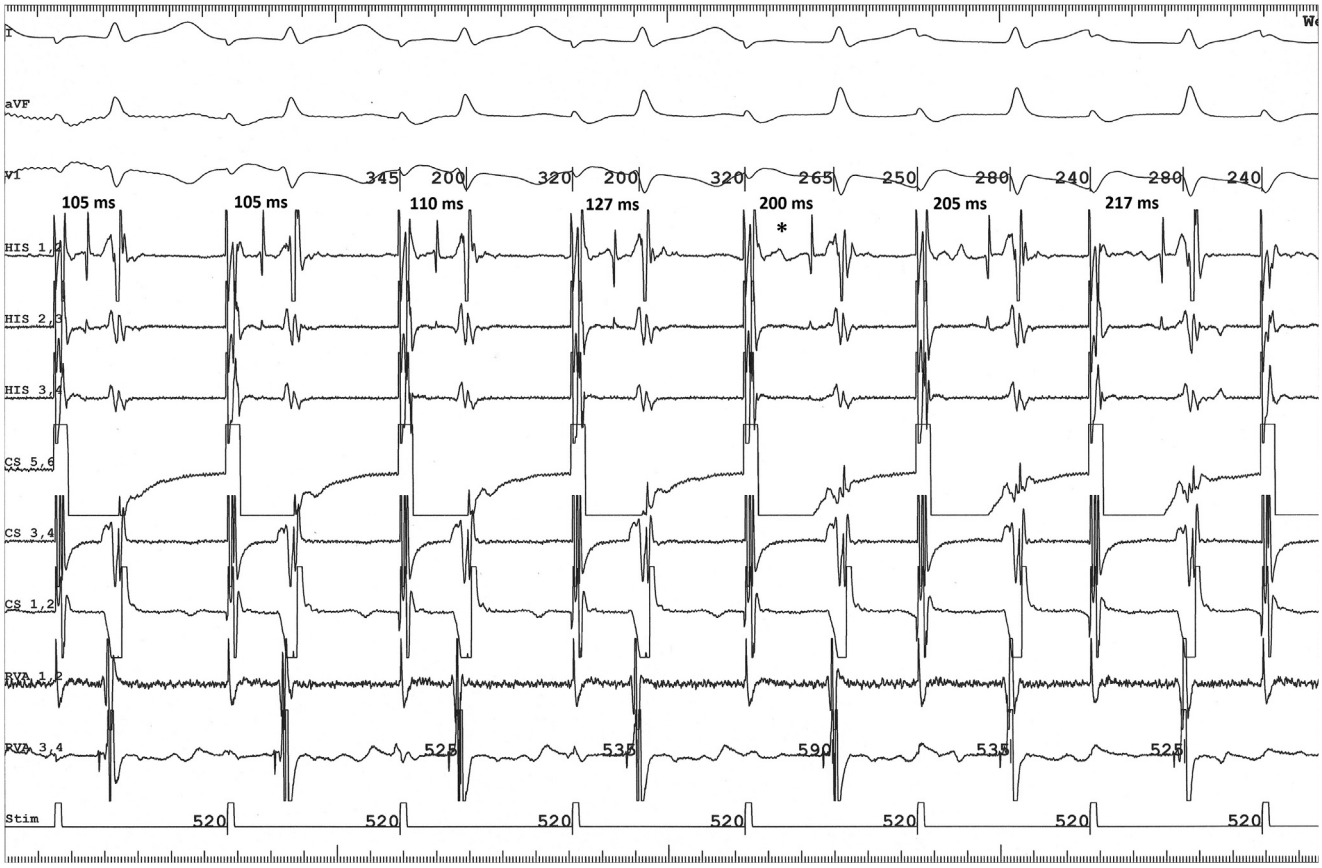

Fig. 1. Rapid atrial pacing at a drive cycle of 520 ms. Gradual increase of the AH interval is well visible. However, the fifth AH interval (marked by *) shows a sudden prolongation by 73 ms due to switch of the conduction over the slow AV nodal pathway. Subsequently further increase of the AH interval is gradual again.

≥50 ms. The prolongation of the AH interval during the burst atrial stimulation was measured within the same pacing cycle and not compared to the previous or following one, thus obviating the need for 10-ms decrements in the pacing cycle (Fig. 1). Jump in the AH interval during rapid pacing was searched for even in the pacing cycles where the Wenckebach point of the slow AV nodal pathway conduction had been reached (Fig. 2).

Analyzed was the presence of AH jump during burst and during programmed atrial stimulation, as well as the correlation between the two pacing modes for induction of sudden AH jump-like prolongation and for induction of AVNRT.

2.3. Statistical analysis

Continuous variables were expressed as a mean ± SD. The frequency rates of induction of AH jump by pacing mode used and also the burst cycle length and the coupling interval inducing the jump were compared by the Wilcoxon signed ranks test. Spearman's bivariate correlation coefficient was used for all other analyzes. P-value < 0.05 was considered statistically significant. Statistical analysis was performed using SPSS software version 19.0 for Windows.

3. Results

All 304 patients had undergone EP study using atrial and ventricular burst and programmed pacing with at least 1 extrastimulus as described in Methods.

The age of the studied population was 48.5 ± 15.7 years (12-85 y); males were 117 (38.5%). AVNRT was induced in all patients – in 110 (36.2%) by programmed pacing only, in 66 (21.7%) by burst pacing only and in 128 (42.1%) by both types of pacing. Besides AVNRT, one patient had also AV reentrant tachycardia using concealed accessory pathway induced. AH jump of ≥50 ms was induced in 246 patients (81%) during burst atrial pacing and in 237 patients (78%) during programmed atrial stimulation, P = 0.366.

The induction of AH jump by pacing mode is shown on Table 1. While programmed pacing was sufficient to induce a jump in 78% of all study patients, only burst pacing was able to induce it in additional 17.8%. Thus the two pacing modes combined diagnosed the presence of dual AV physiology in 95.8% of the patients. In the 192 patients with AH jump induced by both pacing modes, the burst cycle length at which an AH jump was first observed was longer than the coupling interval during programmed pacing, 387 ± 70 (260-600) vs 333 ± 70 (180-650) ms, P < 0.001. The correlation between the burst cycle length at the time of the AH jump (387 ± 70, 260-600 ms) and the Wenckebach cycle length (345 ± 47, 240-500 ms) was positive and significant at the 0.01 level (ρ = 0.72, P<0.001).

The induction of AVNRT by pacing mode is shown on Table 2. Programmed atrial pacing was more effective in inducing the tachycardia than burst pacing (P < 0.001). Nevertheless, AVNRT was induced by burst pacing only in 21.7% more of the patients.

After excluding the 13 patients without AH jump inducible by any mode, the correlation between the two pacing modes for induction of AH jump was found to be negative and significant at the 0.01 level (ρ = −0.204, P<0.001). Even after excluding the patients on antiarrhythmic drugs this result was reproducible, ρ = −0.211, P = 0.001 at the 0.01 level. The correlation between AH jump induced by any method and sex or age was weak and not

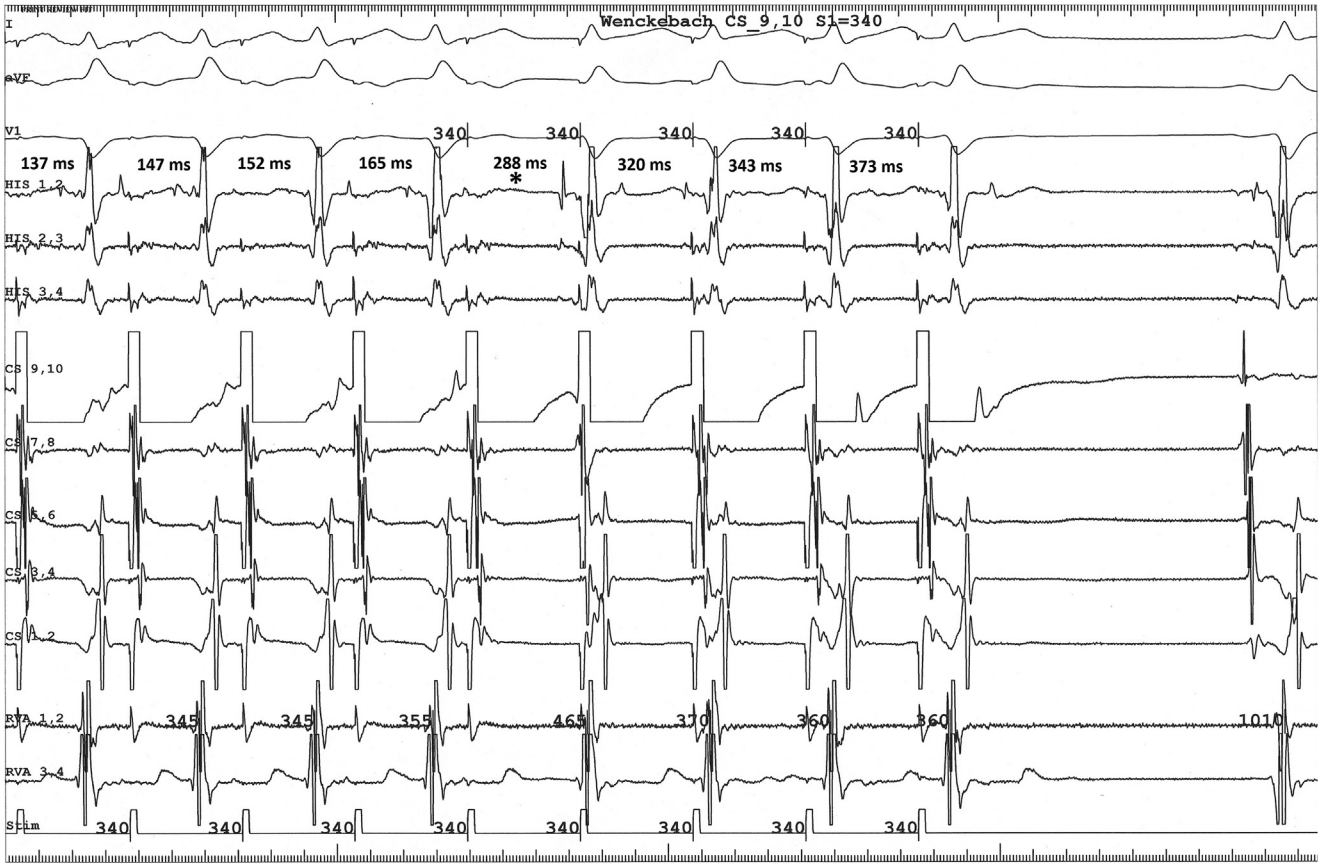

Fig. 2. Rapid atrial pacing at a drive cycle of 340 ms. The fifth AH interval (marked by *) shows a sudden prolongation by 123 ms. The next 3 stimuli are conducted with very long AH interval. The last stimulus is blocked, i.e. the Wenckebach point of the slow AV nodal pathway conduction is reached within this stimulus train.

Table 1
Induction of AH jump by pacing mode.

	Burst +	Burst −
Programmed +	192 (63.2%)	45 (14.8%)
Programmed −	54 (17.8%)	13 (4.2%)

+: AH jump induced by the respective pacing mode.
−: AH jump not induced by the respective pacing mode.
The numbers in front of the brackets depict absolute counts; the incidence is shown in the brackets in %.

Table 2
Induction of AVNRT by pacing mode.

	Burst +	Burst −
Programmed +	128 (42.1%)	110 (36.2%)
Programmed −	66 (21.7%)	0 (0%)

+: AVNRT induced by the respective pacing mode.
−: AVNRT not induced by the respective pacing mode.
The numbers in front of the brackets depict absolute counts; the incidence is shown in the brackets in %.

statistically significant (for burst atrial pacing, age and sex, respectively: $\rho = 0.006$, $P = 0.918$, $\rho = 0.051$, $P = 0.384$; for programmed atrial stimulation, age and sex, respectively: $\rho = 0.042$, $P = 0.48$, $\rho = -0.033$, $P = 0.57$). There was also negative correlation between the modes of induction of tachycardia (burst vs. programmed pacing), $\rho = -0.397$, $P < 0.001$ (significant at the 0.01 level). After excluding the patients on antiarrhythmic drugs the result was reproducible, $\rho = -0.403$, $P < 0.001$ at the 0.01 level.

4. Discussion

The main finding of this retrospective study is that the AH jump induced by burst atrial pacing is a valid criterion for the presence of dual AV nodal physiology and contributes for its correct electrophysiological diagnosis in an added number of patients.

Despite ongoing arguments of its fundamental nature, current views on AVNRT dictate that dual AV nodal physiology must be theoretically present for the tachycardia to exist, be that duality anatomic or functional [1,7]. Though extensive research on the electrophysiological characteristics of AVNRT has been conducted, and many aspects have been studied at length, the presence of dual AV nodal physiology as defined by the traditional criterion of a AH jump of at least 50 ms during programmed atrial stimulation continues to be cited at frequency rates of 53–100% [8,9]. At different times authors have suspected that such a varying degree of frequency may be due to imperfections of the standard pacing protocol of programmed atrial stimulation to demonstrate the phenomenon every time in every patient [6,9,10]. To that effect, different modes of stimulation [3,8,10] have been tested in hopes of elucidating the single perfect stimulation protocol to help achieve stable and reliable EP diagnosis of dual AV nodal physiology. It remains therefore curious that no direct comparison has been conducted between the two basic modes of atrial pacing that have emerged together and continue to be most widely used to establish dual AV nodal physiology [1,4].

The correct diagnosis of dual AV nodal physiology has practical implications as well. In some patients with previously documented paroxysmal supraventricular tachycardia the induction of the arrhythmia during EP study is impossible even with pharmacological potentiation. Nevertheless, even in those patients ablation therapy could be done if dual AV nodal physiology is present and other arrhythmogenic substrates are excluded [11,12].

In an attempt to simplify the practical application of a previously described phenomenon during rapid atrial pacing [13], where the conduction times of the atrial stimuli lengthen to a point where it seems as if two ventricular responses follow a single atrial stimulus – which intrinsically depends on the presence of dual AV nodal physiology and therefore proves it, Baker et al. [8] offer the use of a PR/RR ratio of >1 to demonstrate the presence of a dual AV nodal physiology during incremental atrial pacing. While the idea is theoretically sound, in our practice, during incremental pacing AV block usually occurs before the prolongation of conduction necessary for the ratio to go > 1 is achieved, and subsequently, we have only rarely witnessed it.

Another interesting protocol constructed by Kantharia et al. [3] suggests the use of a train of six atrial extrastimuli, delivered in a decremental ramp fashion, to induce AVNRT or other supraventricular tachycardia, instead of the usual array of atrial and ventricular rapid and programmed stimulation. While the results presented seem promising and the idea to replace several pacing protocols with a single one might be tempting, implementing the suggested protocol with some programmable stimulators would present a certain practical challenge in the EP lab. Besides, this pacing protocol is aimed at inducing any supraventricular tachycardia and would then necessitate further diagnostic maneuvers.

In our retrospective study of 304 patients the traditional pacing technique with an atrial extrastimulus accounted for a clearly demonstrable AH jump in 78% of all patients. The grand majority of the remaining patients (54 out of the remaining 67, or 17.8% more of the entire group) achieved the 50 ms prolongation of AH conduction only with the use of burst atrial pacing. The rationale behind this approach is simple. Long atrial pacing bursts up to and beyond the Wenckebach point of the fast pathway would allow the AH conduction to switch over the slow pathway and to exhibit a jump in the AH interval. In our population of patients the two modes had similar frequency of success when taken separately, regardless of the age or sex of the patients. They had, however, a negative correlation, allowing the combination of the two methods to prove diagnostic of dual AV nodal physiology in almost 96% of the studied population. A possible reason for this finding could be that during burst pacing the larger number of repeated stimuli more effectively reaches the Wenckebach point of the fast pathway in part of the patients, while with the extrastimulus technique the refractory period of the fast pathway cannot always be reached before the refractoriness of the distal common AV nodal pathway is elicited.

We have found also moderate negative correlation between the modes of induction of AVNRT. We hypothesize that in patients with AVNRT inducible by only burst pacing, the critical conduction delay in the slow pathway achieved by programmed atrial stimulation (even with two or three extrastimuli) may not be enough to allow for uninterrupted retrograde conduction over the fast pathway and for re-entry in the upper common pathway of the circuit. On the other hand, during the long atrial bursts the impulses might gradually penetrate retrogradely the fast pathway increasingly higher until the tachycardia starts.

In the opposite scenario, when AVNRT is inducible by only programmed pacing, the long atrial bursts might lead to continuous concealed antegrade invasion of the proximal part of the fast pathway blocking continuously the retrograde conduction over it; with the extrastimulus technique this obstacle may be overcome as the fast pathway is not flooded antegradely by repeated impulses.

Whatever the underlying mechanism, our results show that burst pacing is needed to exhibit AH jump and/or to induce the tachycardia in approximately one out of five patients with AVNRT.

The presence of two simple and widely employed methods to prove dual AV nodal physiology with a close to perfect degree of certainty is especially valuable in situations where demonstrating dual AV physiology remains the sole basis for performing ablation of documented but non-inducible supraventricular tachycardia – an infrequent but decidedly challenging scenario [11,12]. Indeed, the decision to ablate the slow pathway in such a scenario may be rather difficult given the fact that the procedure carries small but relevant risk of AV block, the endpoint of non-inducibility is unusable, and part of the patients remain symptomatic after the ablation. Hence, it may be justified in patients with recurrent documented episodes of antiarrhythmic drug-refractory paroxysmal supraventricular tachycardia with strong ECG suspicion for AVNRT, and at least one inducible AV nodal echo.

4.1. Limitations

Due to the retrospective nature of this study, no randomization in pacing mode sequence could be achieved. However, the functional state of the AV nodal conduction and the influence of autonomic tone on it are complex enough and simple randomization hardly would have corrected for any possible confounding. Some of the patients did not stop antiarrhythmic medication until the hospital admission, however the results were consistent even after excluding those from the analysis.

5. Conclusion

In conclusion, we present a first side-by-side comparison of the two most commonly used methods of atrial stimulation to demonstrate the presence of dual AV nodal physiology. Burst and programmed atrial stimulation when taken separately prove the presence of dual AV nodal physiology in about 80% of the patients respectively. The correlation between the two pacing modes is negative, thus allowing the combination of these to diagnose the presence of dual AV nodal physiology in 96% of the patients. The AH jump induced during burst atrial pacing has additional diagnostic value and can serve as a reliable diagnostic criterion for the presence of dual AV nodal physiology.

Conflicts of interest

None.

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