Obese children and myocardial deformation changes: some discrepancies but a large number of common points

We read with interest the article entitled ‘Alterations of left ventricular myocardial strain in obese children’ by Labombarda et al. regarding the use of two-dimensional (2D) strain imaging to study whether severely overweight children show early abnormalities in myocardial function.

First of all we would like to point out that we published an article with a similar population and similar results 2 years ago. We also would like to make some comments with the intention to help to understand the results. In our study, we used three-dimensional wall motion tracking (3D-WMT) technology (Artida system, Toshiba Medical Systems, Japan) instead of 2D speckle tracking technology. We found a decrease in left ventricular longitudinal and circumferential strains but an increase in radial strain, with a maintained left ventricular ejection fraction. As the authors say in the article, when using the 2D strain technology, ‘radial strain is calculated perpendicularly at the longitudinal motion and is very influenced by the longitudinal out-of-plane motion, much more than the field of circumferential motion’. We think they are completely right as it is one of the main limitations of the 2D speckle tracking technology. The new 3D-WMT technology allows us to avoid this problem as it follows the speckles in a volume and not in a plane. From our point of view, this could be the reason why we found an increase in the radial strain in obese children and Labombarda et al. did not. A decrease in the longitudinal and circumferential strain might be accompanied by an increase in the radial strain in order to maintain the global left ventricular ejection fraction. This fact probably represents a compensation mechanism in early stages of disease. Early impairment of left ventricular subendocardial layers may be the reason for this reduction in the longitudinal and circumferential strain. Nevertheless, radial strain is less dependent on this subendocardial region and may increase to maintain the left ventricular ejection fraction. Keeping in mind all these concepts, we think that our results explain how a decrease in the longitudinal strain plus a decrease in the circumferential strain may coexist with a preserved left ventricular ejection fraction, as we and Labombarda et al. show in our respective results.

Finally, we would like to congratulate the authors for their excellent work, and all the data they provide for a better understanding of heart disease associated with obesity.

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References

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Obese children and myocardial deformation changes: some discrepancies but a large number of common points: reply

We thank Saltijeral et al. for their comments regarding our recent work entitled ‘Alterations of left ventricular myocardial strain in obese children’. We agree our findings are consistent with the results of their previous study. A three-dimensional myocardial strain is an undeniable step forward for quantifying volume changes in the entire cavity, overcoming limitations inherent to two-dimensional deformation imaging such as out-of-plane motion. However, a thorough validation of three-dimensional myocardial strain measurements based on three-dimensional echocardiography has yet to be conducted. Preliminary studies show reasonable correlations between true and estimated longitudinal and circumferential deformations. These studies also show that the radial strain remains an important challenge, with a lower accuracy than longitudinal and circumferential strains. For the case of apical acquisitions, as used in the study of Saltijeral et al., speckle tracking in the radial direction, perpendicular to the beam, is hampered by lower spatial resolution and signal-to-noise ratio. Several possibilities exist for overcoming these limitations: (i) segmenting the endocardium and epicardium, the latter being difficult as this area is very heterogeneous and (ii) using the rule of incompressibility to deduce the radial strain from longitudinal and circumferential strains. In both cases it is necessary to be aware that the calculation of the radial strain remains an information to be taken with caution and that, in its current form, may not reflect a physiological reality.

References

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Fractional flow reserve is a useful reference standard for myocardial perfusion studies with limitations

Plein and Motwani, in their editorial comment on a study by Ebersberger et al. in the journal, state that because the ischaemia threshold of fractional flow reserve (FFR) was initially ‘validated’ in patients with unequivocal stress-induced
ischaemia by stress SPECT/echocardiogram (ECHO), subsequent studies using FFR as a reference standard to assess the accuracy of other non-invasive methods of assessment of stress-induced myocardial ischaemia such as CMR are somewhat paradoxical and create questionable circular arguments.

I suggest an alternative conclusion based on the following reasoning: the initial ‘validation’ study was really to determine the ischaemia threshold of FFR in patients with large and severe enough stress-induced ischaemic defects unequivocally detected by a known technique, stress SPECT/ECHO. Once this was determined, FFR outperforms stress SPECT/ECHO in at least three scenarios, where it is currently useful clinically. First, the pressure wire can inherently accurately measure FFR in smaller vessels supplying smaller myocardial segments than in the initial validation study, below the detection limits of either stress SPECT/ECHO of high quality without artefact. Secondly, in patients susceptible to artefacts with either SPECT/ECHO, the accuracy of FFR remains unaffected. Thirdly, in balanced three-vessel obstructive coronary artery disease (CAD) underestimated by SPECT because it measures intersubject relative flow reserve, the accuracy of FFR remains unaffected because it measures vessel-specific relative (to assumed maximal) flow reserve.

Therefore, it is not a tautology to use FFR as a reference standard in assessing accuracy of non-invasive studies including stress CMR/SPECT/ECHO in other patient subgroups, after initial determination of the ischaemia threshold of FFR by stress SPECT/ECHO.

They list serial lesions in the same vessel as a limitation of FFR. Actually, FFR is uniquely able to determine the individual contributions of each of serial lesions by a careful pull back study to determine which lesion is significant and if stented both predict and confirm the improvement by repeat FFR post-stenting.

The real limitation of FFR as a reference standard is that its concordance for ischaemia with CFR (the basis of stress perfusion imaging) varies with the degree of the accompanying diffuse disease (DD) and microvascular dysfunction (MVD) in CAD patients by differentially affecting CFR and FFR, both decrease CFR, but DD decreases CFR and MVD increases FFR. Thus, the ischaemia threshold of FFR varies from ~0.65 in a theoretical model without DD/MVD to ~0.75–0.8 in CAD patients. The discordance between FFR and CFR by both invasive or non-invasive methods is ~40% in CAD patients, but in selected patients with single-vessel CAD presumably with mild DD/MVD, the concordance is much higher at r = 0.87. This can only be overcome by simultaneous measurement of coronary flow as with a hyperaemic stenosis resistance index.

The gold standard is a vessel-specific reliable absolute maximal myocardial blood flow measurement and positron emission tomography is the most developed today.

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References


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Fractional flow reserve is a useful reference standard for myocardial perfusion studies with limitations: reply

We are grateful for the comments by Dr Ramanna, which add important further detail to the issues discussed in our recent editorial in the European Heart Journal – Cardiovascular Imaging. We would like to emphasize that it was not our intention to devalue the established clinical role of fractional flow reserve (FFR), but rather to highlight possible limitations of its use as an endpoint for non-invasive imaging studies. Determining myocardial ischaemia in vivo remains challenging, as also acknowledged by the investigators of the FFR validation studies, who stated in their seminal publication that: ‘It is thus difficult to establish the value of any new method to assess the functional severity of coronary artery disease, because there is no single unequivocal or gold standard.’ The investigators therefore used a combined imaging and functional endpoint, and only considered defects as ischaemic where perfusion improved after revascularization. Clearly, this was a very stringent definition of ischaemia, and was probably the best achievable at the time.

However, we maintain that in the absence of a true in vivo reference standard for ischaemia, it is problematic to first validate a new test (FFR) against established yet imperfect tests (non-invasive imaging), and then to use the same new test (FFR) to validate modifications of the established tests (non-invasive imaging). Any clinical tool has potential artefacts and limitations. For FFR, some of these were listed in our editorial, and more have been described elsewhere. Importantly, invasive and imaging tests suffer from different limitations and may not deliver identical assessments of ischaemia—regardless of whether absolute values or thresholds are used to define pathology. Comparing tests that use similar methodology with another may reduce discordance and we therefore suggest, in agreement with Dr Ramanna’s final sentence, that absolute myocardial blood flow quantification with positron emission tomography (PET) and cardiovascular magnetic resonance (CMR) imaging, which have both been validated in animal and clinical studies, may be a better reference standard for imaging studies of ischaemia than FFR.

With regard to specific limitations of FFR and imaging noted by Dr Ramanna, we accept that FFR is valuable in interrogating smaller vessels, however, newer imaging tests, especially CMR, are less likely to miss small perfusion defects in view of their better in-plane spatial resolution compared with single-photon emission computed tomography (SPECT)—so an ‘out-performance’ of FFR against all imaging for small areas of myocardial ischaemia remains debatable. The same is true for multi-vessel disease, which is more accurately detected by CMR and PET than by SPECT or stress echocardiography, especially when quantitative analysis is added to visual interpretation.