Assessment of myocardial ischemia and viability using tissue Doppler and deformation imaging: the lessons from the experimental studies

Evaluation de l’ischémie myocardique et de la viabilité par le Doppler tissulaire et l’imagerie de déformation : leçons tirées des études expérimentales

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Summary
Tissue Doppler imaging and strain rate imaging are quantitative methods for assessing myocardial function and have been shown to overcome the limitations of current ultrasound methods in assessing the complex changes in regional myocardial function that occur in differing ischemic substrates. Tissue Doppler imaging (TDI) measures in real time the myocardial velocity gradient which is an index of myocardial deformation. Strain and strain rate (SR) imaging has been shown to be a sensitive technique for quantifying regional myocardial deformation. Strain rate is less load-dependent that strain and provides therefore a better measure of contractility.

In the setting of ischemia, experimental studies have shown that strain imaging was an accurate method for quantitative evaluation of regional myocardial function and may yield important physiological data. In myocardial infarction, transmural extension of scar distribution in the infarct zone is proportionally related to the reduction in systolic function measured by the radial transmural velocity gradient or by strain rate imaging. Measurement of both systolic and post-systolic deformation both at rest and during a graded dobutamine infusion may help to distinguish between transmural and non transmural infarcts.

In conclusion, strain imaging has the ability to evaluate of regional myocardial function. Strain rate has not replaced conventional grey-scale imaging in the assessment of regional left ventricular function and the implement of these new indices in the routine clinical practice will need additional clinical and large-scale studies.

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Coronary heart disease is the leading cause of death worldwide. After an acute myocardial infarction, early and successful reperfusion is the most effective strategy to reduce the size of myocardial infarction, to salvage myocardium and therefore improve the clinical outcome. However, the different stages from ischemia to reperfusion are complex since myocardial injury occurs during ischemia but also during reperfusion. The complete knowledge of myocardial structure, metabolism, perfusion and function is crucial to understanding the response of the heart to injury such as ischemia.

The effects of ischemia and reperfusion on heart based on studies in experimental models of coronary artery occlusion have been extensively described (1-5). Brief periods of ischemia of less than 20 minutes followed by reperfusion are not associated with development of necrosis (reversible injury). If duration of coronary occlusion is extended beyond 20 minutes, a wavefront of necrosis extends from sub-endocardium to sub-epicardium over time. Reperfusion before three hours of ischemia salvages ischemic but viable tissue. Reperfusion beyond three to six hours does not reduce myocardial infarct size. Late reperfusion may still have a beneficial effect on reducing or preventing myocardial infarct expansion and left ventricular remodelling.

Therefore, during an episode of transient ischemia, regional left ventricular wall motion abnormalities develop, because myocardies cease contracting within seconds of the onset of acute ischemia. After relief of ischemia, the post-ischemic but viable myocardium requires hours to days before function is fully restored. This slow return of cardiac function after resolution of ischemia has been called stunning and the length of time for function to return is dependent on a number of parameters, including the duration and the severity of the original ischemic insult and the adequacy of the return of the arterial flow. Stunned myocardium is defined as “prolonged, post-ischemic dysfunction of viable tissue salvaged by reperfusion.” Hence, an important aspect of stunned myocardium is that there is a flow-function mismatch. At a time when coronary blood flow has been restored to normal or near normal and ischemia is resolved, the myocardium still does not contract (1, 3).

The analysis of fiber thickening across the different layers of myocardial walls is important to take into account to differentiate the various patterns of contractile abnormalities that may occur during acute ischemia, hibernation, or stunning. Conventional assessment of contractile function is based on the measurement of the transmural thickening and does not provide information regarding the transmural distribution of contractile performance. Thus, there is a need for a quantitative approach to study the regional changes in deformation and their timing induced by ischemia.

Tissue Doppler imaging and strain rate imaging have been introduced as quantitative methods for assessing myocardial function and have been shown to overcome the limitations of current ultrasound methods in assessing the complex changes in regional myocardial function that occur in differing ischemic substrates. Tissue Doppler imaging (TDI) analyzes in real time endocardial and epicardial velocities, and measures the myocardial velocity gradient which is an index of myocardial deformation (6-8). Strain (e) / strain rate (SR) imaging has been shown to be a sensitive technique for quantifying regional myocardial deformation compared with other cardiac imaging modalities (9-10). In addition, strain rate is less load-dependent that strain and therefore improve the clinical outcome. However, the different stages from ischemia to reperfusion are complex since myocardial injury occurs during ischemia but also during reperfusion. The complete knowledge of myocardial structure, metabolism, perfusion and function is crucial to understanding the response of the heart to injury such as ischemia.

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Tissue Doppler imaging (TDI) is a technique which displays the velocity of myocardium as pulsed wave Doppler spectrogram or as colour coding of the image (Figure 1). Therefore, accurate timing and velocity measurements may be performed. When the velocity of the tissue is known, several other parameters can be derived such as the displacement which is the integral of the velocity over time (Figure 2).

Strain and strain rate are measures of changes in shape and therefore represent deformations. For 1-D deformation (i.e. shortening or lengthening), the conventional strain describes the relative change in length between two states from an initial length L0 that is stretched or compressed to a new length L. For 2-D or 3-D deformations, the concept of strain becomes more complex since shear strains add changes that might not occur in any of the coordinate directions. Therefore, if one aligns the coordinate system to the principal strain directions, the shear strains won’t be taken into account.

The strain rate is the temporal derivative of the strain and indicates therefore the rate of the deformation. This measurement is closely related to parameters of myocardial contractility.

However, Doppler-derived velocity and deformation data are one-dimensional since only the velocity and deformation...
tion component along an image line can be assessed, resulting in an angle dependency of the measurements. Therefore, new approaches have been developed in order to assess the two-dimensional motion and deformation based on speckle tracking. Indeed, this speckle pattern characterises the underlying myocardial tissue acoustically and is assumed to be unique for each myocardial segment. It can therefore, serve as a fingerprint of the myocardial segment within the ultrasound image. Tracking of the acoustic pattern during the cardiac cycle allows to follow the motion and the deformation of this myocardial segment.

**Experimental validation of TDI and SR parameters**

Tissue Doppler imaging and strain rate imaging offer the equivalent of the high-resolution deformation information that is obtainable only by sonomicrometry in the experimental environment. Implanting intramyocardial microcrystals has shown that systolic deformation in the subendocardial layer is higher than deformation in the subepicardial layer (12, 13, 14). Experimental studies have demonstrated that a reduction in coronary flow causes reductions in systolic shortening in the subendocardial layer that far exceed those in the subepicardial layer (13).

The sequence of changes in regional myocardial function consistently induced by acute ischemia has been well defined by both experimental sonomicrometric (14-16) and cardiac ultrasound studies (17-19). Acute ischemia induces both an early systolic thinning, a decrease in maximal systolic thickening and a delay in the onset of systolic thickening. Concomitant with the decrease in maximal systolic thickening, an abnormal ischemia-related thickening of the myocardium occurs after aortic valve closure. This abnormal phenomenon has been termed “post-systolic thickening or shortening”. Several experimental studies have demonstrated that the decrease in the rate and degree of systolic thickening was related to the myocardial blood flow.
Experimental studies who examined the complex interaction of changes in myocardial function and flow in post-ischemic myocardial segments with differing flow reserves, have demonstrated the superiority of deformation indices such as myocardial velocity gradient, strain rate and strain, over motion indices (myocardial velocity and displacement).

Indeed, our group investigated whether pulsed DTI could accurately identify the alterations of myocardial wall motion induced by graded reduction of left anterior descending coronary artery blood flow in open chest pigs and compared the changes in velocities to those in segment lengths as measured by sonomicrometry and in regional myocardial blood flow as assessed by radioactive microspheres (17). Ischemia resulted in a significant rapid reduction of systolic velocities, a marked increase in isometric relaxation velocity (indicative of post-systolic motion) and an early decrease in the ratio of early to late diastolic velocities. Although the decrease in systolic velocity significantly correlated with both systolic shortening and regional myocardial blood flow during reduction of coronary artery blood flow, systolic velocities slightly overestimated the degree of regional wall motion abnormalities and failed to distinguish ischemia from reperfusion-induced contractile dysfunction. With respect to this, estimation of myocardial perfusion through measurement of myocardial wall velocities must be done cautiously and is valid only in situations of ischemia but not reperfusion.

In a closed chest experimental model, Jamal et al. (18, 19) analyzed the spectrum of regional myocardial function changes during and after a transient ischemia of the left ventricular posterior wall induced by an acute total 20-second occlusion or by 30 minutes of a severe hypoperfusion followed by 60 minutes of reperfusion of the left circumflex coronary artery territory. These authors measured the changes in radial strain and strain rate profile of the ischemic posterior wall during ischemia, stunning, and subsequent dobutamine infusion (5 to 20 mg/kg per minute). During total ischemia and left circumflex coronary artery hypoperfusion, strain and strain rate profiles were consistently modified, showing a delayed onset and a decreased magnitude in regional systolic thickening as well as an increased postsystolic thickening of the posterior wall. Interestingly, end-systolic strain could differentiate total ischemia from severe hypoperfusion (10 mL/min). Occlusion release after 30 seconds allowed deformation indices to normalize. However, after 30 minutes of total left circumflex coronary artery reperfusion following 30 minutes of a severe hypoperfusion, systolic thickening partially recovered but remained abnormal because of stunning. During dobutamine infusion, systolic thickening of the posterior wall increased incrementally, whereas postsystolic thickening decreased progressively and was not detectable at 20 μg.kg\(^{-1}\).min\(^{-1}\). After the dobutamine administration was discontinued, deformation returned to the pre-dobutamine stunning profile.

Therefore, these authors demonstrated that as regional flow was progressively decreased, systolic thickening was progressively reduced whereas post-systolic thickening concomitantly increased, thus confirming that changes in regional systolic strain and strain rate paralleled stepwise reductions in coronary flow.

Using M-mode TDI, our group demonstrated that M-mode TDI allowed interrogation of intramural velocities to quantify LV radial contraction by measuring the transmural myocardial velocity gradient between endocardial and epicardial layers as an index of radial deformation. This latter parameter was able to differentiate between ischemia- and reperfusion-induced contractile dysfunction (8). During left anterior descending coronary artery occlusion, TDI data were closely related to sonomicrometric measurements, and both endocardial and epicardial velocities were markedly and uniformly decreased during ischemia, resulting in the disappearance of the myocardial velocity gradient (20). After reperfusion, wall motion in the distribution of the left anterior descending coronary artery remained severely depressed, indicative of stunning but M-mode TDI was able to detect a slight but significant increase in the myocardial velocity gradient. This increase was related to a greater improvement in endocardial than in epicardial velocities early after reflow, likely a consequence of the hyperemic response to the preceding ischemic insult. These data are in close agreement with those from a study by Bolli et al that reported comparable time course of non-uniform transmural functional recovery after reflow in dogs submitted to 15 minutes of LAD occlusion followed by 7 days of reperfusion (21).

To summarize, although TDI measurement of regional function by peak systolic ejection velocity is easy to perform, reproducible, validated (22) and typically reduced during ischemia, many experimental studies have demonstrated its limited ability to differentiate between different grades of ischemic dysfunction and to distinguish ischemic from post-ischemic dysfunction. This important limitation of TDI is related to the fact that velocities in one myocardial segment are determined by function in other segments as well, which is due to tethering between segments and cardiac translational motion. Therefore, using motion to represent function has two important drawbacks: (1) peak velocity measurement is dependent on the angle at which the region of interest is imaged; and (2) overall heart motion, cardiac rotation, and contraction in adjacent segments can influence regional velocity estimates.

To overcome the latter problems, strain and strain rate imaging (each reflecting differing aspects of myocardial deformation and both relatively independent of overall heart motion), has been developed and is used to represent regional contractile function. Previous reports indicate that strain imaging in principle provides better quantification of regional function than velocity imaging (23-25).

Recently, Skulstad et al investigated if grading of ischemic dysfunction by strain imaging was superior to velocity imaging for quantification of regional myocardial dysfunction in a dog model of acute ischemia, using segmental shortening by implanted ultrasonic crystals (from sonomicrometry) and segmental work (from pressure-segment length loops) as reference methods for regional function (26). This elegant study was the first one to directly compare the different TDI modalities (strain, tissue velocity, and displacement imaging). The authors demonstrated that strain was superior to velocity and displacement for grading of myocardial segmental dysfunction during experimental ischemia and they extended their observations in patients with anterior wall infarctions. Peak systolic velocity could not differentiate between hypokinetic and dyskinetic myocardium, while systolic strain was an excellent tool for quantification of function in non-ischemic as well as ischemic myocardium and for defining the anatomical extension of dysfunctional myocardium.
Therefore, strain and strain rate imaging has great promise to improve objective quantification and characterization of regional function in the setting of ischemic cardiomyopathies. However, currently, few centers have adopted strain imaging in their routine clinical practice since some obstacles currently limit the adoption of strain and strain rate imaging including the following:

- current assessment of regional function by strain imaging is one-dimensional and does not take into account the complexity of the spatial configuration of myocardial fibers responsible for a strain occurring in 3 dimensions and involving longitudinal shortening, radial thickening, and rotation
- Tissue Doppler strain is angle-dependent. Because tissue Doppler strain is derived from relative changes in velocity along a single ultrasound scan line, it can only accurately assess shortening or thickening when this principal vector is aligned with the ultrasound beam.
- High-quality imaging is necessary. Although the tissue Doppler velocity signals are robust, any potential errors above become apparent when strain or strain rate imaging is used with below-average image quality.
- Reproducibility needs to be improved before current clinical applications.

With the development of the speckle tracking technique, one may expect to resolve some of these limitations and to improve strain-imaging data quality and analysis.

**Experimental acute ischemia**

In addition to their ability to quantify myocardial function, tissue Doppler and strain imaging have a good temporal resolution that enables to analyse the consequences of acute ischemia during the whole cardiac cycle including isovolumic periods. Measurement of function during ejection is the most widely used parameter for quantifying regional function during ischemia. However, a number of studies suggest that analysis of function during the isovolumic LV phases provides additional important diagnostic information.

**Pre-ejection or isovolumic contraction period (IVC)**

Vogel et al. proposed that myocardial IVC acceleration (IVA) would be a load-independent measure of contractility as opposed to peak systolic ejection velocity that is preload and afterload dependent (27). However, these authors demonstrated that IVA reflected myocardial contractility, and appeared to be load independent in a non ischemic animal model, and measurements were taken near the mitral ring, which means they measured global LV function. In a recent experimental study, Lysøeggen et al analysed IVA as a measure of regional function during myocardial ischemia. This study confirmed that IVA was related to global left ventricular contractility, but IVA did not reflect function in the ischemic myocardium (28). Thus, IVA appears to have limited potential to serve as a measure of regional function during ischemia.

Experimental studies suggest that longitudinal IVC velocities may determine the degree of myocardial dysfunction during ischemia (29). In ventricles with preserved systolic function there is a dominantly positive longitudinal velocity during IVC, with only a minor negative velocity component. With progressive ischemia the positive velocity component diminishes, and the negative component increases. During severe ischemia the positive component is lost and replaced by a large negative IVC velocity which is a reflection of the early systolic lengthening, and therefore a hallmark of severe ischemia. In addition to these experimental findings, Penicka et al have shown that a positive IVC velocity after revascularization predicted recovery of function in the reperfused area in patients with myocardial infarction (30). This study suggests that measurement of IVC velocities may provide important diagnostic information with regard to myocardial viability after coronary reperfusion.

**Post-ejection or isovolumic relaxation period (IVR)**

Experimental ultrasound studies have demonstrated that with increasing severity of acute ischemia, the myocardium shortens and thickens after the aortic valve closure (31-33). Post-systolic or post-ejection shortening (LV long axis) and postsystolic thickening (short axis) are characteristic features of ischemic myocardium with a concomitant reduction in maximal systolic contraction.

Post-systolic shortening can be imaged by velocity imaging, and is in the long axis represented by a positive velocity component during isovolumic relaxation. Post-systolic shortening can be measured directly with strain Doppler echocardiography and is measured as myocardial shortening that occurs after cessation of aortic forward flow (Figure 3).

In non-ischemic myocardium, virtually all contraction occurs during systole with very little post-systolic shortening. Voigt et al demonstrated that minor degrees of postsystolic shortening occurs in normal myocardium, and is not pathologic unless it exceeds a substantial fraction (> 20%) of total myocardial shortening (34). The mechanism of post-systolic shortening in normal myocardium is not defined, but may be related to the LV shape changes and untwisting motion that occur during IVR.

In ischemic myocardium, post-systolic shortening has been introduced as a potentially useful marker of ischemic dysfunction (26). Post-systolic strain, however, does not help in grading of ischemic dysfunction, as there is substantial overlap between post-systolic strain values in segments with moderate and severe dysfunction (35).

To better characterize ischemia-related myocardial dysfunction and to normalize the post-systolic shortening values, a “post-systolic strain index” expressed as ratio between postsystolic shortening and systolic shortening has been proposed by Kukulski et al (36). They showed that this index was a highly sensitive and specific marker of myocardial dysfunction during acute myocardial ischemia.

More recently, Skulstad et al evaluated the ratio between systolic strain and combined systolic and post-systolic strain. This ratio differentiated better between different levels of ischemia than just measuring systolic or postsystolic strain. In contrast, calculating a similar ratio for displacement and velocity did not improve grading of ischemic dysfunction (26).

These experimental results have direct clinical implications. In the setting of stress echocardiography, when postsystolic shortening is absent during baseline, but appears during dobutamine it is a marker of myocardial ischemia. Voigt et al demonstrated that the ratio of post-systolic thickening to
maximal segmental deformation was the best quantitative parameter to identify stress-induced ischemia (37).

In ischemic myocardium, debate exists whether such an ischemia-induced post-systolic shortening represents an active or passive event. From a clinical perspective the differentiation between active and passive post-systolic shortening is critical, since active contraction implies viable myocardium.

Post-systolic shortening is nonspecific with regard to tissue viability since it may occur in entirely passive or necrotic myocardium as well as in actively contracting ischemic myocardium. In passive and dyskinetic myocardium, necrotic segments are stretched in systole by nonischemic segments and recoil during isovolumic relaxation when nonischemic myocardium relaxes and the stretching force drops abruptly.

However, when post-systolic shortening occurs in the absence of systolic lengthening, passive recoil can be excluded, and therefore the post-systolic shortening may represent delayed active contraction. Skulstad et al suggested that active contraction also contributes to post-systolic shortening when the post-systolic shortening far exceeds the systolic lengthening in magnitude in a dyskinetic segment (38). Thus, these authors proposed that the ratio between systolic lengthening and combined late systolic and post-systolic shortening may serve as a marker of active as opposed to passive post-systolic shortening. The rationale for this association is that active wall tension will limit systolic lengthening and enhance active post-systolic shortening (38).

Myocardial Infarction

The noninvasive differentiation of transmural from non transmural infarcted myocardium is important in clinical practice in regard with assessment of viability. A few experimental studies have demonstrated that in both acute and chronic myocardial infarction experimental models, the noninvasive measurement of deformation properties can accurately characterize the transmural extension of scar from subendocardial to subepicardial layers. The transmural extension of scar distribution in the infarct zone was proportionally related to the reduction in systolic function measured either by the radial transmural velocity gradient (39) or by strain rate imaging (40).

![Figure 3](image_url). Example of longitudinal velocity and strain curves before and after induction of a myocardial infarction. The basal portion of the interventricular septal wall (yellow curve) is necrotic whereas the mid (blue curve) and apical (red curve) segments are non ischemic. In the basal portion, the strain curve displays a reduced systolic strain and a post-systolic shortening, measured as myocardial shortening that occurs after cessation of aortic forward flow (AVC : aortic valve closure). Note that the velocity profiles are not significantly modified between baseline and ischemia.
In addition, the measurement of both systolic and post-
systolic deformation both at rest and during a graded dobu-
tamine infusion helps to distinguish between transmural
and non transmural infarcts [40].

A non transmural infarct will have markedly reduced sys-
tolic deformation at rest with some post systolic shorte-
ning. During a low-dose dobutamine challenge, it will exhi-
bite an increase in post systolic shortening associated with a
reduction or no change in systolic strain and strain rate.
Conversely, a transmural infarction is characterized by either no measurable systolic deformation or the presence of abnormal thinning/lengthening at rest, with no inducible increase in thickening/shortening during a dobutamine challenge.

Therefore, assessment of post systolic shortening and
dobutamine-induced enhancement of post systolic shorte-
ning along with a reduction of systolic thickening differenti-
ates non transmural from transmural chronic infarctions and might help in patient triage in acute myocardial infarc-
tion, in particular when thrombolysis has been the primary treatment and additional percutaneous angioplasty is consi-
dered.

Recently, Lyseggen et al found that the ratio of systolic
lengthening to combined late and post-systolic shortening
identified viable myocardium, and decreases in myocardial compliance (systolic lengthening/systolic pressure rise) defined necrotic myocardium in an open-chest animal model of coronary occlusion of variable duration to induce differing degrees of transmural necrosis (41). Partially active myocardium was differentiated from passive increases in myocardial length with pressure-dimensional loops. When the ratio between early systolic lengthening and total shortening (L-S ratio) approached 1, the segment was entirely passive and generated essentially no active force. When the L-S ratio was <0.5 and shortening domina-
ted over lengthening, there was a component of active con-
traction, consistent with preserved tissue viability. Second, in entirely passive segments (L-S ratio approaching 1), low systolic myocardial compliance, calculated as systolic lengthening divided by systolic LVP, proved to be a marker of necrosis. This relation was in part accounted for by mar-
ed tissue edema, which caused stiffening of necrotic myo-
cardium. Reperfusion of necrotic myocardium caused no change in the L-S ratio but resulted in a rapid, further reduction in compliance. Reperfusion of viable myocar-
dium, however, caused an immediate reduction in the L-S ratio. These observations suggest that the myocardial L-S ratio and systolic compliance by strain imaging may diffe-
rentiate between necrotic and viable myocardium and iden-
tify reperfusion in acute coronary occlusion.

Conclusion

In the setting of ischemia, experimental studies have
demonstrated that strain imaging has proved to be an accu-
rate method for quantitative evaluation of regional myocar-
dial function and may yield important physiological data.

So far, strain rate has not replaced conventional grey-
scale imaging in the assessment of regional left ventricular function and the implement of these new indices in the rou-
tine clinical practice will need additional clinical and large-
scale studies.

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