Abnormal myocardial deformation properties in obese, non-hypertensive children: an ambulatory blood pressure monitoring, standard echocardiographic, and strain rate imaging study: reply

We thank Song very much for the interest in our work.1 In our study, we demonstrated a significant increase in left ventricular (LV) circumferential end-systolic stress, in agreement with previous studies,1-5 and a significant correlation between myocardial deformation properties, insulin levels, and HOMA, in agreement with previous studies.6

We agree with Song that we did not suggest any further hypotheses about putative mechanisms that link obesity, insulin levels, and myocardial disturbances. However, this was not the aim of our study, and several hypotheses on this topic, as he reported, have already been formulated in other papers.7-9

Conversely, the subclinical abnormalities in LV function, described in previous studies, although important, may exclusively reflect the role of comorbidities that contribute to LV dysfunction (e.g. hypertension, diabetes, coronary artery disease, and obstructive sleep apnea) as well as altered loading, especially, as conventional echo-Doppler measures are load-dependent.

In our view, the uniqueness of our study is the evaluation of the effect of obesity in a clinical model potentially able to exclude the effect of other comorbidities on ventricular function using the more sensitive ultrasonic-derived strain and strain rate imaging.

Indeed, we studied myocardial deformation properties in healthy children with excess weight who have no other clinically appreciable cause of heart disease, in whom hypertension was excluded using both office and ambulatory blood pressure measurements.

About our methodology, unfortunately from the letter, it is not clear which methodological aspect of our study needs to be clarified.

Further studies, specifically designed, are needed to better describe the pathophysiological mechanism linking obesity and abnormal LV function.

References


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Myocarditis in hypertrophic cardiomyopathy

We read with considerable interest the paper by Frustaci et al. We only wish to point out to the readership that these authors have reported a group of patients with hypertrophic cardiomyopathy (HCM) who differ distinctly in clinical profile from all other cohorts of patients with this disease reported anywhere in the world, including Italy—a country with an intense interest in HCM over many years. We refer specifically to the fact that Frustaci et al. have described 119 patients they regard as having HCM in whom fully 35% required emergency admission to the hospital for acute and profound heart failure (average age, 39; due to myocarditis in two-thirds of the cases based on endomyocardial biopsies), and also 20% who experienced sustained episodes of ventricular tachycardia. Both these disease expressions, so common in the experience of Frustaci et al., are in fact so rare within the broad HCM disease spectrum as to be virtually unheard of by most investigators.1,2 Also, the occurrence of the end-stage of HCM at 20% is fully 10 times greater than that reported by other investigators.3 It is particularly interesting that after almost 50 years of intense study, and reports consisting of literally thousands of HCM patients from around the world, a dramatic subset such as this would suddenly emerge at this time in a confined geographical area. It would be of interest if the authors would explain how and why this circumstance may have come about.

For these reasons, we must sound a note of skepticism regarding the identity of the disease that Frustaci et al. have reported, so tightly linked with acute myocarditis.

References

1. Maron BJ, McKenna WJ, Danielson GK, Kappenberger LJ, Kuhn HJ, Seidman CE, Shah PM, Spencer WH, Spirito P, ten Cate FJ,
The bias selection of HCM patients with acute instability, clearly stated in the discussion section of the article, was prompted by the aim to approach those disease manifestations most likely to be clarified by an endomyocardial biopsy study. With specific regard to myocardial inflammation in HCM, several original pathological studies have reported the observation of inflammatory infiltrates in the context of severely hypertrophied and disorganized cardiomyocytes. The recent introduction of advanced techniques, particularly immunohistochemistry for the phenotypic characterization of the inflammatory cells and polymerase chain reaction for the identification of viral genomes, have remarkably improved our ability to diagnose a myocarditic process. In contrast, the discourage-ment to an invasive study of critical patients with HCM may prevent myocarditis to be diagnosed and eventually treated. Actually, various therapeutic strategies are available including antiviral agents as interferon, immunosuppression, and immunoadsorption procedures with potential impact on recovery of cardiac function.

Skepticism toward new observations on unclarified entities risk to be an obstacle to the comprehension of new insights, if not followed by the aim to confirm or disprove them by further studies.

References

