

Table 2
Technical Aspects of the Procedure

Lesions with previous failed PCI, n (%)	16 (52)
Arterial access, n (%)	
Radial	11 (35.5)
Femoral	1 (3.2)
Two arterial sites	19 (61.3)
Type of laser catheter by diameter, n (%)	
0.9 mm	27 (87.1)
1.4 mm	2 (6.5)
1.7 mm	1 (3.7)
No catheter (failure to pass guidewire)	1 (3.7)
Mean laser fluence, mJ/mm ²	66.43 ± 7.76
Mean laser frequency, Hz	67.16 ± 8.77
Mean number of pulses	3.9 ± 1.47
Laser success at first attempt, n (%)	29 (93.5)
Combined use of other atherectomy therapies, n (%)	
Rotablation	0
Cutting balloon	0
Scoreflex	3 (9.7)
Mean contrast used, cc	211.74 ± 87.33
Mean fluoroscopy time, min	33.69 ± 14.31
Guide catheter used, n (%)	
XB or EBU 3.5	5 (16.1)
XB or EBU 4	14 (45.2)
Amplatz	10 (32.2)
JR	2 (6.5)
Mean number of guidewires used	2.10 ± 1.04
Microcatheter use, n (%)	
Finewire	13 (41.9)
Corsair	5 (16.1)
Other	3 (9.7)
Not used	10 (32.26)
Mean number of stents placed	2.22 ± 1.20
Percentage DES, %	100
Final length treated, mm	42.40 ± 26.05

DES, drug-eluting stent; PCI, percutaneous coronary intervention.

The results presented here allow us to recommend ELCA as a safe adjuvant therapy in complex PCI, when there is failure to cross or dilate the lesion as it significantly increases the success rate of the procedure.

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Late Phrenic Nerve Stimulation in a Super-responder to Cardiac Resynchronization Therapy. The Toll of Success?



Estimulación frénica de aparición tardía en paciente superrespondedor a la terapia de resincronización. ¿El precio del éxito?

To the Editor,

We report an unusual case of late-onset phrenic nerve stimulation (PNS) in a super-responder to cardiac resynchronization therapy (CRT).

The patient was a 58-year-old woman with a history of hypertension and bronchial hyperreactivity. Four years earlier, she had been diagnosed with nonischemic dilated cardiomyopathy and left bundle-branch block (Figure 1A). She remained stable while receiving optimal medical treatment, with 40% left ventricular ejection fraction (LVEF) in New York Heart Association (NYHA) functional class II until her status worsened to NYHA III. An echocardiogram (video 1 of the supplementary material) revealed

a spherical left ventricle (LV) with pronounced asynchrony, end-systolic volume of 128 mL, and LVEF (by Simpson method) of 26%. Consequently, a CRT device with defibrillator was implanted in June 2012. At that time, a bipolar lead was placed in a position with a long electrical delay (LV QRS interval of 180 ms). The LV capture threshold in bipolar pacing was 0.75 V at 0.4 ms, whereas impedance pacing was 460 Ω and R wave was 7 mV, without PNS (10 V output at 0.5 ms). Electrocardiogram showed simultaneous biventricular pacing, atrioventricular interval of 130 ms, and bipolar LV pacing with a QRS complex of 120 ms (baseline, 188 ms) and evident fusion between biventricular pacing and native conduction through the right branches (Figure 1B). Figure 2A shows the lead position on radiography in a posterolateral branch of the coronary sinus.

The patient's clinical and echocardiographic progress was excellent. At 9 months postimplantation, the patient was in NYHA I and the echocardiogram showed disappearance of the spherical LV shape, noticeably decreased volumes (end-systolic volume, 32 mL), and normal LVEF (59%) (video 2 of the supplementary material). However, shortly thereafter, she consulted for PNS in

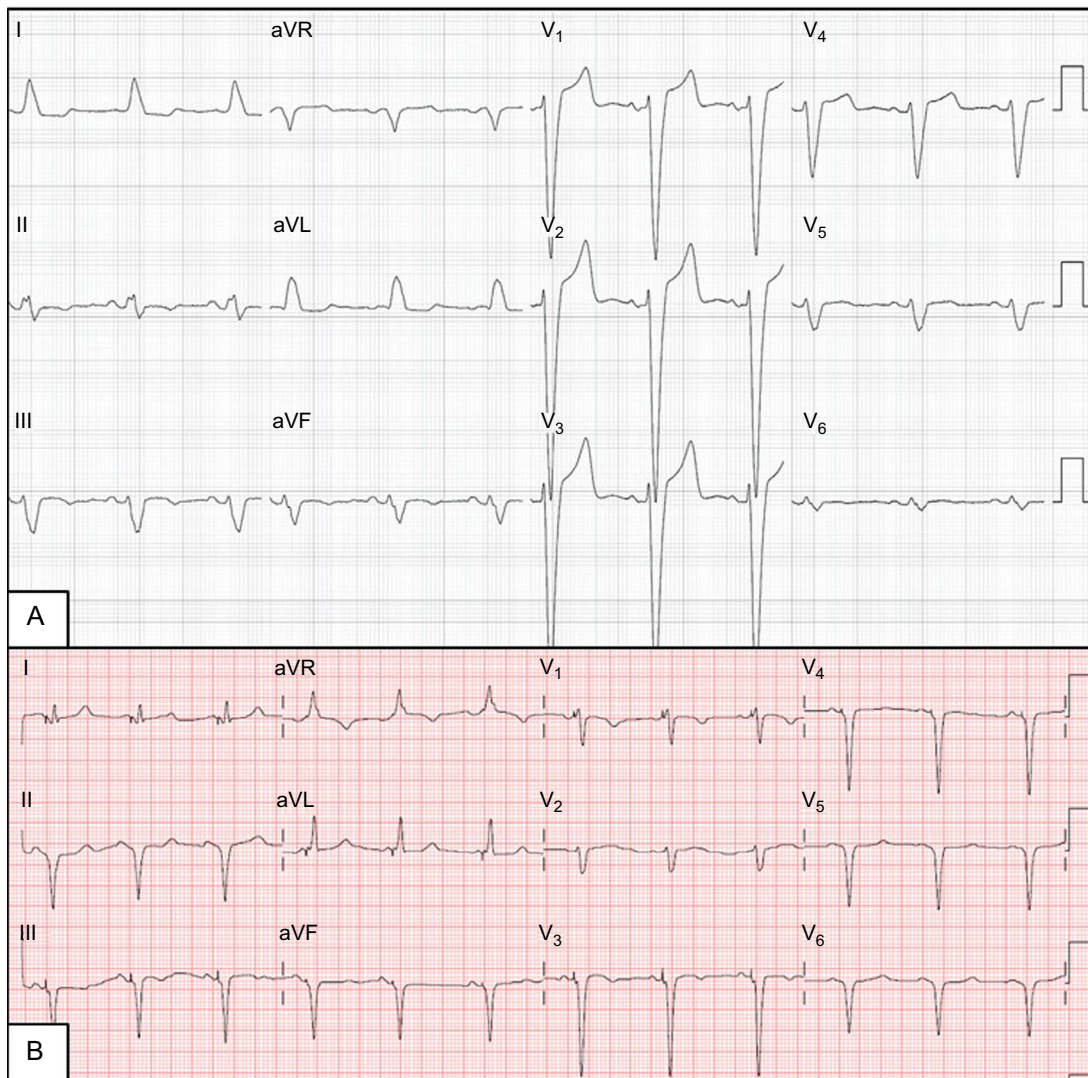


Figure 1. A: Baseline electrocardiogram showing complete left bundle-branch block. B: Electrocardiogram with biventricular pacing with signs of fusion between biventricular pacing and right bundle-branch activation, with narrow QRS.

certain postures. The LV pacing threshold was still 0.75 V at 0.4 ms, there were no changes to the impedance or R wave detected, and LV lead dislodgement was ruled out by chest radiography (Figure 2B). The pacing configuration was changed from LV to

pseudo-bipolar (proximal annulus/right ventricle), which led to disappearance of PNS.

Two years later, the patient consulted again for PNS. The radiologic and electric stability of the LV leads was rechecked, but

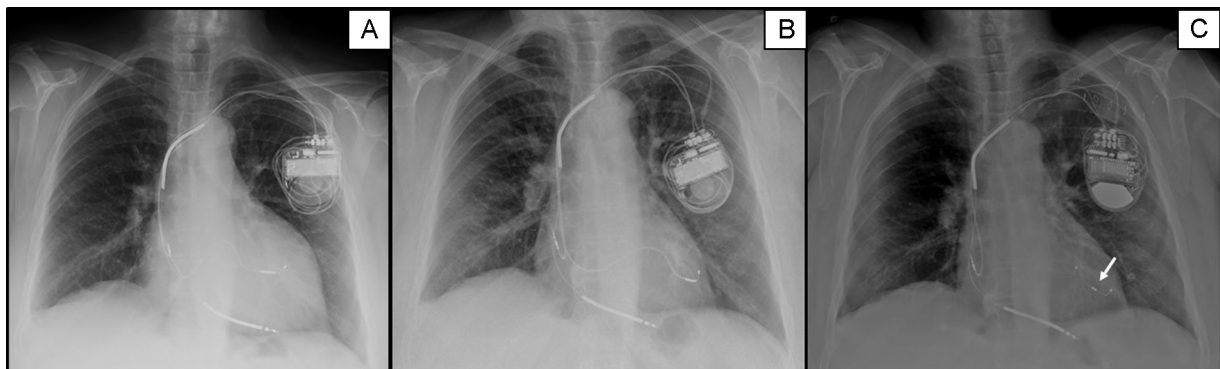


Figure 2. A: Posteroanterior chest radiography immediately after implantation of the defibrillator with resynchronization therapy. B: Chest radiography 9 months after implantation, when phrenic nerve stimulation appeared; the distance is clearly shorter between the distal pole of the left ventricle lead (A) and the edge of the cardiac silhouette; C: Chest radiography after implantation of the new quadripolar lead in the same vein; proximity between leads 2 and 3 (arrow) minimizes phrenic nerve stimulation.

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 sinus² 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](https://doi.org/10.1016/j.rec.2016.04.035).

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Pseudobradycardia-dependent Left Anterior Fascicular Block. A Case Report



Bloqueo fascicular anterior izquierdo pseudobradicardia 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