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LATE RUPTURE OF EXPANDED POLYTETRAFLUORO-ETHYLENE NEOCHORDAE USED FOR MITRAL VALVE REPAIR To the Editor:

In the February issue of the Journal, Farivar and colleagues¹ described a case of late rupture of expanded polytetrafluoroethylene (ePTFE) neochordae after mitral valve repair. In the "Discussion," they state that there was only 1 previous case reported in the literature of ruptured neochordae, presented by Buttany and colleagues,² suggesting their case as the second reported. This is not correct. In 2007, the Journal of Heart Valve Disease published our report of 2 cases of acute mitral regurgitation caused by ruptured ePTFE neochordae.3 Our patients presented with symptoms of acute heart failure caused by sudden rupture of the chordae, which was different from the rare clinical manifestations, hemolysis, and hematuria experienced by the patient reported by Farivar and colleagues.

We first used ePTFE routinely to correct ruptured or elongated chordae tendineae during the late 1980s and have since implanted it in more than 500 patients without a single known case of ePTFE failure necessitating reoperation, until these 2 late events occurred. After a thorough literature search regarding the possible mechanisms behind this unusual occurrence, we analyzed our histopathologic findings. We found that there was likely pannus formation and collagen infiltration through the porous structure that could account for progressive disruption before calcification ensued, which could be postulated as a form of repair/scarring.

In addition to the degenerative causes that could be responsible for the late rupture, we believe that the use of forceps and other metal instruments in the manipulation of the ePTFE could interrupt its electronegative charge and weaken the material. We currently avoid any kind of metallic manipulation.

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BIVENTRICULAR PACING AFTER CARDIAC SURGERY To the Editor:

We read with interest the article by Evonich and colleagues¹ in the October issue of the *Journal*. They performed a randomized study of pacing modes ("normal care," synchronous atrial-right ventricular pacing, and synchronous atrial–biventricular pacing) in 40 patients with impaired left ventricular function after cardiac surgery. They included both a crossover study of acute hemodynamic variables early after operation (in 29 patients) and an assessment of clinical end points, such as intensive therapy unit and hospital stay and mortality. In contrast with experience in chronic heart failure, they showed no advantage to biventricular pacing in any area, although the trial was underpowered for the clinical end points.

They suggest this was the first randomized study in surgical patients but did not cite our 2005 publication.² We performed a similar randomized crossover study, with hemodynamic assessment of different pacing modes in a similar number of patients (25) early after surgery. We demonstrated a clear advantage to atrial–left ventricular pacing and regard this modality as an important addition to the postoperative care of the compromised patient.

Closer analysis of the results in the article by Evonich and colleagues¹ suggests that there is a serious error or that they too found an advantage but did not recognize it. In the second paragraph of the section titled "Hemodynamic Testing," they state a statistically significant difference in stroke volume, cardiac output, and left ventricular stroke work index between AAI and atrial-right ventricular pacing. But, in Table 2, the values they refer to in the text are in the column under atrial-biventricular pacing pacing. If Table 2 is to be believed (and there is inconsistency in nomenclature, does AAI equate to "usual care"?), these investigators did see an advantage to biventricular pacing.

We believe biventricular pacing is of advantage to the postsurgical patient. Even if we are mistaken in our interpretation of this article, surgeons are left with 2 series of patients with contrasting results, perhaps because both have small numbers. This calls for a larger definitive study.

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