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We have read with great interest the recent article by Oksuz et al. [1] entitled “Atrial electromechanical delay analysed by tissue Doppler echocardiography is prolonged in patients with generalised anxiety disorders”. The authors showed prolonged intra- and inter-atrial electromechanical delay and increased P-wave dispersion in patients with anxiety disorders, and found a positive correlation between these abnormalities and the severity of the disease, assessed by Hamilton Anxiety Rating Scale.

P-wave dispersion is considered to reflect the discontinuous and inhomogeneous propagation of sinus impulses and the prolongation of atrial conduction time, and it has been shown to be an independent risk factor for atrial fibrillation (AF) development [2].

Over many years, our research group has studied the electrocardiographic and echocardiographic indexes of AF risk in some other clinical conditions, such as obesity [3], beta-thalassemia major [4–7], congenital heart diseases [8], respiratory disease [9], and muscular dystrophies [10–12]. We showed that electrocardiogram (ECG) may be a feasible and low-cost method to early detect the presence of arrhythmogenic substrate and to identify the high-risk subgroup patients in need of careful electrocardiographic monitoring.

Furthermore, it should be noted that in other clinical scenarios, P-wave parameters other than P-wave dispersion are risk predictors of supraventricular arrhythmias and notably of stroke [13–16], as reported in the literature. Specifically, we refer to interatrial block (IAB), which is defined as prolonged conduction time between right and left atria due to impulse delay or blockage, probably most often, but not exclusively, in Bachmann bundle, resulting in prolonged P-wave duration (≥120 ms), often with a bifid notch representing the electrical gap between the two-atrium activation. We suggest the authors to analyse IAB, taking the opportunity to firstly report the prevalence of IAB in patients with generalised anxiety disorders.

The authors performed all electrocardiographic measurements manually. In our experience manual measurement on standard paper-printed ECGs is of limited accuracy and reproducibility. To achieve greater precision, we suggest that the authors scan and digitise the ECGs from paper records, in order to display and magnify them to 400% on a high-resolution computer screen. This measurement method allows the measurement of P-wave duration with the use of computer software from all 12 ECG leads without significant intra- and inter-observer coefficients of variation.

In order to integrate and complete the arrhythmological evaluation of patients with generalised anxiety disorders, we suggest that the authors assess the QT, JT, and Tpeak-end dispersion, non-invasive electrocardiographic parameters, to measure the inhomogeneity of regional and transmural ventricular repolarisation, which may represent the electrophysiological substrate for malignant arrhythmias in many clinical conditions [17–22]. Finally, it might be interesting to detect the occurrence of supraventricular arrhythmias in patients with generalised anxiety disorders through a 30-day external loop recorder monitoring and to evaluate a possible correlation between non-invasive risk parameters and arrhythmias. Thus, it might strengthen the data obtained in this valuable study.

Conflict of interest: none declared

References
Response to the letter concerning the article: “Atrial electromechanical delay analysed by tissue Doppler echocardiography is prolonged in patients with generalised anxiety disorders”


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We thank Russo et al. [1] for their interest in our article entitled “Atrial electromechanical delay analysed by tissue Doppler echocardiography is prolonged in patients with generalised anxiety disorders” [2]. In our study, we investigated the atrial electromechanical delay (AEMD) by tissue Doppler imaging (TDI) echocardiography in patients suffering from generalised anxiety disorders, and found that this patient group has prolonged AEMD compared to healthy subjects. Additionally, we found prolonged P-wave dispersion (PWD) in the patient group, and this was correlated with the severity of the disease, which we assessed using the Hamilton Anxiety Rating Scale (HAMA). In light of the letter published by Russo et al. [1], we would like to make additional comments about this topic.
Measurement of AEMD by TDI echocardiography is a non-invasive, practical, and alternative method to electro-physiological study [3]. In addition, prolonged PWD, which can be measured easily by electrocardiography (ECG), is known to predict atrial fibrillation (AF) development [4]. However, it is well known that P-wave duration is affected by autonomic nervous system changes and is not a fixed variable because it may change significantly under different autonomic conditions [5]. Therefore, we chose to evaluate atrial delay by means of echocardiography, firstly. In this context, measurement of interatrial block was out of the purpose of this study. However, we believe that this parameter should be evaluated in this patient group in further studies. In addition, although this was a study aiming to evaluate the echocardiographic parameters primarily, we agree with the suggestion that ECG measurements should be done on a computer screen. The investigation of ventricular arrhythmia development in patients with generalised anxiety disorders was not the aim of this study, but we believe that this possibility should be kept in mind and should be investigated in further studies. Finally, as we mentioned in the limitations section of the article, it would be better if we had followed-up the patients with rhythm Holter or external loop recorder monitoring. Nevertheless, this issue should be the aim of further studies.

Arrhythmogenic risk of generalised anxiety disorder patients is a matter of debate in cardiology, and these patients need to be followed-up with ECG and echocardiography monitoring, regularly. PWD measurement by ECG and AEMD measurement by TDI are simple and practical methods to evaluate the risk of AF development in generalised anxiety disorder patients. Further studies are needed to confirm both atrial and ventricular arrhythmia development and the underlying mechanism in this patient group.

Conflict of interest: none declared

References

CORRIGENDUM TO “Protective effects of anti-oxidant supplementations on contrast-induced nephropathy after coronary angiography: an updated and comprehensive meta-analysis and systematic review”

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In the article titled “Protective effects of anti-oxidant supplementations on contrast-induced nephropathy after coronary angiography: an updated and comprehensive meta-analysis and systematic review” [1], the name of the first author was given incorrectly as Sadeh Ali-Hassan-Sayegh. The author’s name should have been written as Sadeq Ali-Hasan-Al-Saegh. The revised authors’ list is shown above. These two spelling are belonging to an author.

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