NEXT IMAGING OF CARDIAC MECHANICS

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I. **TISSUE:** Myocardial Mechanics and Deformation Imaging

II. **FLUID:** Blood dynamics and Flow-Tissue Interaction
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II. **FLUID:** Blood dynamics and Flow-Tissue Interaction
The advent of speckle tracking has open the possibility of evaluating the myocardial mechanics in its deeper details (in principle)

HAS THIS TECHNOLOGY REFLECTED IN A BETTER UNDERSTANDING OF LV MECHANICS?

Many *related* indicators are used *independently* to describe the heart function
Many related indicators are used to describe the heart function (the same heart).

Are they technically interchangeable?

Which one best describes LV “longitudinal function”? 

Which one works best in clinical practice?

Annulus M-mode

Global Strain curve

Mathematically, they’re nearly equivalent

Clinically, reproducibility makes a difference!

Left Ventricle Longitudinal Deformation Assessment by Mitral Annulus Displacement or Global Longitudinal Strain in Chronic Ischemic Heart Disease: Are They Interchangeable?

Ola Gjesdal, MD, Trond Vartdal, MD, Einar Hopp, MD, Ketil Lunde, MD, Torunn Haugstvedt, MD, Truls Thaale, MD, Hans-Jørgen Smith, MD, PhD, and Thor Edvardsen, MD, PhD, Oslo, Norway

JASE 2009;22:823-830

Thorstensen et al. (HUNT Study) EJE 2010;11:149-156.
Many related indicators are used to describe the heart function (the same heart).

**Table 2** Mean left ventricular circumferential and radial peak systolic segmental strain values calculated from 60 healthy subjects aged 39±15 years by Hurlburt et al.¹⁵

<table>
<thead>
<tr>
<th>LV segment (short axis view at a basal level, just below mitral valve)</th>
<th>Mean peak systolic circumferential strain (%)+SD</th>
<th>Mean peak systolic radial strain (%)+SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>−24±6</td>
<td>39±16</td>
</tr>
<tr>
<td>Lateral</td>
<td>−22±7</td>
<td>37±18</td>
</tr>
<tr>
<td>Posterior</td>
<td>−21±7</td>
<td>37±17</td>
</tr>
<tr>
<td>Inferior</td>
<td>−22±6</td>
<td>37±17</td>
</tr>
<tr>
<td>Septal</td>
<td>−24±6</td>
<td>37±19</td>
</tr>
<tr>
<td>Anteroseptal</td>
<td>−26±11</td>
<td>39±15</td>
</tr>
</tbody>
</table>


\[ \text{St}_c = \left( \frac{D_{\text{Sys}} - D_{\text{Dia}}}{D_{\text{Dia}}} \right) \]

\[ D_{\text{Sys}} = D_{\text{Dia}} \times (1 + \text{St}_c) \]

In a normal heart with \( D_{\text{dia}} = 55\text{mm} \)

<table>
<thead>
<tr>
<th>St(_c)</th>
<th>D(_{\text{Sys}}) [mm]</th>
<th>EF % (Teicholz)</th>
</tr>
</thead>
<tbody>
<tr>
<td>-21%</td>
<td>43.45</td>
<td>42%</td>
</tr>
<tr>
<td>-23%</td>
<td>42.25</td>
<td>45%</td>
</tr>
<tr>
<td>-25%</td>
<td>41.25</td>
<td>49%</td>
</tr>
</tbody>
</table>

Two dimensional speckle tracking echocardiography: basic principles

Hermann Blessberger and Thomas Binder

*Heart* 2010 96: 716-722

Congruence between different measures must be always verified and ensured.
Conclusions: A transmural gradient exists in circumferential strain and torsion, with higher values in the subendocardial layer. It might be reduced when systolic function is impaired. Therefore, the multi-layer approach of 2D speckle tracking imaging provides further information on assessment of myocardial diseases.

Table 2
Comparison of circumferential strain in normal controls and patients with heart diseases.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Normal control</th>
<th>ACS</th>
<th>RVA pacing</th>
<th>SHF</th>
</tr>
</thead>
<tbody>
<tr>
<td>ε-circum-18-endo, %</td>
<td>−25.7 ± 4.1</td>
<td>−24.1 ± 4.6</td>
<td>−22.3 ± 6.6</td>
<td>−10.6 ± 3.2</td>
</tr>
<tr>
<td>ε-circum-18-epi, %</td>
<td>−18.7 ± 3.8*</td>
<td>−16.9 ± 4.0*</td>
<td>−15.9 ± 4.7*</td>
<td>−7.9 ± 2.4*</td>
</tr>
</tbody>
</table>

Now, take a normal heart with $D_{dia}=55$mm and $h_{dia}=9$mm ($L_{dia}^{EPI}=73$mm).

ENDO: $D_{sys} = D_{dia} \times (1 + St_c) = 41$mm

EPI: $D_{sys}^{EPI} = D_{dia}^{EPI} \times (1 + St_c^{EPI}) = 59$mm

ZERO WALL THICKENING IN NORMALS??
Many related indicators are used to describe the heart function (the same heart).

LV rotations were determined as **AVERAGE** angular displacements of 6 myocardial segments. If rotation is not uniform **WHAT** is rotation?

**Physically**, **TORSION** is just another component of LV deformation: the base-apex component of **shear**.
Many related indicators are used to describe the heart function (the same heart).

What is Atrial Strain?

How can it say something different from ventricular strain? (what’s the physics?)
Barriers to the clinical uptake of this technique include lack of consensus regarding the **superiority of any one among a number of potential measurements** for different applications.

Revolutions come and go, and the only constant is that they bring headache for many and a need for a few diligent people to **clean up the mess**.

All measures give inter-related information, in the same picture describing cardiac function. **Physics-based assessment of myocardial mechanics can help to progress with conceptual rigor and improve clinical reliability**.
3D Tissue Tracking

Myocardial mechanics is three-dimensional and assessment based on 3D information is theoretically more complete, reliable and reduces potential misinterpretations.

DANGER!! …the number of arbitrary parameters may EXPLODE!

Physics-based SYNTHESES are feasible and they are mandatory.
**3D Strain Analysis:** with reference to longitudinal and circumferential directions

A physical process described in terms of:
- Longitudinal STRAIN
- Circumferential STRAIN
- Base-Apex Shear (Torsion)
- Lateral Shear (Rocking)

**Technical Note 1:** The radial strain (thickening), can be recovered by incompressibility

$$\text{St}_{\text{radial}} = - (\text{St}_{\text{long}} + \text{St}_{\text{circ}})$$

**Technical Note 2:** Only the one shear value (the sum) enters in the deformation, because the other (the difference) represents a rigid rotation with no deformation.
Strain Analysis: with reference to longitudinal and circumferential directions

Deformation Process

Strain (shortening)

Shear (angular deformation)

3 quantities (depending on the long/circ definition)

\( S_{\text{long}} \)

\( S_{\text{circ}} \)

reduction of area balanced by thickening for incompressibility

Pure deformation, no area/volume change
**Principal Strain Analysis:** same deformation seen along different reference

Deformation Process

Detect Principal Strain Direction (effective direction of contraction)

Principal Strain (no shear!)

2 quantity (principal and secondary strain)

Strain lines pattern

\[
\begin{array}{c|c|c|c}
\text{initial} & \text{final} & \text{Deformation Process} & \text{Detect Principal Strain Direction} \\
\hline
\text{St}_{\text{Princ}} & 0 & \uparrow \downarrow & (\text{effective direction of contraction}) \\
0 & \text{St}_{\text{Secon}} & \uparrow \downarrow & \text{Principal Strain} \\
\end{array}
\]

from incompressibility thickening = \(-\text{St}_{\text{Princ}} - \text{St}_{\text{Secon}}\)
**Principal Strain Analysis:** same deformation seen along different reference

The same physical process composed by:

- **Principal STRAIN** (The most important for contraction because shortening is driven by 1D fibers)
- **Secondary STRAIN** (Less important, small, the first that shows changes because “sacrificable”)

![Diagram showing the combination of Long+Circ Strain (contraction), Shear (volume preserving), and Princ+Secon Strain (no shear).]
Preliminary Results

Principal strain color-map & Strain-line pattern

Average
Normal
Set
Principal Strain-lines at end-systole resemble epicardial fibers. Principal Strain-lines represent the functional direction of contraction, to be related and integrated with the anatomical direction of fibers.

Principal Strain reflects the contraction of fibers (epicardial?).
- It is the “contraction” function
- Must be maintained: last to be lost.

Secondary Strain is transversal to those fibers, initially stretches, and contracts later (with endocardial fibers?).
- It is weak (<10%) but represents the “consistency” of contraction.
- It can be sacrificed earlier
- It is an early indicator of sub-optimal contraction → of remodelling?

They represent physically distinct phenomena in LV contraction

Preliminary Results

Secondary systolic contraction is easily lost

Normals

HCM

DCM

Htn+aged
Conclusion – Part I

All deformation measures are parts of a same picture
- Must be congruent and self-supporting.
- Best to focus more on their integration than on which one is better.

Principal/Secondary Strain analysis is the deepest analysis available for tissue deformation
- Principal strain is strong: a loss means completely compromised function.
- Secondary strain is weak: it is the first function loss at early stages of diseases.
  Loss of secondary strain means that fibers tend to separate \(\rightarrow\) remodeling?
- Strain-lines give the functional pattern of contraction.
- Physics-related measures.

Limitations
- 3D tracking is a new technique and care (multiple readings) are required to ensure reliability.
- Best to focus on global strain, regional properties may be not reliable. yet.

Translational process: rigorous physics applied to cardiac function
The same physics used in centuries of technological progress, nothing to invent, just to apply.
I. **TISSUE:** Myocardial Mechanics and Deformation Imaging

II. **FLUID:** Blood dynamics and Flow-Tissue Interaction
Cardiology is about flow. The primary purpose of the cardiovascular system is to drive, control, and maintain blood flow to all parts of the body. Flow dictates the form and function of the heart and blood vessels through ontogenetic and phylogenetic development, the structural and functional consequence of repair, and in its end stages, remodeling and response to failure. Flow should therefore be a primary focus by which we explain where lesions form, why they degrade and decompensate, and how we grade the extent of restoration of function after vascular intervention. Yet this is not the case. Flow is not a standard part of our clinical lexicon. Few reliable and consistent means of measuring flow exist. Despite early use of surrogate flow markers Flow through the heart interacts with the mobile contours of the myocardium, valve, and vessels. Blood flowing through the sequence of compartments is subject to changes in direction and luminal diameter, and as a consequence, flow is multidirectional and vortical with a tendency to curl or spin in the cardiac chambers during various phases of the cardiac cycle (1). The valves, chamber geometry, and wall motion modify the flow patterns to produce a hemodynamic environment comprising normal or pathological adaptation. Therefore, analyzing the spatial and temporal distribution of blood flow in the cardiovascular system may provide diagnostic and prognostic information. However, acquiring and visualizing flow through the volumes of the heart cavity is a complex task.
1. **Theory**: Cardiac Fluid Mechanics Basis

2. **Methods**: How to assess Cardiac Flow in vivo

3. **Application**: What Cardiac Flow may tell us
In a vessel, flow is higher in the center, and lower near the wall.

- A **BOUNDARY LAYER** with *high shear* is present near the wall.

In curved wall, the boundary layer may detach *separate* from the wall.

**BOUNDARY LAYER SEPARATION** → **VORTEX FORMATION**
A vortex forms from the tip of anterior mitral leaflet, propagates along the posterior wall of the left ventricle, reaches rapidly the apex, and washes-up the whole cavity smoothly accompanies the flow from the mitral inlet toward the outflow tract.

Echo acquired by: G. Tonti
Example of an ideal “normal” flow (healthy man at rest EF=55%)

From:
Pedrizzetti et al. Phys Rev Lett 2005

Vorticity longitudinal section

Vorticity 3D (λ₂)
During **diastole**, vortices develop from the mitral valve trailing edges...

The strong mitral jet (~1 m/s) enters into a closed few cm cavity, has to turn 180° to exit...

...flow should produce turbulence, dissipation, overpressure!

...**intraventricular vortex** smoothly accompanies blood in this intense flow.

1. **Vortices here are a low dissipation structure**
   - to maintain kinetic energy
   - to redirect the flow to the aorta

2. **Heart is not** an uniquely efficient pump but has
   - **Extraordinary adaptability**
   - **Self-regulatory mechanisms**
1. **Theory**: Cardiac Fluid Mechanics Basis

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How to assess cardiac flow in vivo: **MRI (phase-encoded)**

Directly acquired 3D flow! (although average from many heartbeats)

*Unique quality for research, difficult for clinical routine*

MRI data courtesy provided by: Lon Simonetti (Ohio State Univ)

How to assess cardiac flow in vivo: color-Doppler 2.0

Limited to the velocity component toward/away from transducer (1D)
Reconstruct velocity vector by fluid dynamics concepts (with some assumptions)

Aloka Doppler data courtesy provided by: A. Frazer & T. Uejima (Univ. Cardiff, UK)

GE Doppler data courtesy provided by: P. Sengupta, G. Caracciolo (Mt Sinai, NY)
PIV (Particle image velocimetry) is an optical method of flow visualization used to obtain instantaneous velocity measurements and related properties in fluids.

The fluid is seeded with tracer particles sufficiently small to assume that they faithfully follow the flow dynamics. The motion of the seeding particles is used to calculate speed and direction (the velocity field) of the flow being studied.
How to assess cardiac flow in vivo: **Echo-PIV**

Track contrast bubbles: Real-time, and up to high frame-rate (200Hz here)
Moderately complex acquisition procedure
Optimal for clinical research, feasible for clinical routine
How to assess cardiac flow in vivo: **Echo-PIV**

**Potential extension to multi-plane and 3D reconstructions**

*from: Sengupta, Pedrizzetti, Narula. JACC Img 2012*

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**Aquired image**

**Velocity field**

**3D reconstruction**

Pedrizzetti et al. 2013, Cover Image - Nature Cardiology
1. **Theory**: Cardiac Fluid Mechanics Basis

2. **Methods**: How to assess Cardiac Flow in vivo

3. **Application**: What Cardiac Flow may tell us
   
   Vortices, Energetics, Pressure and Stress
   
   → Remodeling
Patient after mitral valve substitution (Bi-leaflet mechanical)

Circulation reversed from normal!

Over 80 cases, almost 100%!

Poor flow performance, excess energetic consumption and uneven stresses, may induce adaptation. A fluid dynamics basis of post-surgical remodeling?
Clinical determinants of LV remodeling

<table>
<thead>
<tr>
<th>Condition</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic Cardiomyopathy</td>
<td>52%</td>
</tr>
<tr>
<td>Dilated Cardiomyopathy</td>
<td>17%</td>
</tr>
<tr>
<td>Valvular disease</td>
<td>12%</td>
</tr>
<tr>
<td>Hypertension</td>
<td>11%</td>
</tr>
<tr>
<td>Other</td>
<td>8%</td>
</tr>
</tbody>
</table>

Fluid Dynamics determinants of LV remodeling

- **Excess energy consumption**
  - Energy dissipation, Turbulence (irregular vortices)
- **Anisotropic intra-ventricular forces**
  - Pressure gradients push (misplaced vortices)
Understanding of remodeling must explain why a segmental ischemia may lead to deformation of the entire chamber: how the segment communicates with other regions.

Every segment is in touch with the whole chamber through blood!

Di Donato & Menicanti, Milan, Italy
Normal flow (stable state)

Consistent with a model of diastolic function where the apex recoiling during early diastole contribute to filling by actively drawing blood into the LV.

Base-Apex pressure gradient...

Pressure gradient is aligned base-to-apex accordingly to LV geometry and curvatures.

Systole  E-Wave  A-wave

Base to Apex
Apex to Base

suction  gentle  deceleration

Pressure gradient color-map

RED=High Pressure ; BLUE=Low Pressure
AMI flow (unstable state?)

**Higher energy dissipation:** Flow more irregular with multiple interacting vortices $\rightarrow$ weak turbulence

**Base-Apex pressure gradient:** sharper peaks due to blockage of the flow $\rightarrow$ peak stresses

An adaptive system (heart) may tend to regulate (remodel?) to reduce such negative fluid dynamics behavior.
DCM flow (meta-stable state?)

LOW energy dissipation: regular flow (almost stagnant), no turbulence and peak stresses.

Pressure gradient distribution

Still transversal pressure gradients but wall stresses are reduced because of walls are curved

Dissipation has reduced and stresses have reduced… (meta-)stable because further adaptive possibilities are limited.
The same pathway applies to other sharp changes (discontinuities) to heart function.
An improper tissue motion, that does not gently receives and decelerates the inflow, that present high energy dissipation (small increases of rigidity, minor dysynchrony, valvular jet deviation etc... hard to detect and assess from tissue dynamics)

Gives rise to sharp pressure peaks and deviation of pressure gradients (well noticeable)

- That gives unnatural hammering stresses on tissue;
- That modifies the geometry and dynamics of tissue;
- That further leads to:

Blood dynamics can help to early evidence this pathway
Cardiac flow is a missing brick in cardiac imaging

Flow provides an **immediate measure of the quality** of heart function, and can be helpful for assessment of long term tendencies (**functional stability**) after functional changes.

...like (for example):

- Remodeling risk stratification at early stages of pathologies (MI, Diastolic dysfn, valv disease...)
- Cardiac Resynchronization Therapy optimization
- Evaluation of surgery (MV Repair, MV Implant, LV Reshaping)
- Congenital heart defects
- ...
Conclusions

→ **Myocardium Deformation Analysis** can be taken at a deeper level to uncover the more intimate features of mechanical contraction/release.

→ **Cardiac Fluid Dynamics** can provide novel integrative information about the quality and the stability of the cardiac function.

Such physics-based analyses can help the improved understanding of cardiac patho-physiological function and support refinements of *diagnostic strategies and therapeutic solutions*.

Translational advances require the effort of a deep integration between illuminate clinicians and supportive engineers.
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