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ed significant improvement in pulmonary arterial pressure after the operation (5). We thought there is a need to the extensive studies for detailed evaluation of the subject.

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Does bilirubin level have an effect on cardiac parameters?

To the Editor,

We read the article "The effect of Gilbert's syndrome on the dispersions of QT interval and P-wave" written by Cüre et al. (1) published in *The Anatolian Journal of Cardiology* with great interest.

The authors aimed to investigate the effects of bilirubin levels on noninvasive electrocardiographic parameters. They concluded that increased bilirubin levels are associated with decrease in HR, Pd and QTd in Gilbert's syndrome (1). Thanks to the authors for their contribution.

We know that P wave and QT dispersion are used for the prediction of atrial and ventricular arrhythmias (2). Increased P wave dispersion gives us information about intraatrial and interatrial conduction delaying (3). Several studies have shown that an increased QT dispersion and/or QTc dispersion could be a marker for arrhythmic events, myocardial infarction, and sudden death.

P-wave and P-wave dispersion measurement of distances must be very sensitive. While some centers can measure automatically measurements are usually made manually. Therefore, analysis of intraobserver and interobserver differences are important. Increase of P wave

duration resulting from the increase of interatrial and intraatrial conduction time due to atrial expansion causes a predisposition for arrhythmias. In a study, a mathematical formula has been developed associated with left atrial expansion and P wave duration as left atrial diameter (cm)= $2.47 \pm 0.29 \times p$ wave duration (mm) (4). So P and QT measurements would be supported by echocardiographic measurements of the left atrium and left ventricle.

It is also important to measure the QT duration and QT dispersion but the calculated QT corrected according to the heart rate is able to provide more accurate information. The authors should pay attention to these issues.

The cardioprotective effect of bilirubin is well known but the relationship between cardioprotection and bilirubin levels are unknown (5). It would be useful evaluating the correlation between bilirubin levels and Pd, HR and QTd in this study.

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Author's Reply

To the Editor,

We thank Authors for the interest they have shown in our article published the *Anatolian Journal of Cardiology* (1).

Firstly, all electrocardiographic measurements were performed by a cardiologist and an internist who were not aware of the diagnosis of the patients. Clearly, the measured values of the QT interval were based on the shape of the descending part of the T wave. T wave offset determined by manual method is very unreliable. Unfortunately, available automatic methods have not been shown to have any advantage (2). In our study, echocardiography (echo) was not performed in the patients. So we can not make a comparison between P wave dispersion (Pd) and QT dispersion (QTd) with echo findings. In fact, echo findings might have enriched the study. Our study was performed as a pilot study.

Secondly, several studies failed to find correlation between heart rate and the dispersion of ventricular recovery times measured with QTd. The precise relation between the heart rate and the dispersion of recovery times is still an unresolved issue. However, QTd measured in the standard 12-lead ECG is not based on (and thus should not be corrected for) the heart period in the same way as the QT interval (2). Also, a previous study showed that QTd remains unchanged during atrial pacing at heart rates up to 120 beats/min in individuals without structural heart disease and in patients with a history of sustained ventricular tachycardia (3). In our study, corrected QTd was not calculated since the previous studies have shown that rate correction of parameters of repolarization dispersion is probably unnecessary and may even distort the values and predictive usefulness of QTd.

Thirdly, in the current study, correlation analysis was not performed. According to the author's suggestion, Pearson correlation analysis was performed for indirect (I) bilirubin (B). IB had a negative correlation with QTd ($r^2=0.047$, $p=0.003$) and Pd ($r^2=0.090$, $p=0.001$), but had no correlation with heart rate. B may decrease the risk of arrhythmias with unknown mechanism. However, the mechanism is still not fully understood.

B is a well-known antioxidant. Small dense low density lipoprotein and oxidative stress markers have been found to be low in patients with Gilbert's syndrome (GS). Additionally, in a previous study, it has been reported that B decreases the release of large and active thrombocytes to peripheral blood stream by decreasing proinflammatory cytokines so may prevent arterial and venous occlusive cardiac diseases such as myocardial infarction (4). Also, in another study, high B level has been found to be negatively correlated with epicardial adipose tissue thickness (EAT) and elevated adiponectin levels which has anti-atherosclerotic effect (5). Decreased EAT may lead to lower release of proinflammatory cytokines and lower atherosclerotic heart disease. Another study has displayed the anti-atherosclerotic effect of mild elevation of B due to lower pulse wave velocity in GS patients (6). There is a need for further studies investigating the mechanisms of cardioprotective effects of B.

We, sincerely thank the authors for their contribution to our work.

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Real-time three dimensional transesophageal echocardiography has an incremental value in delineation of paravalvular leakages

To the Editor,

We have read with great interest the article entitled "The relation between location of paravalvular leakage and time to reoperation after mitral valve replacement: an observational study" published in *Anadolu Kardiyol Derg* 2013 Sep 10. (1). The authors aimed to evaluate any potential link between location of paravalvular leakage (PVL) and time to reoperation in patients undergoing redo mitral valve surgery. Thanks to the authors for their contribution of the present study. On the other hand, we want to make essential criticisms about this study from different aspects.

First of all, there is major concern regarding the methodology of the study. The study group was divided into 2 groups; Group 1 (Leaflet) and Group 2 (Commissural). This classification is vague and to the best of our knowledge this has not been described and reported in the literature previously. Recently, most authors (both cardiologists and cardiovascular surgeons) have used clock-wise format to describe the localizations of PVLs (2-4). It would have been much better if the authors had taken into consideration the terminology of PVL localizations in an understandable manner.

In discussion and conclusion sections, the authors concluded that echocardiographic evaluation should include location of the paravalvular leakage during follow-up of patients with PVL after mitral valve replacement. However, due to methodology of the study only transthoracic echocardiography (TTE) was performed for assessment of PVLs preoperatively and during follow-up and transesophageal echocardiography (TEE) was only performed at the end of the surgery for assessment of residual PVL (TEE was not performed neither preoperatively nor during the follow-up). TTE is certainly the initial choice of evaluation of prosthetic valves and complications but is unable to delineate localization of PVLs. Use of both 2D TEE and particularly real time three dimensional transesophageal echocardiography (RT-3D TEE) is mandatory for defining location and size of the paravalvular leakage during follow-up. Recently, RT-3D TEE has been introduced into clinical practice which has permitted assessment of PVLs precisely by 'en face view' from atrial (surgical) side of view (4, 5). Since the authors did not perform any TEE examination during follow-up of PVLs which may stand for a major limitation, it causes a conflict with the authors' conclusions. Furthermore, the authors did not consider this as a major limitation of the study.

Another noteworthy issue is the mortality rates for reoperation in Group 2 (Commissural PVL) which is reported as 0%. In current literature the mortality rates for reoperation of paravalvular leaks is much higher