Author`s Reply

To the Editor,

We would like to thank to the colleagues for their interest in our article (1). First, postoperative atrial fibrillation (POAF) in patients with isolated surgical aortic valve replacement (SAVR) is far from being completely elucidated (2), and novel predictors and algorithm still cause substantial debate. This is very important because POAF has an important impact on patient's recovery and hospitalization duration, requires additional interventions or medications with possible side effects, and can result in major complications such as stroke or death (2, 3). The assessment of predictors for POAF is important not only for increasing morbidity of this arrhythmia but also for increasing related costs in these patients (2).

Second, in this study, we found six variables associated with high postoperative arrhythmic risk using multivariate analysis, namely advanced age, body mass index (with a cut-off value of 27 kg/m^2), moderate tricuspid regurgitation, prolonged ventilation, longer intensive care unit stay, and increased left atrium volume (>35 mL/m^2). The parameters included in preoperative risk assessment (by EuroScore II) are factors with a high risk for AF (age, NYHA class, renal impairment, systolic left ventricular dysfunction, diabetes mellitus, etc). In our patients, EuroScore II was significantly higher in patients with POAF (9.00±2.87 vs. 5.78±1.97; $p<0.001$). We also found that prolonged ventilation, stroke, neurological complications, and acute renal failure were significantly more frequent in the AF group. We consider that these complications were determined by a higher surgical risk. It is not ethically (4) and scientifically appropriate to exclude these patients from the AF group. In addition, in real life, these are the patients who are referred for SAVR. We consider that a higher risk of POAF in these patients could be related to a pre-existing substrate for AF (atrial enlargement and structural atrial remodeling due to the chronic diastolic dysfunction).

Clinically meaningful AF requires the presence of both a trigger and a vulnerable atrial substrate (3). Obviously, there are also incriminated acute factors such as inflammation, atrial oxidative stress, high sympathetic tone, electrolyte changes, and volume overload (3, 5). Beyond the perioperative status of these patients (which ideally should be almost the same) is the degree of proarrhythmic substrate (which is different among patients). We could emphasize that those postoperative acute factors are trig-

gers for AF (as the main substrate for AF). However, the pathophysiology of POAF is unclear. Therefore, a better preoperative control of factors incriminated as substrate in POAF could be very important. For example, the use of statins is associated with a 22%–34% lower risk of POAF (3).

In the third line, we have noted the following statements in study limitations (1): "The underlying mechanisms of POAF are yet to be determined. Future models should include parameters of the inflammatory response and other new variables derived from these findings." Also, in discussions, we have noted the following: "In our opinion, future studies should include variables that describe the inflammatory response to surgery, such as C-reactive protein or interleukin 6." We understand that authors of this letter agree with us.

Finally, the suggestion "to select a standardized patient population between with and without AF group" is not in accordance with the patient population with need for SAVR in real life. However, estimating individual risks for POAF in patients undergoing SAVR is important for applying prophylactic strategies only in patients with high arrhythmic risk, thereby avoiding excessive cost and unwanted side effects in low-risk individuals. Therefore, we consider that our tree model based on chi-squared automatic interaction detection for patients with POAF is very important.

Alina Cristina Iliescu[1], Delia Lidia Salaru[1,2], Ionut Achitei[1], Mihaela Grecu[1], Mariana Floria[2,3], Grigore Tinica[1,2]
[1]Institute of Cardiovascular Disease; Iaşi-*Romania*
[2]Grigore T. Popa University of Medicine and Pharmacy; Iaşi-*Romania*
[3]Sf. Spiridon Emergency Hospital; Iaşi-*Romania*

Reference

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Address for Correspondence: Mariana Floria, MD,
From 3rd Medical Clinic and
Grigore T. Popa University of Medicine and
Pharmacy 16 University Street,
Iaşi-*România*
Phone: +40 232 301 600
Fax: +40 232 211 820
E-mail: floria_mariana@yahoo.com