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**REPLY FROM AUTHOR:
ATRIAL FIBRILLATION
AND FUNCTIONAL
TRICUSPID OR MITRAL
REGURGITATION:
WHICH COMES FIRST,
THE EGG OR THE CHICKEN?**



Reply to the Editor:

We read with interest the Letter to the Editor from Babokin and colleagues.¹ Pulmonary artery denervation has been recently described as an alternative method to treat pulmonary hypertension in patients with left heart failure,² but despite the efficacy and safety of such procedures, it remains to be established in the long term and it is also debatable whether these procedures are superior to the standard of care pharmacologic therapies for pulmonary hypertension.

Atrial fibrillation (AF) is a silent disease with catastrophic consequences, from heart failure, stroke, to sudden death. Its association with valvular disease, right- or left-sided, has long been demonstrated as well as the negative impact even after surgical correction of the valvular problem. Moreover, the latest American and European guidelines have considered it beneficial to perform AF ablation for symptomatic patients with paroxysmal or persistent AF who are undergoing valvular/cardiac surgery.^{3,4}

The causal effect of AF in the genesis of tricuspid (TFR) or mitral (MFR) functional regurgitation has been disputed, and for a long time AF was merely considered as a natural consequence of the valvular disease. However, in recent times, the concept of “atrial” MFR and TFR has gained terrain in opposition to the well-established “ventricular” functional regurgitation, where the dilatation of the left or right ventricle was seen as the precursor of the valvular regurgitation, respectively. Interestingly, the common ground of this nosological entity is the presence of AF. In regard to atrial MFR, which has been reported to be between 3% and 15% in patients with AF, MFR can occur among patients with significant dilatation of mitral annulus and

left atrium.⁵ Nevertheless, other important triggers of MFR, in the presence of the latter anatomical changes, have been identified, such as reduced annular contractility, increased valve stress by flattened saddle shape of the annulus, and left atrial dysfunction. In addition, “atriogenic” leaflet tethering and imbalance of annulus area to leaflet area can result from insufficient leaflet remodeling. There is little evidence thus far on therapeutic options for the management of atrial FMR, with rhythm-control strategies from pharmacologic therapies to catheter or surgical ablation being plausible solutions. In respect to atrial TFR, long-standing AF has been associated with the existence of tricuspid regurgitation despite normal right ventricular geometry and function. The pathophysiology involves the interaction among tricuspid annulus remodeling, right atrium dilatation, and loss of function. Nevertheless, the temporal relationship between tricuspid regurgitation and AF remains to be clarified.⁶

Hence, one can say that there is sufficient evidence to believe that AF comes first, and valvular regurgitation is the natural negative evolution of this disease. Notwithstanding, the therapeutic challenge can be different from the “ventricular” functional regurgitation. In atrial functional regurgitation, rhythm control (pharmacologic, catheter or surgical AF ablation) and or surgical correction should be tailored to the specific pathophysiology found in each patient. Isolated annuloplasty may not be appropriate when there is severe atrio-genetic tethering or inadequate leaflet remodeling causing annulus area-leaflet area imbalance, and leaflet patch augmentation could be necessary along with the implantation of an annuloplasty ring.

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