

## The risk of developing AF after cardiac surgery

### *Kalp cerrahisi sonrası AF gelişme riski*

We would like to congratulate the authors for their original and interesting study (1). Numerous different markers have been demonstrated for AF development in many studies conducted about atrial fibrillation (AF) which is the most common arrhythmia after cardiac surgery (2). Interestingly, there is no consensus on some of (a considerable number of) these markers. For example, in this study by Çetin et al. (1), female gender was reported as a risk factor for AF, while in some other studies male gender is stated as a risk factor (2). Other relevant examples to give are cardiopulmonary bypass time and cross-clamp time. While Çetin et al. (1) did not show these parameters as risk factors, these operative data were stated as very strong risk factors in many other studies (2). We would like to state that we wonder the views of the authors about the causes of the differences in these similar parameters.

The main theme of this article, effect of the preoperative electrocardiographic (ECG) data on postoperative AF development is a really original subject. In few studies on this subject, generally P wave amplitude and PR interval on ECG were studied (3-5). In one of these studies, preoperative P wave to be longer than 110 msec was stated to be a risk factor for AF development (4), while in another study PR interval to be longer than 120 msec and P wave than 110 msec were reported to be risk factors (5). In contrast, there are several studies indicating that negative P wave is also a risk factor (3). In this context, we think that any ECG data available out of the fragmented QRS complexes will add value to the study if specified.

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### Author's Reply

To the Editor,

We thank the authors for their constructive comments on our article in their letter entitled as 'The risk of developing AF after cardiac surgery'. They criticized that the study is focused only on the fragmented QRS, but other some electrocardiographic (ECG) parameters such as p wave duration and amplitude or PR interval may also important to predict postoperative atrial fibrillation (POAF). In addition, it is also said that male gender rather than female and cardiopulmonary bypass time and cross-clamp time are found as predictors for POAF in previous studies.

We accept that it could be included additional ECG signs besides fQRS and performed a comparison among the parameters in a multivariate analysis. While p wave and PR interval are related to diastolic phase, fQRS is related to systolic phase of the cardiac cycle. Therefore, these signs on surface ECG would have different mechanisms on development of AF, and to know more important sign may provide more important mechanism and target to prevent POAF.

On the other hand, we selected patients from a limited population and excluded patients who have additional comorbidities, thus our study population has relatively a low EUROSCORE. Therefore, our results do not apply to all patients, and gender and difference in inotropic support time for prediction of POAF may be related to above mentioned factors.

Based on previous arguments, we believe that further studies on ECG signs are needed to clarify more accurately the mechanisms of individual different POAF rates and to confirm the importance of modulating real underlying mechanism to improve clinical outcome.

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## Coronary collateral development might be impaired by decreases in glomerular filtration rate

### *Koroner kollateral gelişimi glomerular filtrasyon hızındaki azalmalardan etkilenebilir*

We have greatly enjoyed reading the article by Zorkun et al. (1) entitled "Determinants of coronary collateral circulation in patients with coronary artery disease". In that well-designed study, the authors aimed to define the demographic and baseline determinants of coronary collateral development (CCD), and found that male gender, prior statin usage, and elevated levels of high-sensitive C-reactive protein (hs-CRP) are associated with development of coronary collateral. They

excluded patients with renal failure in this study in spite of the fact that they do not give estimated glomerular filtration rate. However, it is well-known that cardiovascular disease is the leading cause of morbidity and mortality in chronic kidney disease (CKD) and CKD is associated with poor CCD, even in patients with mild to moderate renal insufficiency (2). More importantly the predictors of poor CCD in patients with CKD is not known exactly.

Angiogenic adaptation (formation of new vessels) and arteriogenic adaptation (maturation of these new vessels) are pivotal steps in the development of coronary collaterals. Endothelial cells play a pivotal role in all steps of collateral development and nitric oxide (NO) also plays a critical role in the maintenance of normal endothelial function, angiogenesis and arteriogenesis. Asymmetric dimethylarginine (ADMA) is an endogenous inhibitor of nitric oxide synthesis (NOS), competes with L-arginine for the active site of endothelial NOS, decreases the production and bioavailability of NO, and thus decreases the vessel compliance, increases vascular resistance and limits the blood flow. NO deficiency and endothelial dysfunction due to the existence of multiple potential anti-angiogenic factors such as increased plasma ADMA level in patients with CKD has been found to impair the microvascular adaptation and ischemic tolerance of tissues (2). It was previously reported that plasma ADMA level was found to be higher in patients with renal failure (uremic toxin) (3). Today, cardiovascular risk factors known to cause endothelial dysfunction such as hypertension, hyperlipidemia, hyperhomocysteinemia, smoking and diabetes are found to be closely related to increased plasma ADMA levels (4). In our previously published study, we found that the L-arginine/ADMA ratio (relatively increased plasma ADMA level) was higher in patients with glomerular filtration rate (GFR) > 60 mL/min/1.73 m<sup>2</sup> and good CDC than in patients with GFR < 60 mL/min/1.73 m<sup>2</sup> and poor CCD, and suggested that presumably because of the adverse effect of decreased L-arginine/ADMA ratio on endothelial cells and angiogenesis, CCD was worse in patients with GFR < 60 mL/min/1.73 m<sup>2</sup> than in those with GFR > 60 mL/min/1.73 m<sup>2</sup> (5).

In conclusion, CCD can be impaired by a decrease in GFR, and the poor CCD can be explained by impaired angiogenesis (impaired ischemic tolerance) due to endothelial dysfunction in patients with renal failure. We strongly believe that GFR values should be kept in mind when analyzing determinants of coronary collateral circulation in patients with coronary artery disease.

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## Author's Reply

To the Editor,

We thank Çelik et al. (1) for their valuable comments and thoughtful remarks, appreciate their interest in our work (2), and congratulate for their study.

In their letter to the editor, the authors highlighted the importance of reduced glomerular filtration rate as a potential indicator of poor coronary collateral development in patients with chronic kidney disease.

We agree with Çelik et al. (1) that "the predictors of poor coronary collateral development in patients with chronic kidney disease is not exactly known." In spite of the insufficient literature discussion about it, a reduced glomerular filtration rate may be an important pathophysiological mechanism (3, 4).

We would like to reply to the issue raised in their letter. The main difference is due largely to the design of the study. As we have stated in our methodology section, all patients with previous diagnosis of renal failure were excluded. Having a small study group was the main reason of this exclusion, as the number of patients was not sufficient to show all factors to predict improved or impaired coronary collateral development.

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