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Echocardiographic and Electrical Reverse Remodeling in Cardiac Resynchronization Therapy

Remodelado inverso ecocardiográfico y eléctrico en terapia de resincronización cardiaca

To the Editor,

Cardiac resynchronization therapy (CRT) has been shown to be an effective, economically viable tool[1] in patients with severe acute heart failure and intraventricular conduction disorders.

Recent publications suggest the presence of electrical remodeling in patients with smaller ventricular volumes after CRT.[2] We undertook a pilot study to determine the potential relationship between ventricular and electrical remodeling.

The study included 20 patients with idiopathic dilated cardiomyopathy and an indication for CRT, in whom the QRS duration and ventricular volumes were measured before implantation and after 6 months. The study excluded patients with atrial fibrillation or previous pacing by a pacemaker, as well as when the patients' own rhythm precluded measurement of the native or intrinsic QRS width. The study complied with all principles of the Declaration of Helsinki.

Left ventricular reverse remodeling was defined as a decrease ≥10% in end-systolic volume at 6 months, and electrical remodeling as a decrease in intrinsic (unpaced) QRS width.

Of the 20 patients included (age, 61 [10] years; 40% women), 15 (75%) had echocardiographic evidence of reverse remodeling. These patients showed a significant decrease in intrinsic or unpaced QRS on follow-up (169 [15] vs 154 [12] ms; P=.032) compared to the others (180 [23] vs 180 [16] ms; P=.977), in addition to a significant decrease in left ventricular end-diastolic volume (P<.01) and an improvement in ejection fraction (P=.02). Both groups showed similar clinical and echocardiographic profiles at baseline and similar device programming characteristics, but the patients who presented a decrease in QRS on follow-up were characterized by a shorter paced QRS achieved with CRT implantation (121 [15] vs 146 [24] ms; P=.021) (Table).

The main finding of this pilot study is a significant reduction in the intrinsic or unpaced QRS width in patients who present a decrease in ventricular volumes on follow-up, which is consistent with the findings of recent publications that report an improvement in intraventricular conduction among patients who present reverse remodeling.[2] This finding differed from the results reported by Stockburger et al.,[3] who found no such relationship, but the series was much smaller and included patients with ventricular dysfunction of various etiologies, unlike this study which specifically analyzed patients with idiopathic dilated cardiomyopathy, a characteristic that could explain the different results obtained. The presence of mitral regurgitation has also been associated with the appearance of intraventricular conduction disorders.[4] In our study we observed a decrease which was not statistically significant (probably due to sample size) but could partly be due to an improvement in electrical conduction after CRT. Another noteworthy finding is the possible relationship between the presence of ventricular and electrical reverse remodeling with the duration of the QRS complex achieved with implantation. It has been extensively reported in the literature that patients with wider

Table
Comparative Analysis of Clinical, Electrical, and Echocardiographic Variables According to Presence or Absence of Left Ventricular Reverse Remodeling at the 6-Month Follow-up

	LV reverse remodeling (n=15)		Absence of reverse remodeling (n=5)	
	Baseline	Follow-up	Baseline	Follow-up
Age, years	61 (11)	—	62 (8)	—
Women, %	47	—	20	—
Ejection fraction, %	21 (7)	39 (8)[a]	25 (4)	24 (13)[b]
LVEDD, mL	190 (83)	130 (57)[a]	264 (62)	284 (73)[b]
LVESD, mL	146 (65)	81 (35)[a]	191 (57)	215 (82)[b]
ERO, cm^2	0.15 (0.13)	0.06 (0.02)	0.27 (0.16)	0.13 (0.20)
Intrinsic QRS, ms	163 (15)	153 (31)[a]	180 (23)	182 (15)[b]
Paced QRS	121 (15)	122 (17)	146 (15)[c]	140 (14)
AV, ms	145 (22)	—	125 (7)	—
VV, ms	–20 (15)	—	–12 (02)	—

AV, programmed atrioventricular delay; ERO, effective regurgitant orifice area; LV, left ventricle; LVEDD, left ventricular end-diastolic volume; LVESD, left ventricular end-systolic volume; VV, programmed interventricular delay.
Data are expressed as mean (standard deviation).
[a] P<.05 compared to intragroup follow-up.
[b] P<.05 compared to intergroup follow-up.
[c] P<.05 compared to intergroup baseline.

QRS before resynchronization present a higher response rate to therapy although an adequate cut-off point has not yet been achieved; however, the degree of QRS narrowing in the implant[5,6] is probably more important for prognosis than the actual duration of the baseline complex. Our findings support those results and underscore the importance of adequate left ventricular lead placement during implantation, as well as accurate programming to obtain the narrowest paced QRS possible.

The inherent limitations of sample size and the descriptive nature of the study should be pointed out. The new data we present may be useful in routine clinical practice, although clinical trials with more patients are needed. Because the series contains patients with idiopathic dilated cardiomyopathy, the results cannot be extrapolated to other etiologies.

In patients with idiopathic dilated cardiomyopathy, the decrease in left ventricular end-systolic volume after CRT is related to electrical remodeling. This phenomenon appears to be determined by the smaller paced QRS width in the implant.

Julia Fernández-Pastor,* Fernando Cabrera-Bueno, Antonio L. Linde-Estrella, Jose L. Peña-Hernández, Alberto Barrera-Cordero, and Javier Alzueta-Rodríguez

Unidad de Arritmias, Servicio de Cardiología, Hospital Clínico Universitario Virgen de la Victoria, Málaga, Spain

*Corresponding author:
E-mail address: juferpas@secardiologia.es (J. Fernández-Pastor).

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Fatal Presentation of a Cardiac Myxoma

Presentación fatal de un mixoma cardiaco

To the Editor,

A 37-year-old woman with no relevant history came to the emergency room reporting dizziness, acute abdominal pain, and weakness as well as pain and dysesthesia in the lower limbs. The physical examination revealed sinus tachycardia, sweating, altered mental state, and hypotension. A cranial computed tomography (CT) scan and substance abuse screening of the urine showed no abnormalities. The examination of the lower limbs showed no right femoral pulse, a weak left femoral pulse, and signs of poor skin perfusion in both limbs. The chest and abdominal CT scans revealed severe occlusion of the infrarenal aorta (Fig. 1A), acute occlusion of the 2 common iliac arteries, left renal artery thrombosis showing images consistent with infarction of the ipsilateral kidney (Fig. 1B), and segmental infarctions in the spleen and the right kidney (Fig. 1C). Intravenous heparin sodium was started, the patient was intubated and connected to mechanical ventilation, and vasoactive drug therapy was initiated to treat the shock. Transthoracic echocardiography showed left ventricular dilatation with an ejection fraction of 15%, severe mitral regurgitation due to restriction of the posterior leaflet, and a mass in the left atrium (1.9×2.2 cm). The transesophageal echocardiographic study (Figs. 1D and E) confirmed the presence of a hypermobile mass with multiple finger-link projections; the mass was adhered to the interatrial septum and highly consistent with cardiac myxoma. The patient underwent proximal and distal percutaneous embolectomy with bilateral femoral access as well as multiple compartment fasciotomy of both limbs (Fig. 2A). The intravascular material fragments removed (Fig. 2B) were sent for histologic study, which confirmed cardiac myxoma embolism (Figs. 2C and D). The patient experienced hyperpotassemia (7.1 mEq/L) and an increase in creatine kinase concentration (121 500 IU/L) secondary to the reperfusion lesion, which required venovenous hemofiltration. The patient died a few hours later due to multiorgan failure.

Primary tumors of the heart are rare, with an estimated incidence between 0.0017% and 1.9% of unselected patients in autopsy studies. Among malignant tumors, sarcoma is the most common type, followed by angiosarcoma.[1] Atrial myxoma is the most common benign tumor of the heart (approximately 50% of primary cardiac tumors). These tumors occur slightly more often in women, and usually present in patients aged 30 to 60 years. Although histologically benign, they are occasionally fatal due to their strategic intracardial position. Three-fourths of all myxomas are found in the left atrium.[2] The tumors are often underdiagnosed due to the lack of specific symptoms. The clinical manifestations can include congestive heart failure, hemoptysis, cough, transient cyanosis, syncope, dyspnea, neurologic symptoms or pain in ischemic limbs due to cardiac obstruction (as a result of tumor fragmentation or, less often, complete tumor rupture), or arrhythmias. Atrial myxomas are a rare cause of acute peripheral arterial ischemia.[3] Ventricular dysfunction occasionally occurs and can be caused by myocardial infarction due to tumor fragment embolism in the coronary arteries or an acute increase in peripheral resistances resulting from complete acute occlusion of the infrarenal aorta (in our patient, both mechanisms were possible). In conclusion, in young, healthy patients, cardiac myxoma should be considered in the differential diagnosis of peripheral arterial embolism.