

Automated Multimodal Measurements of Curling Motion in Patients with Mitral Valve Disorders

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Abstract

Background

Systolic curling of the mitral annulus is a qualitative hallmark of the arrhythmic mitral valve syndrome (AMVS), yet it has lacked standardized, quantitative characterization.

Objective

We present the first fully automated framework for the quantitative assessment of basal posterior and lateral systolic curling dynamics from transthoracic echocardiography (TTE) and cardiac magnetic resonance (CMR), enabling objective, reproducible, and large-scale evaluation of this phenomenon.

Methods

By combining TTE's high temporal resolution with CMR's spatial precision, we captured curling as a dynamic event defined by angular deformation, velocity, and regional displacement of the myocardium relative to the annular plane. Metrics including the annulo-ventricular angle ($\zeta_{\min}$) and its first derivative ($\zeta'_{\min}$) were analyzed across controls and patients with mitral valve disease (MVD), specifically mitral annular disjunction (MAD) and mitral valve prolapse.

Results

MVD patients exhibited a significantly smaller $\zeta_{\min}$, and more negative $\zeta'_{\min}$ indicating accentuated, and abrupt posterior curling dynamics. Both metrics correlated inversely with MAD length, confirming their sensitivity to disjunction severity. Temporal analysis revealed that peak curling occurs most commonly just after end-systole, during early diastole, suggesting a post-systolic recoil rather than active contraction. Spatial mapping localized this recoil to the basal lateral wall. Posterior leaflet prolapse further amplified the deformation, while anterior prolapse had minimal impact.

Conclusion

Automated multimodal quantification reveals that curling is a dynamic, localized, post-systolic recoil phenomenon driven primarily by MAD.

1. Introduction

Mitral valve disease (MVD) represents a significant and growing source of cardiovascular morbidity¹. Within this spectrum, mitral valve prolapse (MVP) and mitral annular disjunction (MAD), an atrial displacement of the mitral hinge point², are common abnormalities. While often benign, a growing body of evidence highlights a subset of patients predisposed to malignant arrhythmias and sudden cardiac death (SCD)³⁻⁵. These patients are recognized under the umbrella of arrhythmic mitral valve syndrome (AMVS)¹.

Among the more recent clinical hallmarks of MVD thought to be related to AMVS is the systolic curling motion, a systolic deformation of the inferobasal LV wall and posterior mitral ring (Fig. 1). Curling can be visualized clinically on both transthoracic echocardiography (TTE) and cardiac magnetic resonance (CMR) cine imaging, where it manifests as a transient kinking or posterior displacement of the basal myocardium during systole. In normal physiology, the mitral basal myocardium contracts, resulting in a subsequent change of the annular diameter, moving anteriorly and inferiorly during systole ⁶. In contrast, and first described by Gilbert et al. in 1976 ⁷, MVD patients may demonstrate a curled systolic motion of the posterior annulus, hypothesized to be driven by prolapse and annular disjunction ⁸. Several studies imply that curling may represent a marker of dynamic annular dysfunction and a potential precursor to arrhythmogenesis in MVD ⁹⁻¹¹.

[INCLUDE FIGURE 1 HERE]

Figure 1 Caption: Mitral Valve Prolapse (MVP), Mitral Annular Disjunction (MAD) and curling motion visualized by echocardiography and cardiac magnetic resonance imaging. A: A parasternal long-axis (PLAX) echocardiogram. B: Corresponding 3-chamber (3CH) cardiac magnetic resonance image from the same patient. The images show posterior leaflet MVP (red line) beyond the annular plane. Associated MAD is outlined (yellow lines). Static representation of the curling motion is also presented (green arrows). Anatomical landmarks including the anteroseptal (ANT-SEP) and inferolateral walls (INF-LAT) are indicated.

Currently, there is no established approach for the direct quantitative assessment of curling motion. Clinically, curling motion has been estimated from CMR long-axis views by measuring a perpendicular distance at end-systole (ES), from the most inferior point of the mitral annulus to a reference line drawn between the crest of the inferobasal LV wall and the junction of the posterior mitral leaflet with the left atrium (LA) ¹². However, this technique may be limited by its operator dependence and its simplification of a dynamic movement into a single, static, linear measurement. Clinical visual assessment alone, as is currently employed, may be insufficient, as curling varies considerably between patients.

Recent geometric metrics, such as the Mitral Annulus-Inferobasal Rotation Angle (MIRA) ¹³ and the semi-automatic Mitral Annulus to Infero-Basal wall Angle (MAIBA) ¹⁴, improve predictive power but still require manual keypoint initialization, introducing significant subjectivity. These challenges highlight the remaining need for a fully automated approach that can provide objective and reproducible quantification of curling motion. Fortunately, recent developments in computer vision technologies have increased the feasibility of fully automated solutions.

Furthermore, while transthoracic TTE and CMR are highly complementary, offering excellent temporal resolution and 3D tissue characterization, respectively ^{15,16}, no prior study has integrated both modalities for a unified assessment of curling mechanics.

We aimed to introduce a fully automated, multimodal approach to map the spatio-temporal mechanics of curling motion in patients with MVD and related phenotypes. In this context, the present study aimed to: **1**: develop quantitative, automated, and interpretable methods to assess curling motion using TTE and CMR; **2**: evaluate curling motion association with structural abnormalities detected on CMR, including MAD and MVP; and **3**: use the curling metrics to investigate spatio-temporal patterns on a population level. Our goal is to determine whether curling can be measured as a mechanistically relevant, reproducible biomarker for identifying high-risk MVP/MAD phenotypes.

2. Methods

2.1 Study Population and recruitment

In this retrospective study we included patients with MAD and/or MVP. The main study cohort was recruited from Oslo University Hospital and Drammen Hospital, and all eligible patients underwent comprehensive evaluation at Oslo University Hospital and included in an MVP/MAD cohort, as previously described ^{2,5,17,18}. Baseline evaluation consisted of clinical examination, 12-lead electrocardiogram, 24-hour Holter monitoring, exercise stress test, and coronary angiography when clinically necessary to rule out coronary artery disease. CMR imaging was performed in eligible patients.

For a comparative analysis of echocardiogram-derived metrics, a control group of healthy subjects was randomly selected from the publicly available Trøndelag Health Study (HUNT) registry, a study of individuals examined with echocardiography as part of their assessment ¹⁹. The subjects from the HUNT dataset were consequently screened to select a representative PLAX echocardiogram. The included subjects were not reported to have no more than trace or mild mitral valve regurgitation, and no mitral valve stenosis.

The study complied with the Declaration of Helsinki and was approved by the Regional Committee for Medical Research Ethics (2015/596/REK Nord). All study participants provided written informed consent.

2.2 Medical Imaging

2.2.1 Echocardiography

Transthoracic echocardiography was performed using a commercial cardiac ultrasound system (Vivid 7, Vivid E9 or Vivid E95; GE Vingmed Ultrasound, Horten, Norway). Images were analyzed offline by a team blinded to the clinical data (EchoPac, version 202, GE Vingmed Ultrasound, Horten, Norway). Standard parasternal long- and short-axis images, as well as apical 4-, 2-, and 3-chamber views, were acquired.

2.2.2 Cardiac Magnetic Resonance Imaging

As previously published ¹⁷, CMR imaging protocol was performed using a 3 T scanner; the images were then analyzed with Sectra Workstation IDS7 v1 v18.1. The protocol included standard cine sequences

and modified Look-Locker inversion recovery sequences to assess tissue properties. Key measurements and assessments included MV morphology for MVP and mitral annulus for MAD. MAD was defined as a disjunction of at least 1 mm, measured at ES. This measurement was taken from the junction of the left atrial wall and the valve leaflet to the top of the left ventricular wall. The assessment included all available locations using both echocardiography and CMR, covering the circumferential extent as measured by CMR. MVP was defined as a superior displacement of any part of the mitral leaflet of 2 mm or more beyond the mitral annulus, using the long-axis view on CMR⁵. All patients presented MAD, while a portion of them also had concomitant MVP (see Table 1).

MVD was in our cohort defined as the presence of MAD and/or MVP. Mitral regurgitation was quantified according to established guidelines²⁰ and graded as mild, moderate, or severe.

2.3 Reference Measurements for Method Validation

Speckle tracking echocardiography (STE) was performed on the apical 3-chamber view using EchoPAC v203 (GE Healthcare, Horten, Norway). The basal inferolateral segment (segment 5) was manually selected, and its peak longitudinal strain (PLS) was recorded to assess local myocardial deformation.

Additionally, the Pickelhaube sign²¹ was assessed using pulsed-wave Tissue Doppler Imaging (TDI) in the apical 4-chamber view. The sample volume was placed at the lateral mitral annulus, and the peak systolic velocity was measured.

2.4 Automatic Methods for the Quantification of Curling Motion

2.4.1 A Framework for Curling Quantification using Transthoracic Echocardiography

The proof of concept framework used in this study for fully automated measurements of curling motion from echocardiograms is described in Fig. 2A & B. In short, the framework performs cardiac event timing, point initialization with tracking, and metric estimation.

[INCLUDE FIGURE 2 HERE]

Figure 2 Caption: Overview of the automated curling measurement framework for echocardiography (A, B) and CMR (C, D). **A:** The anterior and posterior mitral annulus points a(ED) and p(ED) and 10 points in the inferolateral basal wall are initialized automatically in the end diastole (ED) frame using a segmentation neural network. **B:** The keypoints trajectories are computed through systole using optical flow. The optimal basal point trajectory is selected automatically for further use, based on the criteria of minimizing the annulo-ventricular angle at ES. **C:** Point initialization in CMR via a segmentation neural network. The hinge points a(ES) and p(ES) and the are initialized in the end systole (ES) frame. **D:** The keypoints are tracked backwards to ED, resulting in trajectories used for metric computation.

A representative parasternal long axis (PLAX) recording served as input for the framework. A previously described neural network for cardiac event timing²² detected ED and ES time points in the recording, and the recording was trimmed to one systolic contraction by removing frames during the diastolic phase. The first frame of the trimmed recording was used to initialize the keypoints, which were the annular hinge points, and automatically defined points along the myocardial wall.

First, a segmentation network²³ segmented the LV wall and the LA. The annular hinge points were identified at the intersection of the LV endocardium and the LA. A 40-point spline was fitted to the segmentation boundary of the lateral wall. The basal inferolateral wall points were chosen as the 10 points of the curve on the basal half of the lateral endocardium (Fig. 2A). The keypoints initialized at ED were tracked through systole until ES using Lucas-Kanade (LK) Optical Flow²⁴. Smoothing and regularization were applied to ensure the displacement of a point did not exceed 10 mm, and that the basal points never moved closer than five mm to the posterior hinge point. Invalidated points were replaced with the point from the previous frame.

Out of all the basal point trajectories, we selected the best basal point trajectory by choosing the one that minimized the annulo-ventricular angle at the ES (Fig. 2B, $b(t)$). The trajectory $b(t)$ was selected for further analysis, while the rest were discarded.

2.4.2 Curling related metrics

To quantitatively formalize curling motion, we defined a set of metrics that could be derived from the keypoint tracking trajectories. For the purpose of this study, we explicitly define curling as the abnormal, transient inward kinking and posterior displacement of the basal inferolateral left ventricular wall relative to the mitral annular plane during systole. We defined the annulo-ventricular angle, $\zeta(t)$, as the angle between the annular plane and the inferolateral basal wall keypoint at time t , as described in Fig. 3A. $\zeta(t)$ was calculated as the smallest angle between the inferolateral basal wall point $b(t)$ and the anterior and posterior hinge points ($a(t)$ and $p(t)$, respectively).

[INCLUDE FIGURE 3 HERE]

Figure 3 Caption: Quantitative metrics for systolic curling derived from point tracking. A: The annulo-ventricular angle $\zeta(t)$ at ED and ES. B: Transverse and longitudinal displacements of the anterior hinge point.

We expected $\zeta(t)$ to be smaller on average in ES for MVD patients than for non-curling controls. Intuitively, a smaller angle indicates that the basal ventricular myocardium folds inward toward the ventricular cavity, reflecting a more pronounced curling of the ventricular base. The *annulo-ventricular angle difference*, which is the difference in annular-ventricular angle between ED and ES time points, was also used as a metric. A greater degree of curling should correspond to a greater annular-ventricular difference.

Moreover, we measured the *annulo-ventricular angular velocity* $\zeta'(t)$ and the *annulo-ventricular angular acceleration* $\zeta''(t)$ by computing the numerical time derivatives of $\zeta(t)$. We hypothesized severe cases of curling to be associated with greater angular velocity and acceleration.

Using our framework, we could measure the *annular opening* $d_{ap}(t)$, which was the distance between hinge points a and b at time t, and the *annular opening velocity* $d'_{ap}(t)$, which could be derived from the annular hinge point trajectories.

We also characterized curling by the directional motion of the annulus (see Fig. 3B), quantified by measuring the *transverse and longitudinal displacements* $\mathcal{D}_{trans}$ and $\mathcal{D}_{long}$. Tortuosity τ is a measure of the trajectories' curvature and elongation relative to the straight-line distance between their endpoints, which was also assessed. An increased tortuosity for the posterior annulus hinge point and the inferolateral basal wall point was interpreted as a curling motion.

2.4.3 An Automated System for the Detection of Curling Motion in CMR

We adapted and applied a quantitative framework to CMR cine imaging similar to the framework for echocardiograms.

2.4.3.A CMR Point Tracking

CMR images were processed using the biv-me framework, an open-source, end-to-end pipeline for generating segmentations and 3D + t models from CMR DICOMS²⁵. For each patient dataset, the first step was systematic view selection of the relevant CMR long-axis cines, followed by automated segmentation of the LV blood pool and myocardium. Segmentation predictions were post-processed to ensure spatial continuity across frames. MV insertion points were defined by the point on the LV endocardial contour that was closest to the left atrial contour.

The point tracking process was initiated from the manually selected ES frame (Fig. 2C). A fixed set of 30 equidistant myocardial points was sampled from base to apex. From the ES frame, point tracking was executed towards ED (Fig. 2D). Smoothing and motion correction was applied to ensure consistent tracking.

Following stabilization, ζ was computed for each of these 30 point-trajectories. Subsequent analysis was constrained to the mid-to-base segment (the 15 most basal points).

2.4.3.B Spatio-temporal analysis

To visualize how curling mechanics evolve across the entire patient population, we generated spatio-temporal maps. To overcome differences in heart size and heart rate, we applied a two-step standardization process. Firstly, we normalized the anatomy, mathematically scaling the tracked points so that every patient's lateral wall was mapped to a uniform length from the mitral annulus to the apex. Secondly, we normalized the timing to account for heart rate variability. Finally, to focus specifically on

the dynamic deformation, we measured the change in the annulo-ventricular angle relative to its resting state in diastole.

2.5 Statistical Analysis

Continuous data were presented as mean $\pm$ SD or median (interquartile range), and categorical numbers with (percentages), as appropriate. Continuous variables were compared by means of the Mann-Whitney U-test. Subgroup analyses were conducted excluding cases with missing variables. Correlations with reference measurements were examined using the Pearson correlation coefficient (PCC). *P*-values were two-sided and values < 0.05 were considered significant. Statistical analysis was conducted using the `scipy.stats` library in Python ²⁶.

3. Results

3.1 Baseline characteristics

We included 149 patients with MVD (mean age 47 years $\pm$ 15, 59% female). 98 (67%) of the patients had a corresponding CMR. From the HUNT dataset, we included 106 healthy subjects (mean age 59 years $\pm$ 15, 51% female), none of which had a corresponding CMR. Table 1 contains descriptive statistics of the dataset, after exclusion according to Fig. 4.

[INCLUDE FIGURE 4 HERE]

Figure 4 Caption

Study population flow chart describing exclusion criteria for echocardiograms and CMR recordings. Tracking performance was assessed on the correctness of point initialization, tracking drift and cardiac event timing accuracy. Automatic failure detection rejected 20 echocardiograms based on failed segmentation or timing.

[INCLUDE TABLE 1 HERE]

Table 1 caption

Descriptive statistics of the dataset.

3.2 TTE Tracking Comparison to Controls

The automatic framework was applied to both the control and MVD cohorts (255 total recordings processed). The framework was executed on a NVIDIA RTX PRO 6000 Blackwell Series GPU, with an average processing time of 2.2 seconds per recording. Two expert clinicians, one imaging expert, and two experienced cardiologists assessed all the exams in both the control and MVD cohorts (255 total recordings processed). After evaluation, 20 patients were excluded due to automatic system failure

detection, 27 patients due to poor image quality, and 43 patients due to poor tracking performance (Fig. 4).

[INCLUDE TABLE 2 HERE]

Table 2 Caption

Comparison of different features to quantify curling between control and mitral valve disease (MVD) groups. Mean and standard deviation (SD) values are reported. Significant p-values ($p < 0.05$) from the Mann-Whitney U test are shown in bold.

[INCLUDE FIGURE 5 HERE]

Figure 5 Caption

Comparison of controls to mitral valve disease (MVD) cohort across curling quantification metrics. $\zeta_{\min}$ represent the annulo-ventricular angle, $\zeta'_{\min}$ the annulo-ventricular angle velocity, $\mathcal{D}$ the displacement ratio for the posterior (p) and anterior tracked (a) points and d_{ap} the annular diameter difference. P -values are indicated for each boxplot.

[INCLUDE FIGURE 6 HERE]

Figure 6 Caption: Atrio-ventricular angle ζ in MVD patient vs. control. A: MVD patient displaying a high degree of curling with a steep decrease in $\zeta(t)$ and spike in derivative. B: Healthy control with gradual decrease.

Quantitative metrics revealed distinct alterations in annular motion (Table 2). Patients with MVD exhibited a significantly smaller minimum annulo-ventricular angle ($\zeta_{\min}$, $p = 0.0023$) and a significantly more negative minimum angular velocity ($\zeta'_{\min}$, $p = 0.03$) compared to controls. The temporal behavior of the angle differed notably between the cohorts with controls displaying a gradual decrease in ζ , while MVD patients demonstrated a steep decline corresponding to a pronounced peak in the derivative (Figs. 5 & 6).

Analysis of annular opening demonstrated a trend toward larger difference in diameter and higher opening velocities in MVD, with differences reaching statistical significance for the d_{ap} ($p = 0.037$). Regional displacement analysis indicated significantly increased posterior transverse displacement alongside reduced anterior displacement in both transverse and longitudinal directions in the MVD group. Consequently, the posterior transverse-to-longitudinal displacement ratio was significantly higher, indicating a dominance of transverse motion in the posterior annulus. Tortuosity indices did not differ significantly between the groups.

3.3 Association with Reference Measurements

The PCC and p -values for $\zeta_{\min}$ and its derivatives correlated with reference measurements (Table 3). The metrics correlated with inferolateral MAD length measured on CMR and with inferolateral basal segment strain. $\zeta_{\min}$ and $\zeta'_{\min}$ (echocardiography) correlated inversely with inferolateral MAD length ($p = 0.020$ and $p = 0.062$ respectively for CMR derived inferolateral MAD length; see Table 3).

[INCLUDE TABLE 3 HERE]

Table 3 Caption

Pearson correlation coefficients (PCC) and associated p -values between annulo-ventricular angle metrics and other measures associated with curling. Correlations at significance level $p < 0.05$ are emphasized in bold.

3.4 Association Between Curling Metrics and Valve Pathologies

To analyze subgroup specificities, we stratified the cohort based on the presence (+) or absence (-) of specific valve phenotype: Anterior Prolapse (AP), Posterior Prolapse (PP), MVP, and MAD. Comprehensive results are presented in Table S1 and Table S2.

3.4.1 Association to Mitral Valve prolapse and Mitral annular disjunction features

$\zeta_{\min}$ was significantly reduced in several subgroups, with the most pronounced result in the inferolateral MAD group. Posterior transverse displacement ($\backslash \text{varvec} D^P_{\text{trans}}$) was higher in AP+, PP+, and inferolateral MAD related subgroups. The ratio of posterior transverse to longitudinal displacement ($\backslash \text{varvec} D^P_{\text{ratio}}$) was significantly increased in presence of AP and inferolateral MAD ($p = 0.0087$ and $p = 0.011$ respectively).

Patients without AP (AP-) showed a lower mean $\zeta_{\min}$ compared to controls ($p = 0.0011$). Similarly, the PP + and PP- groups exhibited significant decreases in $\zeta_{\min}$ ($p = 0.0072$ and $p = 0.026$, respectively), while the MVP+ group also displayed a lower mean $\zeta_{\min}$ ($p = 0.0016$). The most pronounced result in $\zeta_{\min}$ was found in the inferolateral MAD group ($p = 0.00052$). Annular opening was significantly larger in patients with MAD ($p = 0.038$).

Significant alterations were also observed in displacement metrics. Posterior transverse displacement ($\backslash \text{varvec} D^P_{\text{trans}}$) was higher in several subgroups, including AP+ ($p = 0.019$), PP- ($p = 0.072$), and inferolateral MAD ($p = 0.042$). Conversely, anterior transverse displacement ($\backslash \text{varvec} D^A_{\text{trans}}$) was lower in multiple groups: PP+ ($p = 0.0068$), MVP+ ($p = 0.0062$), AP- ($p = 0.022$), and MAD ($p = 0.0085$). Furthermore, the ratio of posterior transverse to longitudinal displacement ($\backslash \text{varvec} D^P_{\text{ratio}}$) was

significantly increased in the presence of AP ($p = 0.0087$) and inferolateral MAD ($p = 0.011$), indicating enhanced posterior translation relative to longitudinal shortening.

For the subgroup of patients with MAD, we observed a larger magnitude of $\zeta'_{\min}$ with marginal significance ($p = 0.053$); similarly, the annular opening speed (d'_{ap} , Table 4) was significantly higher in MAD+ ($p = 0.041$).

3.4.2 Association between curling metrics and combination of valve phenotypes

To further investigate the features of inferolateral MAD, we performed a sub-analysis where MAD patients were further stratified by their co-existing prolapse status (MVP+, AP-, PP-, and PP+).

A significant reduction in $\zeta_{\min}$ was observed across all analyzed MAD+ subgroups when compared to controls. Analysis of annulo-ventricular angle derivatives revealed a significantly more negative minimum second derivative ($\zeta''_{\min}$) in the MAD + AP- group compared to controls. The minimum first derivative ($\zeta'_{\min}$) approached significance in the MAD + PP- group ($p = 0.073$).

While the total annular opening difference was not significantly different for any subgroup, the maximum annular opening speed d'_{ap} was significantly higher in both the MAD + MVP+ group and the MAD + AP- group ($p = 0.033$) compared to controls.

Displacement metrics showed significant differences, particularly in the anterior and posterior regions. Posterior transverse displacement ($\backslash \text{varvec} D^P_{\text{trans}}$) was significantly increased in the MAD + MVP+ group ($p = 0.014$) and the MAD + PP- group ($p = 0.042$) compared to controls. Conversely, anterior displacement was significantly reduced in both transverse and longitudinal directions for several subgroups. Anterior transverse displacement ($\backslash \text{varvec} D_{\text{trans}}$) was lowest in the MAD + PP+ group ($p = 0.005$) and also significantly reduced in the MAD + MVP+ ($p = 0.014$) and MAD + AP- ($p = 0.019$) groups, relative to controls. Similarly, anterior longitudinal displacement ($\backslash \text{varvec} D_{\text{long}}$) was significantly reduced in the MAD + PP+ and MAD + MVP+ ($p = 0.023$) subgroups compared to controls.

Finally, the posterior displacement ratio ($\backslash \text{varvec} D^P_{\text{ratio}}$) was found to be significantly increased in the MAD + MVP+ subgroup ($p = 0.032$) compared to controls, indicating a greater proportion of transverse motion relative to longitudinal motion for the posterior annulus in this group.

3.6 Myocardial Tracking and Trajectory Analysis with CMR

The tracking framework was successfully applied to CMR cine series; 86 (88%) patients were further processed, while 12 (12%) patients were excluded due to bad initialization of the MV insertion points or poor tracking. We generated 2D trajectories for all the points along the lateral wall, with all trajectories subsequently passing visual inspection for anatomical plausibility and temporal continuity. From this dataset of individual point trajectories, we first analyzed the cohort-level dynamics.

3.6.1 Spatio-temporal distribution of the curling motion:

A spatio-temporal map visualized the median annulo-ventricular angle difference relative to ED (Fig. 7). Temporally, the motion was tightly phased around ES. The minimum of median ζ was reached during early diastole, at approximately 10% of a heartbeat following ES. Spatially, the profile demonstrated a pronounced longitudinal gradient: ζ was maximally negative at the most basal points and progressively attenuates towards the mid-ventricle. The *hotspot* concentrated in the basal ventricle during the early post-systolic phase.

[INCLUDE FIGURE 7 HERE]

Figure 7 Caption: Spatio-temporal distribution of curling motion mechanics. The central heatmap displays the cohort-median annulo-ventricular angle difference relative to ED (ζ), visualizing the magnitude of curling in both space (y-axis: normalized position from base to mid-wall) and time (x-axis: beat fraction relative to ES). Warmer colors (yellow) indicate more negative ζ values, corresponding to more severe curling deformation. Marginal panels illustrate the median (solid gray line) and interquartile range (shaded region) for the spatial and temporal profiles.

3.6.2 Annulo-ventricular angle in CMR and association with MAD and MVP

CMR-derived $\zeta_{\min}$ was significantly associated with the presence of MAD (80.9° vs 90.8° , $p = 0.030$) and MVP (78.0° vs 89.4° , $p = 0.005$). No significant difference was found between patients with posterior-only prolapse and other prolapse types.

4. Discussion

Systolic curling has long been recognized as a qualitative marker of AMVS, yet its clinical utility has been hampered by a lack of standardized quantification.

To our knowledge, this is the first study to apply a fully automated, multimodal framework to quantify systolic curling motion. This is a proof of concept study, and while its methodology can be further refined, we prioritized a fully explainable approach allowing for human oversight given the lack of prior standardized quantitative assessment of curling motion. By integrating the temporal resolution of echocardiography with the spatial precision of CMR, we demonstrated that curling is a hallmark of dynamic, asymmetric mechanical recoil. Our central finding is that this deformation is primarily associated with MAD and occurs most intensely during the early post-systolic phase.

To validate our automated framework, we compared our results against recent semi-automated data and established echocardiographic indices (see Figure S1). Our TTE-derived angular metrics showed trends consistent with those reported by Bergo et al.²⁷. The larger variance and group overlap observed in our cohort reflect our broader definition of curling – a more generalized abnormal posterior motion, rather

than the strict *ballerina foot* morphology^{12,28,29} – as well as the greater phenotypic heterogeneity of our population, suggesting utility beyond highly selected study groups.

Furthermore, we observed significant correlations between our angular metrics ($\zeta_{\min}$) and inferolateral basal segmental strain, confirming that the algorithm correctly captures local myocardial deformation. We also observed a moderate correlation with the Pickelhaube sign²¹. This association validates that our method detects the high-velocity dynamics of the annulus, yet offers the distinct advantage of measuring rotation independent of the Doppler angle limitations inherent to the Pickelhaube sign, making it a more robust tool for routine screening.

4.1. The Dynamic Nature of Curling

While previous semi-automated indices have attempted to quantify curling using static systolic angles^{12,27}, the proposed method analyses the full cardiac cycle variation of the annulo-ventricular angle and its dynamical properties (i.e. velocity, displacements), revealing that the curling phenomenon is fundamentally dynamic.

MVD patients exhibited not only a significantly smaller annulo-ventricular angle ($\zeta_{\min}$) but also a more negative angular velocity ($\zeta'_{\min}$) compared to controls. This confirms that the *curling* observed visually is a rapid, whip-like motion rather than a gradual systolic shortening.

This motion appears to be asymmetric. We observed a posterior dominance in tissue displacement, where the posterior annulus translates excessively in the transverse direction while the anterior annulus remains restricted as compared to controls. This asymmetric pattern parallels prior three-dimensional echocardiographic studies reporting posterior annular expansion, anterior restriction in prolapsing valves, and overall increased dilatation of the annular ring in MAD, hereby confirmed in this study³⁰.

Overall, this uncoupling suggests that the presence of MAD essentially releases the posterior hinge point, converting normal longitudinal shortening into aberrant transverse motion.

4.2. Pathophysiology: Post-Systolic Recoil

A key insight from our CMR spatio-temporal analysis is the timing of the deformation. Peak curling magnitude was consistently observed *after* ES (10–15% of the cardiac cycle after ES), during the early isovolumic relaxation phase. This timing suggests that curling is not an active contraction but a passive mechanical recoil.

This behavior supports the proposed mechanism whereby the inferobasal myocardium stores elastic energy during late systole as a result of opposing ventricular pressure and the mechanical pull of the prolapsing leaflets on the PMs^{31–33}. As ventricular pressure drops and aortic valve closure occurs, this energy is released, causing the basal wall to snap back toward its resting configuration. This observation aligns with recent strain imaging studies describing "double-peak" strain patterns in MVP³³, reinforcing

the concept of a distinct mechanical load acting on the myocardium during the vulnerable phase of repolarization³⁴.

4.3. Structural Drivers and Clinical Implications

Our subgroup analysis clarifies the hierarchy of structural drivers. Inferolateral MAD appears to be the primary prerequisite for curling, with the angle of deformation correlating inversely with MAD length (Table 3). However, the mechanics are most severely deranged when MAD coexists with PP. In these combined phenotypes, the prolapsing leaflets likely act as a lever, amplifying the traction on the disjointed annulus.

Clinically, this localized, repetitive mechanical motion could potentially have implications for the arrhythmic substrate. The spatial gradient of curling we mapped on CMR—maximal at the base and attenuating toward the mid-ventricle—overlaps with the inferobasal fibrosis patterns frequently identified in AMVS^{2,4,9,35}. We can speculate that this rapid post-systolic recoil occurs during a period of ventricular repolarization, when mechanical stretch may trigger mechano-electrical coupling via stretch-activated channels³⁴. While a direct causal link remains to be established, curling-associated recoil might not only represent a marker of altered mechanical coupling, but could also theoretically contribute to arrhythmogenic vulnerability in MAD and MVP.

5. Limitations

This study is retrospective, and while the automated tracking minimizes observer bias, it remains subject to image quality limitations inherent to 2D optical flow. While the optical flow tracking used here is robust, its performance can be degraded by poor image quality and limited long-range temporal consistency. Learning-based tracking has the potential to improve robustness³⁶. However, due to the limited dataset size of our MVD group we decided to use all the data for testing, rather than reserving a significant portion for training learning based models. Minimizing potential confounding effects in subsequent research by incorporating age-matched patient and control groups could further benefit the analysis. Future longitudinal studies are required to determine if quantitative curling metrics can independently predict arrhythmic risk beyond traditional markers like fibrosis.

6. Conclusion

Automated multimodal quantification reveals that curling is a rapid, localized, post-systolic recoil driven primarily by mitral annular disjunction. By identifying this motion as a specific mechanical failure of the basal reservoir rather than a simple contraction abnormality, these metrics provide a novel, objective marker for mechanical instability in patients with MVD.

Abbreviations

MAD: mitral annular disjunction, MVP: mitral valve prolapse, AP: anterior mitral valve prolapse, PP: posterior mitral valve prolapse, MVD: mitral valve disease, CMR: cardiac magnetic resonance, TTE: transthoracic echocardiography, AMVS: arrhythmic mitral valve syndrome, ES: end systole, ED: end diastole, LVEF: left ventricular ejection fraction, BMI: body mass index, MR: mitral regurgitation

Declarations

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During the preparation of this work the authors used Gemini in order to improve readability and language. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

Data availability

Data may be made available upon reasonable request to the authors.

Competing interests

There are no competing interests to declare.

Contributions

- **Giulia Monopoli:** Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Data Curation, Writing - Original Draft, Writing - Review & Editing, Visualization, Project administration
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- **Molly Maleckar:** Co-study conceptualization, Writing - Review & Editing, Project Management, Funding.

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Tables

Table 1 caption: Descriptive statistics of the dataset.

	All (n = 165)	MVD (n = 99)	Control (n = 66)
Female, n (%)	82 (50)	48 (64)	34 (51)
Age at inclusion, years	52 ± 23	46 ± 15	58 ± 17
BMI	25 ± 5	23 ± 3	26 ± 4
LVEF, %	59 ± 11	57 ± 7	60 ± 8
MAD, n (%)	80 (48)	80 (94)	0 (0)
Inferolateral MAD, n (%)	66 (49)	66 (79)	0 (0)
MVP, n (%)	75 (165)	75 (75)	0 (0)
Anterior MVP, n (%)	48 (29)	48 (48)	0 (0)
Posterior MVP, n (%)	65 (39)	65 (65)	0 (0)
Bileaflet MVP, n (%)	52 (32)	52 (52)	0 (0)
MR; None, n (%)	72 (44)	24 (24)	48 (74)
MR; Mild, n (%)	36 (22)	33 (33)	3 (5)
MR; Moderate, n (%)	12 (7)	11 (11)	1 (2)
MR; Severe, n (%)	7 (4)	7 (7)	0 (0)

Table 2 Caption: Comparison of different features to quantify curling between control and mitral valve disease (MVD) groups. Mean and standard deviation (SD) values are reported. Significant p-values ($p < 0.05$) from the Mann-Whitney U test are shown in bold.

Feature	Control Mean (SD)	MVD Mean (SD)	p-value
Annulo-Ventricular Angle			
$z_{\min}$ (°)	76.81 (13.41)	70.45 (13.10)	0.0023
$z_{ES} - z_{ED}$ (°)	42.85 (14.58)	45.82 (14.58)	0.448
$z'_{\min}$ (°/ms)	-0.33 (0.14)	-0.38 (0.16)	0.03
$z''_{\min}$ (°/ms ²)	-0.0040 (0.0021)	-0.0045 (0.0027)	0.309
Annular Opening			
d_{ap} (mm)	5.92 (5.07)	7.54 (4.11)	0.037
d_{ap}' (mm/ms)	0.07 (0.03)	0.08 (0.04)	0.06
Tortuosity			
τ_P	1.85 (2.04)	1.64 (0.69)	0.13
τ_A	1.57 (1.03)	1.54 (0.94)	0.956
τ_B	2.01 (1.18)	1.64 (0.55)	0.209
Displacement			
D^P_{trans} (mm)	10.52 (4.23)	11.96 (4.17)	0.023
D^A_{trans} (mm)	7.01 (3.48)	5.44 (2.59)	0.005
D^B_{trans} (mm)	10.49 (3.95)	9.78 (3.83)	0.209
D^P_{long} (mm)	19.50 (5.24)	19.10 (6.20)	0.628
D^A_{long} (mm)	14.37 (4.00)	12.76 (4.12)	0.0009
D^B_{long} (mm)	20.30 (6.35)	19.63 (6.02)	0.41
Displacement Ratios			
D^P_{ratio}	0.55 (0.23)	0.67 (0.26)	0.003
D^A_{ratio}	0.50 (0.22)	0.47 (0.30)	0.105
D^B_{ratio}	0.58 (0.30)	0.56 (0.31)	0.66

Table 3 Caption: Pearson correlation coefficients (PCC) and associated p-values between annulo-ventricular angle metrics and other measures associated with curling. Correlations at significance level $p < 0.05$ are emphasized in bold.

Metric	MAD length (CMR) PCC	MAD length (CMR) p-value	Basal Seg. Strain PCC	Basal Seg. Strain p-value	PicklehaubePCC	Picklehaube p-value
$z_{\min}$	-0.12	0.36	-0.28	0.026	-0.21	0.24
$z'_{\min}$	-0.26	0.062	-0.26	0.035	-0.31	0.08
$z''_{\min}$	-0.36	0.0083	-0.12	0.35	-0.3	0.082
MAD length (echo)	-0.32	0.02	-0.31	0.014	-0.27	0.11

Figures

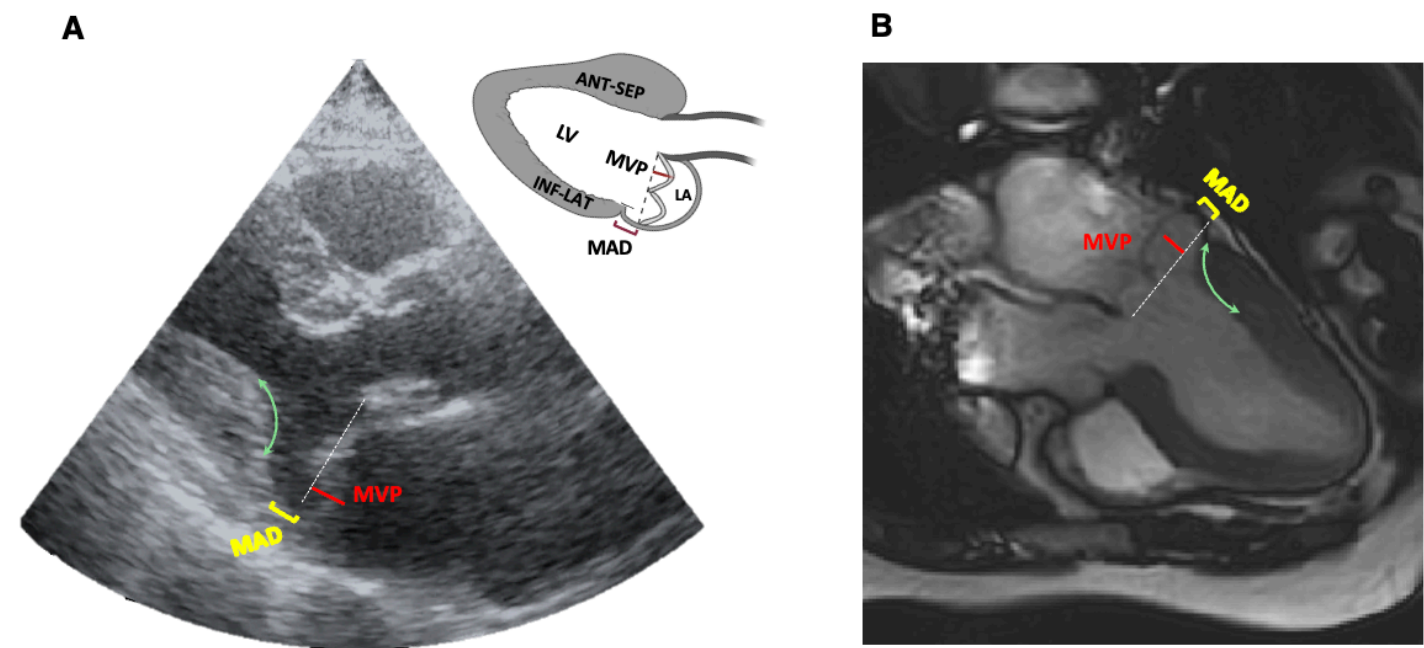


Figure 1

Mitral Valve Prolapse (MVP), Mitral Annular Disjunction (MAD) and curling motion visualized by echocardiography and cardiac magnetic resonance imaging. A: A parasternal long-axis (PLAX)

echocardiogram. **B:** Corresponding 3-chamber (3CH) cardiac magnetic resonance image from the same patient. The images show posterior leaflet MVP (red line) beyond the annular plane. Associated MAD is outlined (yellow lines). Static representation of the curling motion is also presented (green arrows). Anatomical landmarks including the anteroseptal (ANT-SEP) and inferolateral walls (INF-LAT) are indicated.

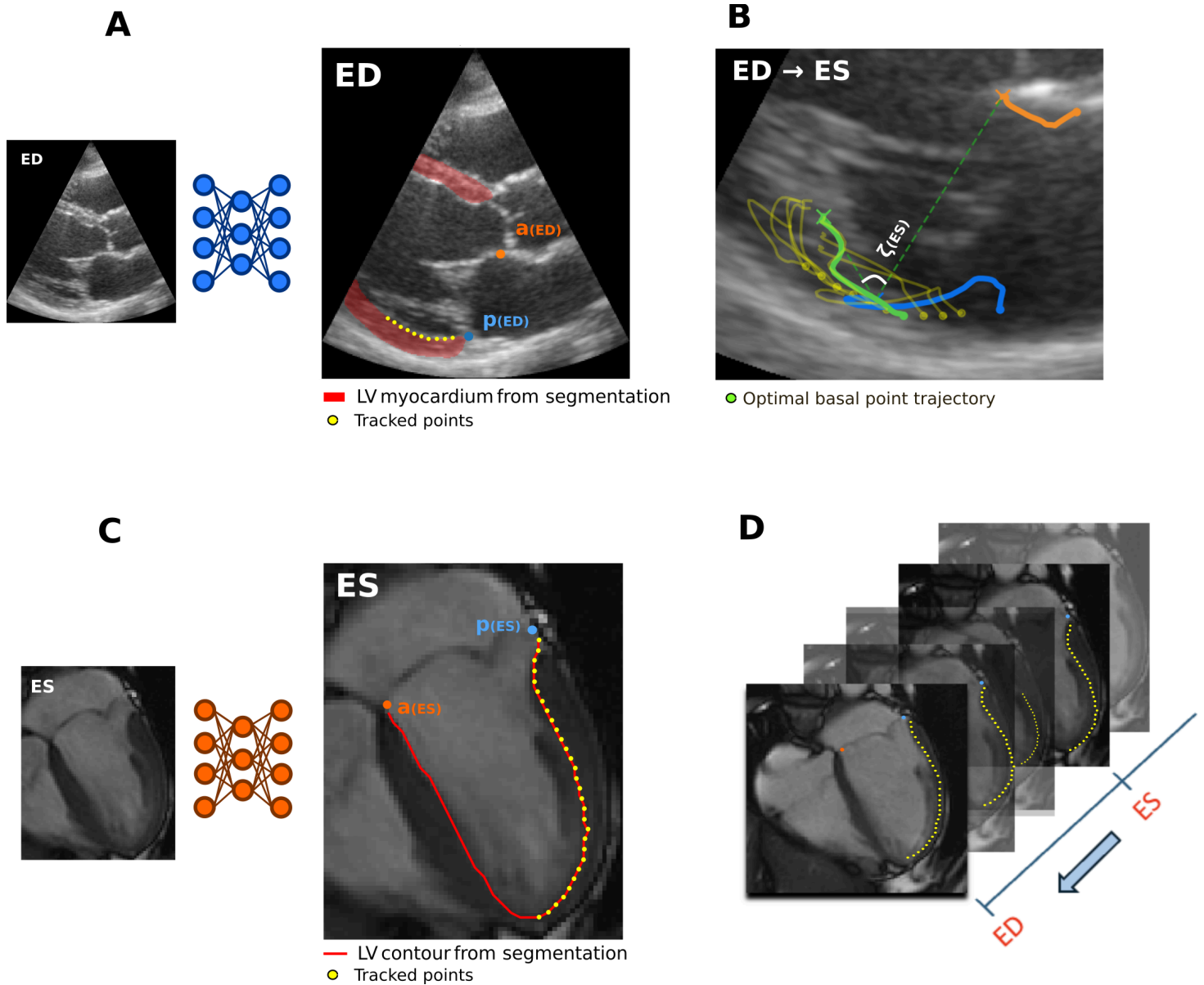


Figure 2

Overview of the automated curling measurement framework for echocardiography (A, B) and CMR (C, D). **A:** The anterior and posterior mitral annulus points $a(ED)$ and $p(ED)$ and 10 points in the inferolateral basal wall are initialized automatically in the end diastole (ED) frame using a segmentation neural network. **B:** The keypoints trajectories are computed through systole using optical flow. The optimal basal point trajectory is selected automatically for further use, based on the criteria of minimizing the annulo-ventricular angle at ES. **C:** Point initialization in CMR via a segmentation neural network. The

hinge points a(ES) and p(ES) and the are initialized in the end systole (ES) frame. **D**: The keypoints are tracked backwards to ED, resulting in trajectories used for metric computation.

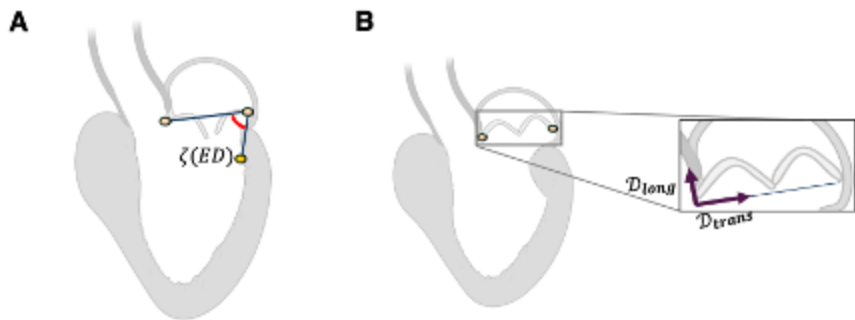


Figure 3

Quantitative metrics for systolic curling derived from point tracking. A: The annulo-ventricular angle $\zeta(t)$ at ED and ES. B: Transverse and longitudinal displacements of the anterior hinge point.

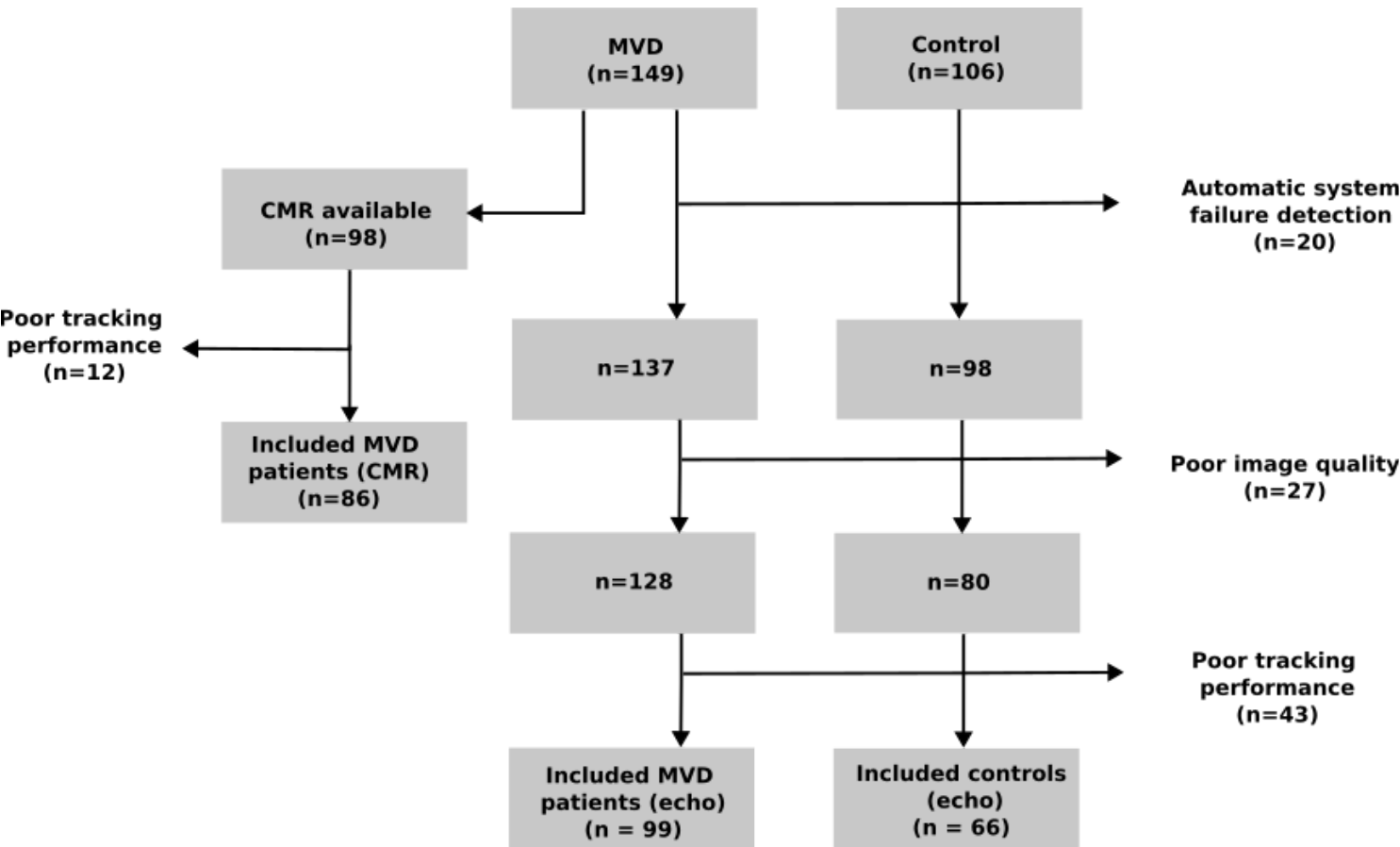


Figure 4

Study population flow chart describing exclusion criteria for echocardiograms and CMR recordings. Tracking performance was assessed on the correctness of point initialization, tracking drift and cardiac

event timing accuracy. Automatic failure detection rejected 20 echocardiograms based on failed segmentation or timing.

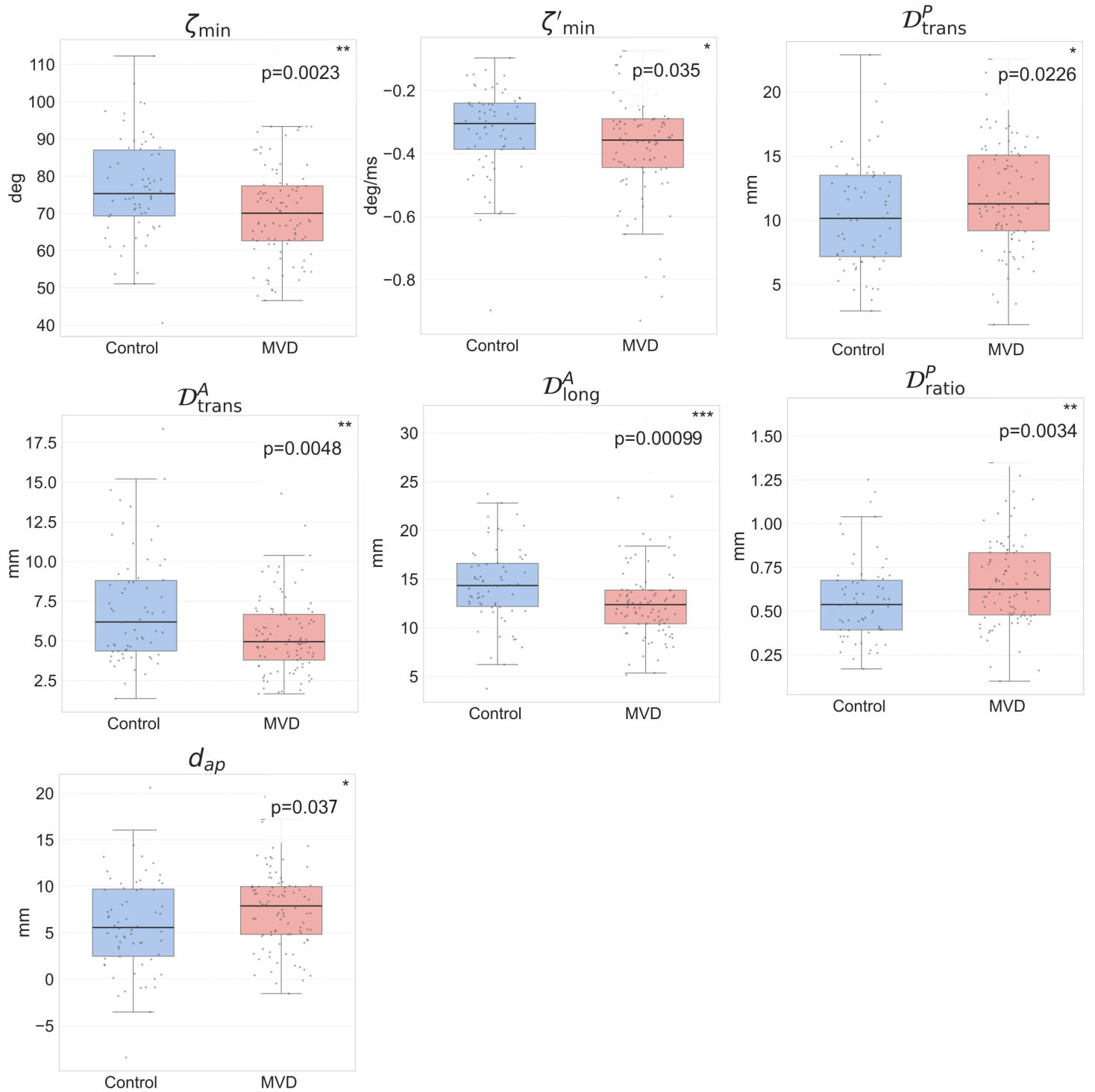


Figure 5

Comparison of controls to mitral valve disease (MVD) cohort across curling quantification metrics. $\zeta_{\min}$ represent the annulo-ventricular angle, $\zeta'_{\min}$ the annulo-ventricular angle velocity, $\mathcal{D}$ the displacement ratio for the posterior (p) and anterior tracked (a) points and d_{ap} the annular diameter difference. P -values are indicated for each boxplot.

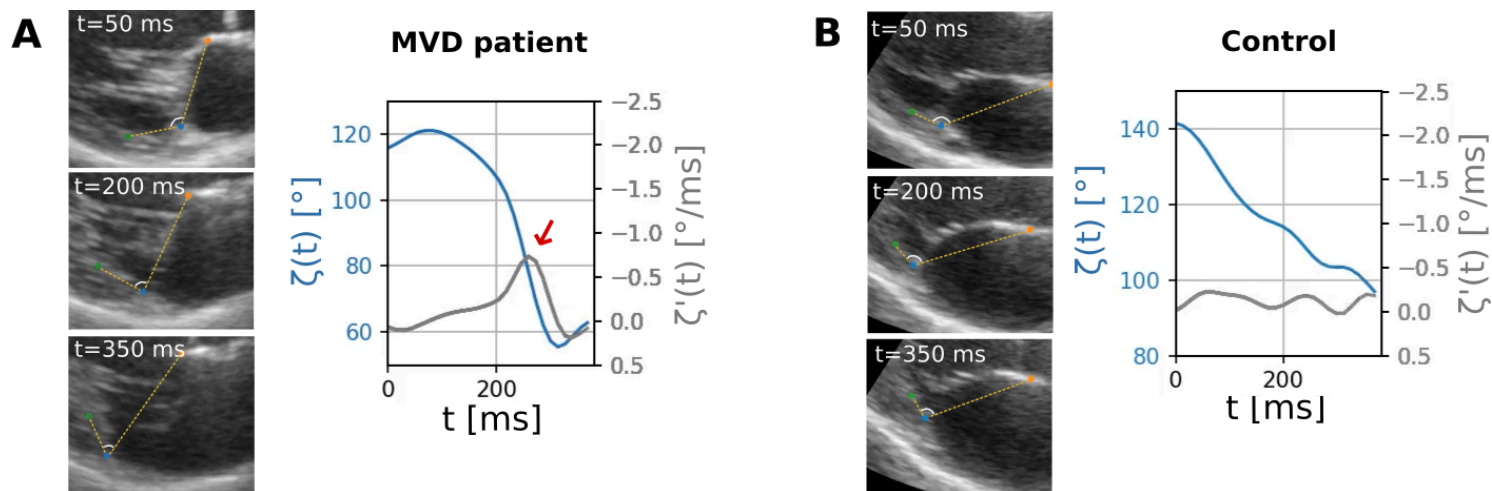


Figure 6

Atrio-ventricular angle ζ in MVD patient vs. control. A: MVD patient displaying a high degree of curling with a steep decrease in $\zeta(t)$ and spike in derivative. B: Healthy control with gradual decrease.

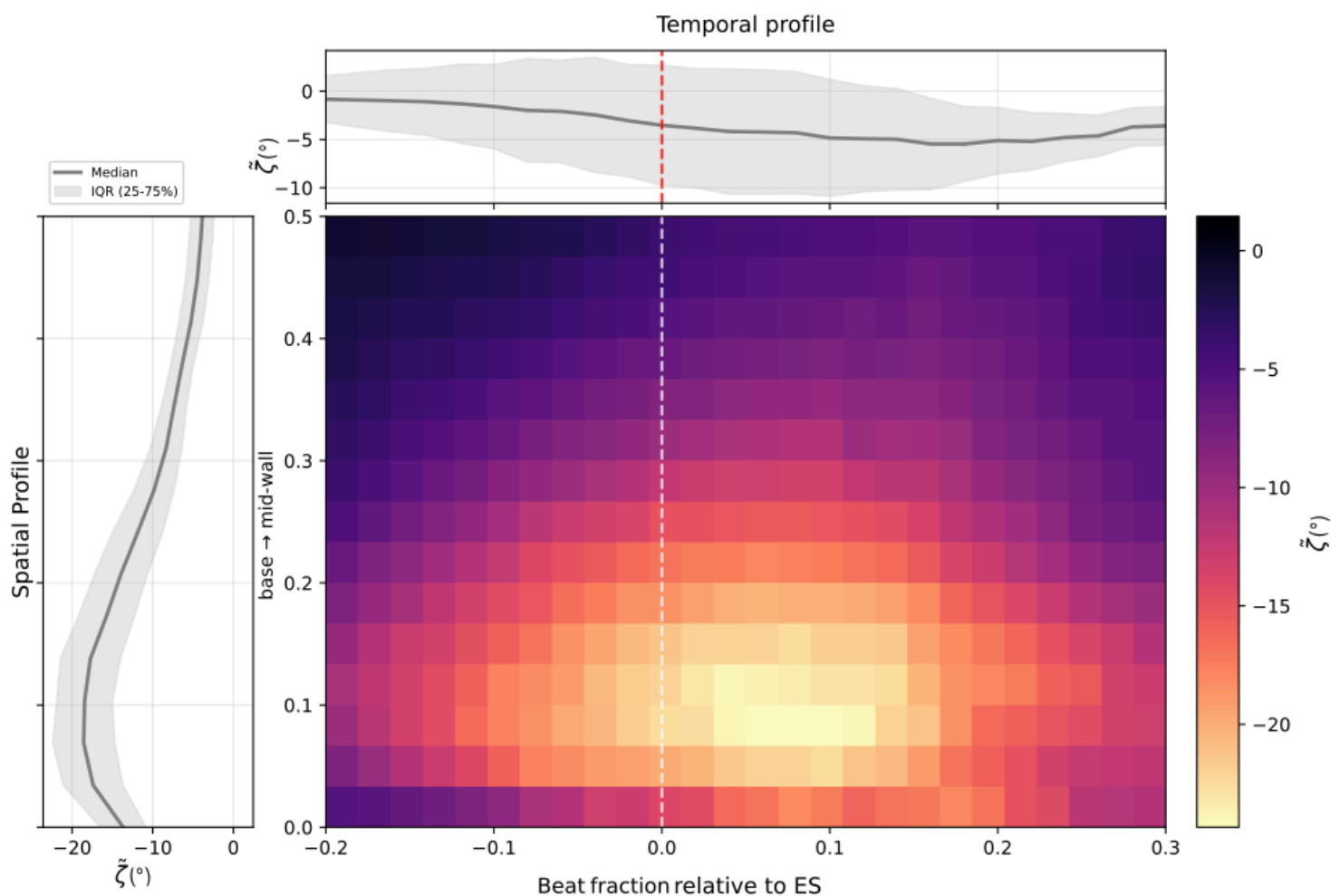


Figure 7

Spatio-temporal distribution of curling motion mechanics. The central heatmap displays the cohort-median annulo-ventricular angle difference relative to ED (ζ), visualizing the magnitude of curling in both space (y-axis: normalized position from base to mid-wall) and time (x-axis: beat fraction relative to ES). Warmer colors (yellow) indicate more negative ζ values, corresponding to more severe curling deformation. Marginal panels illustrate the median (solid gray line) and interquartile range (shaded region) for the spatial and temporal profiles.

Supplementary Files

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