Anterior Mitral Basal 'Stay' Chords are Essential for Left Ventricular Geometry and Function

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Background and aim of the study: **Among the anterior mitral basal chords, two particularly strong and thick stay chords (SC) remain under tension during the entire cardiac cycle. Collagen fibers of the anterior mitral leaflet (AML) are oriented from insertion of the SC on the AML to the fibrous trigones (FT), suggesting that local stress is directed from the papillary muscles (PM) over the SC and AML to the FT, maintaining left ventricular (LV) geometry.**
Methods: **Sonomicrometry crystals were implanted in sheep at the LV apex (A), the anterior (AW) and septal (SW) LV wall, the PM tips (M1 and M2), the SC insertion into the AML (S1 and S2), the posterior (PMA) and lateral (P1 and P2) mitral annulus, the FT (T1 and T2), the tips of the anterior (AL) and posterior (PL) mitral leaflets, and the base of the aortic right coronary sinus (RCS). Changes in distances, areas, and volume were time-related to aortic flow and LV and ascending aorta pressures. Recordings were taken at baseline and after transection of the SC.**
Results: **After transection of the SC, the systolic distance from M1-T1 increased by +0.96 ± 0.41 mm ($p < 0.05$) and from M2-T2 by +0.97 ± 0.42 mm ($p < 0.05$). The LV length increased at T1-A by +1.14 ± 0.60 mm ($p < 0.05$) and at T2-A by +0.97 ± 0.37 mm ($p < 0.05$). The aortomitral angle narrowed at end-systole by –3.26 ± 0.85° ($p < 0.05$). Transection of the SC reduced dP/dt by –11.20 ± 5.29% ($p < 0.05$), maximum aortic flow by –16.89 ± 7.86% ($p < 0.05$), and maximum pressure-volume ratio by -10.83 ± 3.36% ($p < 0.05$).**
Conclusion: **Transection of the anterior mitral SC did not result in mitral regurgitation but induced significant changes in LV geometry, including narrowing of the aortomitral angle and subsequent deterioration of LV function. The SC are essential for maintaining normal LV geometry and function.**

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It is becoming increasingly apparent that the mitral valve is an integral part of the left ventricle. It is no longer tenable that the mitral valve consists simply of the annulus, leaflets, chords and papillary muscles (PM). Numerous studies have shown the importance of maintaining annulopapillary continuity if left ventricular (LV) systolic function is to be preserved (1,2), and this fact has been advanced as one of the main reasons for the superiority of valve repair over replacement (3). Insights into functional mitral regurgitation (where an anatomically normal valve can become insufficient because of altered ventricular geometry) reinforce the concept of a single functional structure (4).

The function of the marginal mitral valve chords is to maintain mitral leaflet apposition during valve closure (5). The function of the anterior basal chords is not clear, but has been shown to support the belly of the anterior mitral leaflet (AML) (6). Among these basal chords, there are two particularly thick, tendon-like chords that stretch from the PM to the undersurface of the AML close to the anterior mitral annulus. The description in isolated perfused pig hearts by van Rijk-Zwikker et al. (7) that these two chords remained tense during the entire cardiac cycle led the present authors to use the nautical term of 'stay chords', which support the mast under any wind direction when sailing (8). In the isolated working pig heart, Obadia et al. (5) have shown that transection of all anterior basal chords was

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immediately followed by a significant decrease in aortic flow and a reduction in LV anterior wall shortening fraction without mitral regurgitation or change in leaflet coaptation.

Materials and methods

Animals

Six Targhee sheep (mean ±SD) bodyweight 58 ± 18 kg) underwent implantation of 15 ultrasonic crystals (Fig. 1) using cardiopulmonary bypass (CPB) (cross-clamp time 81 ± 23 min). All animals received humane care in accordance with the *Principles of Laboratory Animal Care* formulated by the Animal Welfare Act and the *Guide for Care and Use of Laboratory Animals* prepared by the Institute of Laboratory Animal Resources and published by the National Institutes of Health (NIH publication #85-23, revised 1996). This project was also reviewed and approved by the Institutional Animal Care and Use Committee (IACUC) of The University of Montana.

Surgical protocol

Anesthesia and surgical preparation of the sheep was performed as previously described (9). Thirteen 2-mm ultrasonic crystals (Sonometrics, London, Ontario, Canada) were implanted and secured with 5-0 polypropylene sutures at the LV apex (A), the origin of the SC on the PM (M1 and M2), the anterior LV endocardial wall between the tips of the PM (AW), the septal LV endocardial wall at the level of the PM tips (SW), the midpoint of the posterior mitral annulus (PMA), the lateral mitral annulus (P1 and P2), both fibrous trigones (FT) (T1 and T2), the insertion of the SC on the AML (S1 and S2), and the lowest point of the right coronary sinus (RCS). Additionally, two 1-mm ultrasonic crystals were implanted at the midpoint of the free edge of the anterior (AL) and posterior (PL) mitral leaflets (Fig. 1).

Electrodes of the crystals placed in the mitral annulus, trigones, SC, and mitral leaflet tips were exteriorized through the left atriotomy, and those in the PM and the anterior and posterior LV wall were exteriorized through the LV apex. Two insulated wires were placed around the SC close to their leaflet insertion, and the part of the wire in contact with the chords was denuded. Both of these wires were exteriorized through the LV septum and the right ventricular outflow tract to prevent injury to the other chords when diathermy was applied.

High-fidelity, catheter-tipped pressure transducers (Model 510; Millar Instruments, Houston, TX, USA) were placed within the lumen of the proximal ascending aorta and the LV cavity through the apex. A flowmeter ring (Transonic Flowmeter T206; Transonic Systems, Inc., Ithaca, NY, USA) was placed around the ascending aorta.

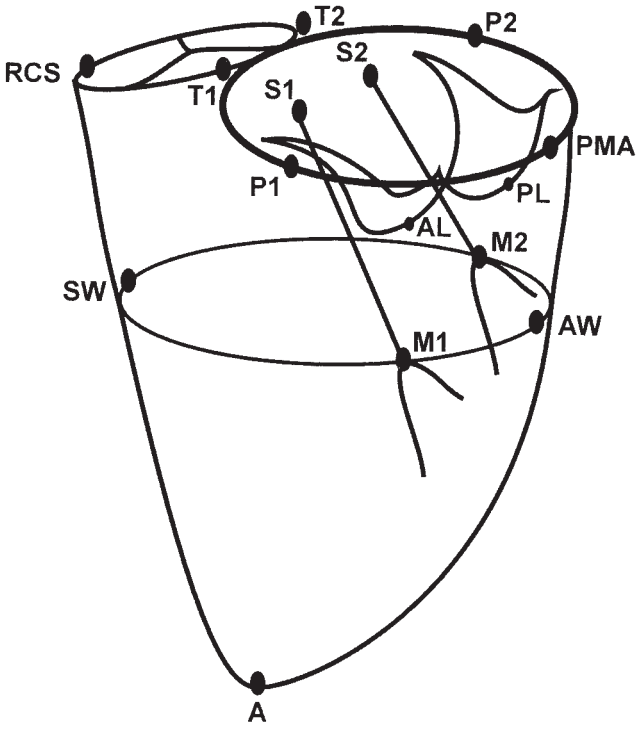

Figure 1: Diagram showing crystal locations at: A: Apex; SW: Endocardial septal wall; AW: Endocardial anterior wall; M1: Anterolateral papillary muscle tip; M2: Posteromedial papillary muscle tip; RCS: Right coronary sinus; T1: Anterior trigone; T2: Posterior trigone; P1: Anterolateral mitral annulus; P2: Posterolateral mitral annulus; PMA: Midpoint of the posterior mitral annulus; S1: Insertion of anterior stay chord on anterior mitral leaflet; S2: Insertion of posterior stay chord on anterior mitral leaflet; AL: Tip of anterior mitral leaflet; PL: Tip of posterior mitral leaflet.

After weaning from CPB, the venous and arterial cannulae were removed. Under hemodynamically stable conditions (at least 30 min after weaning from CPB) and after closing the pericardium, baseline recordings were taken. Sectioning of the two SC was achieved in the beating heart by applying diathermia to the two wires placed around the SC close to their leaflet insertion. Epicardial, two-dimensional (2D) color echocardiography was performed to determine mitral competence. Subsequently, a new set of recordings was acquired, paying attention to maintain similar filling pressures in all sheep.

Definition of cardiac cycle phases

Geometric changes were time related to each phase of the cardiac cycle defined from the aortic and LV pressure curves. End-diastole, or beginning of systole,

was defined as the point of increasing LV pressure tracing (dP/dt > 0). End of isovolumic contraction was defined as the beginning of ejection at the crossing point of the left ventricle and aortic pressure curves (gradient aortic/LV pressure = 0). The dicrotic notch in the aortic pressure curve defined end-systole. End-diastole was defined as the lowest point of the LV pressure tracing after ejection (dP/dt = 0) (10).

Definition of anatomic regions and calculations

Distances between crystals were explored in a coordinate-independent analysis (11). Insertion of the SC on the PM was defined as the origin of the SC (M1 and M2). Their attachment to the undersurface of the AML was determined by pulling on each SC to show a dimple in the atrial surface of the leaflet. The anterior (T1) and posterior (T2) FT were identified by pulling on the midpoint of the AML, which created folds terminating at the trigones (12). The endocardial anterolateral wall (AW) was represented by the crystal placed on the anterior LV wall between the PM tips, and the endocardial LV septal wall (SW) was represented by the crystal in the midseptum at the level of the PM tips. The mitral annulus plane was defined as the plane that included both FT (T1 and T2) and the middle of the PMA. The aortic annulus plane was defined as plane incorporating both FT (T1 and T2) and the lowest point of the right coronary sinus (RCS) (Fig. 1). Both planes defined the base of the heart with a hinge at the axis between the FT (T1 and T2). The aortomitral angle was defined as the angle formed by the aortic and mitral annulus planes, and was calculated by a line traced between the RCS and the midpoint between T1 and T2 and another traced between PMA and the midpoint between T1 and T2 with the apex of the angle at the axis between T1 and T2 (Figs. 3a and b). The maximum

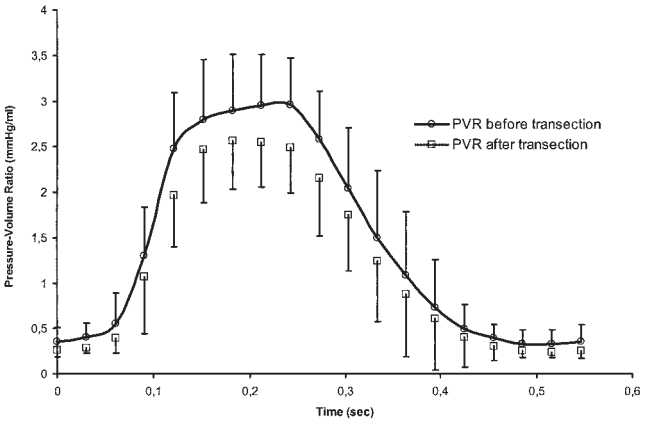

Figure 2: Mean time-varying pressure-volume ratio (PVR) before and after transection of the stay chords.

pressure-time quotient (dP/dt) was calculated from end-diastole (dP/dt > 0) as the steepest LV pressure-time slope (10). Maximum pressure-volume ratio (MPVR) was calculated by instantaneous LV pressure and an estimation of instantaneous LV volume (13), assuming that the left ventricle was a general ellipsoid (14).

Data acquisition

Distances were measured with the Sonometrics Digital Ultrasonic Measurement System TRX Series using transmitter/receiver crystals. A post-processing program (Sonometrics Corporation, London, Ontario, Canada) was used to examine each individual length tracing between crystals and for three-dimensional (3D) reconstruction of the crystal coordinates. The data sampling rate was 200 Hz. Millar pressure transducer control units TCB 600 and MIKRO-TIP pressure transducers (Millar Instruments) were used to obtain the LV

Table I: Hemodynamic measurements and mitral regurgitation by echocardiography before and after transection of the stay chords.

Hemodynamic measurement	Baseline	After transection
Heart rate (beats/min)	108.72 ± 14.31	110.13 ± 14.35
Central venous pressure (mmHg)	10.17 ± 1.60	10.83 ± 1.17
Left atrial pressure (mmHg)	9.33±1.21	9.83 ± 1.33
LV systolic pressure (mmHg)	95.49 ± 10.97	97.99 ± 24.35
LV end-diastolic pressure (mmHg)	14.75 ± 5.19	16.53 ± 1.55
Stroke volume (ml)	44.49 ± 11.45	41.23 ± 10.74
dP/dt (mmHg/s)	1,774.90 ± 601.23	1,592.41 ± 610.59*
Maximum aortic flow (ml/min)	21,888.85 ± 4,086.26	18,179.60 ± 4,840.35*
Maximum pressure-volume ratio (mmHg/ml)	3.01 ± 0.51	2.68 ± 0.47*
Systemic vascular resistance (dynes/cm^5/m^2)	1,245.14 ± 847.19	1,252.22 ± 833.16
Mitral regurgitation by echocardiography	0	0

Values are mean ± SD.
*p ≤0.05, versus baseline.

and aortic pressures. All other pressure measurements were taken directly by inserting a 20-gauge needle and using a conventional pressure transducer. Aortic flow was recorded using a Transonic flowmeter T206 and a 20A ultrasonic flow probe (Transonic Systems, Inc., Ithaca, NY, USA) placed around the ascending aorta. All distances, pressures, and flows were displayed and recorded simultaneously on the same screen by the Sonometrics system. This ensured that all data were synchronized and recorded on the same timeline. Epicardial, 2D echocardiography with pulsed-wave and color Doppler was used to determine the presence of mitral regurgitation before and after SC transection.

Measurement and statistical analysis methods

After close examination of the data, three consecutive heartbeats that contained the least amount of noise were chosen for analyses. The summary statistics are reported as mean ± SD. Hemodynamic and geometric measures were compared using the two-tailed *t*-test for paired comparisons, with a significance level of $p < 0.05$ (corrected by Stepdown Bonferroni for multiple pair wise comparisons). Statistical analyses were performed using the SAS/STAT software MULTTEST procedure (SAS Institute, Inc., Cary, NC, USA).

Results

Before and immediately after transection of the SC, no mitral regurgitation was detected by echocardiography. Post-mortem examination of the heart showed crystals in the correct position and the SC transected approximately 2-3 mm from their leaflet insertion. The other basal and marginal chords were intact.

Hemodynamic changes

Transection of the SC induced an immediate reduction in dP/dt by –11.20 ± 5.29%, from 1,774.90 ± 601.23 mmHg/s to 1592.41 ± 610.59 mmHg/s ($p < 0.05$). Maximum aortic flow was reduced by –16.89 ± 7.86%, from 21,888.85 ± 4,086.26 ml/min to 18,179.60 ± 4,840.35 ml/min ($p < 0.05$). The MPVR was reduced by –10.83 ± 3.36%, from 3.01 ± 0.51 mmHg/ml to 2.68 ± 0.47 mmHg/ml ($p < 0.05$) (Fig. 2). None of the other hemodynamic parameters revealed any significant differences after transection of the SC (Table I).

Sonomicrometry results

At baseline (Fig. 3a), most distances between crystals shortened significantly during systole. Only the distances between the PM tips and insertion of the SC on the AML (M-S) and the PM tips to the FT (M-T)

Table II: Distances at baseline and after transection of stay chords at end-diastole and end of systole and change in distance from end-diastole to end of systole.

Distance	Baseline			After transection		
	End-diastole	End of Systole	Change in distance	End-diastole	End of systole	Change in distance
M1-T1 (mm)	28.88 ± 5.32	30.47 ± 5.61	+1.58 ± 0.65*	29.73 ± 5.31	31.42 ± 5.93	+1.91 ± 1.54*
M2-T2 (mm)	33.37 ± 2.08	34.38 ± 1.88	+1.00 ± 0.45*	34.14 ± 1.92	35.35 ± 1.93	+1.20 ± 0.58*
M1-S1 (mm)	21.15 ± 2.65	22.68 ± 3.05	+1.57 ± 0.64*	22.25 ± 3.13	23.09 ± 9.98	+1.59 ± 0.65*
M2-S2 (mm)	22.93 ± 1.10	24.22 ± 1.27	+1.29 ± 0.68*	24.23 ± 1.76	26.96 ± 1.62	+2.73 ± 1.29*
T1-A (mm)	93.91 ± 11.75	88.77 ± 12.00	-5.14 ± 1.85*	94.95 ± 11.64	89.91 ± 11.23	-5.04 ± 2.41*
T2-A (mm)	89.86 ± 11.96	85.83 ± 11.40	-4.03 ± 1.79*	90.88 ± 12.00	86.80 ± 12.15	-4.08 ± 1.52*
P1-A (mm)	90.80 ± 11.10	83.93 ± 9.81	-6.87 ± 2.05*	91.18 ± 11.23	84.13 ± 9.23	-7.05 ± 2.67*
P2-A (mm)	83.87 ± 11.9	78.28 ± 10.34	-5.59 ± 1.81*	84.96 ± 11.16	78.39 ± 9.77	-6.30 ± 2.99*
PMA-A (mm)	84.79 ± 12.75	77.82 ± 11.79	-6.98 ± 2.29*	85.64 ± 12.83	78.49 ± 11.16	-7.14 ± 3.24*
RCS-A (mm)	94.89 ± 13.43	89.28 ± 12.71	-5.61 ± 1.97*	95.03 ± 13.30	89.01 ± 12.18	-5.94 ± 2.29*
T1-PMA (mm)	25.39 ± 3.54	22.45 ± 4.84	-3.77 ± 1.58*	25.71 ± 3.99	22.99 ± 4.59	-3.58 ± 1.37*
T2-PMA (mm)	29.80 ± 5.21	26.49 ± 5.51	-3.31 ± 1.20*	31.71 ± 5.02	28.14 ± 5.73	-4.23 ± 0.67*
P1-P2 (mm)	35.51 ± 5.55	32.42 ± 5.72	-3.09 ± 0.63*	35.62 ± 5.74	31.94 ± 6.39	-3.68 ± 0.87*
M1-M2 (mm)	29.29 ± 6.20	20.05 ± 3.44	-8.42 ± 3.52*	30.20 ± 6.12	19.90 ± 0.59	-10.30 ± 3.40*
AW-SW (mm)	37.60 ± 4.91	26.58 ± 7.97	-11.02 ± 2.84*	37.64 ± 4.64	26.00 ± 3.64	-11.63 ± 3.76*
Aortomitral angle (°)	150.73 ± 15.48	139.66 ± 16.87	-11.06 ± 6.86*	146.57 ± 14.95	136.40 ± 17.22	-10.17 ± 6.83*

Values are mean ± SD.
*$p \leq 0.05$, versus baseline.
A: LV apex; AW: Anterior endocardial LV wall; M: Papillary muscle tip; P: Lateral mitral annulus; PMA: Posterior mitral annulus; RCS: Right coronary sinus; S: Insertion of stay chord on anterior mitral leaflet; SW: Septal endocardial LV wall; T: Trigones

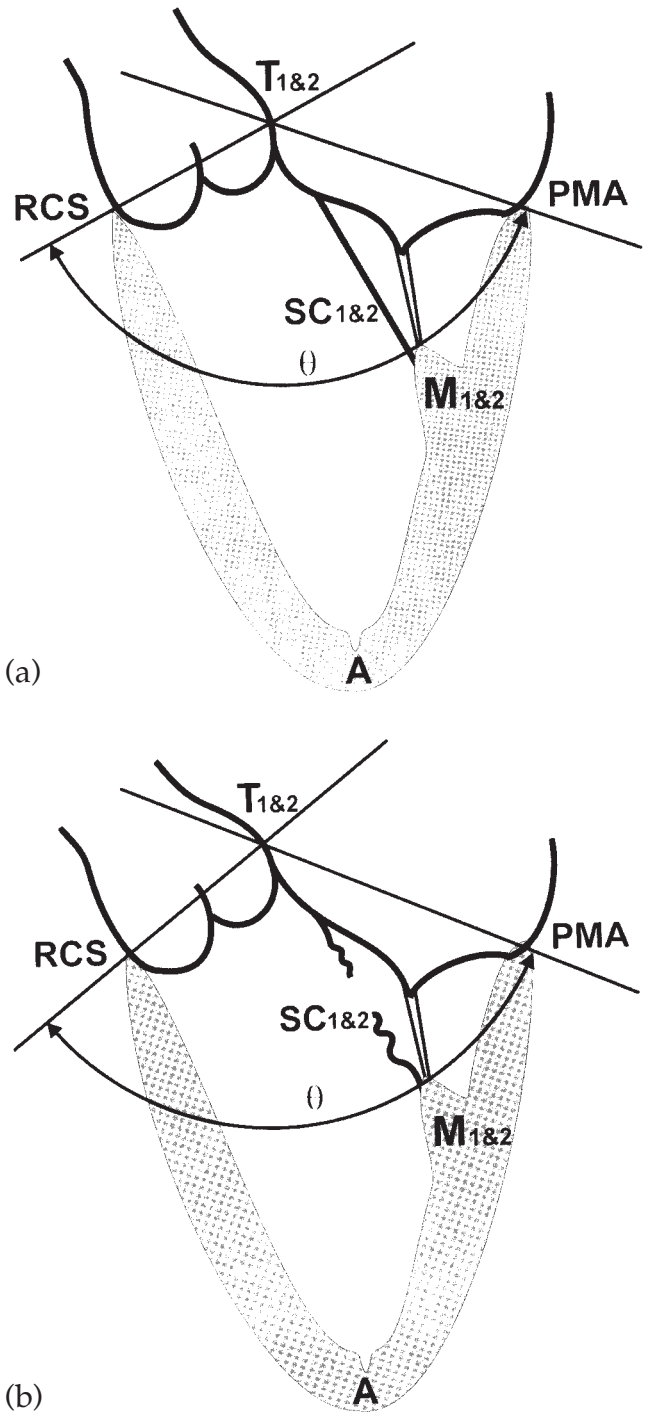

Figure 3: a) Left ventricle and aortomitral angle (θ) at baseline (diastole: 150.7 ± 15.5°, systole: 139.7 ± 16.9°). b) Left ventricle and aortomitral angle (θ) after transection of the stay chords (SC). The aortomitral angle narrowed after transection of SC in diastole by –4.2 ± 1.3° and in systole by –3.3 ± 0.8°. SC1: Anterior stay chord; SC2: Posterior stay chord; A: Apex; M1: Anterolateral papillary muscle tip; M2: Posteromedial papillary muscle tip; RCS: Right coronary sinus; T1: Anterior trigone; T2: Posterior trigone; PMA: Midpoint of the posterior mitral annulus.

increased. These findings represent normal systolic tension on the SC (15) (Table II).

Immediately after transection of the SC (Fig. 3b), the distances between the PM tips and insertion of the SC on the AML (M1-S1 and M2-S2) increased during the entire cardiac cycle (Table II). The distance M1-S1 increased by +0.34 ± 0.69 mm at end-diastole, and by +0.41 ± 0.16 mm at end-systole ($p < 0.05$). The distance M2-S2 increased by +1.30 ± 1.01 mm at end-diastole, and by +2.74 ± 0.57 mm at end-systole ($p < 0.05$). Transection of the SC resulted in a significant increase in systolic and diastolic distances between the PM and the FT. The distance M1-T1 increased by +0.85 ± 0.35 mm at end-diastole ($p < 0.05$), and by +0.96 ± 0.41 mm at end-systole ($p < 0.05$). The distance M2-T2 increased by +0.77 ± 0.26 mm at end-diastole ($p < 0.05$), and by +0.97 ± 0.42 mm at end-systole ($p < 0.05$) (Table III).

After transection of the SC, the distance between the mitral annulus plane (T1-T2-PMA) and the AML at the level of the insertion of the SC (S1 and S2) decreased. The distance was reduced by –0.34 ± 0.17 mm at S1 ($p < 0.05$), and by –3.09 ± 1.17 mm at S2 ($p < 0.05$). The distance from the AML tip (AL) to the plane of the mitral annulus did not change significantly. This indicates a change in AML shape during systole, including billowing of the lateral aspects of the AML (particularly at S2) (Table IV).

Stay cord transection was followed by an increase in LV length. The distance LV apex to trigones (T-A) increased at T1-A by +1.04 ± 0.53 mm at end-diastole ($p < 0.05$), and by +1.14 ± 0.60 mm at end-systole ($p < 0.05$). Similarly, T2-A increased by +1.02 ± 0.38 mm at end-diastole ($p < 0.05$), and by +0.97 ± 0.37 mm at end-systole ($p < 0.05$). However, this overall increase in LV length was not uniform. The distances between the LV

Table III: Change in distances from baseline to after transection at end-diastole and end of systole.

Distance	End-diastole	End of systole
M1-T1 (mm)	+0.85 ± 0.35*	+0.96 ± 0.41*
M2-T2 (mm)	+0.77 ± 0.26*	+0.97 ± 0.42*
M1-S1 (mm)	+0.34 ± 0.69	+0.41 ± 0.16*
M2-S2 (mm)	+1.30 ± 1.01	+2.74 ± 0.57*
T1-A (mm)	+1.04 ± 0.53*	+1.14 ± 0.60*
T2-A (mm)	+1.02 ± 0.38*	+0.97 ± 0.37*
P1-A (mm)	+0.38 ± 0.45	+0.21 ± 0.74
P2-A (mm)	+0.82 ± 1.14	+0.11 ± 0.77
PMA-A (mm)	+0.84 ± 0.55	+0.68 ± 1.26
RCS-A (mm)	+0.14 ± 0.58	-0.19 ± 0.80
M1-M2 (mm)	+0.91 ± 0.34*	-0.15 ± 0.26
Aortomitral angle (°)	-4.16 ± 1.28*	-3.26 ± 0.85*

Values are mean ± SD.
*$p \leq 0.05$, versus baseline.
Abbreviations as Table II.

Table IV: Minimum distances between the mitral annulus plane and the insertion of anterior (S1) and posterior (S2) stay chords and the anterior mitral leaflet tip (AL).

Distance to mitral annulus plane at end of systole	Baseline (mm)	After stay chord transection (mm)	Change in distance (mm)
S1	5.22 ± 2.48	4.88 ± 2.44	-0.34 ± 0.17*
S2	5.61 ± 2.16	2.52 ± 1.39	-3.09 ± 1.17*
AL	5.52 ± 1.46	5.25 ± 1.44	-0.27 ± 0.37

Values are mean ± SD.
*p ≤0.05, versus baseline.

apex and the tips of the PM, and distances between the apex and the PMA, RCS, P1 and P2 did not change significantly after transection of the SC. Therefore, the increase in LV length was due exclusively to the increase in distance between the PM tips and insertion of the SC and trigones. This resulted in a change of the aortomitral angle, which narrowed by –4.16 ± 1.28° during end-diastole ($p < 0.05$), and by –3.26 ± 0.85° during end-systole ($p < 0.05$) (Table III). After SC transection, distances within the mitral annulus (T1-PMA, T2-PMA and P1-P2) did not change significantly from baseline; however, the interpapillary distance (M1-M2) increased significantly during diastole by +0.91 ± 0.34 mm ($p < 0.05$). These findings suggest that the SC have a restraining effect on diastolic interpapillary distention.

After sacrifice, when the heart was explanted, the aorta was completely transected at the sinotubular junction. In all animals, it was observed that the aorta recoiled cranially. An average force of 1.8 ± 0.2 N was needed to pull the aortic root back to its original position. An incremental force of 1.8 ± 0.1 N was necessary to pull it 10 mm further toward the apex of the heart.

Discussion

Although long recognized in anatomic studies (16), the function of the anterior mitral basal (or second-order) chords is unknown. They are often excised in valve replacement or transferred to the free margin (17). Marcus et al. (6) suggested that their function was to support the leaflet belly, avoiding its ballooning. In the present study, it was also found that transection of the two main SC resulted in an increase in systolic motion toward the mitral annulus or lateral portions of the AML.

Among the basal chords, there are two particularly thick chords described in the anatomic literature as 'principal' or 'strut' chords (18). In a study of 50 adult human hearts, Lam et al. (19) showed that these two chords were the longest and thickest of all chords.

Videofluoroscopy studies in normal sheep have shown that the PM to mitral annulus distance remained constant throughout the cardiac cycle (20). Dagum et al. (11) have shown in sheep that although acutely induced myocardial ischemia resulted in LV dilatation and anterolateral displacement of the PM, the absolute distance from PM tips to mitral annulus remained constant. This occurred despite the presence of mitral regurgitation due to leaflet tethering and mitral annulus dilatation. Although no explanation for these findings was advanced at the time, the observation of a video produced by van Rijk-Zwikker et al. (7) suggested to the present authors that the strut chords must be responsible for this constant annulopapillary distance. The study, performed in isolated working pig hearts, showed the presence of two thick, anterior basal chords that remained under tension during the entire cardiac cycle (although this tension is not linear throughout the cardiac cycle) (21). In every mitral valve anatomically studied or surgically explored, there were two recognizable tendon-like chords termed 'stay chords' (8). In a study of the distribution and direction of the collagen fibers within the anterior mitral valve leaflet, Cochran and associates (22) showed that these fibers are oriented from the region of insertion of the SC toward the FT. This finding suggests that the SC connect the PM to both FT under tension.

The results of the present study provide an anatomic basis for the previously reported constant distance between the PM and the mitral annulus (11,20). Selective transection of the SC significantly increased this distance. This increase in length was larger between the posterior PM and the insertion of the posterior SC (M2-S2), suggesting that the posterior SC might carry a heavier load (Tables III and IV). Also, this increase in distance determined an overall LV lengthening (apex-trigones) without a distance change between the apex and the lateral and PMA or the apex to the RCS.

In both an isolated pig heart preparation and in echocardiograms of normal humans, we have observed that the SC are in a single plane that stretch-

es from the PM tips to the aortic curtain and ascending aorta, including the FT. We also observed in the sheep heart that when the aorta was completely transected at the sinotubular junction, the aorta recoiled cranially, and an average force of 1.8 ± 0.2N was needed to pull the aortic root back to its original position. We hypothesized that this elastic recoil of the ascending aorta must be balanced by the SC, which connect the PM to the ascending aorta through the aortic curtain and the FT, constituting the apex of the aortomitral angle. This mechanism would keep the aortomitral angle constant during the entire cardiac cycle.

In the present study, transection of the SC resulted in significant narrowing of the aortomitral angle because the elastic recoil of the ascending aorta was no longer balanced by the pull of the SC at the trigones. Transection of the SC in a normal ventricle significantly decreased dP/dt, maximum aortic flow, and the MPVR. These findings confirm those of Obadia et al. (5), who showed that transection of all anterior basal chords was followed by significant reduction in maximum aortic flow.

In an acute, open-chest, ovine model, Timek et al. (23) found that transection of the SC induced only slight changes in AML geometry without mitral regurgitation. Hemodynamic parameters did not show significant differences after transection of the SC. In that study, LV volume and ejection fraction were calculated using epicardial markers, potentially missing the endocardial changes and changes in aortomitral angle. Messas et al. (24) used chordal cutting to treat ischemic mitral valve insufficiency. On the basis of the present results, chordal cutting cannot be recommended because it might have a negative effect on LV function. Nevertheless, in the present study, it could be confirmed that transection of the two main basal chords resulted in an increase in the systolic motion of the lateral portions of the AML towards the mitral annulus.

One possible explanation for the deleterious effects on LV performance after transection of the SC might be the lack of support of the aortomitral angle's apex and, therefore, the center of the atrioventricular plane. Alam and Rosenhamer (25) have shown clinically the importance of the atrioventricular plane displacement for LV pumping action. The SC might be more important to LV geometry and function than to the mitral valve. In the present study, only acute changes after transection of the SC were examined, therefore, any long-term effects remain unknown.

Study limitations

The present findings were drawn from a limited number of LV markers, and only three markers were placed on the AML. Present technology limits the number of crystals; moreover, the markers and their electrodes might interfere with normal movements of the different structures. An effort was made to exteriorize the electrodes away from the mitral valve. Variability among animals in the position of the crystals was a concern that was minimized by a single surgeon performing all operations. In addition, the data were obtained in an anesthetized, open-chest, acute animal model studied immediately after CPB and cardioplegic arrest. Although these obviously non-physiological conditions limit the value of the findings, the fact that the results were a comparison between before and after chordal transection under the same conditions advocates for the reliability of the data. It must be emphasized that findings in sheep cannot be necessarily applied to the human heart. Sheep have relatively few secondary chords of the AML, and their SC are relatively thinner than in humans; therefore, the role of the SC may be more pronounced in humans. Species differences and experimental conditions forbid the uncritical transfer of these findings to the clinical setting.

In order to calculate the estimated volume, only implanted crystals were used. These crystals represent the most critical points of the left ventricle in these experiments; thus, changes in volume and the change in the pressure-volume relationship after SC transection might be magnified.

In conclusion, selective transection of the two anterior SC resulted in an immediate decrease in the dP/dt and MPVR. Left ventricular length was increased due to an increase in the distance between the PM tips and the FT. This lack of support by the SC resulted in a narrowing of the aortomitral angle. The AML shape was also changed after chordal transection, with a systolic billowing of the lateral aspects of the AML toward the left atrium. It is concluded that the SC not only participate in conserving the systolic shape of the AML but - more importantly - are also essential in maintaining LV geometry and function.

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Meeting discussion

DR. WILLEM FLAMENG (Leuven, Belgium): You must be aware that the ventricular shape in sheep is very conical, and can be compared slightly to a hypertrophic human heart. Did that have any influence on your measurements?

DR. WOLFGANG GOETZ (Singapore): No, I don't think so. The big difference compared to a human heart is that the stay chords in sheep are very much thinner, so their impact on left ventricular geometry and function might be greater in humans.

DR. D. CRAIG MILLER (Stanford, California, USA): You have shown subtle but real declines in ejection performance, and we have now confirmed that. It's important that we speak as a group, and convince oth-

ers that cutting second-order stay chords carries a cost. This technique for ischemic mitral regurgitation - IMR - is gaining popularity all over the world. I think it probably helps the IMR, but at what cost to an already impaired ventricle? I'm afraid that it's very dangerous. Dr. Nielsen recently showed a decline in regional systolic function in sheep, and Dr. Flameng's point is correct - that it's different from humans. But Dr. Rodriguez has recently shown in sheep that left ventricular systolic performance declines if the stay chords are cut. So, if a surgeon aims to do that, they should be asked, 'At what price?'

DR. CARLOS DURAN (Missoula, Montana, USA): I appreciate those comments. I heard only yesterday of a surgeon in Japan who is cutting the chords for the same indication of IMR - so it's becoming endemic, without enough evidence. Another finding that Dr. Goetz could not show here was that, by cutting the anterior basal chords, the movements of the anterior leaflet change completely - so much so that the likelihood of systolic anterior motion by cutting the chords increases considerably. So, cutting them not only for ischemia but also for chordal transfer can be dangerous. The message is that even if we don't know completely what the cords do, they are thick enough not to be ignored.