Two-dimensionally (2D)-directed echocardiography has arguably become the leading method for assessing left ventricular (LV) function in small animals because it is non-invasive, relatively cost-effective, widely available, and has short acquisition and postprocessing times that allow high throughput. These techniques have proved useful in determining both the interplay between altered cardiovascular gene expression and compensatory physiologic regulation, as well as the effects of genetic, surgical, and pharmacological interventions in rodent models of disease.1–3

However, 2D-directed detection of LV dysfunction by conventional echocardiography (M-mode, 2D, Doppler) is considered a late manifestation of cardiac disease that lacks the sensitivity to identify subclinical disease.4–7 Doppler- and 2D echocardiographic-derived myocardial strain (or deformational) imaging are recent advances in noninvasive cardiac imaging intended, in part, to overcome the limitations that confound the traditional echocardiographic assessment of LV function. Abnormalities of myocardial deformation are seen early in the development of a variety of pathophysiologic states, and thus provide a sensitive means for detecting global and regional myocardial dysfunction.8 Compared with Doppler-determined strain, 2D echocardiographic (2D speckle-tracking echocardiography [STE]) strain is independent of the angle of ultrasound propagation, measures the 3 normal Lagrangian strains (radial strain [RS], circumferential strain [CS], and longitudinal strain), and is less noisy, and more reproducible. 2D STE has been previously described in humans for use in myocardial layer-specific deformation analysis,9 and more recently has been used to quantify ventricular wall motion objectively, but few studies have compared strain measured with echocardiography against magnetic resonance imaging (MRI) in small animals.

**Background**—Myocardial strain imaging using echocardiography can be a cost-effective method to quantify ventricular wall motion objectively, but few studies have compared strain measured with echocardiography against magnetic resonance imaging (MRI) in small animals. 

**Methods and Results**—We compared circumferential strain (CS) and radial strain (RS) measured with echocardiography (velocity vector imaging [VVI]) to displacement encoding with stimulated-echo MRI in 2 mouse models of cardiomyopathy. In 3-month-old mice with gene targeted deficiency of cardiac myosin-binding protein-C (cMyBP-C−/−, n=6) or muscle LIM protein (MLP−/−, n=6), and wild-type mice (n=8), myocardial strains were measured at 3 cross-sectional levels and averaged to obtain global strains. There was modest correlation between VVI and MRI measured strains, with global CS yielding stronger correlation compared with global RS (CS R²=0.4452 versus RS R²=0.2794, both P<0.05). Overall, strain measured by VVI was more variable than MRI (P<0.05) and the limits of agreement were slightly, but not significantly (P=0.14), closer for global CS than RS. Both VVI and MRI strain measurements showed significantly lower global CS strain in the knockout groups compared with the wild type. The VVI (but not MRI) CS strain measurements were different between the 2 knockout groups (~14.5±3.8% versus ~6.6±4.0%, cMyBP-C−/− versus MLP−/− respectively, P<0.05).

**Conclusions**—Measurements of left ventricular CS and RS are feasible in small animals using 2-dimensional echocardiography. VVI and MRI strain measurements correlated modestly and the agreement between the modalities tended to be greater for CS than RS. Although VVI and MRI strains were able to differentiate between wild-type and knockout mice, only global CS VVI differentiated between the 2 models of cardiomyopathy. (Circ Cardiovasc Imaging. 2012;5:776-781.)

**Key Words:** strain ■ speckle-tracking echocardiography ■ magnetic resonance imaging
for phenotyping mice. This imaging technique tracks speckle patterns generated by interference between the ultrasound beam and myocardium on 2D echocardiographic images. It allows a non–Doppler-based assessment of regional myocardial motion, and provides information about segmental as well as global LV function with greater sensitivity and specificity compared with conventional echocardiography. Velocity Vector Imaging (VVI; Siemens Medical Solutions, Mountain View, CA) is a novel imaging technique based on 2D STE, which incorporates speckle-tracking and endocardial contour tracking that allows angle-independent measurements of strain.

With its high spatial resolution and superb imaging quality, magnetic resonance imaging (MRI)-based strain imaging is considered the accepted standard method for measuring myocardial wall strain. However, it is costly, time-consuming, and not widely available, precluding it from routine use in the small animal research arena. Although other studies have compared 2D STE strain measurements with MRI, there are no studies to date that have directly compared VVI strain measurements with MRI-based strain in small animal models of cardiovascular disease. In this study, we report LV strain measurements in normal mice and in mouse models of hypertrophic and dilated cardiomyopathy using VVI and displacement encoding with stimulated-echo MRI, thus providing a direct comparison of the strain measurements in pathological states.

**Methods**

**Experimental Animals**

Adult male (8–10 weeks of age) mice with deficiency of either myosin-binding protein-C, a thick filament-associated protein of the sarcomere (cMyBP-C−/−; n=6), or muscle LIM protein, a promoter of myosin-binding protein-C, a thick filament-associated protein of the sarcomere (cMyBP-C−/−; n=6), and their wild-type littermates of the SV/129 strain (wild-type [WT]; n=8) were used in this study. These groups served as models of hypertrophic and dilated cardiomyopathy, and controls, respectively.

Mice were placed on a standard mouse chow diet and water ad libitum, and housed in a temperature-controlled environment with an alternating 12-hour light/dark cycle. All procedures involving animal care and handling were performed according to institutional guidelines set forth by the Animal Care and Use Committee at Case Western Reserve University.

**Magnetic Resonance Imaging**

Animals were anesthetized with 2% isoflurane with supplemented O₂ in an isoflurane induction chamber, and then moved into the magnet and kept under inhalation anesthesia with 1.5% isoflurane. With electrocardiogram and respiratory gating, cardiac functional MRI studies were performed with a 9.4-T Bruker Biospec (Billerica, MA) horizontal scanner using a volume coil. A series of scout images were first acquired to obtain the horizontal long-axis image from which 3 LV short-axis planes at basal, mid-ventricular, and apical levels were prescribed as perpendicular to the LV long-axis with an interslice distance of 1.5 mm. Two-dimensional myocardial motion was quantified using displacement encoding with stimulated-echo MRI, the details of which has been described previously. Data were captured at a rate of 13 frames/cardiac cycle, resulting in a temporal resolution of ≈9 ms. Image processing (MRI reconstruction) and data analysis were performed offline using custom-built software written in Matlab (MathWorks, Natick, MA). Data acquisition and analysis required ≈3 to 3.5 hours and 1 per animal.

The epicardial and endocardial LV borders were traced using cine images to calculate LV ejection fraction. A 2D displacement map was calculated by means of vector addition of the displacement from 2 orthogonal directions to compute Lagrangian strain tensors at the base, mid, and apex of the LV. The CS and RS measurements at the base, mid, and apex of the LV were averaged to obtain global CS and RS for each animal (Figure 1A).

**Echocardiography**

Echocardiographic studies were performed within 5 days of the MRI. Animals were anesthetized with 2% isoflurane supplemented with O₂ in an isoflurane induction chamber and maintained with 1.5% isoflurane by nose cone. Echocardiography was performed as previously described. Acoustic capture B-mode cine clips (120 Hz) were obtained with electrocardiographic gating using a Sequoia ACUSON System (Siemens Medical Solutions, Mountain View, CA) with a 15-MHz linear array transducer. Image processing and data analysis were performed offline using Syngo Vector Imaging technology software (Siemens Medical Solutions, Mountain View, CA). 2D-directed M-mode images from the mid-papillary short-axis were used to calculate conventional measurements of the LV, which included the LV end-diastolic diameter, end-systolic diameter, anterior and posterior wall thicknesses, and fractional shortening.

**Figure 1.** Displacement encoding with stimulated-echo magnetic resonance imaging (A) and velocity vector imaging echocardiographic (B) vector maps for regional strain in wild type (left) vs knockout (KO) (right). The KO group has decreased strain compared with the wild type, demonstrated by smaller size of the vectors.
B-mode clips were selected based on adequate visualization of the endocardial border and the absence of image artifacts. The epicaldial and endocardial LV borders were manually traced and accurate tracking verified ≥3 cycles in the parasternal short-axis view at end-systole to calculate Lagrangian strain at the base, mid, and apex of the LV. Peak CS and RS values for each segment of the LV were recorded and averaged to obtain global strains for each animal (Figure 1B).

Data acquisition (3 short axes) and analysis (CS and RS) required ≈15 minutes per animal. To ensure good quality images for STE-based strain analyses, image acquisition was performed at a high frame rate by using the smallest possible depth and sector size. All image acquisitions and offline measurements were conducted by a single investigator (SA) who was blinded to animal groups. Using a second investigator (BDH), intraobserver differences of peak regional systolic RS and CS were determined from 20 randomly selected clips as 100× the difference between 2 observations divided by the mean of the 2 observations. Intraobserver differences were determined similarly from 20 randomly selected clips measured 10 days apart by a single investigator (AL). Intra- and interobserver coefficients of variation were estimated as the root mean square of the coefficients of variations and intra- and interleaver intraclass coefficients were calculated using the method of Fleiss.20

Statistical Analysis
The statistical analysis was performed using SigmaPlot 12.0 (Systat Software, Inc., San Jose, CA) and SAS (SAS Institute, Cary, NC) version 9.2 commercial software. All values are expressed as mean±SD. Nondeformation echocardiographic and MRI variables were compared with 1-way ANOVA with post hoc Tukey tests. Strain data were analyzed using linear mixed models, fitting a separate model for the RS and CS at 3 locations (base, mid, and apex) and a model each for global RS and CS. Thus, 8 models were fit; for each, Tukey multiple comparison procedure with a family-wise error rate of 0.05 was performed for pairwise comparisons of the 6 groups defined by the 3 genotypes, and for comparisons between VVI and MRI within a genotype. Bland-Altman plots were constructed to illustrate the agreement between VVI and MRI strain measurements. The Morgan-Pitman test21 was used to test the hypotheses that variability was greater for VVI than MRI, and variability was greater for RS than CS. A P value (2-sided) <0.05 was considered as statistically significant.

Results
Echocardiographic and MRI Volumetric Variables
The LV ejection fraction measured by MRI for the cMyBP-C−/− and MLP−/− groups (33±6% and 29±9%, respectively) was significantly lower than the WT group (69±9%; P<0.05), and was similar between the 2 groups of knockout mice. Similarly, the fractional shortening measured by 2D-directed M-mode echocardiography was significantly lower for the cMyBP-C−/− and MLP−/− groups (38±6% and 19±5%, respectively) compared with the WT group (61±5%), but was significantly lower in the MLP−/− than cMyBP-C−/− group (Table 1). The LV EDD and LV ESD were greater in the MLP−/− group and wall thicknesses were greater in the cMyBP-C−/− group. There were no differences in heart rate among the 3 groups of mice.

Global Strain
The Bland-Altman limits were narrower for global CS compared with global RS (Figure 2A and 2B; however, the difference was not statistically significant P=0.14). Additionally, there was modest correlation between VVI and MRI measured strains, with global CS yielding a stronger correlation compared with global RS (CS R²=0.4452 versus RS R²=0.2794, both P<0.05). The global RS measured by MRI was greater in the wild-type (24.7±1.0%) than knockout mice (cMyBP-C−/−: 12.7±2.0%, MLP−/−: 14.8±3.2%). In contrast, the global RS measured by VVI in the WT mice (23.5±9.2%) was statistically similar to that in the cMyBP-C−/− (16.4±4.6%), but significantly greater than in the MLP−/− mice (6.8±4.0%). The global CS determined with either VVI or MRI was significantly lower in the knockout than WT mice. However, only VVI showed significantly lower CS strain in the MLP−/− mice (−6.6±4.0%) than the cMyBP-C−/− mice (−14.5±3.8%) (Tables 1 and 2).

In comparing the VVI and MRI, only global CS strains in the WT mice were statistically different, with lower strains measured with MRI compared with VVI. Variability of global CS and RS strains was greater for VVI than MRI (both P<0.05), although variability was similar for global CS and RS (P=0.39).

Regional Strain
Values of regional RS comparing VVI and MRI were similar in both the WT and cMyBP-C−/− mice, and only at the LV base of MLP−/− mice were values significantly different when comparing VVI and MRI (Figure 3). Regional strains measured with MRI in both knockouts were lower than those in the WT mice, whereas, only regional RS measured with VVI were significantly lower in the MLP−/− than those in the WT mice. When comparing between the 2 knockout groups, strains measured by VVI were significantly lower in the MLP−/− than cMyBP-C−/− knockouts at the base of the LV; regional RS measured by MRI was similar between the 2 knockout groups.

Values of regional CS comparing VVI and MRI were similar in both the WT and MLP−/− mice, and only at the mid LV base of cMyBP-C−/− mice were values significantly different when comparing VVI and MRI. Regional CS measured by MRI was lower in the cMyBP-C−/− knockout group compared with the WT group at the mid and apex levels, and the MLP−/− knockouts at the mid LV. The regional CS measured by VVI was also lower for both knockout groups compared with WT except...

Table 1. Echocardiographic Variables

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Wild Type (n=8)</th>
<th>cMyBP-C−/− (n=6)</th>
<th>MLP−/− (n=6)</th>
<th>P for ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight, g</td>
<td>25.0±2.1</td>
<td>20.5±1.9</td>
<td>23.0±1.9</td>
<td>0.002</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>436±32</td>
<td>453±8</td>
<td>441±23</td>
<td>NS</td>
</tr>
<tr>
<td>LV EDD, mm</td>
<td>0.35±0.04</td>
<td>0.39±0.04</td>
<td>0.43±0.03*</td>
<td>0.004</td>
</tr>
<tr>
<td>LV ESD, mm</td>
<td>0.14±0.03</td>
<td>0.24±0.02*</td>
<td>0.35±0.02†</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV FS, %</td>
<td>61±5</td>
<td>38±6*</td>
<td>19±5†</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AWTh, mm</td>
<td>0.09±0.01</td>
<td>0.12±0.01*</td>
<td>0.07±0.01†</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PWTh, mm</td>
<td>0.08±0.01</td>
<td>0.10±0.01*</td>
<td>0.07±0.01†</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Global RS, %</td>
<td>23.5±9.2</td>
<td>16.4±4.6</td>
<td>6.8±4.0†</td>
<td>0.0011</td>
</tr>
<tr>
<td>Global CS, %</td>
<td>−23.9±6.4‡</td>
<td>−14.5±3.8‡*</td>
<td>−6.6±4.0‡</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

cMyBP-C−/− indicates gene targeted deficiency of cardiac myosin-binding protein-C; MLP−/− gene targeted deficiency of muscle LIM protein; LV, left ventricular; EDD, end-diastolic dimension; ESD, end-systolic dimensions; FS, fractional shortening; AWTh, anterior wall thickness; PWTh, posterior wall thickness; RS, radial strain; CS, circumferential strain; MRI, magnetic resonance imaging; and NS, non significant.

Data are mean±SD.
*P<0.05 vs wild type.
†P<0.05 vs cMyBP-C−/−.
‡P<0.05 vs MRI strain (Table 2) for pairwise comparisons.
for regional strain at the mid LV in cMyBP-C−/− mice (Figure 4). Similar to regional RS, VVI regional CS measurements (in this instance, mid LV) was significantly lower in the MLP−/− group compared with cMyBP-C−/− group.

**Interpretative Variability**

The interobserver differences for CS and RS were −3.5±14.7 and −3.9±21.9, respectively. Intraobserver differences for CS and RS were 4.7±9.2% and 4.7±13.0%, respectively. The intra- and interreader coefficients of variations were 9.6% and 15.3% for RS and 7.2% and 10.4% for CS, respectively. The intra- and interreader intraclass coefficients were 0.986 and 0.955 for RS and 0.990 and 0.932 for CS, respectively.

**Discussion**

This is the first study comparing echocardiographic strain imaging using VVI and MRI in the wild-type and genetically altered mouse. The principal results of this study are (1) correlations between MRI and VVI strains are modest and are greater (and agreement tends to be greater) for CS than RS; (2) strains measured with VVI are more variable than those measured with MRI and the variability is similar for RS and CS; and (3) VVI-measured strains can be used to rapidly

![Figure 2](image-url)

**Figure 2.** Bland-Altman plots comparing the magnetic resonance imaging (MRI)- and echo-derived strain. **A.** Circumferential strain measurements. **B.** Radial strain measurements. There is closer agreement between the MRI and echo imaging modalities for circumferential strain compared with radial strain measurements. Upper and lower dotted lines are the 95% limits of agreement.

![Figure 3](image-url)

**Figure 3.** Regional radial strain; *P<0.05 echo vs magnetic resonance imaging (MRI), †P<0.05 wild type vs knockout (KO). Regional radial strain measurements using MRI and echo in the wild-type group are similar. Regional radial strain measurements by MRI in the KO groups are significantly lower compared with the wild-type group but more variable via echo. cMyBP-C−/− indicates deficiency of cardiac myosin-binding protein-C; MLP−/−, deficiency of muscle LIM protein; and WT, wild-type.
phenotype mouse models of cardiomyopathy. Although MRI is currently considered the accepted standard for noninvasive myocardial strain imaging, in part because of the ability to evaluate strain in 3 dimensions, long acquisition and postprocessing times, expense, and limited availability preclude its routine use in the small animal laboratory. Thus, echocardiographic strain is a potentially advantageous method for the objective assessment of global and regional LV function and for high-throughput phenotyping of murine models.

Bauer et al previously reported speckle-tracking strain measurements in WT mice, which were higher than values obtained in our study. The previous study used speckle-tracking algorithm supplied by VisualSonics (VevoStrain, VisualSonics, Toronto Canada), whereas echocardiographic strain measurements in our study were obtained by VVI algorithm supplied by Siemens Medical Solutions (Syngo Vector Imaging technology software, Siemens Medical Solutions, Mountain View CA). The proprietary speckle-tracking software from the 2 companies and the superior sampling rates in the VisualSonics software may account for the difference in the strain measurements. Importantly, in this study, the VVI strains are similar to those obtained with MRI and the latter measured in the WT mice were similar to MRI strains previously reported.

Bansal et al previously reported validation of VVI strain with harmonic phase MRI in humans with a very modest correlation between the 2 modalities, greater with CS than RS (CS $R^2=0.12$ versus RS $R^2=0.005$). Similarly, in our study, the correlation between VVI and MRI strain was higher with CS than RS (CS $R^2=0.4452$ versus RS $R^2=0.2794$), but both CS and RS correlations in our study were much greater than in that validation study involving human subjects and were similar to both Bansal et al (CS $R^2=0.397$ and RS $R^2=0.348$, both $P<0.05$) and Cho et al (CS $R^2=0.26$ and RS $R^2=0.36$) using a different speckle-tracking algorithm, automated functional imaging (GE Medical Systems, Milwaukee WI). Nevertheless, our correlations are disappointingly less robust than those reported in a validation study in 5 mice after myocardial infarction and 2 control mice using the VisualSonics instrument with EKV (CS $R^2=0.81$ and RS $R^2=0.72$), which acquires data from multiple cardiac cycles and constructs an image sequence composed of $>100$ images per cardiac cycle.

LV contraction is a complex process involving deformation resulting in shortening in 3 normal directions; longitudinal, CS, and RS. Longitudinal strain, which is a sensitive indicator of subendocardial fiber dysfunction and an early marker of ischemia and increased wall stress, was not evaluated in this study as we were comparing directly with MRI as it is measured in small animals in our institution. In addition, advantages of strain imaging compared with conventional echocardiographic parameters could not be demonstrated in this study, which was designed to compare strain imaging with MRI in fully developed models of cardiomyopathy. In a previous study in mice, longitudinal strains were sensitive to changes early after experimental myocardial infarction and were able to predict LV remodeling; CS and RS were not as consistent. Longitudinal strain is typically obtained from an apical view (although the parasternal long-axis view was used by Bauer et al), which is difficult to obtain reliably in small animals. CS and RS are more influenced by transmural fiber dysfunction (especially the mid-myocardium), and are generally more suited for identifying dysfunction in ventricles with reduced LV systolic function.

Both MRI and VVI strain measurements were able to differentiate between the WT group and the genetic models of cardiomyopathy, reinforcing the potential usefulness of strain measurements in mouse models of LV dysfunction. However, only VVI CS strain differentiated between the 2 models of cardiomyopathy, which corresponded with differences in their echocardiographic LV fractional shortening.

One potential difficulty is that VVI had greater variability in strain measurement compared with MRI, and was particularly problematic in measurement of RS. This may be because of the need to track both epicardial and endocardial borders, the former more difficult to identify with a resultant decrease in accuracy. The greater variability has been reported in previous studies. Although MRI RSs are generally more variable than CS, in this study, their variability was similar.

Limitations

Several limitations of this study merit consideration. First, for logistic reasons, echocardiography and MRI were not performed on the same day for all the animals. This may be responsible, in part, for the modest correlations between the 2 techniques. Second, the echocardiographic images were obtained using 2D rather than 3D imaging used in MRI; translation of the heart remains a problem using 2D acquisition methods as error is introduced to strain measurements when the heart swings out of the imaging plane. In addition, out-of-plane motion occurs because of rotation and motion of the heart; as a result, only a portion of the real motion can be detected. Third, potential sources of variation include the quality of the images obtained for strain analysis. High quality of the 2D images at high frame rates is essential for accurate VVI strain measurements. Moreover, poor quality of images may hinder the investigator’s ability to perform tracing of the endocardium and epicardium which may affect the strain values, thereby introducing another source of variability. Fourth, while temporal resolution for the echocardiograms (120 Hz) was similar (8.3 ms) to the MRI, the accuracy of tracking speckles may theoretically be jeopardized at

![Figure 4. Regional circumferential strain: *P<0.05 echo vs magnetic resonance imaging (MRI), †P<0.05 wild type vs knockout (KO), (**)P<0.05 MyBP-C−/− vs MLP−/− (echo only). Regional circumferential strain measurements using MRI and echo in the wild-type group are similar. Regional circumferential strain measurements by MRI in the KO groups are significantly lower compared with the wild-type group but more variable via echo. cMyBP-C−/− indicates deficiency of cardiac myosin-binding protein-C; MLP−/−, deficiency of muscle LIM protein; and WT, wild-type.](image-url)
these frame rates and may have resulted in a significant underestimation of strain values; higher frame rates were reported using different instrumentation and algorithms.\textsuperscript{6,15} Finally, changes in the imaging angle of incidence can result in capturing different fiber layers at different levels and may introduce variability, particularly because we analyzed exclusively short views which are sensitive to small differences in image angle.\textsuperscript{6}

**Conclusion**

Despite these limitations, we demonstrate that measuring LV CS and RS is feasible in small animals using 2D echocardiography with VVI. VVI and MRI strain measurements correlated modestly and the correlation was greater (and agreement tended to be greater) for CS than RS. VVI had greater variability in strain measurement compared with MRI. Although global VVI and MRI strains were able to differentiate between WT and knockout mice, only VVI strain differentiated between the 2 models of cardiomyopathy.

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**Disclosures**

Dr Hoit is a speaker for Philips Medical. The other authors have no conflicts to report.

**References**


**CLINICAL PERSPECTIVE**

Although 2-dimensional echocardiographic strain imaging cost-effectively and objectively quantifies ventricular wall motion, only 1 small study has directly compared strain measured with echocardiography against magnetic resonance imaging (MRI) in mice. Using a different speckle-tracking algorithm (velocity vector imaging [VVI]), we compared circumferential (CS) and radial strain (RS) to displacement encoding with stimulated-echo MRI in 2 genetic mouse models of cardiomyopathy. CS and RS were measured in groups of wild-type mice and mice with gene targeted deficiency of cardiac myosin-binding protein-C or muscle LIM protein. There was modest correlation between VVI and MRI measured strains, with global CS yielding a stronger correlation compared with global RS (CS $R^2=0.4452$ versus RS $R^2=0.2794$, both $P<0.05$). Overall, strain measured by VVI was more variable than MRI and the limits of agreement were slightly, but not significantly, closer for global CS than RS. Both VVI and MRI strain measurements showed significantly lower global CS strain in the knockout groups compared with the wild-type, but only the VVI CS strain measurements were different between the 2 knockout groups. These data demonstrate that measurements of LV CS and RS are feasible in mice using VVI, although correlations and agreement were modest. VVI may complement conventional methods that objectively assess global and regional LV function and be particularly useful for high throughput phenotyping of murine models.
Comparison of Velocity Vector Imaging Echocardiography With Magnetic Resonance Imaging in Mouse Models of Cardiomyopathy

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