Strain og strain rate
klinisk bruk og praktisk demonstrasjon

Charlotte Björk Ingul
Lege, Dr. med., Postdoc
Inst. for sirkulasjon ogbildediagnostikk, NTNU

Ultrasound for deformation

Quantitative analysis of segmental myocardial deformation
(mechanics = pathology)

Global motion
v₁ = v₂
Deformation
v₁ ≠ v₂

Deformation estimation =
motion estimation
+ post-processing

Courtesy: J. D’Hooge

Strain estimation scheme

The quantitative assessment of regional myocardial function remains an important goal in clinical cardiology

Velocities Strain rate Strain
Δ
v₁ v₂ ε

Calculate spatial gradient Integrate temporally

The quantitative assessment of regional myocardial function remains an important goal in clinical cardiology

Myocardial velocity and strain rate/strain

Spatial derivation of velocity

Shortening

No change

Elongation

Temporal integration

Strain %
Strain rate s⁻¹

Normal -1.5 -1.0 -20 -15
Hypokinesia -1.0 0 -15 0
Akinesia 0 0
Dyskinesia + values + values

Strain and strain rate

Strain
-deformation relative to its original length
-shorting (compression) in systole is negative strain and lengthening (stretching) in diastole positive strain
-calculated as the time integral of strain rate, most often using end-diastole as reference, and is a dimensionless quantity

Strain rate
-means deformation rate and reflects how fast regional myocardial shortening or lengthening occurs
-calculated from myocardial Doppler velocities (V₁ and V₂) measured at two locations separated by a distance (L)
equals the instantaneous spatial velocity gradient and has units of sec⁻¹: SR = (V₂-V₁)/L

Regional systolic function
Peak diastolic strain rate

Peak diastolic strain rate E
2.30 s⁻¹

Peak diastolic strain rate A
1.38 s⁻¹

(young, healthy)

Pitfall: angle dependency

Limited strain data

Meaningful data can only be acquired in the following segments:

- Longitudinal velocity/deformation
- Radial velocity/deformation
- Circumferential velocity/deformation

Courtesy: J. D’Hooge

Not all strain components can be assessed for all LV segments

Sector LV, ROI size and position

Half strain length outside ROI, not shown

Strain length

4 mm  12 mm default  20 mm

Tracking of ROI
Lateral (spatial) resolution

Reverberation

Reverberation

Angle deviation alignment beam, segment

Sample filtering Gaussian averaging

Drift compensation strain

Drift compensation off
Linear compensation
Reset at every cycle
Cine-compound

Average of 2 cycles
Cycle 1-2
Average of 3 cycles
Cycle 1-3

Automated analysis of strain and SR

- Landmark detection (a), endocardial detection (b-c) and segment border marker placement are automatic (d)
- After the segment border markers are placed, the locations can be manually adjusted
- Having determined the segment border markers in end-diastole, tracking can be used to find the location of the markers in all frames of the cardiac cycle

Automated analysis of strain and SR

- Feasible, time-saving and more accurate than conventional wall motion scoring
- The automated methods define the region of interest and objective traces are obtained as there is no possibility of searching for a suitable curve.
- Feasibility is lower than for manual analysis
- Increase accuracy compared to wall motion score for dobutamine stress echocardiography and to be a stronger predictor for the prognosis of all cause mortality.

Combination of TDI and speckle tracking

- Axial tracking using TDI
- Lateral tracking using a sum of absolute differences (SAD) speckle tracking algorithm
- TDI tracking of a marker at a given frame consists of calculating the displacement (in axial direction) of that marker from the velocity at that location.
- B-mode intensity based speckle tracking is performed in the lateral direction
Myocardial infarction- recovery

Strain rate imaging can quantify early contractility improvement due to stunning recovery after AMI. Peak systolic strain rate (SRs) in infarcted segments increased significantly from day 1 to 7, but not after day 7.

Post-systolic shortening disappeared mainly between 7 days and 3 months, probably due to fibrosis and reduced elasticity.

Diastolic function is relatively less reduced in the acute phase than systolic, but with no subsequent recovery.

AND NOW TO OUR STRESS TEST!

START

I WANT MY MONEY NOW!

“You never help at home!”

“I'M RUNNING AWAY”

SRI during dobutamine stressecho

Do we need SRI for interpretation of DSE

• The cardiac cycle is a complex motion
• Need to be an expert to interpret DSE by conventional WM
• So yes we need quantitative measurements as an adjunctive to WM
• Need to be an expert in SRI to be used in DSE?
• Basic understanding necessary because of pitfalls
How to obtain good data

Before scanning optimize the following settings
- PRF
- Image - adjust sector width and depth, to cover only the left ventricle, frame rate above 100 for systole (higher for if IVc, diastole)
- Velocity, minimum 16 cm/s (aliasing)
- Protocol, 3 cine loops
- Second harmonic strain rate

During scanning
- Apical views optimize for best alignment, remember angle deviation, keep walls aligned with ultrasound beam
- Don’t move probe during scanning

Pitfalls in SRI interpretation
- Low frame rate
  - May miss peaks
  - Averaging not possible
- Probe movement during scanning
  - Prevents cine compound (average of 2-3 cardiac cycles)
- Misaligned areas
  - To measure in a misaligned area you will pick up the wrong strain rate component
- Artifacts
  - Misinterpretation if not recognized
  - Reverberations common! Use CAMM to reveal them, change of color to opposite

Optimal parameter for stress induced ischemia, clinical studies
- Bjork Ingul Bjork Ingul et al European Heart Journal 2005; Vol.26(Abstract Supplement):230; SRs, increased sensitivity compared to WMS (87 vs. 75%), AUC 0.90 (cut-off -1.3 s⁻¹) (n=197)
- Hanekom (European Heart Journal 2005;Vol.26 (Abstract Supplement):210);
  - SRs may improve specificity of DSE, AUC 0.78 (cut-off -0.95 s⁻¹) (n=207)
- Voigt (Circulation 2005);
  - PSS strain index optimal parameter, AUC 0.90 (cutoff 35%), sens 82%, spec 85%
  - semi-quantitative by CAMM (strain rate) increased sensitivity (86 vs. 81%) and specificity (96 vs. 82%)(n=44)

Strain rate and strain normal values

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Peak</th>
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</thead>
<tbody>
<tr>
<td>Strain rate s⁻¹</td>
<td>-1.4 (0.4)</td>
<td>-2.6 (0.7)</td>
</tr>
<tr>
<td>Strain %</td>
<td>-18.4 (5.8)</td>
<td>-18.2 (5.4)</td>
</tr>
</tbody>
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- 110 patients a normal response to DSE
- age >70, SBP >160, HR <140, use of beta-blocker therapy had significantly lower SRs However, beta-blockade was the only independent predictor of SRs
- a different normal range (-2.4 ± 0.7 s⁻¹) should be considered for patients on beta-blockers.

Normal response to DSE by SR CAMM
- Baseline HR 64 Low-dose HR 87 Peak HR 110

Normal response to DSE by SR traces
- Baseline SR -1.2 s⁻¹ Low-dose SR -2.5 s⁻¹ Peak SR -3.5 s⁻¹
Normal response to DSE by strain traces

Apical ischemia, traces at peak

Baseline strain 26%  Low-dose strain 35%  Peak strain 32%

Strain rate  Strain

CAMM SR septum

Vertical longaxis  Horizontal longaxis

stress Tc-tetrofosmin myocardial perfusion scintigraphy reveals an apical perfusion defect

rest septum lateral

2 ch 4 ch

Staylen/Dahle

Conclusion

• Use SRI in addition to WM, never without!
• Always CAMM (SR) of all walls in the three apical views
• Strain rate and strain traces where your WM is suspicious
• Clinical studies have shown SRI (SR, CAMM, PSI)
  – Increased sensitivity (ischemia)
  – Increased specificity (ischemia)
  – Increased sensitivity (viability)
  – Incremental value on prognostic information on mortality to WM