Revista Portuguesa de Cardiologia 41 (2022) 309–310

Portuguese Society of CARDIOLOGY

Revista Portuguesa de
Cardiologia
Portuguese Journal of Cardiology
www.revportcardiol.org

EDITORIAL COMMENT

The challenge of assessing left ventricular function after repair of aortic coarctation: Can we do better?

O desafio da avaliação da função ventricular esquerda após reparação da coartação da aorta: podemos fazer melhor?

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Available online 17 January 2022

When studied in long-term follow up, a large fraction of patients who have undergone repair of coarctation of aorta (COA) have arterial hypertension,[1,2] which has been attributed to increased aortic stiffness.[3] A similar mechanism may explain the enhanced exercise-induced increase in systolic arterial pressure in this group of patients.[4] As shown by Egbe et al.[5] COA-treated patients have higher LV diastolic stiffness, impaired LV relaxation and slightly elevated LV end-diastolic pressure.

Studies of LV systolic function in patients with successful COA repair have demonstrated reduced LV global longitudinal strain (GLS), whereas LV ejection fraction (EF) was normal in the majority of patients.[6,7] As shown by Kutty et al.[6] in a study which used cardiac magnetic resonance imaging, the reduction of GLS was most marked in the subgroup of patients with LV hypertrophy, suggesting that impairment of LV systolic function may be coupled to remodeling.

The traditional method for measuring LV contractile function is by EF, which has proven very useful in patient management. An important limitation of this parameter, however, is in heart failure (HF) diagnostics, as it fails to identify systolic function in nearly one half of all HF patients, a condition named HF with preserved EF (HFpEF). One reason why EF does not identify systolic dysfunction in all patients is that ventricles with concentric hypertrophy may have small LV cavities, and therefore even a small contraction by a dysfunctional ventricle leads to normal or even supernormal EF. Furthermore, in the early phase of LV systolic dysfunction, there is often impairment predominantly of longitudinal shortening, which is measured as reduced GLS. This is presumably because myofibres that account for longitudinal shortening are located mainly in the vulnerable subendocardium. As shown by Stokke et al.,[8] EF reflects predominantly circumferential LV shortening, and is less sensitive to reductions in longitudinal shortening. Therefore, GLS appears to be a more sensitive measure than EF to identify mild systolic dysfunction and is a promising method to diagnose subclinical HF and HFpEF.[9] Whereas LV GLS is useful in HFpEF diagnostics, it does not completely solve the problem since GLS is normal in almost one half of all patients with HFpEF.

Afterload dependency of GLS and EF is a significant limitation, since elevation of afterload can lead to reductions in both parameters, which may be misinterpreted as a reduction in LV contractility. More recently, a myocardial work index was introduced as a potentially more robust measure of LV systolic function since it incorporates LV afterload.[10]

Non-invasive myocardial work can be estimated as the area of the LV pressure-strain loop, globally or for each LV segment. Since work is based on measurement of force and length rather than pressure and strain, it is an index rather than a direct measure of work as conventionally defined. Computation of the pressure-strain-loop areas requires

DOI of original article:
https://doi.org/10.1016/j.repc.2021.02.024
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https://doi.org/10.1016/j.repc.2021.12.005

inputting brachial artery systolic by the cuff method, setting valvular event times, and measuring LV longitudinal strain using speckle tracking echocardiography.

In a study presented in the current issue of the journal, Timóteo et al.[11] investigated LV systolic function in patients who had undergone COA repair in childhood, the majority through surgery. The study included a group of 42 adult patients, 64% of whom had a bicuspid aortic valve and about one half were treated for arterial hypertension. Left ventricular systolic function measured as myocardial work index, was slightly reduced in repaired COA patients, suggesting impaired LV contractility. The authors concluded that patients with repaired COA had signs of LV dysfunction with a reduction in global myocardial work index, despite normal EF and strain. There was, however, numerically lower EF and GLS in the COA group than in the reference population, but these differences did not reach statistical significance. The observations in this study illustrate how the myocardial work method may be applied to identify subclinical LV dysfunction and may provide added clinical value to EF and GLS, which are standard clinical methods for evaluation of LV contractility.

The study by Timóteo et al.[11] is somewhat limited due to its relatively small size and the use of a historical group of control patients. The authors, however, should be acknowledged for their innovative approach and attempt to improve assessment of systolic function in COA patients. It would be interesting to further explore their concepts in a larger study with direct comparison with control group of healthy individuals, and also a group of COA patients with arterial hypertension.

There is a need for more insight into how to manage patients with repaired COA when they present with LV remodeling and impairment of LV systolic and diastolic function. Studies should be conducted to explore whether these patients may benefit from more aggressive antihypertensive therapy to reduce long-term risk of cardiovascular complications. Although the myocardial work index is a promising parameter of LV systolic function, more studies are needed to define its potential superiority or added value to EF and GLS in clinical routine.

Conflicts of interest

OAS is co-inventor of the ''Method for myocardial segment work analysis'', has filed a patent on ''Estimation of blood pressure in the heart'', and has received one speaker honorarium from GE Healthcare.

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