LETTER TO THE EDITOR

# Myocardial Fibrosis Is the Key Component of Hypertrophied Myocardium That Cause Fragmented QRS in Aortic Stenosis

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**Ann Noninvasive Electrocardiol 2015;20(5):513**

To the Editor,

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

In his letter, Dr. Eyuboglu discussed that patients with fQRS may have more severe AS and thus they should have higher left ventricle mass (LVM) and left ventricle mass index (LVMI) in our study. He concluded that presence of fQRS in these patients may be due to the left ventricular hypertrophy (LVH) rather than AS itself and inclusion of LVM and LVMI might affect the results of the study.[2] As the authors, we found the logic behind treating AS and LVH as two unrelated entity questionable. LVH is an expected result of significant AS and myocardial fibrosis is the key component of hypertrophied myocardium which causes inhomogeneous electrical activation of the myocardium resulting fQRS.[3–5] Thus, it would be nonsense to say that LVH is the cause of the fQRS but the isolated AS is not. In fact, having a sensitivity of 51%, fQRS is a poor indicator of LVH.[6] In the light of this finding, we think that histological changes such as myocardial fibrosis are more important than degree of LVH for development of fQRS complexes. So, we doubt that inclusion of LVM and LVMI would alter our results.

In conclusion, fQRS is a marker of myocardial fibrosis secondary to AS and it is associated with severity of AS.

## REFERENCES

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DOI:10.1111/anec.12295