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Review Article
Published: 26 Jan, 2018

Journal of Cardiology Forecast

How to Improve the Interpretation of Dobutamine Stress Echocardiography?

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Introduction

Dobutamine stress echo is well validated, non-invasive test for diagnosis of myocardial ischemia and viability by increasing the heart rate and myocardial oxygen demand so precipitate ischemia in presence of a flow limiting coronary artery stenosis, but interpretation of the test depends mainly on subjective analysis of wall motion abnormality which is not easy especially with rapid heart rate at peak dose of Dobutamine in addition to the known limitation of 2 D analysis that it cannot differentiate passive from active motion.

Ischemia is not only decreases the amplitude of contraction but also slows its onset and velocity as well as delaying the onset of relaxation so deformation analysis provides the additional advantage of being able to assess the timing parameters of systolic shortening.

So the role of adjunctive echo modalities is important in this setting as it will provide objective and quantitative evaluation of the myocardial response to stress.

There are multiple adjunctive echo modalities to improve the interpretation of dobutamine stress echo but here the discussion will focus on quantitative parameters.

Tissue Doppler Image (TDI)

Tissue Doppler uses filter that bypass the velocity of blood which is high velocity and low amplitude and records the velocity of tissue which is the opposite (low velocity and high amplitude) Figure 1.

- All tissue Doppler derived measurement; velocity, strain and strain rate depend on assessment of the longitudinal motion of the heart Figure 2.
- Strain is a measure of tissue deformation and is defined as the change in length normalized to the original length. The rate at which this change occurs is called strain rate.
- Myocardial velocity decreases form base to apex but strain and strain rate are uniformly distributed in the myocardium.
- Tissue doppler velocity imaging detects motion so may be influenced by overall heart motion, cardiac rotation, and tethering from adjacent segments. On the other hand; local deformation indices such as TDI derived strain and strain rate imaging less affected by tethering and translation motion.
- The normal response of the myocardium is incremental increase of systolic tissue velocities, strain and strain rate with dobutamine infusion Figure 3. This response is blunted in areas with induced ischemia Figure 4 and biphasic in viable myocardium.
- Changes in strain precede those in wall motion or tissue velocity during dobutamine stress and can differentiate stunned from ischemic myocardium. Strain rate correlates with regional myocardial perfusion during dobutamine stress [1].
- Strain-rate improves the sensitivity for prediction of functional recovery after revascularization.

But TDI-derived measurements are sensitive to angulation issues like other Doppler techniques. During acquisition, every effort should be taken to align the tissue direction parallel with the beam direction.

Speckle Tracking Echocardiography (STE)

- The interaction of ultrasound with the myocardium produces unique acoustic patterns,

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Received Date: *03 Dec 2017*
Accepted Date: *15 Jan 2018*
Published Date: *26 Jan 2018*

Citation: *Mostafa SA. How to Improve the Interpretation of Dobutamine Stress Echocardiography?. J Cardiol Forecast. 2018; 1(1): 1001.*

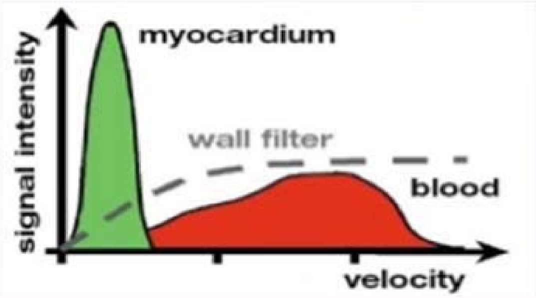
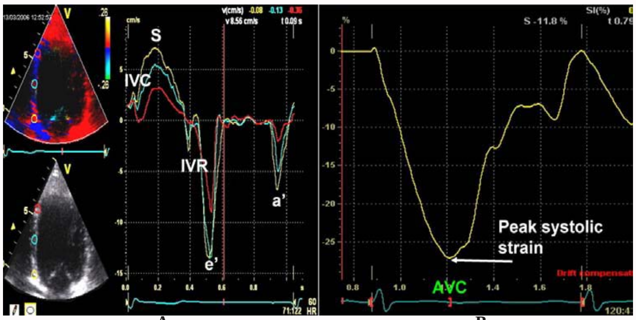
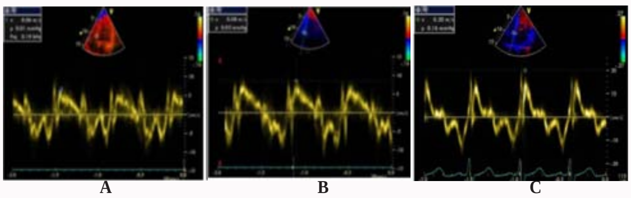


Figure 1: Principle of tissue Doppler image.



A B

Figure 2: (A): Color coded tissue Doppler image shows 3 waves and 2 time intervals; (B): tissue Doppler derived strain shows peak systolic strain. (IVC: Isovolumetric Contraction S: Systolic Activation, IVR: Isovolumteric Relaxation, e´ early diastolic wave, a´ late diastolic wave) [2].



A B C

Figure 3: Tissuedoppler derived velocity shows incremental increase in velocity of S wave from 7m/sec at resting (A) To 14m/sec at low dose; (B) To 20 m/sec at peak dose (c)

or "speckles" Two-dimensional speckle-tracking echocardiography (STE) is based on frame-by-frame tracking of tiny echo-dense speckles within the myocardium and subsequent measurement of LV deformation not only in the longitudinal but also in the radial and circumferential directions.

- Speckle tracking is angle independent because it is not based on the Doppler principle and less susceptible to translational motion and tethering, which lead to better evaluation of stress echo images

- Because speckle tracking is automated and allows the generation of bull's eye plots of longitudinal segmental strain beside segmental curves of strain.

- It is performed at much lower frame rates (40 to 90 frames per second) and may not be as accurate in timing mechanical events as Doppler-based imaging (100 to 250 frames per second) [4] (Figure 5).

Analysis of strain curve should include

- End-systolic strain: the value at end-systole (ES).

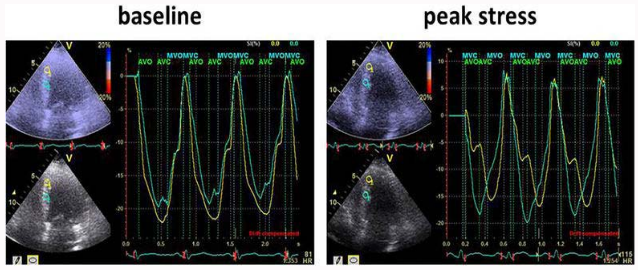
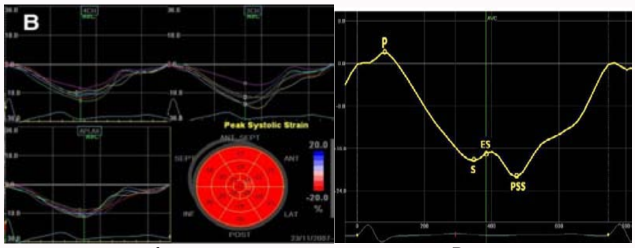


Figure 4: If there is any doubt about regional function, tissue Doppler strain is the fastest means of regional function analysis in a stress echocardiogram. **Right panel:** the yellow region of interest shows reduced systolic strain and marked post-systolic shortening at peak stress, suggesting inducible ischemia. This becomes particularly obvious if compared to the green region of interest which shows a normal strain curve. Note that the overall strain amplitude is similar in both segments. **Left panel:** inducible ischemia is distinguished from a pre-existing abnormality by demonstrating that the yellow region of interest shows a normal strain curve at rest. AVO, AVC, MVO, MVC – aortic and mitral valve opening and closure [3].



A B

Figure 5: (A) Bulls eye speckle tracking; (B) strain curve by speckle tracking.

- Peak systolic strain: the peak value during systole (S).

- Positive peak systolic strain (P).

- Peak strain: the peak value during the entire heart cycle. The peak strain may coincide with the systolic or end-systolic peak, or may appear after aortic valve closure (AVC) (post systolic strain)

Post systolic shortening occurs in segments where peak shortening occurs after aortic valve closure, it must be noted, that PSS of a minor extend can be found in normal hearts, particularly at the apex and base of the inferior, septal and anteroseptal walls.

Criteria to define pathologic PSS include

- Transient PSS (occurring during and resolving after ischemia),

- The presence of decreased systolic function (peak systolic strain>-7%.

- In case of moderately reduced systolic function (−7%> peak systolic strain >−18%).

- The presence of PSS >20% of peak strain.

- PSS that occurs >90 ms after aortic valve closure.

As for tissue Doppler imaging, speckle tracking increased with stress and decline with ischemia and biphasic for viable myocardium in addition to unique differentiation of transmral and non-transmural ischemia (Figure 6); in subendocardial ischemia only longitudinal strain is reduced but other strain components (particularly circumferential strain) remain unaltered but in transmural infarction there is reduction in multidirectional strain.

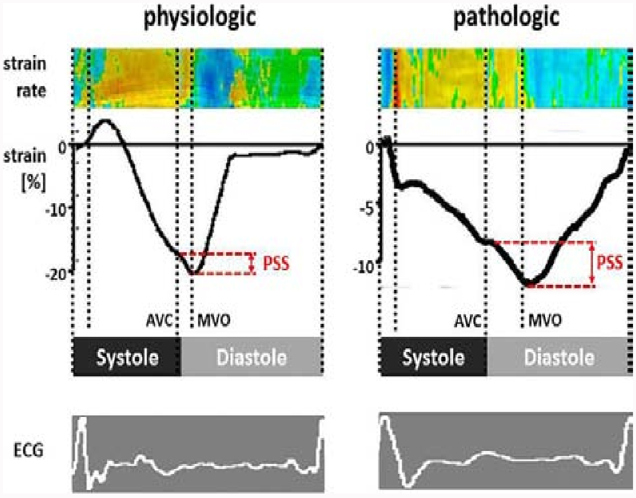


Figure 6: Colour coded strain rate curved M-modes (top) and strain curves (middle) in 2 myocardial segments both showing post-systolic shortening (PSS). **Left panel:** physiologic PSS in an apical septum of a normal heart. Peak systolic strain exceeds −18% and PSS is less than 20% of systolic shortening. **Right panel:** pathologic PSS in a partially scarred segment post infarction. Peak systolic strain is reduced to −10.1% while post systolic strain is increased to 39% of systolic strain. AVC - Aortic valve closure; MVO - mitral valve opening [5].

3D Echo

3D echo is feasible during stress echo and provide better quantitative assessment of the left ventricular volume and function and offers shorter aquestation time beside the ability of 3DE to identify wall motion abnormalities in the apical region so higher sensitivity for the left anterior descending coronary artery territory.

Conclusion

Objective and quantitative assessment of regional and global myocardial response to stress is feasible by adjunctive echo techniques to improve interpretation of Dobutamine stress echo.

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