An 85-year-old previously healthy woman with symptomatic complete atrioventricular block, baseline QRS duration 166 ms, slow ventricular escape rhythm and normal ventricular systolic function underwent successful dual-chamber permanent pacemaker implantation with right ventricular (RV) apical (RVA) lead placement. One week later, plasma B-type natriuretic peptide (BNP) increased from 260 to 1,020 pg/ml, and cardiothoracic ratio was increased on chest X-ray (63%>59%). In addition, echocardiography showed new left ventricular (LV) wall motion abnormalities involving the mid and apical segments despite lack of serial change on 12-lead electrocardiogram (ECG) under ventricular pacing, or creatine phosphokinase elevation. Coronary angiography showed normal coronary arteries without vasospastic diathesis, while left ventriculogram showed apical ballooning. (Figure 1A). Cine magnetic resonance imaging (MRI) also showed apical ballooning with slow flow artifact, but myocardial edema was not detected. (B, Top) Corresponding late gadolinium-enhanced magnetic resonance imaging (LGE-MRI) showed subendocardial scar at the apical ballooning (red arrows).

Figure 1. (A) Coronary angiography showed normal coronary arteries without vasospastic diathesis, while left ventriculogram showed apical ballooning. (B, Top) Black-blood T2-weighted imaging in 4-chamber view and 2-chamber view showed apical ballooning with slow flow artifact, but myocardial edema was not detected. (B, Bottom) Corresponding late gadolinium-enhanced magnetic resonance imaging (LGE-MRI) showed subendocardial scar at the apical ballooning (red arrows).
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Ballooning, and it is possible that RVA pacing augmented and prolonged takotsubo cardiomyopathy. Indeed, there are a few previous reports of persistent takotsubo cardiomyopathy associated with pacemaker implantation, whereas apical ballooning typically returns to normal in a few weeks. In the present case, however, the onset of apical ballooning was not preceded by emotional or physical stress, and there was no ST-segment elevation or deep T wave inversion on 12-lead ECG during the acute phase under RVA pacing. Furthermore, the present cardiac MRI findings were different to those of typical takotsubo cardiomyopathy.

Chronic RVA pacing frequently induces myocardial perfusion defects that are associated with apical wall motion abnormalities. In the present case, resting myocardial scintigraphy showed perfusion defects in the area around the lead placement site. LGE-MRI showed residual subendocardial enhancement in the apical region.

We encountered a case of apical ballooning shortly after the initiation of RVA pacing. Very interestingly, it persisted for 3 months until the lead was repositioned to the septal site. RVS pacing immediately improved LV performance, and lead repositioning to the septal site led to resolution of the apical ballooning, hence the patient was finally diagnosed with RVA pacing-induced apical ballooning. It was assumed that RVA pacing directly induced LV mechanical dyssynchrony with apical stretching during systole and subsequent apical ballooning. Apical ballooning shortly after RVA pacing, however, is an unusual presenting form of pacing-induced wall motion abnormalities. Unrecognized microvascular spasm or latent myocardial damage at the segment of lead placement might also contribute to the onset of and/or persistence of apical ballooning, although baseline echocardiography before RVA pacing did not show any wall motion abnormalities. Catecholamine-induced acute myocardial stunning such as takotsubo cardiomyopathy is another important cause of apical ballooning, and it is possible that RVA pacing augmented and prolonged takotsubo cardiomyopathy. Indeed, there are a few previous reports of persistent takotsubo cardiomyopathy associated with pacemaker implantation, whereas apical ballooning typically returns to normal in a few weeks. In the present case, however, the onset of apical ballooning was not preceded by emotional or physical stress, and there was no ST-segment elevation or deep T wave inversion on 12-lead ECG during the acute phase under RVA pacing. Furthermore, the present cardiac MRI findings were different to those of typical takotsubo cardiomyopathy.

Given that serial echocardiography showed persistent apical ballooning with 100% ventricular paced rhythm after 3 months, RVA pacing-induced cardiomyopathy was clinically suspected, and therefore LV pressure-volume measurement was used to assess the acute hemodynamic effects of RV septal (RVS) pacing compared with baseline RVA pacing. During RVS pacing, LV stroke work dramatically increased from 658 to 2,463 mmHg·ml and end-diastolic pressure decreased from 8.3 to 3.8 mmHg (Figure 2C). Therefore, at 3 months the patient underwent lead repositioning to the RV septum, with resolution of apical ballooning (Figure 2B Top), accompanied by dramatic improvement of BNP and myocardial perfusion on scintigraphy (Figure 2B Bottom). In contrast, LGE-MRI indicated residual subendocardial enhancement in the apical region.

Although the precise mechanisms of persistent apical ballooning remain unknown, the speed and extent of wall motion recovery can vary depending on the amount of metabolic and structural myocardial tissue damage incurred before the lead repositioning. We speculate that persistent apical ballooning for 3 months during RVA pacing caused substantial myocardial tissue damage that impeded the prompt recovery of apical wall motion. In addition, septal pacing can negatively affect the resolution of LV wall motion abnormality, despite being

Figure 2. (A,B) Cine magnetic resonance imaging (Top), regional wall thickening map using a 16-segment model (Middle), and polar display myocardial perfusion using $^{201}$Tl myocardial scintigraphy at rest (Bottom) during (A) right ventricular (RV) apical (RVA) pacing and (B) at 3 months after change to RV septal (RVS) pacing. Apical ballooning and perfusion defect in the corresponding myocardial area were observed during RVA pacing. Both improved after change to RVS pacing, with a remaining small perfusion defect at the apical-inferior segment. (C) Left ventricular (LV) pressure-volume loops (blue, RVA pacing; red, RVS pacing). RVS pacing immediately improved stroke work and decreased end-diastolic pressure.
better than RVA pacing. The present patient was closely observed for signs of spontaneous recovery of the apical ballooning during the 3 months of RVA pacing. In hindsight, however, prompt lead repositioning to the septal site could have led to earlier resolution of wall motion abnormalities.

This is the first case of apical ballooning shortly after RVA pacing, in which acute hemodynamic effects of RVS pacing were confirmed on LV pressure-volume measurement. In addition, in the present case myocardial tissue characterization was assessed using cardiac MRI after pacemaker implantation, which excluded takotsubo cardiomyopathy. The presence of apical ballooning after pacemaker implantation should alert the clinician to the possibility of a direct effect of RVA pacing and the necessity for repositioning after thorough evaluation including LV pressure-volume analysis and cardiac MRI.

Disclosures
None of the authors have any conflict of interest or financial disclosures.

References