A 56-year-old woman was transferred to our institution for evaluation of New York Heart Association class IV congestive heart failure that was refractory despite treatment with furosemide, carvedilol, digitalis, spironolactone, and valsartan. She was bedridden before her transfer and was referred for consideration of cardiac transplantation. Prior treatment at another institution included bypass of the left anterior descending coronary artery, a left internal mammary...
graft for unstable angina, and implantation of a dual-chamber cardioverter/defibrillator for ventricular tachycardia. The device was programmed for DDDR pacing, with rate limits of 80 to 130 bpm and an atrioventricular (AV) delay of 160 ms. In this setting, the patient had been paced in the right ventricle 100% of the time for 6 months before she presented at our institution. Deterioration of her condition began after cardioverter/defibrillator placement and persisted despite increasing pharmacotherapy.

At the time of hospital admission, she was in florid congestive heart failure and had hypotension, elevated jugular venous pressure, bilateral lung crepitations, a murmur of mitral regurgitation, and pitting edema of the lower extremities. The initial ECG (at hospital admission) showed AV sequential pacing at 80 bpm with wide, paced QRS complexes that showed a left bundle-branch block configuration (Figure, A). The initial echocardiogram showed a dilated left ventricle, severe mitral regurgitation (MR) (regurgitant volume, 69 mL), and a left ventricular ejection fraction of 25% (Figure, B and Movie I). A chest radiograph confirmed pulmonary congestion and cardiomegaly, and the ventricular pacing lead was in the conventional right ventricular apex position near the septum (Figure, C).

The cardioverter/defibrillator was reprogrammed for DDIR pacing, with an AV delay of 300 ms to promote ventricular activation via the native conduction system. The QRS complexes became narrow (Figure, D), and the severity of MR markedly decreased from severe to trivial (shown by echocardiography on the day of reprogramming) (Figure, E and Movie II). The patient’s symptoms improved immediately, and she was able to ambulate. At the day-15 follow-up examination, the cardiothoracic ratio, measured by chest radiography, had decreased from 0.7 to 0.6 (Figure, C and F). Four months later, she had maintained her improved condition and had New York Heart Association class II symptoms that were associated with atrial pacing and ventricular activation via the native conduction system (Figure, G). An echocardiographic examination showed a further decrease in left ventricle size, improvement in ejection fraction to 40%, and absence of MR (Figure, H and Movie III). No marked change in the left atrial volume was noted at presentation (75 mL) or at last follow-up (78 mL). The calculated pulmonary artery pressures were 35 mm Hg on admission and 37 mm Hg after the pacemaker was reprogrammed for DDIR pacing. The cardiothoracic ratio, measured by chest radiography, had decreased to 0.5 (Figure, I).

This case illustrates iatrogenic severe mitral regurgitation and congestive heart failure that was due to abnormal activation of ventricles that were paced from the right ventricular apex position. The disappearance of MR after reprogramming suggested that right ventricle apical pacing caused dyssynchronous contraction of the papillary muscles (Figure, E and H). Reprogramming the cardioverter/defibrillator to DDIR pacing with prolonged AV delay resynchronized ventricular function by restoring native ventricular activation (Figure, D and G). This provided a normal QRS duration with no intraventricular conduction defect and was associated with dramatic improvement of symptoms. The patient no longer needed an upgrade procedure (for cardiac resynchronization therapy) or a cardiac transplant. In patients with iatrogenic left bundle-branch block (due to ventricular pacing, absence of AV block, and narrow QRS ventricular activation), restoration of native conduction by AV interval reprogramming should be attempted before therapies such as surgical upgrade to biventricular pacing are considered.

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