Letter to the Editor
Heart rate correction should be kept in mind during measurements of ventricular repolarization parameters

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Key words: Magnesium sulfate ; tracheal intubation ; QT dispersion ; Tp-e ; transmural dispersion.

We read the article entitled “A comparison of the effects of lidocaine or magnesium sulfate on hemodynamic response and QT dispersion related with intubation in patients with hypertension” by KIRACI et al. with interest (1). The authors investigated the effects of magnesium on the hemodynamic, QT interval and QT interval dispersion (QTd) changes during anesthesia induction in hypertensive patients that compared to lidocaine. Finally, they concluded magnesium sulfate may be useful for patients who develop QTd increase during tracheal intubation, especially in coronary artery disease patients. We would like to thank to the authors for their valuable contribution. On the other hand, we would like to report a few concerns regarding this study from a methodological point of view.

The QT interval is dependent on the heart rate. It is inversely proportional to heart rate: The QT shortens at faster heart rates and lengthens at slower heart rates. In order to improve the detection of increased ventricular arrhythmia risk, some formulas are used for QT correction such as Bazett’s and Fredericia’s (Bazett Formula: Corrected QT(QTC) = QT/(RR)^0.5 and Fredericia Formula: Tc = QT/(RR)^0.333). If the authors had used these corrected QT formulas, that would make the study more precise.

In addition to QTd, transmural dispersion of repolarization (TDR) quantifies myocardial inhomogeneity (2). Isolated cells from different layers of the myocardium revealed that myocardium consists of three different myocyte types: i) endocardial, ii) epicardial, and iii) midmyocardial (M) cells (3). These myocyte types have various electrophysiological characteristics. This heterogeneity may cause to electrical instability and are measurable on surface ECG. Epicardial repolarization phase ends at the peak of the T-wave, M cells repolarization continues until the end of the T wave (3). Thus, the distance between the peak and end of the T wave is entitled as Tp-e interval, which reflects TDR. Previously, we presented that TDR was increased in patients with obstructive sleep apnea and chronic arsenic exposure via drinking water (4, 5). It has been also demonstrated that TDR is increased in ST-elevation myocardial infarction (3). Adding TDR measurements might have been better for determining the arrhythmic risk during tracheal intubation in hypertensive patients.

We believe that the paper of KIRACI et al. will lead to further studies concerning myocardial heterogeneity and instability. However, assessment of Tp-e and corrected QT interval should be kept in mind to provide more comprehensive data.

References

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