LETTER TO THE EDITOR

Reply to the Letter from Canpolat and Colleagues

Eser Açıkgoz, M.D.,* Belma Yaman, M.D.,* Sadık Kadri Açıkgoz, M.D.,†
Salih Topal, M.D.,* Yusuf Tavil, M.D.,* and Nuri Bulent Boyacı, M.D.*

From the *Cardiology Department, Gazi University Faculty of Medicine, Ankara, Turkey and †Cardiology Clinic, Türkiye Yüksek İhtisas Education and Research Hospital, Ankara, Turkey

Ann Noninvasive Electrocardiol 2015;20(1):100–101

We thank Dr. Canpolat and his colleagues for their interest and comments about our study which demonstrated the independent association of fragmented QRS (fQRS) and severity of aortic stenosis (AS).¹

In their letter, Canpolat and colleagues mentioned the importance of presence of symptoms in AS.² We also thought presence and severity of symptoms are of great importance in AS and investigated the association of fQRS with presence of manifest heart failure, which we defined as presence of New York Heart Association Class III–IV symptoms or history of hospitalization for heart failure. As a result, we found out that presence of fQRS was independently associated with manifest heart failure.¹

Canpolat and colleagues next discussed the possibility of presence of aortic regurgitation in our patients and its role in fQRS.² Although not written one by one in methods section, patients with all other severe valvular disease including aortic regurgitation were excluded from our study. Furthermore, we mentioned the exclusion of other severe valvular disease as one of the limitation of our study.¹

Canpolat and colleagues also point out the link between body mass index (BMI), metabolic syndrome (MetS) and left ventricular hypertrophy (LVH) in AS and its possible impact in analysis of AS severity.² It is known that BMI and

MetS are independently associated with LVH in patients with AS.^{3,4} In our study, we aimed to investigate the relationship of fQRS with LVH and other indices of AS rather than predictors of LVH in AS. Since our patients were already evaluated for LVH by ECG and echocardiography, and rates of hyperlipidemia, hypertension, and diabetes mellitus were similar between groups, we are not in an opinion that inclusion of BMI or MetS parameters would alter our results. Notwithstanding we agree with Canpolat and colleagues in that addition of data about BMI and MetS parameters would be more informative.

Lack of LV diastolic dysfunction parameters and biomarkers such as hs-Troponin-I and BNP is another subject discussed by Canpolat and colleagues in their letter.² As the authors, we agree with them in that LV diastolic dysfunction parameters and biomarkers like hs-Troponin-I and BNP might have a role in determining the extent of LVH and fibrosis in AS. However, we simply did not intend to use them and we think their use is out of the purpose of our study. Since none of the major guidelines oblige their routine use in evaluation of the severity of AS, we are not in an opinion that it is a limitation for our study.^{5,6}

The authors of the letters finally discuss the possible role of the number of leads with fQRS.² It was proposed that the number of leads with fQRS is an indirect indicator of myocardial fibrosis and

Address for correspondence: Eser Açıkgoz, M.D., Gazi Üniversitesi Tıp Fakültesi, Kardiyoloji AD, 06500, Beştepe, Ankara, Turkey. Fax: +90 312 3124120; E-mail: dreacikgoz@gmail.com

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DOI:10.1111/anec.12235

predictive of arrhythmic events in patients with arrhythmogenic right ventricular cardiomyopathy and response to cardiac resynchronization therapy.^{7,8} The Achilles' heel of this theory is that unlike precordial leads, limb leads are not independent and six limb leads are calculated by electrocardiographs from limb leads I and II. For instance, $III = II - I$ and $aVF = II - 1/2 I$ according to the lead equations.⁹ As a result of this, if one of the I or II has fQRS, other derived limb leads should have also so and it is meaningless to count them as separate leads with fQRS. For this reason, we did not include the number of the leads with fQRS to our study.

In conclusion, fQRS is an easily available marker of myocardial fibrosis which may have a role in determination of the severity of AS. We strongly agree with Canpolat and colleagues in that limitations of our study should be kept in mind while interpreting its results and prospective studies are needed to determine the prognostic value of fQRS in AS.

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