no pacing configuration was able to prevent PNS at that time. A second operation was performed to remove the bipolar LV leads, and a quadripolar lead was implanted in the same bundle branch after confirmation that no other branches were suitable. The lead position for LV pacing was similar to the previous position (Figure 2C), but allowed more options for pacing configuration. Pacing by electrodes 2 and 3, which are very close to each other, had a threshold of 0.5 V at 0.4 ms and no PNS at 10 V and 0.5 ms. Eight months later, the patient had experienced no further symptoms of PNS and maintained good response to CRT.

Due to the close anatomic relationship between the phrenic nerve and LV, PNS is a common problem that limits CRT.[1] In 80% of patients with PNS, it occurs close to the lateral and posterior branches of the coronary sinus[2] and, therefore, often appears at the anatomic site considered optimal for resynchronization. Up to 35% of patients display it during implantation,[3] which often makes it necessary to switch the lead sites to suboptimal positions for CRT. Around 15% of patients experience PNS during follow-up, but it usually appears in the first few weeks and rarely de novo after 6 months postimplantation.[3] Pacing tests with high voltage outputs during implantation make it possible to avoid sites that produce PNS, even though this maneuver can only be used with the patient in the supine position. This explains the appearance of PNS shortly after implantation, when the patient adopted different postures in her daily life, even in the absence of lead dislodgement or microdislodgement, which is usually accompanied by an increased LV threshold.

In our patient, it is difficult to explain late-onset PNS in view of the stable radiologic position and lack of changes in electrical parameters. It appears that strong reverse remodeling with smaller LV and morphologic changes led to a progressive change in the anatomic relationships between the cardiac veins and the phrenic nerve, which would explain the late onset and progression of PNS until making it inevitable with programming changes. Implantation of a quadripolar lead (even in the same vein) with the electrodes very close together, made it possible to maintain CRT and avoid PNS.

The phenomenon of PNS due to CRT-induced reverse remodeling has not been described to date and could explain some cases of late onset during follow-up in responders. This association should be confirmed in future studies.

SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found in the online version available at doi:10.1016/j.rec.2016.04.035.

Javier Molina-Martín de Nicolás, María López-Gil,* Adolfo Fontenla, Rafael Salguero, Belén Díaz-Antón, and Fernando Arribas

Servicio de Cardiología, Hospital Universitario 12 de Octubre, Madrid, Spain

*Corresponding author:
E-mail address: mlopezgil@secardiologia.es (M. López-Gil).

Available online 12 July 2016

REFERENCES

1. Biffi M, Moschini C, Bertini M, Saporito D, Ziacchi M, Diemberger I, et al. Phrenic stimulation: a challenge for cardiac resynchronization therapy. Circ Arrhythm Electrophysiol. 2009;2:402–10.
2. Spencer JH, Goff RP, Iaizzo PA. Left phrenic nerve anatomy relative to the coronary venous system: Implications for phrenic nerve stimulation during cardiac resynchronization therapy. Clin Anat. 2015;28:621–6.
3. Biffi M, Exner DV, Crossley GH, Ramza B, Coutu B, Tomassoni G, et al. Occurrence of phrenic nerve stimulation in cardiac resynchronization therapy patients: the role of left ventricular lead type and placement site. Europace. 2013;15:77–82.

http://dx.doi.org/10.1016/j.rec.2016.04.035

Pseudobradycardia-dependent Left Anterior Fascicular Block. A Case Report

Bloqueo fascicular anterior izquierdo seudobradicardia dependiente. Presentación de un caso

To the Editor,

The term aberrant conduction refers to transient branch block not due to previous QRS abnormalities, accessory pathway conduction, or unwanted drug effects.[1] The block can occur at any level of the His-Purkinje system and may be due to different mechanisms. Phase 3 block (tachycardia-dependent) is due to invasion of tissue during the effective refractory period and can be a physiological or pathological phenomenon. A special form of this block is acceleration-dependent block, which is due to changes in the heart rate. Phase 4 block (bradycardia-dependent or pause-dependent) is almost always pathological. It occurs after the end of the refractory period due to decreased membrane potential, because of increased His-Purkinje automaticity or partial depolarization of the myocardial lesion. The fourth and last aberrant mechanism is due to hidden conduction, which is defined as the propagation of an impulse within the specific conduction system and can only be recognized by its effect on the impulse, the interval, or the following cycles.[2] As indicated by its name, this phenomenon cannot be observed on surface electrocardiogram (ECG).

We present the case of an 86-year-old woman who was admitted to the emergency department for palpitations and dyspnea. Some years before, she had been assessed by a cardiologist for asymptomatic sinus bradycardia, for which she was not receiving treatment. Physical examination revealed irregular low-intensity heart sounds without murmurs and bibasal crackles with no other findings of interest. On admission, ECG showed atrial fibrillation with a ventricular response of around 100 bpm, with left anterior fascicular block (LAFB), alternating with beats with a narrower QRS complex (Figure 1A and Figure in the supplementary material). During her stay in the emergency department, the patient was administered 2.5 mg atenolol intravenously and achieved sinus rhythm at a rate of 39 bpm, with normalization of QRS morphology (Figure 1B and Figure in the supplementary material). The patient was discharged without antiarrhythmic medication. At 3 weeks, she was admitted with marked asthenia and documented sinus bradycardia at 35 bpm, for which she received a DDD pacemaker.

Careful analysis of the ECG obtained during the episode clearly showed 2 types of QRS: *a)* QRS with LAFB morphology (120 ms), alternating with *b)* narrow QRS with small variations in axis and

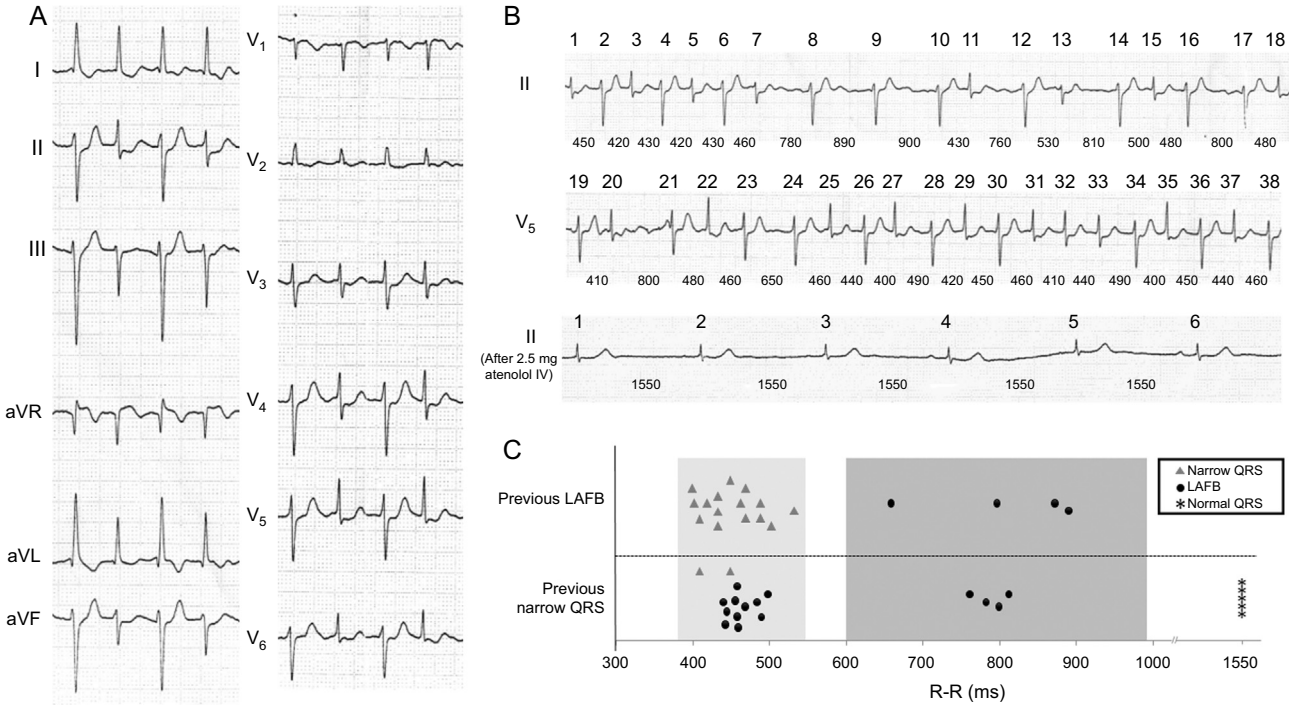

Figure 1. A: 12-lead electrocardiograph. B: Complete ECG tracing at admission. Beats 1–38 are consecutive despite being in 2 different leads (II and V_5). Cycle lengths between beats are shown in milliseconds. The lower panel shows the QRS morphology after intravenous administration of 2.5 mg atenolol and conversion to sinus rhythm. C: The relationship of the QRS morphology with the cycle length and the morphology of the preceding beat. LAFB, left anterior fascicular block.

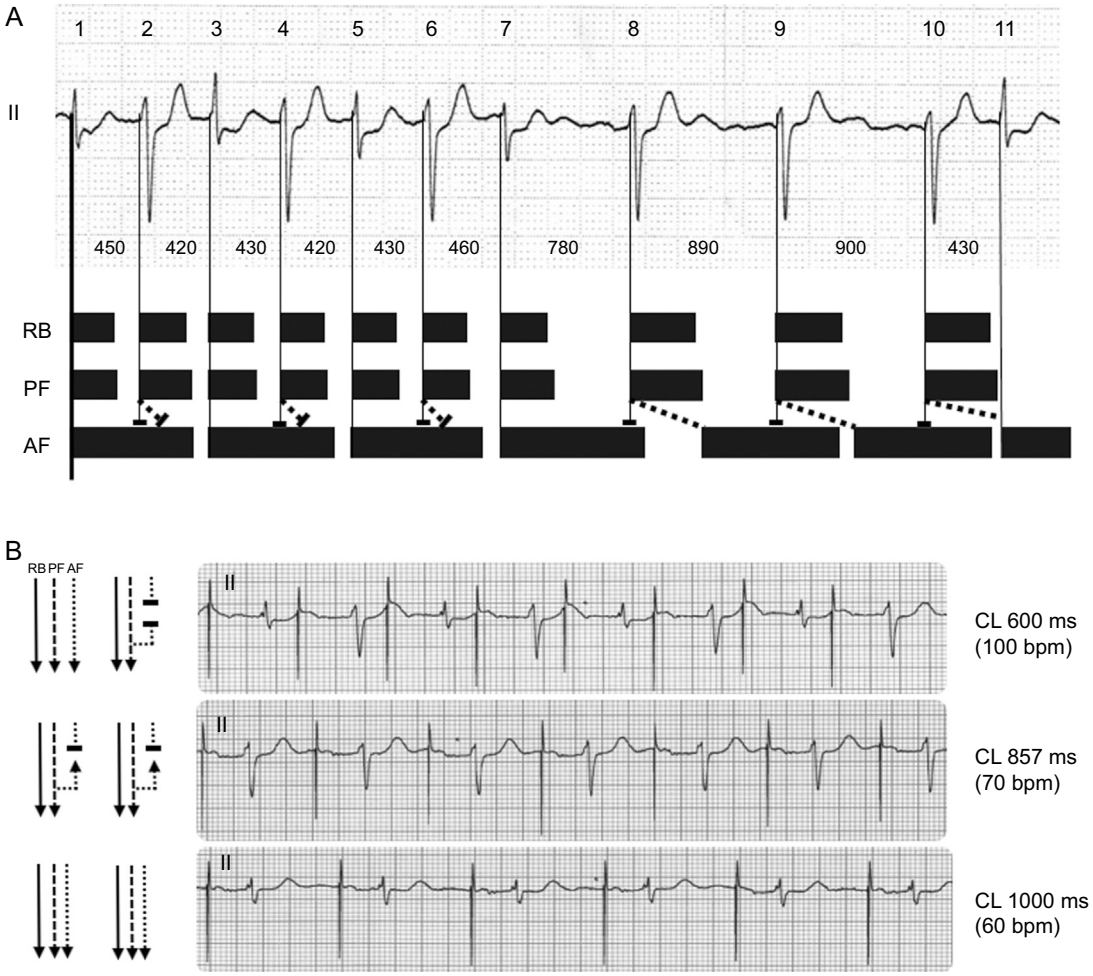

Figure 2. A. Proposed mechanism of the electrocardiographic findings. Black bars represent the theoretical effective refractory periods, and oblique dotted lines represent hidden retrograde interfascicular conduction. B. Confirmation of the mechanism with atrial pacing (AAI). AF, anterior fascicle; CL, cycle length; PF, posterior fascicle; RB, right branch.

duration (90–100 ms). Furthermore, at longer R–R intervals, beats always had LAFB morphology. Two areas can be clearly differentiated in Figure 1C, which compares the morphology of the QRS with the cycle length and the previous QRS complex.[3] In zone 1 (R–R, 400–530 ms), the QRS morphology depends on the previous beat, (ie, if the previous beat is narrow, the following beat will have LAFB morphology). The only exceptions to this rule are beats 32–33, which could be explained by the penetration of the impulse in the supernormal conduction phase of the anterior fascicle.[3,4] However, in zone 2 (R–R >600 ms), the QRS complex always has LAFB morphology independently of the morphology of the previous beat, which is suggestive of bradycardia-dependent block. The curious aspect of this case is that, in contrast to what would be expected in this type of block, after a much longer R-R interval(>1500 ms), the QRS becomes normal.

Figure 2 shows the proposed mechanism for these findings. With short R–R intervals (zone 1), an anterograde and retrograde block occurs in the anterior fascicle, which makes the following impulse able to conduct anterogradely since it has time to repolarize. In this way, the small variations in narrow QRS complexes could be explained by their occurring at different moments in their relative refractory period, with a higher or lower degree of latency (eg, beats 3 and 7, or 13 and 15). With very long cycle lengths, tissue recovery and permanent anterograde conduction take place. Cohen et al[5] described this phenomenon at the end of the 1970s and called it pseudobradycardia-dependent branch block alternans (ie, a phase 3 block). For this to occur, the retrograde effective refractory period of the anterior fascicle should be less than the anterograde effective refractory period and thus favor hidden retrograde conduction.[4]

We were able to confirm this mechanism (Figure 2B) because our patient had been implanted with a DDD pacemaker. Alternating LAFB was produced by AAI pacing at 100 bpm, at 70 bpm all beats were conducted with LAFB morphology, and at 60 bpm all beats were narrow, which confirmed tachycardia-dependent block.

SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found in the online version available at doi:10.1016/j.rec.2016.04.036.

Bruno Bochard-Villanueva,[a,b,*] Aurelio Quesada-Dorador,[a,c] Víctor Palanca-Gil,[a] Javier Jiménez-Bello,[a] Natalia Chacón-Hernández,[d] and Salvador Morell-Cabedo[d]

[a]*Unidad de Arritmias, Servicio de Cardiología, Hospital General Universitario de Valencia, Valencia, Spain*
[b]*Fundación para la Investigación, Hospital General Universitario de Valencia, Valencia, Spain*
[c]*Universidad Católica de Valencia San Vicente Mártir, Valencia, Spain*
[d]*Servicio de Cardiología, Hospital General Universitario de Valencia, Valencia, Spain*

* Corresponding author:
E-mail address: bbochard@gmail.com (B. Bochard-Villanueva).

Available online 4 July 2016

REFERENCES

1. Issa Z, Miller J, Zipes D. Clinical arrhythmology and electrophysiology: a companion to Braunwald's heart disease. 1st ed. Philadelphia: Saunders-Elsevier; 2009. p. 143.
2. Josephson M. Josephson's clinical cardiac electrophysiology: techniques and interpretations. 5th ed. Philadelphia: Wolters Kluwer; 2016. p. 125.
3. Carbone V, Marafioti V, Oreto G. Changes in QRS morphology during atrial fibrillation: what is the mechanism? Heart Rhythm. 2014;11:901–3.
4. Ortega-Carnicer J, Blanco J, Ceres F. Pseudobradycardia-dependent bundle branch block alternans. J Electrocardiol. 2003;36:273–5.
5. Cohen HC, D'Cruz I, Arbel ER, Langendorf R, Pick A. Tachycardia and bradycardia-dependent bundle branch block alternans. Clinical observations Circulation. 1977;55:242–6.

http://dx.doi.org/10.1016/j.rec.2016.04.036

Clinical Profile of Arrhythmogenic Right Ventricular Cardiomyopathy With Left Ventricular Involvement

Perfil clínico de la miocardiopatía arritmogénica del ventrículo derecho con afección asociada del izquierdo

To the Editor,

The clinical profile of arrhythmogenic cardiomyopathy (ACM) with left ventricular (LV) involvement appears to be distinct from ACM isolated to the right ventricle (RV)[1,2], with a higher incidence of ventricular arrhythmias and sudden cardiac death (SCD) shown in some studies.[3] However, in Spain, there are few published series correlating LV involvement with an increased risk of arrhythmia and SCD.

Our aim was to analyze the differentiating clinical and morphological characteristics of biventricular ACM compared with ACM isolated to the RV in our series. This was a cross-sectional study that included 30 patients with ACM from 20 families; 17 were probands (56.7%) and 13 were relatives. All met the Task Force criteria for ACM. Participants were divided into 2 groups according to whether they had involvement of the RV alone or biventricular involvement (the LV was considered involved when the ejection fraction on echocardiography was < 50%).

Data were collected on age and sex, as well as clinical information on functional class, syncope, ventricular arrhythmias, atrial fibrillation, implantable cardioverter-defibrillator (ICD) shocks, heart transplantation, and death due to end-stage heart failure. All participants were studied with 12-lead ECG, echocardiography, and where possible, cardiac magnetic resonance (MR). Genetic study was performed in 27 patients (90%).

The statistical analysis was performed by comparing these variables in relation to the presence of isolated RV involvement or biventricular involvement, and nonparametric tests (Mann-Whitney *U* test) were used for the study of the mean.

Biventricular involvement was predominant in the probands and relatives. Of a total of 55 relatives studied, 18.2% received a new diagnosis of ACM. Follow-up time from diagnosis was similar in both groups (Table 1). In addition, in the group with biventricular involvement, 89% of the patients already had LV involvement at the time of diagnosis. No significant differences were found in the presence of epsilon waves and bundle branch block on ECG, but there was a trend ($P = .07$) toward an increased presence of inverted T waves in the precordial leads in the group with biventricular involvement.

Table 1 shows the clinical characteristics of the groups with isolated RV involvement and with biventricular involvement. The functional class was clearly more advanced in the biventricular group. Although the presence of syncope and ventricular arrhythmias was similar, the burden of family history of SCD