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Role of pericardium in the maintenance of left ventricular twist

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ABSTRACT

Background The role of pericardium in left ventricular (LV) twist has not been directly investigated. We sought to determine the role of pericardium in maintenance of LV twist function in an animal experiment, before and after pericardial opening.

Methods 13 mongrel dogs were initially operated on, but two dogs were excluded from the final analyses owing to poor speckle tracking. Intraoperative echocardiography for conventional and speckle tracking measurements was performed at baseline with intact pericardium, and after pericardial opening. Using the speckle tracking technique, LV twist and strains were obtained before and after pericardial opening in 11 animals and additionally after pericardial repair in five animals.

Results LV twist was significantly decreased after pericardial opening (10.1° (5.1°) to 7.4° (6.4°), p = 0.001). LV twist and untwist rate were also decreased (115.0° (99.6°)/s to 66.7° (42.5°)/s for twist rate, −127.6° (74.3°)/s to −84.2° (734°)/s for untwist rate, p = 0.015 and 0.009, respectively). LV stroke volume and ejection fraction were similar irrespective of pericardial opening, but radial strain measured at the mid ventricular level was significantly increased (31.7% (17.4%) to 32.3% (24.0%), p = 0.024) after pericardial opening without changes in circumferential and longitudinal strains. LV twist degree was restored after pericardial repair.

Conclusion The pericardium is an important structure for maintaining LV twist. Given no significant impact of the presence or absence of pericardium on LV systolic performance, an increase in LV radial strain serves as a compensatory mechanism to preserve LV systolic function despite a decrease in LV twist in the absence of pericardium.

The pericardium is an avascular fibrous sac surrounding the heart and great vessels. It has a significant role in maintaining the position of the heart via attachments to the sternum, diaphragm and mediastinum. The role of the pericardium is not restricted to maintenance of cardiac position. It also protects the heart from the mechanical stress generated by adjacent organs and acts as a barrier that prevents the dissemination of infection. The pericardium is also believed to prevent excessive dilatation of cardiac chambers and to secrete immunological mediators. The role of the pericardium in the maintenance of ventricular function, and hemodynamic changes associated with removal of pericardium has been investigated.1 2

The presence of pericardium was reported to affect the interventricular interaction,3 although it was found to be insignificant in clinical situations.4 5

Left ventricular (LV) twist is a complex myocardial motion that is generated by the dynamic interaction between oppositely wound epicardial and endocardial myocardial fibre helices.6 7 From the functional perspective, LV twist with ensuing untwist motion has been reported to contribute significantly to maintain LV systolic and diastolic functions.8–15 Recently, a decrease in LV twist was reported in a small number of patients with congenital total absence of pericardium.6 We believed that this finding requires confirmation by human study or animal experiment, because in this earlier study an indirect comparison of changes in the extent of LV twist were conducted in completely different populations—that is, in normal controls and in patients with congenital total pericardial absence.5 Therefore, the purpose of the current work is threefold. The first objective was to investigate and confirm the previous finding5 regarding a drop in the magnitude of LV twist in the absence of pericardium. To achieve this objective, we employed an animal model and tried to investigate directly the impact of pericardium on LV twist mechanics before and after pericardial opening. The second purpose of this study was to evaluate whether LV systolic and diastolic function can be preserved even with a significant decrement in the LV twist, because LV twist is closely linked to the LV systolic function according to the previous study, as measured by LV EF7 Thus, the third purpose of this study was to elucidate how LV performance can be maintained without the support of pericardium.

METHODS

Animal preparation

Thirteen mongrel dogs, weighing 17–22 kg, were anaesthetised with thiopental sodium (20 mg/kg intravenous) for induction, after which they were intubated and mechanically ventilated using an artificial respirator in the supine position. Anaesthesia was maintained with 1–2% isoflurane with supplemental oxygen. Arterial oxygen saturation was monitored to remain over 95% and PCO2 was maintained in the physiological range by adjusting respiratory rate. Arterial blood pressure and ECG were monitored continuously.

The animal’s heart was exposed through a median thoracotomy. The pericardium was opened after baseline echocardiography had been performed. An incision was made through the vertical line along the anterior portion of the interventricular groove, from just proximal to the atrioventricular groove to the posterior side of LV mid wall in an attempt to expose the whole LV as much as possible. After pericardial opening, the
animals were allowed to remain stable for 15 minutes and then echocardiography was repeated. In the last five of the 11 animals, pericardium was repaired with care, so as not to produce pericardial constriction, and then echocardiography was performed once again.

All the animals were housed at the Laboratory Animal Facility of our Clinical Research Institute and the study protocol was approved by our Institutional Animal Care and Use Committee.

Echocardiographic image acquisition

Echocardiographic Epiphrenic echocardiography was performed using a commercially available echocardiography machine (Vivid i, GE Medical System, Horten, Norway) in the supine position by single researcher. Saline bath was used to avoid the near field artefact. A 2D, M-mode and Doppler echocardiographic examination was performed to obtain data on LV systolic and diastolic dimensions, wall thickness, LV ejection fraction (EF) and pulsed-wave Doppler in mitral inflow. LV EF was calculated using the hipline Simpson’s method. Peak early (E) and late (A) diastolic velocities of the mitral inflow were measured using a pulsed-wave Doppler at the tip of mitral leaflets. Systolic (S’), early (E’) and late (A’) diastolic mitral annular velocities were acquired at the septum in apical four-chamber view. Time velocity integral (TVI) at the LV outflow tract were obtained using a pulsed-wave Doppler at the apical five-chamber view and then LV stroke volume was calculated using LV outflow tract diameter and TVI. LV spasticity index, defined as the ratio of the long-axis and short-axis LV dimensions, was measured from the apical four-chamber view in end diastole as previously described.14

Techniques used for the speckle tracking measurements were described in detail previously.17 In brief, scanning of apical, mid and basal short-axis planes was performed in each animal without using a dual-focusing tool. Frame rate (range 80-100 frames/s) was adjusted for optimal image acquisition. Sector width and image depth were optimised to maintain an adequate, highest frame rate without losing the 2D image quality. Basal, mid and apical levels were defined as the point of the tips of mitral valve leaflets, papillary muscle level and just proximal to the level with LV luminal obliteration at the end-systolic period, respectively.14 Maximal effort was made to make the LV cross-section as circular as possible. To obtain reliable values, three consecutive heart beats were digitally stored in cine-loop format and analysed offline. Image acquisition was performed repetitively using the same protocol before and after pericardial opening, and after pericardial repair procedures.

Speckle tracking analysis

Image analysis was performed by one independent cardiologist using a customised dedicated software package (EchoPac 7.05 for PC, GE Medical Systems). LV endocardial borders were manually traced during the end-systolic phase for speckle tracking echocardiography analysis. The reliability of tracking was confirmed using the reliability parameter in the EchoPac system (V=valid tracking, X=unacceptable tracking), and was again visually checked. In the case that the reliability of speckle tracking was confirmed by EchoPac software, but was not satisfactory by visual assessment, we continued to perform the same analysis procedure repetitively until we got the most appropriate speckle tracking. LV rotations and rotation rates at the basal or apical short-axis planes were determined as average angular displacement of six myocardial segments. Curves of the averaged basal and apical rotation/twisting rate in six segments and LV twist/twisting rate can be directly generated from this version of the EchoPac program. LV twist, apical and basal rotation, peak twisting rate and peak untwisting rate were measured using curve from the EchoPac program. LV torsion was calculated using LV twist divided by LV end-diastolic long axis as previously described.7 Radial and circumferential strains were obtained from the mid-ventricular short axis plane by averaging the peak radial and circumferential strains of six myocardial segments.

Statistical analyses

All continuous variables were presented as median (IQR). Wilcoxon’s signed ranks test was used to compare the variables before and after pericardial opening and after pericardial repair. All statistical analyses were performed using SPSS 13.0, and p values of <0.05 were considered statistically significant.

RESULTS

Echocardiographic data from two animals could not be analysed owing to poor speckle tracking. Accordingly, data from 11 animals are presented. Haemodynamic data of the 11 animals analysed were unchanged throughout the experiment (table 1).

Effect of the presence of pericardium on LV systolic and diastolic function, and LV shape

LV systolic and diastolic function show no significant difference before and after pericardial opening, as evidenced by the LV EF, S’, E and E/E ratio (table 1). LV ejection fraction displayed a trend towards a decrease after pericardial removal, but the
degree of decrement did not reach statistical significance. $S\prime$, a more sensitive index for LV systolic function, was similar before and after pericardial opening, and likewise, $E$ and $E/E\prime$ ratio did not change significantly. Of note, the LV sphericity index significantly decreased after opening the pericardium, implying that LV shape was transformed into a more globular shape after pericardial opening.

LV mechanics before and after pericardial opening

Changes in the LV twist and twist rate are summarised in table 2. LV apical rotation dropped significantly from $10.1^\circ (3.8^\circ)$ before to $7.0^\circ (5.3^\circ)$ ($p<0.001$) after pericardial opening. A decrement in LV basal rotation was also demonstrated, but with borderline statistical significance ($−4.2^\circ (4.6^\circ)$ before pericardial opening versus $−2.0^\circ (2.0^\circ)$ after pericardial opening, $p=0.07$). Consequently, LV twist was significantly decreased after pericardial opening (figures 1 and 2). Peak LV twist and untwist rates were also decreased after opening of pericardium. LV radial displacement-twist curve (figure 3) showed a figure-of-eight configuration with a linear systolic phase, a rapid early untwisting phase and a late untwisting phase as previously described. The slope of a linear systolic phase was smaller after pericardial opening ($−6.2^\circ (3.8^\circ)/\text{mm}$ before pericardial opening vs $−5.2^\circ (4.6^\circ)/\text{mm}$ after pericardial opening, $p=0.65$).

Of the 11 dogs, five underwent surgical repair of the pericardium to verify restoration of LV twist. Conventional echocardiographic parameters including 2D and Doppler did not change after pericardial repair. Although not exactly the same as the baseline level, LV twist significantly increased after pericardial repair ($14.0^\circ (5.2^\circ)/\text{mm}$ after pericardial repair vs $12.0^\circ (4.2^\circ)/\text{mm}$ before pericardial repair, $p=0.01$).

Figure 1  A representative case demonstrating a decrease in the degree of left ventricular (LV) basal and apical rotations before (A) and after (B) pericardial opening (PO).

Figure 2  Changes in left ventricular (LV) twist are shown in each of the 11 animals. LV twist was found to significantly decrease after pericardial opening (PO). LV twist was restored after pericardial repair in five animals.
Pericardial disease

Figure 3 A serial change in the extent of left ventricular (LV) twist in a dog. Before pericardial opening (PO), LV twist was estimated to be 11.3°. This significantly decreased to 7.4° after PO and subsequently increased to 9.1° after pericardial repair. LV radial displacement-twist loop shows smaller slope of linear systolic phase in LV after PO. Slope of a rapid early untwisting phase was not changed after PO. ○, aortic valve closure; ●, mitral valve opening; ●, mitral closure; ○, aortic valve opening.

In terms of LV strain measurements, LV radial strain increased significantly after pericardial opening, but circumferential and longitudinal strains did not change significantly (table 3). After pericardial repair (n=5), LV radial strain tended to decrease (34.3% (11.7%) before to 28.5% (5.0%) after, p=0.08). Circumferential and longitudinal strains did not show significant differences before and after pericardial repair (table 4).

DISCUSSION

Role of pericardium in the maintenance of LV twist motion

The clinical importance of pericardium has been undermined by the early notion that pericardiectomy does not result in any obvious negative consequences, despite the fact that several important functions of pericardium had been previously described.16 17 According to the earlier studies, pericardium has been suggested to have a possible role in the interventricular interaction,9 although pericardial opening does not significantly affect the LV systolic and diastolic function in clinical situation.4 5 Other researchers suggested that a well-lubricated pericardium is important for normal twist,18 but they did not provide the data about the change of LV twist without pericardium. However, a recent study by Tanaka et al using 2D speckle tracking echocardiography showed that pericardial defects observed in patients with congenital total absence of pericardium leads to a lack of LV twist compared with normal controls.6 Interestingly, they also observed that LV regional myocardial function in these patients with congenital total absence of pericardium was not impaired despite a lack of LV twist motion,7 which is in agreement with earlier findings.4 5 8 Although the work by Tanaka et al provides a very interesting observation on LV twist in relation to the presence or absence of pericardium, their study was limited owing to the indirect nature of the comparisons of LV twist magnitude between patients with congenital total absence of pericardium and normal controls—that is, no comparison was possible before and after pericardial removal. Accordingly, a study comparing the LV twist performance just before and after pericardial opening was needed to clarify the issue. In addition, if LV twist is significantly decreased by pericardial opening along with preservation of LV systolic ejection performance, it should be elucidated which myocardial or ventricular alteration is responsible for a successful compensation of a decline in the LV twist, given that LV twist motion is closely associated with LV ejection performance.7

In the present study, we found that the extent of LV twist, and twist and untwist rate were significantly reduced by pericardial opening, which tended to be restored after pericardial repair. LV EF tended to decrease but did not show statistical significance. The S', E' and E/E' ratio did not show significant alteration. Even though the untwisting rates were decreased after pericardial opening, the slope of a rapid early untwisting phase in twist-radial displacement curve was not decreased after pericardial opening, which suggested that early diastolic suction was preserved after pericardial opening. This results indicate that a pericardial defect or pericardium removal itself can significantly attenuate the extent of LV twist and twist rate without significant LV systolic and diastolic performance. Therefore, our data confirmed that the LV twist mechanics are significantly affected by the presence or absence of pericardium. Importance of pericardium in terms of twist function is further supported by the restoration of LV twist by pericardial repair. If we cannot propose a definite mechanism for the significant decrease in LV twist observed after pericardial opening, but can suggest a possibility. Some researchers have insisted that the geometry of the myocardium should influence LV twist mechanics.19 20 Previous studies have depicted the LV myocardial architecture as a transmural continuum between two helical myocardial fibre geometries, whereby a right-handed helical fibre orientation in the subendocardial region gradually and smoothly changes into left-handed helical fibre orientation in the subepicardial region.7 21 Pericardial opening or removal causes morphological changes in LV shape, as demonstrated by a change in the LV sphericity index (1.61 (0.15) before pericardial opening vs 1.53 (0.14) after pericardial opening, p=0.01), which suggests a change in LV from elliptical to globular. This globular shape of the LV causes the alignments of the counter-directional helical myocardial fibres to be more transverse rather than longitudinal, which may contribute to the increase in radial...

Figure 4 A serial change in the extent of left ventricular (LV) twist in a dog. Before pericardial opening (PO), LV twist was estimated to be 11.3°. This significantly decreased to 7.4° after PO and subsequently increased to 9.1° after pericardial repair.
function. Nevertheless, this study does offer unique insights into the chronic effect of pericardial absence on LV twist or LV strains in animals which underwent pericardial repairs. These findings might show small alterations in LV function or LV twist, as was demonstrated by LV EF, as previously reported.2 4–6 When considering the significant reduction in LV twist, a change of LV systolic performance is relatively small, which suggests the presence of a compensatory mechanism. Based on our findings, the decrement in LV twist induced by pericardial opening appears to be balanced by an increment in LV radial strain. This increase in radial strain is probably the result of a more transverse rather than longitudinal direction of myocardial fibres after the opening of pericardium.

LV geometry change and LV twist

We found that the pericardium has a pivotal role in maintaining LV twist motion, but our data showed preserved LV systolic performance, as was demonstrated by LV EF, as previously reported.2 4–6 When considering the significant reduction in LV twist, a change of LV systolic performance is relatively small, which suggests the presence of a compensatory mechanism. Based on our findings, the decrement in LV twist induced by pericardial opening appears to be balanced by an increment in LV radial strain. This increase in radial strain is probably the result of a more transverse rather than longitudinal direction of myocardial fibres after the opening of pericardium.

Study limitations

Several limitations of this study require consideration. First, the pericardial repair was performed in only a small number of studied animals; thus, although there was a trend, we did not show the full recovery of myocardial mechanics after pericardial repair with statistical significance. Second, a change of LV twist was performed only at resting state. Considering LV torsion reserve with exercise might be an important contributor to normal systolic function,2 4–6 we cannot assume the role of pericardium to maintain LV twist and systolic function during physiological exercise in humans. Furthermore, measurement of invasive haemodynamic monitoring using a manometer system might show small alterations in LV function or LV twist according to loading change, which has not been performed in our study. Additionally, the study provides no information on the chronic effect of pericardial absence on LV twist or LV function. Nevertheless, this study does offer unique insights into the acute effects of pericardial opening, and resulted in a proposal regarding the mechanism underlying LV twist reduction after pericardial removal and regarding the compensatory mechanism that appears to maintain LV ejection performance despite a reduction in LV twist.

CONCLUSIONS

The pericardium is an important structure in terms of the maintenance of the LV twist. Without pericardium, LV twist was significantly reduced and, furthermore, this reduction in LV twist was found to be restored by pericardial repair. Changes in LV morphological constraints are likely to be responsible for this decrease in LV twist in the setting of pericardial opening. In addition, LV radial strain was found to increase in this setting, which appears to be associated with a compensatory change to preserve LV systolic performance.

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Competing interests None.

Ethics approval This is an animal study and the committee of animal ethics approved this study.

Contributors SAC obtained the echocardiographic data and analysed. HKK and SO provided the animal experimental setting for our study. YJK and GYC advised in the interpretation of data.

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