

Differential Diagnosis Between Dual Ventricular Response and Bigeminy Arising From the Bundle of His

Diagnóstico diferencial entre doble respuesta ventricular y extrasistolia hisiana bigeminada

To the Editor,

We have read with attention the scientific letter published by Evertz et al.¹ entitled “Dual Ventricular Response: Another Road to Supraventricular Tachycardia in Dual Atrioventricular Nodal Physiology”. As the authors clearly explain, their case concerned a form of supraventricular “pseudotachycardia” in which they established a differential diagnosis involving 2 conditions: atrial tachycardia (quickly ruled out during the electrophysiological study) and bigeminy arising from the bundle of His. The latter diagnosis is not easily differentiated. The authors ruled out premature contractions of the bundle of His due to the consistent relationship of the His and ventricular action potentials to the preceding atrial action potential. However, in the description of the electrocardiographic recording shown in Figure 1 of their letter, the authors mention certain irregularity of up to 50 ms in the PR2 interval. As they state in the text, the diagnosis of dual nodal physiology was more evident once the dual ventricular response had disappeared following ablation of the slow pathway.

Our group reported a case of frequent extrasystoles arising from the bundle of His in which, as in that described by Evertz et al., the patient had been referred to us with palpitations and supraventricular tachycardia.² Our patient showed a wider variability in the H1-H2 interval, which contributed to the presence of a greater number of beats with aberrant conduction and facilitated the differential diagnosis. Moreover, in our case, the presence of blocked P waves and “pseudoblock” of atrioventricular conduction was incompatible with the existence of dual nodal physiology.

By way of this letter, we wish to stress how difficult it is on occasion to differentiate between these two conditions during the diagnostic stage of the electrophysiological study performed prior to ablation. The diagnosis of premature beats arising from the bundle of His can be confirmed using a detailed map of the region

of this bundle. Recordings proximal and distal to the bundle of His reveal a reversal of the activation sequence of the bundle during the ectopic beat compared to the sinus beat.³ Likewise, a recording of the proximal bundle of His may show a shorter HV interval during the premature beat than during sinus rhythm. These two criteria can help to differentiate bigeminy arising from the bundle of His from dual ventricular response. Like many of the diagnostic criteria employed in electrophysiology, their presence increases the likelihood of a clinically relevant condition.

Claudio Hadid,* Darío Di Toro, Sebastián Gallino, and Carlos Labadet

Servicio de Cardiología, Hospital General de Agudos Dr. Cosme Argerich, Buenos Aires, Argentina

* Corresponding author:

E-mail address: claudio.hadid@gmail.com (C. Hadid).

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Differential Diagnosis Between Dual Ventricular Response and Bigeminy Arising From the Bundle of His. Response

Diagnóstico diferencial entre doble respuesta ventricular y extrasistolia hisiana bigeminada. Respuesta

To the Editor,

We thank the authors for their interest in our article.¹ First, we agree with them and would emphasize the difficulty of making a differential diagnosis between dual ventricular response due to dual atrioventricular nodal physiology and hisian extrasystole in bigeminy. In an attempt to differentiate between ventricular and dual response hisian bigemined extrasystoles, we referred to the article published by Massumi et al.,² where it is stated that—in the case of junctional parasystole—the interval between the junctional extrasystoles tend to be quite fixed and 2 variables namely PR1 and PR2 are seen. We observed slightly variable PR2 intervals as shown in Figure 1; also, the R2R2 intervals were not fixed. However, this does not completely rule out the diagnosis of junctional parasystole, which concurs with the statements in Massumi et al.² Other electrocardiographs of the patient

(not published) showed a similar phenomenon of 2 QRS complexes following 1 P wave. The sinus rates on these electrocardiographs differed from 60 bpm to 73 bpm with an exact doubling of the ventricular rate. This makes junctional parasystole less likely, unless the extrasystoles are triggered by normal conduction. Another electrocardiograph showed a PR2 lengthening until a P wave was followed by only 1 QRS complex, suggesting a type 1 pattern, second degree, atrioventricular block in the slow pathway. This could not be confirmed during the electrophysiology study. Although we did not map the ‘his’ region in detail, we retrospectively reviewed the activation pattern of the ‘his’ bundle. The ‘his’ potential appeared slightly earlier at the proximal bipolar electrode as compared to the distal electrode, both for AH1 and AH2. As stated by the authors and as published by Eizmendi et al.,³ a reversal in the activation pattern would have been expected with junctional extrasystoles.

In conclusion, we agree with the authors that the differentiation between dual ventricular response and junctional parasystole is difficult and that the criteria published to date only increase or decrease this probability. In our case, most of the published criteria pointed in the direction of a dual ventricular response. However, the final diagnosis was based on the

successful termination of the arrhythmia by ablating the posterior aspect of the Koch's triangle; in other words, by ablation of the slow pathway.

Reinder Evertz* and Lluís Mont

*Sección de Arritmias, Departamento de Cardiología,
Instituto del Tórax, Hospital Clínic, Barcelona, Spain*

* Corresponding author:

E-mail address: reinderevertz@gmail.com (R. Evertz).

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