Investigating Cardiac Function Using Motion and Deformation Analysis in the Setting of Coronary Artery Disease

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In the last decade, noninvasive cardiac imaging has played an increasing role in cardiology. As one of the most widespread clinically used techniques, echocardiography has witnessed several technical developments in imaging modalities and image analysis. One of the most recent has been the introduction of velocity-based strain-rate imaging and speckle tracking to quantify regional deformation.1

Coronary artery disease induces important changes in regional myocardial function. Both acute ischemia and chronic ischemia decrease regional wall motion and thickening. Despite recent technical developments in clinical cardiac imaging, the evaluation of regional radial and longitudinal function is often based on visual interpretation of wall motion. This is both qualitative and subjective.

This paper discusses regional myocardial deformation and motion, studied in different (experimental) substrates of coronary artery disease, from acute ischemia to chronic infarction. It will be shown that regional deformation at rest, combined with observations during a dobutamine challenge, can uniquely discriminate the different ischemic substrates.

Motion and Deformation in Normal Myocardium

Both radial and longitudinal regional peak systolic velocities show a significant fall after β-blockade2,3 but no added effect of additional pacing. Regional displacement shows a tendency to decrease with β-blockade. With the addition of pacing a further significant reduction in displacement takes place. The transmynocordial velocity gradient (the difference between epicardial and endocardial peak velocity, divided by their distance4–6) is influenced by β-blockade in the same way as peak velocities and is not altered by changes in heart rate during β-blockade. With a dobutamine infusion, myocardial velocities increase.2 With induced changes in contractility, either by dobutamine or β-blockade, peak systolic velocities correlate well with fractional shortening, regional stroke work, end-systolic and maximal elastance, as well as preload recruitable stroke work.

On the basis of velocities, regional myocardial deformation properties can be assessed noninvasively both in the radial and longitudinal directions. An excellent correlation was shown between the velocity-derived deformation (strain rate) and sonomicrometry.7,8 Deformation shows particular changes with changes in contractility.9–11 Peak systolic strain rate progressively increases with dobutamine. Atrial pacing alone has no effect on peak systolic strain rates over a wide range of heart rates. β-Blockade induces a significant reduction in strain rate, which is not altered by a subsequent increase in heart rate by pacing (Figure 1A). In contrast, end-systolic strain shows a biphasic response to dobutamine. After an initial increase with increasing dose, strain decays and is no longer significantly higher compared with baseline. Atrial pacing induces a trend to an initial increase in strain, but with higher heart rates, which induce a fall in preload, it is significantly reduced. After β-blockade, strain is significantly reduced. The addition of pacing leads to a trend toward a further decay (Figure 1B).

Peak systolic strain rate shows an almost identical behavior as maximal systolic dP/dt (Figure 1, A and C), which suggests that it parallels changes in contractility over a variety of different inotropic states and different heart rates. In contrast to peak strain rate, changes in end-systolic strain parallel changes in ejection fraction or stroke volume (Figure 1, B and D). This can be easily understood because, in order to eject a certain volume, the whole ventricle has to deform and decrease its internal volume. This is done by deformation of the myocardium, hence changing strain values. A similar relationship between strain and stroke volume has been shown for the right ventricle.12

Deformation shows a significant increase after significant volume loading by a saline infusion,7 whereas maximal elastance (an indicator of myocardial contractility) does not change. This shows its load dependency and indicates that this has to be taken into account when strain rate is used as a marker of myocardial contractility. However, this caveat is also valid for sonomicrometric measurements, which have
been accepted as a gold standard for regional myocardial function.

Normal myocardium shows the highest strain rate in the first third of the ejections period, whereas the maximal deformation mostly occurs at aortic valve closure. However, in normal myocardium, post-systolic thickening/shortening (PST) (continuation of deformation after aortic valve closure) can be observed in up to 30% of the segments, but it is always very small (<2% or <10% of total deformation). PST depends on age and loading conditions. This is further discussed in the section on PST.

Thus, deformation is a very sensitive parameter in detecting changes in myocardial performance. However, it should not be used as a direct marker of myocardial contractility in cases where loading is significantly altered (such as in aortic stenosis, hypertension, valve regurgitation, and shunts).

Regional Changes in Motion and Deformation in Acute Ischemia

Invasive Assessment
In early studies, ultrasonic crystals were implanted to assess changes during acute ischemia. It was seen that acute regional ischemia causes rapid, predictable, and reproducible changes in deformation in a segment. A typical example of the time course of these changes is illustrated in Figure 2. During the isovolumetric contraction and early ejection, myocardial thinning (radial) and lengthening (longitudinal) occur, whereas normal segments thicken and shorten. During mid/late ejection, the magnitude of shortening/thickening is decreased compared with normal. After aortic valve closure, an additional or continuing shortening/thickening occurs in the ischemic segment (PST) that is not present in the surrounding nonischemic myocardium.

The magnitude of these changes is proportional to the severity of the acute ischemic insult and flow-related. Figure 2A shows an example of the combined measurement of deformation and blood flow in a segment perfused by a coronary artery that is gradually occluded. Systolic deformation decreases with increasing stenosis, whereas PST becomes more and more prominent. These changes parallel the decrease in transmural perfusion and are mainly determined by subendocardial flow, which shows a linear relation with decrease in deformation, whereas subepicardial flow remains normal and only drops when systolic bulging already exists.

It is also important to note that even during an acute complete coronary occlusion the myocardium in the at-risk zone is not totally akinetic and can show both motion and deformation. This may be caused either by tethering to the adjacent normally contracting segments or changes in segmental wall stress induced by cavity pressure. Thus, deformation parameters can be considerably influenced either by loading conditions or pharmacological interventions that alter contractility in neighboring segments.
demonstrates the influence of afterload on systolic deformation, systolic bulging, and PST at baseline during different degrees of ischemia and reperfusion. The deformation is shown under normal loading condition and with an acute increase in afterload. With increasing preload, both PST and systolic bulging are reduced (Figure 3, right). Another (clinically) relevant finding is that the difference between akinetic and dyskinetic myocardium is not only caused

Figure 3. A, Myocardial deformation (systolic, postsystolic deformation, and systolic bulging) with gradual degrees of coronary occlusion. At each stage, an additional acute increase in afterload was performed (indicated by “L”). B, Changes with preload in ischemic PST and systolic bulging. A is reprinted from Leone et al,19 with permission from The European Society of Cardiology. B is reprinted from Dalmas et al,21 with permission from Cambridge University Press.
by the severity of the ischemia but is also importantly determined by loading (both pre- and afterload).26

Thus:

- Acute ischemia induces systolic bulging, decreases systolic deformation, and increases PST. These changes are directly related to perfusion.
- With acute total vessel occlusion, systolic deformation is totally ablated and replaced by systolic bulging.
- An acute increase in afterload exaggerates the effects on deformation induced by ischemia, whereas an increase in preload diminishes it.
- Reperfusion restores deformation to near normal, but some bulging and PST remains present in the early phase as a result of stunning.

Noninvasive Assessment

Figure 4 shows the typical sequence of changes in M-mode images of the at-risk wall after an acute coronary occlusion.29 As can be observed, changes are identical to those described with an invasive approach: a gradual decrease in systolic thickening (and motion) with a concomitant increase in postsystolic deformation/motion (continuing even after the opening of the mitral valve). Deformation and motion return to near normal after reperfusion (after a short initial period of hypercontractility caused by hyperemia). Similar observations were made using pulsed,30 color M-mode,31 or color Doppler images.31,32

Myocardial velocities have the advantage of being relatively easy to acquire and process. However, local velocities are influenced both by tethering to adjacent segments and overall heart motion. This was illustrated by Urheim et al7: during left anterior descending coronary artery occlusion, the peak systolic velocity of the nonischemic basal segment of the septum decreased significantly as a result of tethering to adjacent ischemic myocardium while systolic strain remained unchanged. Therefore, deformation parameters better reflect regional changes in myocardial function than motion indices, which just represent the way the region of interest is moving within the thorax and is the result of local contraction, overall heart motion, and the influence of neighboring segments.

The initial approach toward noninvasively quantifying local deformation was based on analyzing velocity gradients from M-modes. Derumeaux et al showed that for normal myocardium a gradient exists between endo- and epicardial velocities, which disappeared with an acute total coronary artery occlusion.33 The introduction of color Doppler–based strain (rate) imaging made it easier to obtain local deformation information and results in measurements similar to those obtained using microcrystals.7,8,34 With this approach, deformation can be studied using transthoracic imaging. During acute coronary occlusion, the changes observed in radial strain profiles in the at-risk segment have been shown to be comparable to segmental thickening described from sonomicrometry.35 With a total occlusion, systolic deformation decreases to almost zero and PST becomes dominant (Figure 5A and 5B). This again emphasizes the need to incorporate timing of global events (most importantly aortic valve closure and opening) because both the magnitude and timing of regional changes in deformation are important in ischemia. Global timing information can be obtained from additional data sets acquired during the same examination (taking care heart rate has not significantly changed). Either gray-scale M-modes, showing valve-opening or closure or blood-pool Doppler, including the valve-clicks, can be used. Additionally, aortic valve closure can be obtained from unfiltered myocardial velocity traces from the basal septum.35

Pislaru et al have suggested that it may be of value to quantify the presence of PST by defining an asynchrony parameter: the time from the R-wave on the ECG to the occurrence of compression/expansion crossover.36,37 They showed this increased in ischemic regions and was related to the extent of the myocardium at risk. However, mainly depending on the loading conditions, late systolic thinning
can occur before postsystolic rethickening, which introduces another crossover of compression/expansion.

Skulstad et al also showed similar changes with both microcrystals and velocity-derived parameters. Additionally, they suggested the use of pressure-segment length loops and pressure-strain loops to assess the work performed by a myocardial segment. Although this approach offers important possibilities to study stress-strain relationships, it has to be kept in mind that regional pressure-segment length loops represent both the work performed by the segment as well as the work performed on the segment by its neighbors.

It has also been suggested that PST, especially in an apical segment, is an important determinant of the abnormal intraventricular blood-flow pattern observed during filling in the presence of acute ischemia. During graded ischemia, induced by gradually decreased perfusion, the fall in strain values parallels the sequential reduction in blood flow. With only a slight decrease in perfusion, a decrease in strain is induced which is paralleled by the development of PST.

In clinical practice, the assessment of the regional myocardial response to inotropic stimulation is widely used to detect inducible ischemia and viability. In classic dobutamine stress echocardiography, viable myocardium is identified by the visual detection of residual contractile reserve. Conversely, inducible myocardial ischemia is recognized by a worsening in regional function. However, the visual interpretation of stress echo presents several problems, especially by operators with limited experience. Crystal-based measurements showed that changes in deformation in ischemic myocardium subjected to a dobutamine challenge strongly depend on regional myocardial flow reserve. When resting perfusion and coronary flow reserve are normal, dobutamine initially increases thickening (strain), whereas at higher doses strain decreases as a result of a drop in stroke volume at high heart rates. When resting flow is normal but flow reserve is reduced, only a small initial increase in deformation occurs that does not change further with increasing dobutamine and starts to decrease with higher heart rates. When flow reserve in the ischemic region is absent, dobutamine produces no initial increase in strain. Similar findings were reported using strain (rate) imaging. Figure 5C shows radial strain curves of myocardium supplied with decreased coronary flow and no flow reserve, where systolic thickening is blunted and PST appears. Systolic deformation decreases even more with an increasing dose of dobutamine, whereas PST becomes more prominent (Figure 5C). Note that although systolic deformation clearly decreases, the concomitant increase in PST means that overall deformation remains constant. Without accurate timing of the aortic valve, the shift from systolic to postsystolic events cannot be detected. When the images are assessed visually, deformation would appear to be unchanged. It is also important to be aware that the response to a dobutamine challenge, described above, holds only when infused peripherally. When dobutamine is infused directly (into a nonsevere ischemic territory), local systolic deformation actually (slightly) increases, whereas PST decreases.

**Myocardial Stunning**

When myocardium is ischemic for a prolonged period and subsequently reperfused, deformation remains abnormal despite full restoration of perfusion (Figure 6, bottom, 3rd profile from left). In contrast to ischemic myocardium without flow reserve, which worsens during dobutamine stimulation, stunned myocardium will respond by normaliz-
ing its deformation.\textsuperscript{45} However, although the profile normalizes and PST disappears almost entirely, the amplitude of the deformation under dobutamine stimulation is usually reduced compared with normal myocardium.

Monnet et al\textsuperscript{45a} showed that, in stunned myocardium, systolic deformation returns to normal within a period of 24 hour of reperfusion and that this is paralleled by a decrease in PST. Additionally, they showed that preconditioning resulted in a faster recovery of deformation and less PST with a new ischemic event.

**Acute Infarction and Reperfusion**

In clinical practice, it is accepted that acutely ischemic myocardium should be reperfused as early as possible. However, the benefit of early reperfusion may be obscured by reperfusion injury\textsuperscript{46–48} and extracellular edema.\textsuperscript{49} Reperfusion of an infarct results in an immediate large increase in end-diastolic wall thickness caused by the development of acute, massive edema.\textsuperscript{50–52} Strain (being low because of the prior occlusion) does not recover because of the incompressibility of the edema. PST, which occurs immediately at the onset of occlusion and has the tendency to decrease during the development of the infarction (this decrease has been suggested as a marker of irreversible myocardial damage\textsuperscript{18}) does not change acutely with reperfusion.

Lyseggen et al compared both short occlusions (resulting in viable tissue after reperfusion) and long occlusions (resulting in necrosis).\textsuperscript{52} They confirmed that nonviable segments show acutely increased wall thickness, whereas viable segments did not. Additionally, the area of pressure-segment length loops in the nonviable reperfused tissue was zero, and these segments show an increasing stiffness after reperfusion as a result of the edema (also reported by Pislaru et al\textsuperscript{53}).

Others have demonstrated that no response to dobutamine occurs after reperfusion of acute transmural infarctions,\textsuperscript{54} whereas stunned myocardium (reduced deformation, but without the massive increase in end-diastolic thickness) will increase deformation with dobutamine.\textsuperscript{45}

Derumeaux et al\textsuperscript{55} compared myocardial velocities and velocity gradients in an acute model of reperfusion with either transmural or nontransmural infarction. Deformation parameters in a full-thickness infarct did not recover with reperfusion and remained almost zero. In contrast, a nontransmural infarction did show some improvement in deformation after 60 minutes of reperfusion, although this did not return to baseline values. One could infer that, in the case of reperfusion of a nontransmural infarction, only subendocardial edema is present, which enables the subepicardial myocardium to contract and deform after flow restoration.

**Chronic Ischemia**

In acute ischemia, the properties of the tissue itself do not change in such a way that this has a major influence on deformation. However, in case of an infarct or a critical stenosis (inducing ischemia or repetitive stunning), changes in tissue morphology, mainly myocyte loss and fibrosis, will occur. This will induce alterations in tissue elasticity and thus deformation. Figure 7 illustrates the difference in deformation, at rest, between chronic hypoperfusion (resulting in a subendocardial infarction) and a fully established transmural infarction in an experimental model of chronic ischemia.\textsuperscript{56} Subendocardial fibrosis resulted in a local reduction in systolic deformation and a concomitant induction of PST (Figure 7, left). In addition, a direct relationship exists between the extent of fibrosis and the reduction in systolic deformation: the more fibrosis the more reduction in defor-

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure6.png}
\caption{Changes in radial strain of the at-risk posterior wall and remote normal septum during hypoperfusion, reperfusion, and a dobutamine challenge. Ischemia induces decreased systolic strain and postsystolic thickening, which persists on reperfusion caused by stunning. With a dobutamine infusion, the deformation profile normalizes gradually. After 10 days, segmental function recovers. AVC indicates aortic valve closure.}
\end{figure}
Thus a chronic transmural infarction shows no deformation. Figure 7, right, shows a comparison of the response to dobutamine of strain rate, strain, and PST for normal myocardium and both nontransmural and transmural infarction.

Thus, with chronic ischemia:

- Compared with normal myocardium, at rest, chronic nontransmural infarction shows a decrease in peak systolic strain rate and end-systolic strain with some PST, whereas chronic transmural infarction has no deformation, neither systolic nor postsystolic.
- With a dobutamine challenge, chronic ischemia, with subendocardial necrosis and a residual critical stenosis, will show a small initial increase in strain rate, which decreases at higher doses. End-systolic strain has a tendency to decrease immediately, whereas PST tends to increase. With the critical stenosis present, this response is very similar to that from acute ischemia caused by the response of the nonfibrotic viable subepicardium becoming ischemic after the limited flow reserve is exhausted.
- Transmural chronic infarctions show no deformation at rest and do not respond to dobutamine.

**Postsystolic Thickening: What Is It and Why Does It Occur?**

In the changes in deformation characterizing different ischemic substrates, the development and persistence of postsystolic deformation is an important finding. It was shown that it is not a marginal phenomenon in clinical practice, because it is observed in up to 80% of coronary artery disease patients. Despite much experimental work, the mechanisms underlying PST remain unclear. Figure 2B shows an example of crystal-based deformation in acute ischemia. The normal region shows wall thickening from the onset of ejection, when pressure is rising, until maximal pressure drop, when the wall starts thinning. In the early phase of acute ischemia, this pattern is changing. During ejection, the ischemic wall thickens less than normal. This is followed by some thinning toward the end of ejection while the normal tissue is still thickening. At the moment the pressure drops to almost its lowest value, the ischemic segment starts to rethicken, whereas in the same period the normal wall is thinning. At the moment when the traces of both the rethickening ischemic region and the thinning normal region cross, the ischemic region starts to thin again. Additionally, as discussed in Figure 3, PST can be observed in normal myocardium and is clearly influenced by afterload and preload.

In isolated muscles put in series, of which one was made hypoxic while its loading was forced into a normal physiological pattern, deformation was similar as in an intact ventricle, with PST occurring after the active force started to reduce. A similar setup, but with a constant nonphysiological load, only showed reduced contraction and bulging with no PST. This clearly suggests that the interaction of the
ischemic muscle with its surroundings is crucial for the understanding of its deformation.

To understand PST it is necessary to discuss all factors influencing regional deformation. The relation between forces acting upon an object and the resulting deformation is described by Hooke’s law, which states that forces and deformation are linked by the elasticity. The more elastic an object, the more it will be deformed by a given force. This relation, when applied to myocardium, is illustrated in Figure 8A. However, when myocardium contracts, an additional (contractile) force is developed within the tissue. Hooke’s law remains valid, but, in order to describe the total deformation of the myocardium, all forces acting on it have to be taken into account. Obviously, the internal contractile force (the intrinsic contractility) is the most important, but it has to be kept in mind that any piece of myocardium is always imbedded in a ventricle. This means that external forces are also acting upon it. These forces are described as the loading of the tissue and consist of the local wall stress (caused by the intracavity pressure) and the interaction with neighboring, contracting segments (each contracting neighboring segment will pull the segment under investigation). As for any object, the relation between all acting forces and the resulting deformation is ruled by the regional elasticity, which, for myocardium, translates in the fiber structure and the presence or absence of fibrosis and depositions. Also, elasticity is not a constant because, owing to its matrix structure, the more myocardium is stretched the more difficult it becomes to stretch it even further.

Thus, the main factors that influence regional deformation are (Figure 8B):

- Intrinsic contractility, which is the force developed by the myocardium and acting in the direction of the myofibers (influenced by tissue perfusion and electrical activation). Note that the active tension in normal myocardium does not last throughout the whole time period of systole; it peaks around one-third of the ejection period and is over at aortic valve closure.
- Cavity pressure (often referred to as afterload and influenced by preload) acting perpendicular to the local endocardium, of which the influence is related to the local ventricular geometry (shape and wall-thickness).
- Segment interaction acting in the direction of deformation of the neighboring segment.
- Tissue elasticity (depending on local histology and the amount the myocardium is stretched).

It is clear that the amplitude of active contraction will decrease in ischemia caused by lack of perfusion. It was shown that contraction of an isolated myocyte from a chronically ischemic segment will be reduced but prolonged, indicating that the duration of the active force is of importance for the resulting deformation. As well as the active contraction, tissue properties (mainly elasticity) will change in chronic ischemia because of the development of fibrosis. Because ischemia is often a regional phenomenon, neighboring segments can have different contractile properties, thus changing segment interaction.

To understand what the actual deformation pattern of an ischemic segment should be, the influence of the parameters most likely to be involved was studied using mathematical modeling. A gradual reduction in the contractile force in an ischemic segment, without any other changes (no addi-
tional late contraction) results in a gradual decrease in deformation and a concomitant increase in PST (Figure 9, A and B). When the contractile force is totally absent, an initial thinning of the wall takes place (because of high cavity pressure), followed by some systolic thickening (pulled along by the normal, thickening segment) and marked PST (a result of exaggerated interaction with the thicker normal segment when the pressure has dropped). A prolongation of a reduced active contractile force results in an increase in both systolic and postsystolic thickening (Figure 9, C and D).

Increasing fibrosis (decreasing local elasticity), will blunt all deformation up to the point at which the segment only moves and does not deform (Figure 9, E and F). Similar observations were made when myocardial contracture was created by nonischemic inhibition of myocardial energy metabolism.65

The above findings imply that the presence of deformation, whether systolic or postsystolic, means that the elasticity cannot have been altered significantly. This explains why PST can act as a marker of tissue viability.17,18,66 Whereas some used this as an argument to state that PST is an active process, its presence actually implies that tissue elasticity is preserved, which means that no important, irreversible fibrosis is present, which is of course a condition sine qua non to have viable tissue. PST is very sensitive for the detection of ischemic abnormalities but not very specific because it is influenced by a lot of factors, of which loading and the state of the surrounding tissue are the most important.

With a dobutamine challenge in the presence of ischemia without flow reserve, the ischemic segment itself does not significantly change, but deformation in the neighboring segment will increase, resulting in an increased difference between ischemic and normal segments and thus increasing PST. However, if dobutamine is infused directly in the related artery,24 the surrounding nonischemic myocardium does not alter its contractile state, while (if the flow reduction is not severe) the stimulated ischemic tissue might improve a little its contractile function, thus overcoming part of the pressure force and increasing deformation and reducing PST. Stunned myocardium will develop more active contraction with dobutamine, reducing the difference with the surrounding segment and thus normalizing the deformation pattern.

PST can also occur in normal myocardium,13 especially where increased afterload (aortic stenosis, hypertension), myocardial storage diseases, and abnormal activation are present. With increased afterload, deformation in normal myocardium will be reduced because it has to overcome a higher pressure. The ischemic region will have even more difficulties to thicken against a high pressure (and might even bulge), resulting in a more pronounced difference at aortic valve closure between normal and abnormal tissue and an increase in PST. This (passive) mechanism was also suggested by Skulstad et al.28 To be able to discriminate ischemic PST from other forms, it was suggested that changes in strain rate during isovolumic relaxation could be used.67,68 It was
suggested that, in ischemia, the ratio between maximal strain rate during isovolumic relaxation and systole is larger than unity and that, in the radial direction, maximal strain-rate during isovolumic relaxation is positive in ischemia but negative in other forms of PST.

In summary, PST will be present in all cases where an imbalance exists at the moment left ventricular pressure drops (aortic valve closure), between deformation of neighboring segments, regardless whether this imbalance is caused by reduced contractility as a result of ischemia, high regional wall stress in some segments (mainly basal septum and in the presence of increased afterload), or regionally delayed contraction.

**Active or Passive?**

One of the ongoing discussions with regard to the mechanisms behind PST is whether it is associated with late active (re-)contraction. It was shown that an active contractile force is not required to initiate PST in a segment perfused by a totally occluded artery, where the interaction with neighboring segments can explain PST. Skulstad et al have suggested that PST is associated with active contraction during this period on the basis of the analysis of pressure-segment length loops. However, their analysis looks at the total work from the ischemic segment, containing both the contribution of active contraction and all loading forces, including segment interactions (Figure 8). This implies that regional pressure-segment length loops represent both the work performed by the segment as well as the work performed on the segment by its neighbors. This is in contrast with the global pressure-volume loops that correspond to the whole ventricle without major interaction from the environment.

**Overview of Deformation in the Different Substrates in Coronary Artery Disease**

From the above discussion of regional deformation in the different ischemic substrates and their response to dobutamine, it becomes clear that each substrate can be individually identified on the basis of its baseline deformation (strain rate, strain rate...).
strain, and PST) and how each of these parameters react to an inotropic challenge (Figure 10).

Conclusion
Myocardial deformation is the complex resultant of intrinsic contractile forces and extrinsic loading forces applied to tissue with varying elastic properties. Therefore, loading variations as well as changes in myocardial stiffness are important determinants of myocardial deformation patterns and their magnitude. Thus, strain and strain rate indices are not a direct measure of contractility, but despite some limitation, strain (rate) imaging, especially when combined with a dobutamine challenge, offers a noninvasive imaging approach to resolve the complex process of myocardial deformation during ischemia as it was historically assessed with sonomicrometry. Knowledge from cardiac mechanics and the relation between forces and deformation, combined with imaging data, provides new insights into ischemia related phenomena such as postischemic thickening.

Disclosures
None.

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