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Ejection fraction: a measure of desperation?

Charlotte H Manisty, Darrel P Francis

Ejection fraction is the most widely used measure of left ventricular systolic function, but why? It was initially adopted before the introduction of echocardiography, when ventricular function was assessed by ventriculography at cardiac catheterisation. To avoid the huge effort of calibrating ventriculographic volumes, ejection fraction was born as a quick way of quantifying function.

In this edition of Heart, Maciver and Townsend report their findings from a mathematical model that investigates the separated effects of changes to left ventricular (LV) mass and to longitudinal shortening, on ejection fraction (see article on page 446). They show that the increased radial wall thickness in LV hypertrophy means that ejection fraction may be preserved despite impaired long-axis shortening. This supports the growing evidence that ejection fraction alone may be an insufficient descriptor of systolic function.

As cardiology is no longer bereft of rapid, calibrated measures of systolic function with the availability of tissue Doppler velocity, strain and strain rate, perhaps we can progress from limiting our assessment to ejection fraction.

Assessment of LV systolic function is used to predict morbidity and mortality, in the diagnosis of heart failure, and also to enable or preclude patients from receiving a variety of potentially beneficial procedures including chemotherapy, cardioverter-defibrillator implantation and valve replacement.

LV systolic function was initially quantified from contrast cineventriculography using ejection fraction (the ratio of stroke volume divided by end-diastolic volume) before the introduction of echocardiography and other imaging techniques. Ejection fraction has the advantage of not requiring calibrated measurements of cardiac volumes, and so avoids the complexity and errors associated with volume measurements.

Although three-dimensional echocardiography and magnetic resonance imaging produce more reproducible measurements of ejection fraction than two-dimensional echocardiography or ventriculography, no measure of ejection fraction takes into account regional abnormalities unless several myocardial segments are involved. Many other quantitative techniques are now available for the assessment of both regional and global LV systolic function, which are to be found on most echocardiographic machines and which are quick, easy and reproducible to perform. These include tissue velocity, strain and strain rate by tissue Doppler imaging, which can be used to assess both longitudinal and radial shortening in isolated LV segments.

A series of recent studies have shown that longitudinal systolic function assessed using measures derived from tissue Doppler imaging is reduced in many patients with normal ejection fractions but clinical evidence of heart failure (HFNEF), suggesting that ejection fraction alone is not sufficiently sensitive to pick up early disease.

RELATIONSHIP BETWEEN LONGITUDINAL AND RADIAL CONTRACTILITY IN HYPERTENSVIE HEART DISEASE

In the natural history of hypertension, even before the development of LV hypertrophy, there are changes to longitudinal systolic function that may be accompanied by symptoms and signs of heart failure. Comparisons between hypertensive patients with HFNEF and those with asymptomatic left ventricular hypertrophy (LVH) show that despite similar ejection fractions, the patients with HFNEF had lower systolic tissue Doppler velocities and greater LV mass indices. Greater LV mass therefore enhances ejection fraction allowing depressed longitudinal function to go unnoticed unless measured specifically.

HOW A MATHEMATICAL MODELLING APPROACH CAN DEAL WITH THE QUESTION OF HFNEF IN HYPERTENSIVE HEART DISEASE

Where multiple interacting parameters are involved, simple intuition cannot always determine the effect that a change in one physiological measure may have on another. Mathematical models provide a way of assessing the independent effect of individual parameters.

The mathematical analysis performed by Maciver and Townsend allows a systematic examination of the separate effects of LV wall thickening and reduced longitudinal velocity on ejection fraction and stroke volume. They found that in the presence of LVH with preserved external cardiac dimensions, a reduction in LV long-axis shortening would not be accompanied by a concomitant reduction in ejection fraction, despite a fall in stroke volume.

RELATIONSHIP BETWEEN LV WALL THICKNESS AND EJECTION FRACTION

This important message from Maciver and Townsend’s model can be summarised by applying the relationship, internal volume = external volume – LV wall volume, to the definition of ejection fraction:

Ejection fraction = (End-diastolic internal volume – end-systolic internal volume)/end-diastolic internal volume

Thus if the external cardiac volumes remain constant, then any increase in LV wall volume must necessarily raise the ejection fraction.

INFLUENCE OF LV REMODELLING ON VENTRICULAR FUNCTION

The key physiological difference between patients with HFNEF and systolic heart failure with reduced ejection fractions is ventricular remodelling with consequent LV dilatation. It might therefore be interesting if the authors could expand their current model to investigate the independent effect of increased LV internal cavity dimensions on ejection fraction in patients with LVH.

THE FUTURE FOR THE ASSESSMENT OF LV FUNCTION

Neither the British nor American Societies of Echocardiography have yet updated their guidelines to include measurements of longitudinal systolic function, and with the increasing evidence that longitudinal systolic function reduces before any fall in ejection fraction, we suggest...
that myocardial Doppler systolic velocity should be measured routinely.

In addition to its use in the diagnosis of heart failure, a marker of longitudinal systolic function would give valuable prognostic data for the management of hypertension. Angiotensin-converting enzyme (ACE) inhibitors delay progression toward overt heart failure in hypertensive subjects with asymptomatic LV systolic dysfunction, and reduction of LVH prevents clinical heart failure, independently of changes to blood pressure. Thus in clinical heart failure, independently of heart failure treatment, but may not receive it for systolic function, even despite only borderline hypertension, drug treatment should be started early to prevent heart failure.

CONCLUSIONS

Ejection fraction emerged as a time-saving descriptor of systolic function because routine ventriculography is uncalibrated. Nowadays, ejection fraction is surely only justified as a sole index of cardiac function in situations of desperation when there are no available quantities calibrated in physical units. In LV hypertrophy, the increased radial thickening will necessarily raise ejection fraction, even in the presence of reduced longitudinal systolic function. This means that there must be a group of patients with symptoms of heart failure and impaired longitudinal function who might benefit from heart failure treatment, but may not receive it because they have LVH that raises their ejection fraction into the “normal” range.

We hope that one day we will be no more likely to classify patients with heart failure by ejection fraction than we classify patients with cancer by haemoglobin level. We would encourage clinicians to include a measurement of systolic longitudinal function in their reports of routine echocardiograms—especially in those patients with LVH.

Competing interests: None.

REFERENCES