HCM Deserves Better—Ditch The 16-Segments
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A wind of change is sweeping across the cardiomyopathy landscape. Advanced high-resolution imaging kitted in some cases with artificial intelligence and powered by accessible high performance computing, is redefining established disease concepts, classifications and why not, patient care. Hypertrophic cardiomyopathy (HCM) has seen its entire fluid-electro-mechanical model and its developmental origins, thrown into question by the sheer force of these technologies. The heartbeat phenomenon of HCM is being re-learnt thanks to computational flow dynamics(1), mono and bidomain models of propagating electrical activity(2), and structure/diffusion tensors appraising myocardial microstructural dynamics(3).

In the current issue, Piras et al.(4) elegantly load geometric morphometrics and 4-dimensional (4D) trajectory analysis onto 3D speckle tracking echocardiography to characterise myocardial deformation in a brand new way. Quite boldly they ditch the time-honoured 16-segment bulls-eye plot of mean radial strains and opt for a continuous mapping technique, liberating us from those annoying artificial boundary conditions (the segments) that we have grown accustomed to impose on our cardiac slices. Segment-wise analysis and averaged values may have been all-right ten years ago, but our imaging cameras (echocardiography, cardiovascular magnetic resonance, computerized tomography, etc.) have evolved tremendously and given the resolution we are now capable of, the notion of collapsing a segmental chunk of myocardial data richness into a single number, seems incomprehensible. Yet this is how we are reporting our clinical scans currently–according to an old-fashioned, biased, not fit-for-purpose, 16-segment cardiac model.

To study the myocardial deformation in controls and in patients with overt and subclinical HCM, Piras et al. scrutinised 41,504 landmarks (2,594 at each of 16 homologous time points along the cardiac cycle) per participant by 3D echocardiography. They then sub-selected landmarks of significant deviance for more complex analysis that included principal component analysis and machine learning for HCM classification. The end-result is that they discovered aberrant apical myocardial deformation in subclinical HCM, with sufficient sensitivity and specificity, to distinguish a subclinical HCM heart from a control heart, even in spite of limited numbers.

The jump from 16 to 41,504 myocardial landmarks, with all its attendant risks of over-fitting, potentially signals the advent of personalised computational medicine for HCM. We have been preparing long for this–exciting technologies from inline quantitative perfusion mapping of the myocardium(5), to multi-parametric tissue relaxometry, to tissue phase mapping are now at our finger tips, pervading our clinics and insinuating themselves into our reports.

How much ground-breaking biology and pathophysiology have we missed this past decade, on account of our locked-down 16-segment view of the HCM heart? Perhaps, finally we are being unshackled.
References:


