

If It Is Not There, It Cannot Be Measured

Si no está, no se puede medir

MARIO J GARCÍA, MD

Cardiovascular disease remains the leading cause of death in the world. Coronary artery disease (CAD) affects about one third of the adult population, and its sequelae, myocardial infarction and heart failure (HF) are among the leading causes of the rapidly growing health care expenditure to society. A great emphasis has been placed over the last 2 decades in improving our strategies to increase the detection of CAD in both asymptomatic subjects at risk and in those who present for the evaluation of typical or atypical symptoms. Stress testing with or without imaging has been traditionally used in this setting. Although the results of stress testing remain very useful to establish prognosis and to select patients who may benefit from myocardial revascularization, the ability of stress ECG, myocardial perfusion imaging and/or stress echocardiography to establish the diagnosis of CAD has been historically poor, and many patients undergo unnecessary coronary angiography while others demonstrate significant CAD after a normal stress test result (1). Accordingly, computed tomographic coronary angiography (CTCA) has been proposed as an alternative method to evaluate patients with chest pain, but in large multicenter studies, its application has not led to superior outcomes when compared to stress testing (2).

Among non-invasive methods to evaluate for CAD, stress echocardiography is one of the safest and most cost-efficient, reasons why it has been widely used in Latin America. The detection of changes in global and regional contractility from rest to immediate post-stress is used to determine the presence of obstructive CAD, since changes in contractility appear early during the ischemic cascade. However, these changes are often subtle and transient, and in many patients, wall motion abnormalities that occur at peak exercise disappear by the time post-exercise images are collected. Many investigators have proposed the use of bicycle ergometry or modified treadmill protocols to acquire images at peak heart rate during active exercise, but

these are not easy to acquire in many patients. Thus, in order to increase the accuracy of stress echocardiography, quantitative methods that use tissue Doppler or speckle tracking have been developed and implemented. We, (3) as well as others, (4) have demonstrated that these methods can enhance the detection of CAD, despite some of their limitations: angle dependency for tissue Doppler and low temporal resolution for speckle tracking. Since low frame rates are a limiting factor for speckle tracking when images are obtained at higher heart rates, many investigators have proposed evaluating images at rest or post-peak stress under the hypothesis that ischemic memory may cause persisting subtle reduction of regional contractility caused by repetitive stunning.

In this issue of the *Revista Argentina de Cardiología*, two publications by Gastaldello et al. (5) and Lowenstein et al. (6) investigate the prevalence of abnormal regional strain at rest and post-stress in patients with obstructive CAD. In Gastaldello et al.'s article, the longitudinal strain (LS) values obtained at rest in 62 patients with positive stress echo were found to be similar to those obtained from a normal control group. In their study, patients with abnormal global or regional function by visual analysis on the resting study were excluded. This could explain why their results differed from others previously reported that included a more heterogeneous population (7). Lowenstein et al. (6) analyzed 101 ischemic segments in 21 patients who also had normal wall motion at baseline. In their study, segmental LS significantly decreased from baseline and quickly normalized after 3 minutes, demonstrating that stunning is transient and is not followed by ischemic memory in most cases. In fact, LS values increased above the baseline values immediately after recovery.

In essence, quantitative methods intend to provide a numerical value to what we already perceive, and they rarely identify abnormalities that otherwise could be detected. Myocardial contractility is an en-

REV ARGENT CARDIOL 2016;84:302-303. <http://dx.doi.org/10.7775/rac.v84.i4.9201>

SEE RELATED ARTICLE: *Rev Argent Cardiol* 2016;84:330-335. <http://dx.doi.org/10.7775/rac.v84.i4.8530>

Rev Argent Cardiol 2016;84:351-354. <http://dx.doi.org/10.7775/rac.v84.i4.9062>

Address for reprints: Mario J García, MD - Montefiore Medical Center - 111 East 210th Street - Bronx, NY 10467 - Tel: 718-920-4172
e-mail: mariogar@montefiore.org

¹Chief of Cardiology, Montefiore Medical Center
Professor of Medicine and Radiology, Albert Einstein College of Medicine - Bronx, NY

ergy dependent process that depends on the balance between oxygen supply and demand. Once demand increases during stress, the myocardium shifts from aerobic to anaerobic metabolism. Changes in intracellular pH, calcium overload and other metabolic processes eventually result in a reduction of contractility. These changes rapidly reverse when workload decreases unless there is a severe and sustained reduction in blood flow, such as that seen during acute coronary syndromes. Accordingly, prior studies that have reported abnormal strain values at rest in patients with severe obstructive CAD and/or persisting post-stress abnormalities late into recovery have been most likely biased by the inclusion of patients with prior myocardial infarction, unstable symptoms and/or severe ischemia inducing repetitive stunning.

Conflicts of interest

None declared.

(See authors' conflicts of interest forms in the website/Supplementary material).

REFERENCES

1. Patel MR, Peterson ED, Dai D, Brennan JM, Redberg RF, Anderson HV, et al. Low diagnostic yield of elective coronary angiography. *N Engl J Med.* 2010;362:886-95. <http://doi.org/d8ccd7>
2. Douglas PS, Hoffmann U, Patel MR, Mark DB, Al-Khalidi HR, Cavanaugh B, et al; PROMISE Investigators. Outcomes of anatomical versus functional testing for coronary artery disease. *N Engl J Med.* 2015;372:1291-300. <http://doi.org/46m>.
3. Yamada E, Garcia M, Thomas JD, Marwick TH. Myocardial Doppler velocity imaging--a quantitative technique for interpretation of dobutamine echocardiography. *Am J Cardiol.* 1998;82:806-9, A9-10.
4. Moonen M, Lancellotti P, Zacharakis D, Pierard L. The value of 2D strain imaging during stress testing. *Echocardiography.* 2009;26:307-14. <http://doi.org/cb5wd6>
5. Gastaldello N, Merlo P, Amor M, Alasia D, Galello M, Rousse M y cols. Longitudinal Strain at Rest Does not Predict Stress Echo Results. *Rev Argent Cardiol* 2016;84:XXX-XXX.
6. Lowenstein J, Gastaldello N, Merlo P, Galello M, Rousse M, Darú V. Longitudinal Strain at Rest does not Predict Stress Echo Results. *Rev Argent Cardiol* 2016;84:351-354.
7. Asanuma T, Fukuta Y, Masuda K, Hioki A, Iwasaki M, Nakatani S. Assessment of myocardial ischemic memory using speckle tracking echocardiography. *JACC Cardiovasc Imaging.* 2012;5:1-11. <http://doi.org/fxkkwp>