trial fibrillation (AF) is the most common sustained cardiac arrhythmia that clinicians encounter in daily practice. In addition to the evidence of a growing worldwide AF burden, considering the aging of the Japanese population, the prevalence and incidence of AF are expected to increase, making AF a serious epidemic because it is a highly age-related condition. In addition to systemic embolization, heart failure is one of the most dismal consequences associated with AF. In patients who have left atrial (LA) dilatation caused by chronic AF, but do not have left ventricular (LV) dilatation or systolic dysfunction, mitral regurgitation (MR) without an organic mitral valve (MV) lesion is often seen on echocardiography. Recently, this type of MR has been called ‘atrial functional MR’ to differentiate it from general functional MR (another term is ischemic MR for patients with chronic ischemic heart diseases) mainly caused by LV dysfunction. Similar to other types of MR, significant atrial functional MR might increase cardiovascular mortality and morbidity in AF patients; however, there are many unresolved clinical questions.

It has been generally believed that the only morphological alteration in the MV apparatus is annular dilatation in patients with longstanding AF. However, it has been controversial whether isolated MV annular dilatation could cause significant MR. In this issue of the Journal, the study by Machino-Ohtsuka and colleagues provides novel information of the
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mechanism behind the development of atrial functional MR. Consulting their transesophageal echocardiographic database, the authors retrospectively extracted data from 25 patients with lone AF (with neither LV dysfunction nor MV organic lesion) and who had moderate-to-severe MR. The baseline characteristics of the patients with significant atrial functional MR were compared with those of matched AF patients with mild MR and without MR, as well as normal controls. They found that the patients with significant atrial functional MR had longer AF duration and more severe LA dilation. In their multiple regression analyses of the determinants of effective regurgitant orifice area (EROA), which is the most reliable quantitative measure of MR severity, posterior MV leaflet tethering was a strong independent determinant equivalent to MV annular dilation, which had been believed to be an isolated determinant of MR in AF patients, as mentioned before. On the other hand, in their study anterior MV leaflet tethering was not aggravated, even in patients with significant MR.

This asymmetric posterior MV leaflet tethering in patients with LA dilatation has been recently hypothesized as “atriogenic” leaflet tethering. As shown in Figure, Silbiger proposes that LA dilatation itself possibly exaggerates MV tethering through a mechanism distinct from LV dysfunction. In comparison with well-known mechanisms of functional MR caused by LV dysfunction, a characteristic phenomenon is that the MV posterior leaflet body is tightly pulled against the LV inlet and the free part of the posterior leaflet, which can work for leaflet coaptation, is decreased because of displacement of the posterior MV annulus onto the crest of the LV inlet. Anterior leaflet tethering is relatively small by virtue of fixation of the anterior annulus to the fibrous portion. Traditionally, this is known as “hamstringing of posterior MV cusp” in patients with rheumatic MR and LA dilatation; however, this kind of old-fashioned comprehension might be analogous with the extremely novel concept of “atriogenic” MV leaflet tethering in AF patients. Although the study is a single-center retrospective study with a small sample size, demonstration of this unique hypothesis is so valuable that it could partially resolve the aforementioned controversy. Namely, not only a dilated annulus but also posterior MV leaflet tethering could develop MR in AF patients.

Because there are only a few small studies regarding surgical intervention for this kind of MR, this study is also important from the viewpoint of exploring optimal surgical treatment for atrial functional MR. Asymmetric posterior MV leaflet tethering is sometimes observed in postoperative patients who underwent surgical annuloplasty for functional MR caused by LV dysfunction, which is associated with persistent MR following annuloplasty. Therefore, it could be inappropriate to perform isolated annuloplasty for atrial functional MR. Some adjunctive procedures would be preferable, with the aim of alleviating MV tethering; however, they ought not to be already conducted major techniques for functional MR caused by LV dysfunction (ie, procedures for the subvalvar apparatus or the LV myocardium itself). Combined with MV annuloplasty, other procedures, for example, posterior leaflet augmentation, might be promising. Otherwise, assuming that LA dilation might be a direct inducer, although it has never been reported, we can speculate whether LA volume reduction might be effective for ameliorating “atriogenic” MV tethering. In order to identify an effective treatment, there are some issues to be further clarified. As demonstrated in the study, 3D echocardiographic measurement of the posterior to anterior MV leaflet area was smaller in patients with atrial functional MR than in those without MR; the posterior MV leaflet often appears echocardiographically regressed or shortened in these patients. Thus, it would be crucial to confirm at the time of surgery that significant organic insufficiency of the posterior MV leaflets strictly does not exist.

According to the current clinical guidelines, to begin assessing chronic MR we need to distinguish primary from secondary MR (ie, functional MR). The recommendations for these distinct types of MR are entirely different; first of all, the absolute threshold of evaluating severe MR is quite different because the prognosis of functional MR has been proven to be much worse than that of primary MR. As for the aforementioned EROA, the cutoff of severe degree is 0.40 cm² for primary MR; meanwhile, it is only 0.20 cm² for functional MR. However, functional MR mentioned in the guidelines means “functional MR due to LV dysfunction”. Clinical recommendations for assessing or treating patients with atrial functional MR do not exist to date. Atrial functional MR is an entirely novel concept of the disorder; thus, we need to begin learning about it from now on.

References