Evaluation of Heart Function by Tissue Velocity and Strain (rate) Imaging

E.S. Zegers MD[1]

G.A. Pop MD, PhD[1]

L. Kapusta MD, PhD[2].

Radboud University Nijmegen Medical Center, Heartcenter (670), P.O. Box 9101

6500 HB Nijmegen, The Netherlands, tel: 00-31-(0)24-3614533, fax: 00-31-(0)24-3540537

1. Department of Cardiology

2. Children's Heart Center

Keywords: tissue velocity imaging, echocardiography, strain (rate) imaging

Summary

Tissue velocity imaging has become a useful non-invasive method that complements conventional echocardiographic techniques in the assessment of left ventricular function. Myocardial velocities can be determined in a variety of clinical conditions, such as evaluation of regional left ventricular systolic and diastolic function in cardiomyopathies as well as in ischemic heart disease. Strain and strain-rate are applications derived from tissue velocity imaging data. Strain imaging shows great promise as a means to access myocardial function. A novel approach to quantify regional left ventricular function from routine gray-scale two-dimensional echocardiographic images, known as speckle tracking, calculates myocardial strain independent of angle of incidence. Speckle tracking strain images complement traditional tissue velocity imaging providing a more complete description of cardiac dynamics.

Tissue velocity imaging

Echocardiography is the predominant, non-invasive diagnostic modality, for the evaluation of left ventricular function as well as for the assessment and quantification of valvular heart lesions. However, assessment of regional cardiac dysfunction at rest and during stress with this technique remains subjective and semiquantitative.[1,2]

Tissue velocity imaging (TVI) is an echocardiographic diagnostic method that provides quantitative data about regional myocardial function. Isaaz et al. first reported the use of TVI in the evaluation of left ventricular (LV) function with pulsed Doppler recordings of intramyocardial velocities in the posterior wall.[3] TVI was introduced by McDicken et al. in 1992.[4] The use of color TVI, including M-mode and two-dimensional (2-D) imaging, was further developed by Sutherland et al. in 1994.[5]

TVI is non-invasive, does not yield radiation damage, is user friendly and therefore repeatable. Color TVI is the color Doppler technique modified to colorize the myocardium, in order to depict the velocities of myocardial motion. Myocardial motion may be studied either radially (endo- epicardial direction of motion in parasternal imaging) or longitudinally (cardiac base to apex direction of motion in apical imaging).

The technique is based on the Doppler principle of velocity estimation of structures moving with respect to the ultrasound transducer. The high-pass wall filters that are usually used to eliminate the low-velocity and high-amplitude signals of myocardial walls for detection of blood flow velocities in conventional Doppler/color flow modalities are bypassed for TVI. In addition, thresholding is used to enhance low-velocity myocardial signals and eliminate the blood flow signals within the cardiac chambers.[4-6]

In the 2-D image, each pixel displays one color representing the direction and magnitude of the wall velocity along the scan line.[7] As with all Doppler, the direction of motion should be as close as possible to the scan line direction, and careful attention needs to be paid to gain settings, because excessive gain leads to spectral broadening and overestimation of velocities. Due to its relatively high spatial resolution, TVI provides valuable information on regional myocardial wall motion during different intervals of the cardiac cycle, namely during systole, early diastole and late diastole. In addition, by using the single-gated pulsed-wave tissue Doppler option, myocardial velocity vs. time curves (sonograms or velocity-time waveform displays) can be recorded from locations selected in the 2-D TVI images. Off-line processing enables accurate quantification of regional myocardial motion in both systole and diastole, from multiple sites, with an image dataset being acquired within minutes by an experienced sonographer.

TVI uses standard color coding to depict both velocity and direction of movement, with myocardial motion away from the transducer coded in blue and towards the transducer in red.[5] This information can be displayed as a standard 2-D image (Figure 1) or as a M-mode TVI, where temporal resolution is enhanced. Differences in myocardial velocity from endocardium to epicardium are thus displayed, leading to the recognition of myocardial velocity gradients (MVG).[8,9]

Contraction of the LV occurs towards a central point situated at two-thirds along a long-axis line from the level of the mitral annulus to the apex.[10] Normal myocardial motion includes three components, along the radial, longitudinal and circumferential axes. It is not yet possible to asses all three axis simultaneous by any of the echo techniques. The longitudinal shortening is an integral part of global contractile function, and it is thought to play a role in contributing to the LV ejection fraction.[11-13] Using single-gated pulsed-wave TVI, three (sometimes four) distinct velocity peaks in systole and diastole are observed[14] (Figure 2):

- Systolic myocardial velocity may show two components in some patients, representing isovolumic contraction (S1) and the peak systolic (shortening) velocity (S2, very often titled as S)

- Peak early diastolic myocardial relaxation velocity (De)
- Peak late diastolic myocardial relaxation velocity, associated with atrial contraction (Da)

Several studies aiming at determining normal values in adults were carried out.[3,15-21] The early diastolic myocardial velocities are usually the highest. There is little evidence of a significant difference in velocities between sexes. Increased age has been associated with a gradual fall in myocardial velocities both in systole and early diastole,[15,22-24] increase in late diastolic myocardial velocity, as well as a fall in the ratio De/Da.[25-26] Diastolic myocardial velocities vary with heart rate and phase of respiratory cycle at which they are measured.[27]

Using the parasternal view, normal values for the systolic and early diastolic transmyocardial velocity gradients within myocardial segments were assessed (radial axis).[8,9,18,28] The transmyocardial velocity gradient was obtained, showing a gradual increase in radial shortening velocity from subepicardial to the subendocardial region, independent of age.[18] When imaging from the apex (longitudinal axis) longitudinal contraction and relaxation velocities of the LV myocardium are highest in the basal segments and decrease progressively when moving the measurement site toward the apex .[16,17]

Strain and Strain Rate

Applications derived from TVI data are strain and strain rate (SR). Strain and SR are measures of deformation and rate of deformation that are basic descriptors of both the nature and function of cardiac tissue. [29-30] The term strain was first used in relation to the heart by Mirsky and Parmley to describe myocardial deformation.[31] Strain is defined as tissue deformation due to applied or internal force (stress), normalized to tissue original length. Strain reflects the functional properties of the tissue. It is a dimensionless measure of regional lengthening and shortening of the myocardium. As ventricles contract, muscle shortens in the longitudinal and circumferential dimensions (a negative strain) and thickens or lengthens in the radial direction (a positive strain). SR is the speed at which deformation (i.e. strain) occurs (deformation per time unit). SR is given by the formula SR= (v2-v1) / distance, where v1 and v2 are velocities of myocardium at two points separated by distance; this gives the difference in tissue velocity per unit length.[32] The strain rate is positive during elongation and negative during shortening. Thus, systolic myocardial thickening is positive strain rate. Strain and SR are not influenced by the function of adjacent myocardial segments nor of the global heart movements, and are less dependent upon the direction of shortening and lengthening relative to the scan line.

Like TVI, strain and SR throughout the cardiac cycle may be represented as a cyclic curve of a single point over time (Figure 3) or as a color coded imaging (SRI) over M-mode imaging. Two parts of the heart muscle may have the same amount of strain, but different strain rates.

Strain rate by speckle tracking in grey scale images

A novel approach to quantify regional LV function from routine gray-scale 2D echocardiographic images, known as speckle tracking, calculates myocardial strain independent of angle of incidence. [33-36]

The basic principle of speckle tracking is using the interference of the reflected ultrasound, giving rise to an irregular - random – 'speckle' pattern. The speckles follow the motion of the myocardium so when the myocardium moves from one frame to the next, the position of this fingerprint will shift slightly, remaining fairly constant .Thus, if a region (kernel) is defined in one frame, a

search algorithm will be able to recognise the region with the most similar speckle pattern in the next frame and hence, to find the new position of the kernel. This has been shown to be feasible in flow and strain rate imaging (Figures 4 and 5).

Applications

TVI has become a useful non-invasive method that complements conventional echocardiographic techniques in the assessment of LV myocardial velocities in a variety of clinical conditions, such as evaluation of regional LV systolic and diastolic function in cardiomyopathies as well as in ischemic heart disease.[37,38]

The greatest clinical contribution of tissue Doppler to date has almost certainly been in the assessment of left ventricular diastolic function. TVI offers the ability to measure regional diastolic function, with pulsed-wave TVI allowing quantification of diastolic myocardial velocities from a small sample volume placed in the region of interest.

A related application of TVI to routine clinical practice is the assessment of left ventricular filling pressure.[39] Regional LV function is assessed by color-coded 2-D tissue velocity mapping or position of a sample volume within the myocardium at different sites. The color-coded 2-D tissue velocity map enables immediate appreciation of cardiac asynchrony, e.g. akinetic segments are shown by dark colors representing low tissue velocity, dyskinetic segments may be seen to exhibit colors opposite to those of adjacent normal segments.

Patients with ischemic heart disease often have abnormal LV diastolic function. Color 2-D and color M-mode TVI are used for quantification of systolic myocardial velocities in these patient, both at rest and with pharmacological stress testing. [40-42] TVI enables measurement of regional systolic and diastolic myocardial velocities and furthermore, is particularly useful in the identification of abnormalities of LV diastolic relaxation.

Both isovolumic contraction and isovolumic relaxation phases represent situations in which the afterload to left ventricular contraction and relaxation is very low. This is a privileged situation for myocardial contraction to express itself, especially in cases of stunning or hibernation, in which the low or absent myocardial thickening during ejection phase, i.e. against a very high afterload, becomes possible during the isovolumic contraction phase. Myocardial velocity during IVC decreases to zero or inverts direction of motion in cases of myocardial ischemia. This aspect seems peculiar with the delayed onset of contraction in response to ischemia. Myocardial velocity during IVR appears increased, late-peaking, and prolonged by ischemia. This aspect, called post-systolic thickening (if radial) and shortening (if longitudinal) seems also peculiar with the delayed onset of relaxation in response to ischemia. [43-45] Several studies have demonstrated that De is often reduced. De/Da ratio < 1, and regional isovolumic relaxation times are prolonged in patients with LV segmental dysfunction due to ischemia. [43-46] However, Da velocity remains unchanged, suggesting that ("post systolic") early diastolic changes reflect an active, energy requiring relaxation process, whereas late diastolic filling merely reflects passive stretch after atrial contraction.[41]

TVI is also useful in assessing the severity of LV asynchrony in patients with LBBB with heart failure as well as in evaluating the pacing effects on long-axis function in these patients.[47,48]

Change of velocity and patterns of velocity propagation along the heart walls can be easily observed using the 2-D TVI image sequences. The application in electrophysiology enables recognition of the site of pre-exitation in the Wolff-Parkinson-White syndrome and detecting the origin of other rhythm abnormalities .[49] Bartel et al. demonstrated that using TVI patterns increases the detection of vegetation in infective endocarditis as well as of thrombus formations.[50]

Peak systolic pulsed TVI of the tricuspid annular velocity provides a simple, rapid, and non-invasive tool for assessing right ventricular systolic function in patients with heart failure.[51]

Conclusion

Tissue velocity imaging has become a useful non-invasive method that complements conventional echocardiographic techniques in the assessment of LV myocardial velocities in a variety of clinical conditions, such as evaluation of regional LV systolic and diastolic function in cardiomyopathies as well as in ischemic heart disease. Strain and strain-rate are applications derived from TVI data. Strain imaging shows great promise as a means to access myocardial function. A novel approach to quantify regional LV function from routine gray-scale 2-D echocardiographic images, known as speckle tracking, calculates 2-D myocardial strain, i.e. independent of angle of incidence. Speckle tracking images thus provides a more complete description of cardiac dynamics.

Although not widely applied in routine echocardiography, tissue velocity imaging can be used to assess global and regional systolic and diastolic LV function and to identify abnormal LV relaxation in a variety of conditions. This technology should be understood and training recommended on the basis of the promising future use of this Doppler technology.

References:

1. Hoffman R, Hanrath P. Stress echocardiography: the scourge of subjective interpretation. *Eur Heart J* 1995;16:1458-59.
2. Hoffman R, Lethen H, Marwick T, et al. Analysis of inter-institutional observer agreement in interpretation of dobutamine stress echocardiograms. *J Am Coll Cardiol* 1996;27:330-36.
3. Isaaz K, Thompson A, Ethevenot G, et al. Doppler echocardiographic measurement of low velocity motion of the left ventricular posterior wall. *Am J Cardiol* 1989;64:66-75.
4. McDicken WM, Sutherland GR, Morane CM, et al. Colour Doppler velocity imaging of the myocardium. *Ultrasound Med Biol* 1992;61:651–54.
5. Sutherland GR, Stewart MJ, Groundstroem KW, et al. Color Doppler myocardial imaging: a new technique for the assessment of myocardial function. *J Am Soc Echocardiogr* 1994;7:441-58.
6. Sutherland GR, Bijnens B, McDicken WN. Tissue Doppler echocardiography: historical perspective and technological considerations. *Echocardiography* 1999;16:445-57.
7. McDicken WN, Hoskins PR, Moran CM, Sutherland GR. New technology in echocardiography I: Doppler techniques. *Heart* 1996;75:2-8.
8. Fleming AD, Xia X, McDicken WN, et al. Myocardial velocity gradients detected by Doppler imaging. *Br J Radiol* 1994;67:679-88.
9. Uematsu M, Miyatake K, Tanaka N, et al. Myocardial velocity gradient as a new indicator of regional left ventricular contraction: detection by a two-dimensional tissue Doppler imaging technique. *J Am Coll Cardiol* 1995;26:217-23.
10. Ingels NB Jr, Daughters GT, Stinson EB, et al. Evaluation of methods for quantitating left ventricular segmental wall motion in man using myocardial markers as a standard. *Circulation* 1980;61:966-72.
11. Jones CJ, Raposo L, Gibson DG. Functional importance of the long axis dynamics of the human left ventricle. *Br heart J* 1990;63:215-20.
12. Gulati VK, Katz WE, Follansbee WP, et al. Mitral annular descent velocity by tissue Doppler echocardiography as an index of global left ventricular function. *Am J Cardiol* 1996;77:979-84.
13. Sutherland GR, Lange A, Palka P, et al. Does Doppler myocardial imaging give new insights or simply old information revisited? *Heart* 1996;76:197-99.
14. Oki T, Tabata T, Mishiro Y, et al. Pulsed Tissue Doppler Imaging of left ventricular systolic and diastolic wall motion velocities to evaluate differences between long and short axes in healthy subjects. *J Am Soc Echocardiogr* 1999;12:308-13.
15. Garcia MJ, Rodriquez L, Ares M, et al. Myocardial wall velocity assessment by pulsed Doppler tissue imaging: characteristic findings in normal subjects. *Am Heart J* 1996;132:648-56.
16. Pai RG, Gill KS. Amplitudes, durations, and timings of apically directed left ventricular myocardial velocities: I. Their normal pattern and coupling to ventricular filling and ejection. *J Am Soc Echocardiogr* 1998;11:105-11.
17. Galiuto L, Ignone G, DeMaria AN. Contraction and relaxation velocities of the normal left ventricle using pulsed-wave tissue Doppler echocardiography. *Am J Cardiol* 1998;81:609-14.
18. Palka P, Lange A, Fleming AD, et al. Age-related transmural peak mean velocities and peak velocity gradients by Doppler myocardial imaging in normal subjects. *Eur Heart J* 1996;17:940-50.
19. Miyatake K, Yamagishi M, Tanaka N, et al. New method for evaluating left ventricular wall motion by color-coded tissue Doppler imaging: in vitro and in vivo studies. *J Am Coll Cardiol* 1995;25:717-24.
20. Palka P, Lange A, Fleming AD, et al. Doppler tissue imaging: myocardial wall motion velocities in normal subjects. *J Am Soc Echocardiogr* 1995;8:659-68.
21. Donovan CL, Armstrong WF, Bach DS. Quantitative Doppler tissue imaging of the left ventricular myocardium: validation in normal subjects. *Am Heart J* 1995;130:100-4.

22. Onose Y, Oki T, Mishiro Y, et al. Influence of aging on systolic left ventricular wall motion velocities along the long and short axes in clinically normal patients determined by pulsed tissue Doppler imaging. *J Am Soc Echocardiogr* 1999;12:921-26.

23. Sohn DW, Chai IH, Lee DJ, et al. Assessment of mitral annulus velocity by Doppler tissue imaging in the evaluation of left ventricular diastolic function. *J Am Coll Cardiol* 1997 ;30:474-80.

24. Alam M, Wardell J, Andersson E, et al. Characteristics of mitral and tricuspid annular velocities determined by pulsed wave Doppler tissue imaging in healthy subjects.
J Am Soc Echocardiogr 1999;12:618-28.

25. Henein M, Linqvist P, Francis D, et al. Tissue Doppler analysis of age-dependency in diastolic ventricular behaviour and filling: a cross-sectional study of healthy hearts (the Umea General Population Heart Study). *Eur Heart J* 2002;23:162-71.

26. Garcia MJ, Thomas JD, Klein AL. New Doppler echocardiographic applications for the study of diastolic function. *J Am Coll Cardiol* 1998;32:865-75.

27. Nilsson B, Bojö L, Wandt B. Influence of body size and age on maximal diastolic velocity of mitral annulus motion. *J Am Soc Echocardiogr* 2002;15:29-35.

28. Pellerin D, Veyrat C. Quantitative analysis of tissue Doppler data. *Echocardiography* 1999;16:473-80.

29. Thomas H, Marwick MD. Measurement of strain and strain rate by echocardiography. *J Am Soc Echocardiogr* 2006;47:113-27.

30. Gilman G, Khandheria BK, Hagen ME, et al. Strain rate and strain: a step-by-step approach to image and data acquisition. *J Am Soc Echocardiogr* 2004;17:1011-20.

31. Mirsky I, Parmley WW. Assessment of passive elastic stiffness for isolated heart muscle and the intact heart. *Circ Res* 1973;33: 233-43.

32. Heimdal A, Stoylen A, Torp H, et al. Real-time strain rate imaging of the left ventricle by ultrasound. *J Am Soc Echocardiogr* 1998;11:1013-19.

33. Bohs LN, Trahey GE. A novel method for angle independent ultrasonic imaging of blood flow and tissue motion. *IEEE Trans Biomed Eng* 1991;38:280-86.

34. Bohs LN, Friemel BH, Trahey GE. Experimental velocity profiles and volumetric flow via two-dimensional speckle tracking. *Ultrasound Med Biol* 1995;21:885-98.

35. Kaluzynski K, Chen X, Emelianov SY, et al. Strain rate imaging using two-dimensional speckle tracking. *IEEE Trans Ultrason Ferroelectr Freq Control* 2001;48:1111-23.

36. Amundsen BH, Helle-Valle T, Edvardsen T, et al. Noninvasive myocardial strain measurement by speckle tracking echocardiography: validation against sonomicrometry and tagged magnetic resonance imaging. *J Am Coll Cardiol* 2006;47:789-93.

37. Kapusta L, Thijssen JM, Groot-Loonen J, et al. Tissue Doppler imaging in detection of myocardial dysfunction in survivors of childhood cancer treated with anthracyclines.
Ultrasound Med Biol 2000;26:1099-108.

38. Palka P, Lange A, Donnelly JE, et al. Differentiation between restrictive cardiomyopathy and constrictive pericarditis by early diastolic Doppler myocardial velocity gradient at the posterior wall. *Circulation* 2000;102:655-62.

39. Ommen SR, Nishimura RA, Appleton CP, et al. Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: A comparative simultaneous Doppler-catheterization study. *Circulation* 2000;102:1788-94.

40. Wilkenshoff UM, Sovany A, Wigstrom L, et al. Regional mean systolic myocardial velocity estimation by real-time color Doppler myocardial imaging: a new technique for quantifying regional systolic function. *J Am Soc Echocardiogr* 1998;11:683-92.

41. Garcia-Fernandez MA, Azevedo J, Moreno M, et al. Regional diastolic function in ischaemic heart disease using pulsed wave Doppler tissue imaging. *Eur Heart J* 1999; 20: 496–505.

42. Derumeaux G, Ovize M, Loufoua J, et al. Doppler tissue imaging quantitates regional wall motion during myocardial ischemia and reperfusion. *Circulation* 1998;97:1970-7.

43. Palmes PP, Masuyama T, Yamamoto K, et al. Myocardial longitudinal motion by tissue velocity imaging in the evaluation of patients with myocardial infarction.
J Am Soc Echocardiogr 2000;13:818-26.

44. Rambaldi R, Bax JJ, Rizzello V, et al. Post-systolic shortening during dobutamine stress echocardiography predicts cardiac survival in patients with severe left ventricular dysfunction. *Coron Artery Dis* 2005;16:141-5.

45. Citro R, Galderisi M. Myocardial postsystolic motion in ischemic and not ischemic myocardium: the clinical value of tissue Doppler. *Echocardiography* 2005;22:525-32.

46. Edvardsen T, Urheim S, Skulstad H, et al. Quantification of left ventricular systolic function by tissue Doppler echocardiography: added value of measuring pre- and postejection velocities in ischemic myocardium. *Circulation* 2002;105:2071-77.

47. Ansalone G, Giannantoni P, Ricci R, et al. Doppler myocardial imaging to evaluate the effectiveness of pacing sites in patients receiving biventricular pacing. *J Am Coll Cardiol* 2002;39:489-99.

48. Suffoletto MS, Dohi K, Cannesson M, et al. Novel speckle-tracking radial strain from routine black-and-white echocardiographic images to quantify dyssynchrony and predict response to cardiac resynchronization therapy. *Circulation* 2006;113:960-68.

49. Eder V, Marchal C, Tranquart F, et al. Localization of the ventricular preexcitation site in Wolff-Parkinson-White syndrome with Doppler tissue imaging. *J Am Soc Echocardiogr* 2000;13:995-1001.

50. Bartel T, Muller S, Nesser HJ, et al. Usefulness of motion patterns indentified by tissue Doppler echocardiography for diagnosing various cardiac masses, particularly valvular vegetations. *Am J Cardiol* 1999;84:1428-33.

51. Meluzin J, Spinarova L, Bakala J, et al. Pulsed Doppler tissue imaging of the velocity of tricuspid annular systolic motion; a new, rapid, and non-invasive method of evaluating right ventricular systolic function. *Eur Heart J* 2001:2:340-8.

Figures

Figure 1:

Tissue velocity imaging uses standard color coding to depict both velocity and direction of movement, with myocardial motion away from the transducer coded in blue and towards the transducer in red. This figure shows a 2-dimensional image of a heart failure patient with left bundle-branch block.

Figure 2:

Longitudinal pulsed wave Doppler of the myocardium, 4 chamber view; the sample volume is positioned at the basal level of the interventricular septum. S1 = peak systolic myocardial velocity during isovolumic contraction; S = peak systolic myocardial velocity; De = peak early diastolic myocardial velocity; Da = peak late diastolic myocardial velocity. Tissue velocity imaging shows abnormal relaxation (Da>De).

Figure 3:

Longitudinal strain versus time curves in a heart failure patient with left bundle-branch block. Dyssynchrony is shown as the difference in timing of peak strain from interventricular septum (yellow) and lateral (light blue) segment.

Figure 4:

An example of longitudinal strain versus time curves in a normal control subject from the apical 4 chamber view. Longitudinal strain was calculated by speckle tracking from multiple circumferential points over a cardiac cycle. These data were averaged to 6 strain versus time plots to represent standard segments. The curves are color-coded by the defined myocardial regions as depicted in the figure (yellow-basal septum; red-midseptum; green-apical septum; purple-apicolateral; dark blue-midlateral; light blue-basolateral). Time to peak strain in a normal subject occurs synchronously over a very narrow time range.

Figure 5:

An example of longitudinal strain versus time curves in a heart failure patient with left bundle-branch block. Longitudinal strain was calculated by speckle tracking and averaged to 6 strain versus time plots to represent standard segments. The curves are color-coded by the defined myocardial regions as depicted in the figure (yellow-basal septum; red-midseptum; green-apical septum; purple-apicolateral; dark blue-midlateral; light blue-basolateral). An example of dyssynchrony is shown as the difference in timing of peak strain from earliest (yellow) and latest (light blue) segment.

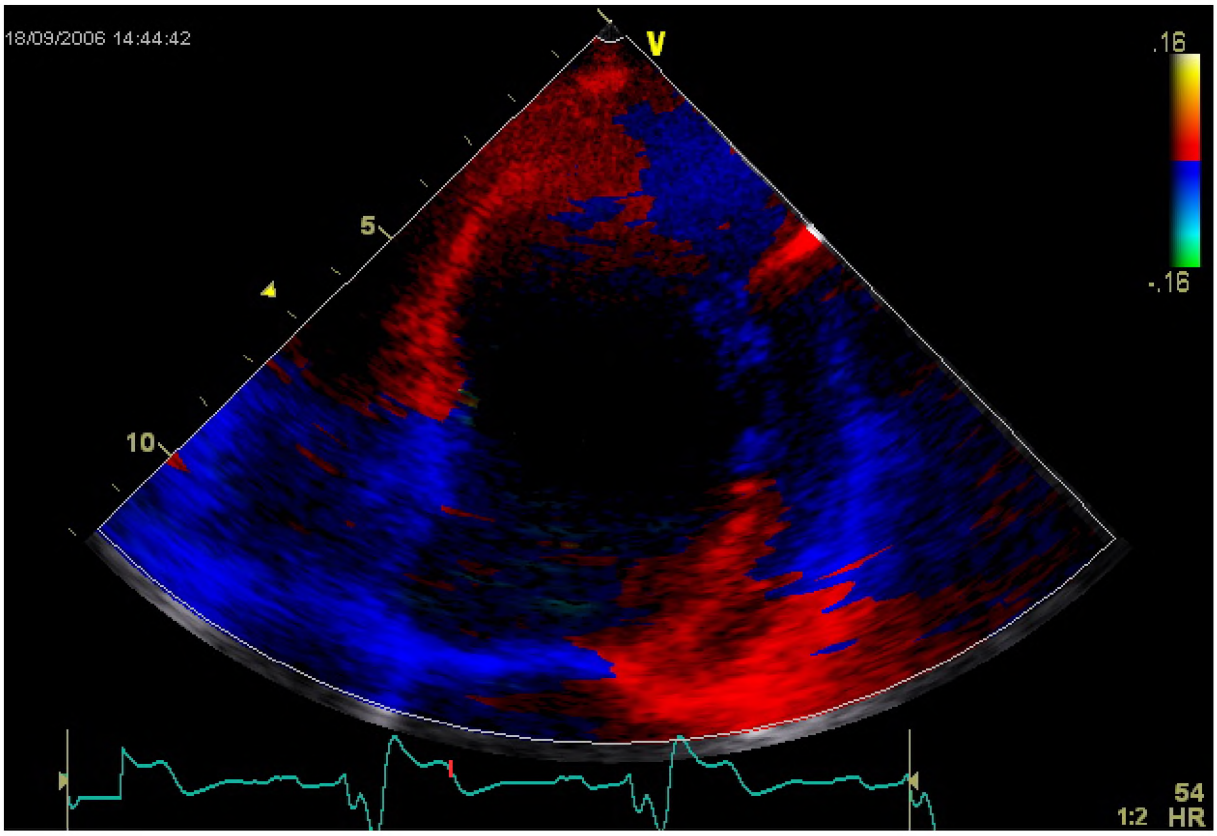

Figure 1

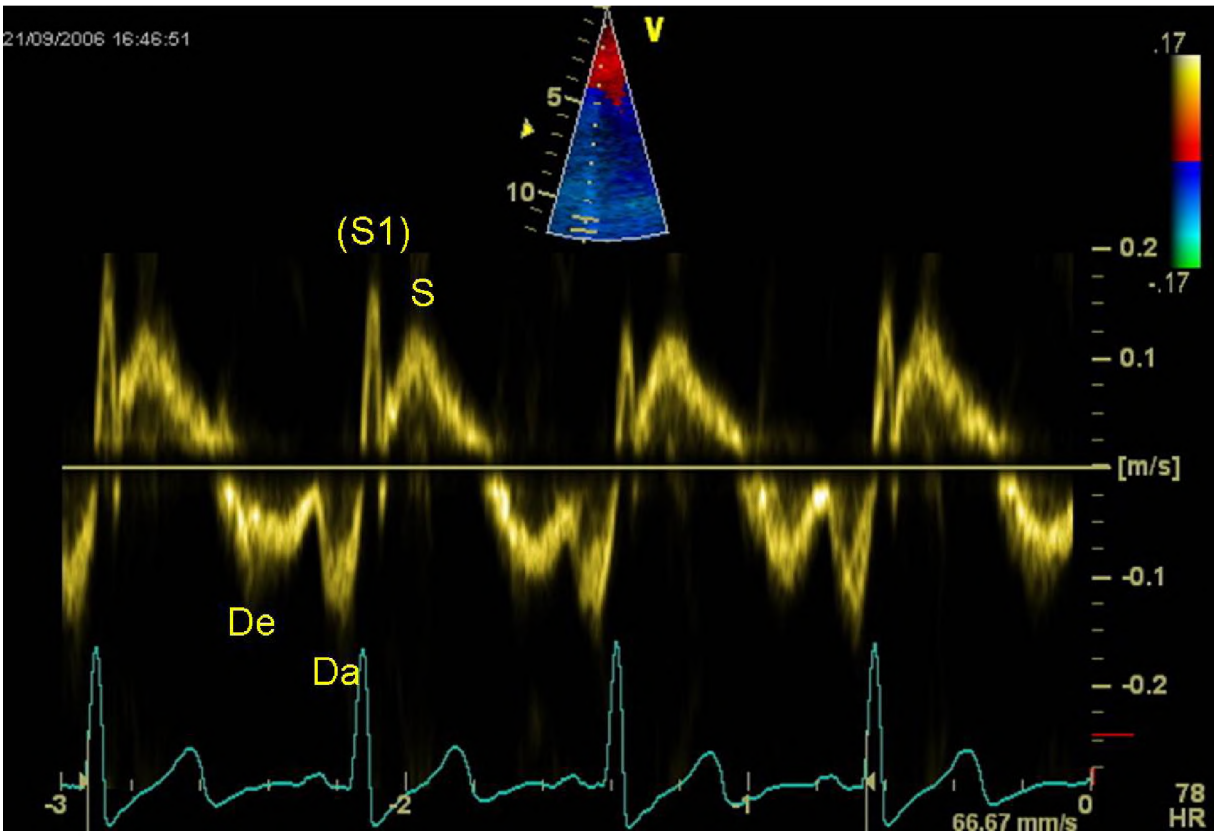

Figure 2

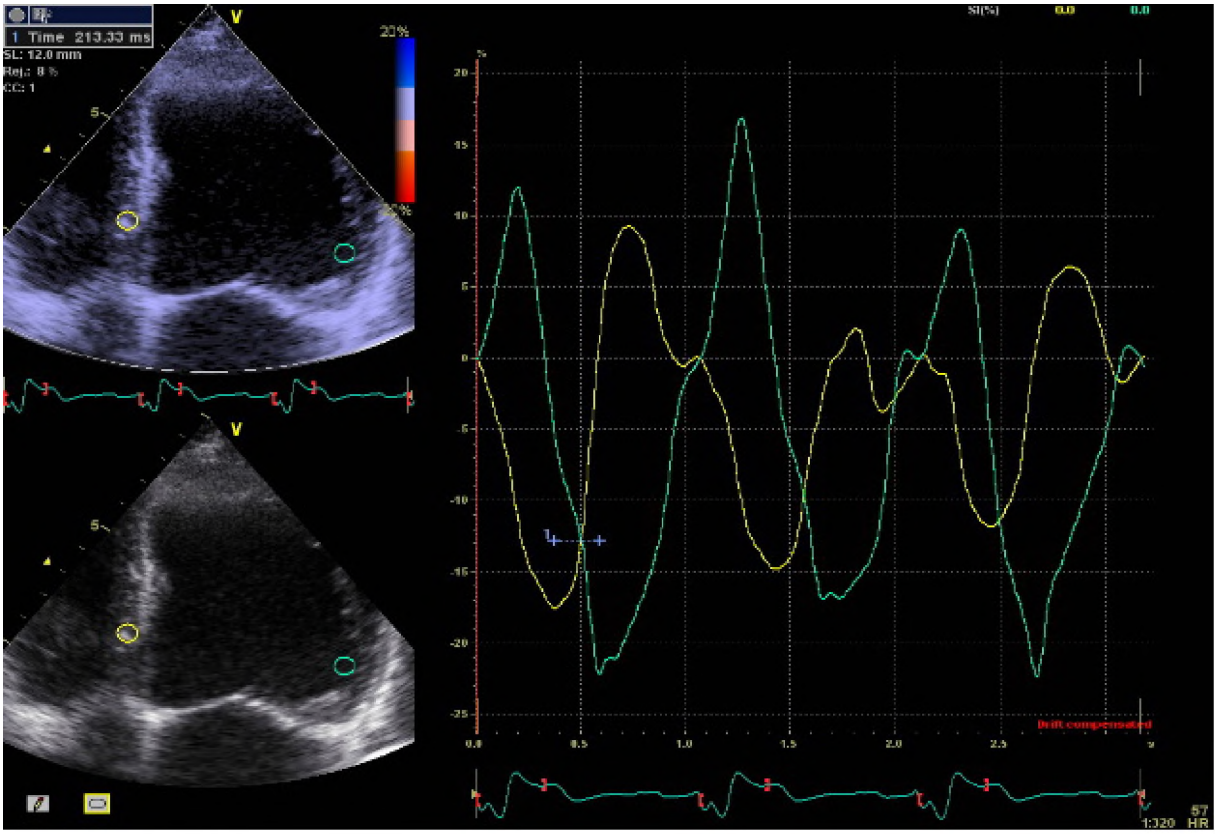

Figure 3 and Figure 4

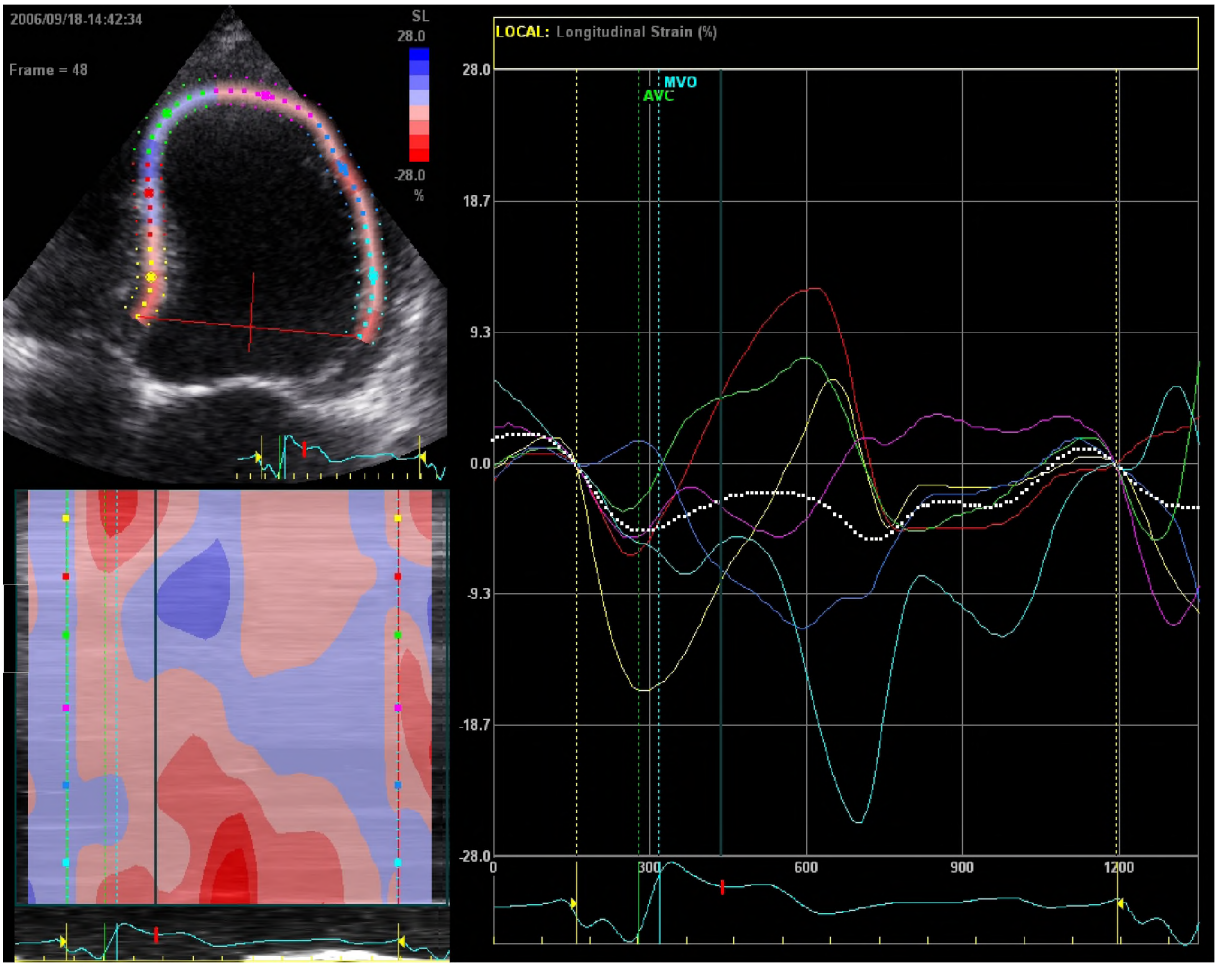
2006/09/18-14:42:34
Frame = 48
SL
28.0
-28.0
%
LOCAL: Longitudinal Strain (%)
MVO
AVC
28.0
18.7
9.3
0.0
-9.3
-18.7
-28.0
0
300
600
900
1200

Figure 5