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Response to the letter 'Dual atrioventricular nodal non-reentrant tachycardia: an often overlooked diagnosis'

We thank Prof. Belhassen for his interest in our case report. The comment he provided draws attention to 1:2 atrioventricular (AV) conduction as

another, frequently unrecognized, cause of pseudotachycardia.¹ The author, indeed, suggested that the arrhythmic presentation of our patient derived from the concomitant anterograde conduction of the sinus beats over both the fast and slow AV nodal pathways with the aberrant QRS complexes occurring after the conduction over the slow pathway. Conduction of the sinus beat over both the fast and slow pathways may lead to a 'manifest' dual ventricular response, showing two QRS complexes after one P wave but can also be 'concealed', when the occult penetration



Figure 1 Twelve-lead electrocardiograms of the patient. (A and B) Junctional extrasystole conducted with left bundle branch block morphology (asterisk) and a dissociated blocked P wave (arrows). (C and D) Junctional extrasystole conducted with right bundle branch block morphology (asterisk) and a dissociated blocked P wave (arrows).

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of both fast and slow pathways leads to alternating block distal to the His bundle [not clearly detectable on the surface electrocardiogram (ECG)].² Invasive electrophysiological (EP) study results differ in the two cases. In the case of a manifest 1:2 conduction, after a sinus P wave leading to an atrial electrogram (A), two his electrograms (H), one with a normal AH interval (conduction through the fast pathway) and one with a long AH interval (conduction through the slow pathway), and two ventricular ECGs (V) with the same HV interval are recorded. In the second case, after an A, two H are recorded but only one is followed by a V while the other is blocked.² This rare modality of dual AV conduction is frequently unrecognized or misdiagnosed with other arrhythmias like atrial fibrillation, ventricular arrhythmias, or even AV block, mostly leading to unnecessary and potentially dangerous treatments. On the other hand, interpolated junctional extrasystoles may mimic double 1:2 AV conduction because both arrhythmias manifest with two QRS following one P wave, and both can cause various degrees of ventricular conduction aberrancies.3 Like Prof. Belhassen, we had also taken into consideration the 1:2 AV conduction due to a dual AV nodal pathway as the cause of our patient's arrhythmic events. However, it seemed unlikely to us that a similar type of AV conduction during sinus rhythm could only occur sporadically and with a few beats. Furthermore, after a careful revision of the ECG traces, we did not find significant diurnal or nocturnal variations of the PR intervals and we collected what we think are junctional extrasystoles with aberrant conduction (or polifocal fascicular extrasystoles) followed by dissociated and blocked P waves (Figure 1). We suggest that this phenomenon may be more likely due to a block of the AV node by a partial retrograde conduction of the extrasystole rather than to a blocked anterograde conduction of both AV nodal pathways following a previous 1:2 conduction. However, we agree with Prof. Belhassen that both interpretations are possible. An EP study with measurement of the Hisian electrogram and the HV interval would have been necessary to precisely define the nature of the arrhythmia. Differently from our case, where the patient refused the invasive EP study and the arrhythmias were very infrequent and suppressed during effort (thus not elicitable with isoproterenol infusion), other clinical settings (i.e. frequent arrhythmias, differential diagnosis with atrial fibrillation or ventricular arrhythmias to avoid overtreatment) would benefit from this invasive and potentially curative approach.

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Data availability

The data underlying this article will be shared on reasonable request to the corresponding author.

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