Surgical reduction of ventricular radius by aspirated plication of the myocardial wall: An experimental study

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At its introduction, partial left ventriculectomy was seen as a promising option.¹ As initially used, its main drawback was its unpredictable effect on cardiac function. The size, shape, and location of the segment to be resected were at the discretion of the surgeon. Once the segment had been resected, any disadvantageous results were irreversible. Resection, and subsequent coaptation of the incision, often compromised coronary perfusion.² Experience showed that insufficient narrowing of the ventricular base could cause late postoperative mitral valvular incompetence.³ Right ventricular function was also impaired by extensive resections from the left ventricle.⁴ A further reported complication is sudden death from ventricular fibrillation, which reportedly occurred within 6 months of the intervention.⁵

Encouraging initial clinical results^{1,6-8} nonetheless have shown that the basic concept of partial left ventriculectomy is sound, at least when conceived as a bridging procedure. To validate the technique and improve its results, we have devised an off-pump strategy that permits reduction of ventricular radius by plication of a chosen segment of ventricular wall. By using aspirator cups, we avoid an open procedure. The technique permits initial sequestration in trial fashion, allowing us to assess ventricular function. Only if it is judged satisfactory do we then perform the definitive ventricular plication. Our new technique of sutured splinting for the definitive plication preserves the coronary vasculature.

Methods

In 8 female pigs weighing 80 ± 7.6 kg, with hearts of normal size and function, we performed left lateral thoracotomies under deep ketamine-halothane anesthesia. The pericardium was split from the base to the apex, and 100 mg lidocaine (Xylocaine) were repetitively injected into the pericardial cradle. An aspirator cup, designed to fit the size and shape of the area to be sequestered, was placed on the left ventricular free wall (Figure 1). Suction was applied, sequestering the segment by plicating it from near the base to the apex. Hemodynamic values were then measured. If the left ventricular filling pressure rose by more than 20 mm Hg, we released the suction and used a smaller sucker. To render the sequestration permanent, two stainless steel splints 1.5 mm in diameter were driven through the protruding bulge parallel to the long edges of the aspirator cup, inserted 3 to 5 mm from the epicardial surface (Figure 1). Transverse sutures were then passed round the two splints, one near the base and the other close to the apex, and pulled tight (Figure 1). Aspiration was released, and the sucker was removed. After final measurements, the chest was closed. All animals received human care in compliance with the *Principles of Laboratory Animal Care* formulated by the National Society for Medical Research and *Guide for Care and Use of Laboratory Animals* prepared by the Institute of Laboratory Animal Resources, National Research Council, and published by the National Academy Press, revised 1996. The protocol was approved by the Federal Committee for Laboratory Animal Care, Bezirksregierung Münster, Ref G 24/2001, on April 25, 2001.

Measurements. Two fluid-filled catheters were introduced into the pulmonary trunk and the left ventricle, respectively, to measure pressures. Myocardial mesh tension was measured with a needle probe⁸ implanted into the superior free wall of the left ventricle. Four microsonometer crystals were implanted to measure ventricular inner and outer diameters, one pair in the superior equatorial wall and the other in the inferior equatorial wall. From diameter data (D), stroke volume (V) was estimated according to a simple cubic formula ($V = D^3$). Measurements were done before and during aspiration of the mural segment and were repeated after definitive sequestration.

Five animals were killed 2 weeks after the intervention. In 3 animals, the hemodynamic measurements were repeated under deep anesthesia. In the other 2, pericardial concretion hampered attempts at measurement. All hearts were removed and weighed. Postmortem macroscopic morphologic evaluation was focused on the extent of the trauma produced. The other 3 animals were killed 1 week after the intervention. From these hearts, we made casts of the right and left ventricular cavities with and without the sutured splints in place.

Statistics. All values were expressed as mean $\pm$ SD. For comparison of paired data, we used the Wilcoxon signed rank test.

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Figure 1. Left ventricular plication. Aspirator cup (*top*), slightly waisted in middle area where foot points of papillary muscles are expected to be located. Interpapillary area of left ventricular free wall with marginal artery (*middle*). Segment has been aspirated and hence is plicated. Two longitudinal splints had been passed through myocardium below aspirator cup from apex to base, and two cross sutures are approaching two splints to one another. Cross section through plicated interpapillary segment (*ASP*) with two papillary muscles (*P*), left ventricular interpapillary lumen (*L*), subepicardial coronary vessels (*X*), and two channels in which splints (*S*) were located (*bottom*). Two arrows indicate directions of compression of tissue interposed between splinted suture. *Labeled area* in subendocardium indicates tissue compartment expected to be distorted and compressed. Interpapillary segment of ventricular lumen is narrowed. Specimen was produced by postmortem aspiration for 24 hours while tissue was fixed in formaldehyde.

Results

Alterations induced by aspiration of a segment proved transient. On release of suction, the aspirated zone was demarcated by slight edematous thickening. After 10 minutes, the epicardial surface looked normal, except for some fine traces of subepicardial bleeding. Placement of the sutured splints caused some extrasystoles in the first 2 animals studied, and life-threatening arrhythmias in the

Figure 2. Cardiac output as calculated from left ventricular dimension multiplied by heart rate. Asterisk indicates $P < .05$ versus control.

Figure 3. Left ventricular equatorial outer and inner diameters during systole and diastole measured before (*control*) and during aspiration, and after securing two transverse sutures (*sequestered*). Asterisk indicates $P < .05$ versus control.

third, but the induced ventricular fibrillation was successfully treated by electrotherapy. In the last 5 animals, after administration of lidocaine on the surface of the heart, no further life-threatening arrhythmias were observed.

Cardiac output, calculated from left ventricular diameter, decreased 34% during aspiration (Figure 2). Left ventricular outer

Figure 4. Heart rate measured before (*control*) and during aspiration, and after completion of sequestration by splinting.

Figure 5. Total peripheral resistance measured before (*control*) and during aspiration, and after completion of sequestration by splinting. Asterisk indicates $P < .05$ versus control; double asterisk indicates $P < .01$ versus control.

TABLE 1. Hemodynamics (n = 8)

	Control	Aspiration	Sequestered
Left ventricular end-diastolic pressure (mm Hg)	7.42 ± 2.54	16.24 ± 6.94*	10.86 ± 5.13*
Left ventricular peak pressure (mm Hg)	112.69 ± 14.75	108.88 ± 10.81†	108.09 ± 8.02
−dp/dt (mm Hg/s)	−1274.76 ± 277.07	−1126.11 ± 272.71*	−1263.94 ± 257.00
+dp/dt (mm Hg/s)	1496.75 ± 281.9	1186.92 ± 166.1†	1339.87 ± 173.4
Pulmonary arterial pressure (mm Hg)			
Diastole	10.11 ± 3.8	16.01 ± 6.5*	13.60 ± 5.2†
Systole	25.46 ± 6.2	29.87 ± 8.1†	26.75 ± 5.8

Values are mean ± SD.

* $P < .05$ versus control.

† $P < .01$ versus control.

diameter decreased by 12% during systole and by 13% during diastole. The inner diameter decreased by 13% during both systole and diastole (Figure 3). Left ventricular peak pressure dropped by 8% (Table 1), whereas end-diastolic pressure increased from 7.42 ± 2.54 mm Hg to 15.86 ± 5.13 mm Hg. The $-dp/dt_{\max}$ decreased by 12%, and the $+dp/dt_{\max}$ decreased by 21% (Table 1). Pulmonary arterial systolic pressure increased from 10 to 15 mm Hg during diastole and from 25 to 30 mm Hg during systole (Table 1). Heart rate did not change significantly (Figure 4). Total peripheral resistance increased by 73% (Figure 5). Developed myocardial force, expressed as the systolic minus the diastolic forces, decreased by 41% (Figure 6). The $+dF/dt_{\max}$ dropped by 47%, and the $-dF/dt_{\max}$ dropped by 25% (Figure 7).

All measurements were repeated 10 ± 2 minutes after permanent sequestration had been achieved by application of the transverse sutures to ensure that the step from plication by aspiration to

final plication by splinting was not associated with conformational changes of the left ventricle. We found that left ventricular diameters had increased insignificantly (Figure 3), whereas heart rate had increased (Figure 4) and total peripheral resistance had decreased (Figure 5), both insignificantly. Calculated cardiac output remained unchanged (Figure 2). Left ventricular systolic pressure had risen, whereas diastolic pressure had fallen, as had pulmonary arterial pressures during both systole and diastole (Table 1). The $-dp/dt_{\max}$ had almost reached the control level, whereas positive $+dp/dt_{\max}$ had risen significantly (Table 1). Developed measured forces had risen significantly (Figure 6), as had $+dF/dt_{\max}$ and $-dF/dt_{\max}$ (Figure 7).

During the first 8 to 10 postoperative days, the animals showed a constrained tolerance to physical stress and an enhanced need for sleep. During the subsequent 4 to 6 days, their feeding behavior and movement within their individual pigsty improved. They re-

Figure 6. Developed force, systolic minus diastolic force, in the myocardial mesh measured before (control) and during aspiration and after completion of the sequestration by splinting.

mained constrained, nonetheless, until the final experiment. Our examinations of the splinting secured by sutures, studied in 3 animals 1 week after the intervention, revealed the interpapillary segment still to be properly constricted. Left ventricular casts taken with and without the splinted sutures in place differed in diameter by $12\% \pm 6\%$.

During the final experiment, 2 weeks after intervention, cardiac output at rest had returned in the mean to the preoperative control state at 10 L/min. Left ventricular pressure had normalized both in systole, at 103 mm Hg, and in diastole, at 5 mm Hg. Heart rate in the mean remained slightly elevated, at 112 beats/min. Pulmonary arterial pressures remained elevated at 32 mm Hg during systole and 21 mm Hg during diastole. Left ventricular outer diameter in diastole had returned to the control state, namely 59 mm, as had the inner diameter in diastole, measured at 46 mm.

Structural alterations. In no case at postmortem investigation did we observe the papillary muscles, or the tendinous cords of the mitral valve, to be damaged or injured by the sutures. Damage was restricted to the central part of the wall, with the coronary arteries being unaltered. Gravimetrically, the mass of sequestered tissue in the mean amounted to $28\% \pm 8.4\%$ of the mass of the left ventricular wall, with the septum being included within these calculations. The two opposing parts of the plicated segment had not formed a solid scar because the two transverse sutures, though supported on both sides by felt, had intruded into the wall.

Discussion

Although once considered a promising procedure, partial left ventriculectomy has fallen from grace because of undue mortality, often blamed on overcorrection of left ventricular diameter.⁴ An-

Figure 7. Maximal velocity of development ($+dF/dt_{max}$) and decay of force ($-dF/dt_{max}$) measured before (control) and during aspiration, and after completion of sequestration by splinting. Asterisk indicates $P < .05$ versus control.

other surgical complication of the procedure has been the concomitant resection of segments of the coronary vascular bed.² The extent of the wound, coupled with high intracavitary pressure, has sometimes resulted in rupture during the early postoperative period.⁷ The most intriguing drawback of the method to date, nonetheless, has been the inability, before definitive surgery, to assess the postoperative effects of the ventriculectomy.

If the procedure is to be placed on a sound methodologic footing, the surgical intervention must be less invasive, the coronary vascular bed must be preserved, and ideally the subsequent result in terms of global ventricular pump-function must be quantified before the definitive sequestration of any segment of the ventricular wall. If it is reproducible in clinical practice, our proposed technique will enable the surgeon to tailor the exact size, shape, and location of the segment to be sequestered without opening the left ventricle, while preserving intact the coronary vasculature.

In the experimental setting, we have shown that the normal left ventricle is able to cope with an important reduction in its radius, although it thereafter exhibits restrictions in its compliance and pump function, resulting in an increase in pulmonary arterial pressure. The improvement in ventricular function that we identified from the early state of aspiration to the final state of sequestration in our opinion heralds the recovery of contractility, which during the early surgical preparation had been suppressed by narcotics and antiarrhythmic medications.

By the time of our final experiments, the left ventricles had returned to control size. This response to the nonphysiologic constriction imposed by our experiments should not be considered analogous to the ventricular redilation sometimes observed after reduction of ventricular radius in previously dilated hearts. As a feature of our experimental conditions, reduction of radius was

carried out in left ventricles of normal size. That the hearts remodeled to their initial state is but a process of physiologic adaptation necessary to readapt pump function to peripheral requirements.

It has been suggested that partial left ventriculectomy interrupts the continuity of an alleged apical spiraling muscle that is part of a "unique myocardial band."⁹ It is argued that on these grounds the approach is incompatible with an improvement in global ventricular function.⁹ If true, this argument would also apply to our experimental model. In fact, the heart is a modified blood vessel, rather than being structured on the basis of skeletal musculature.¹⁰⁻¹² If there were such a unique muscle band, it would be contained within a discrete fascial sheath comparable to the structures seen isolating the skeletal muscles of the limbs and trunk. There is no such fascial compartmentation to be found within the heart.¹⁰⁻¹² The encouraging clinical results obtained by some teams with partial left ventriculectomy,⁶⁻⁸ coupled with our current experimental evidence, argue strongly against structural analysis based on the "unique band."⁹

Our use of longitudinal splints running intramurally should not be confused with the myosplint procedure suggested earlier by McCarthy and colleagues.¹³ Their technique makes use of two horizontal transluminal splints, which reduce left ventricular radius by constricting its anteroposterior extent.

Suckers of variable size have already been used in cardiac surgery to stabilize circumscribed areas during coronary surgery, or as a means of assessing the mechanical properties of the myocardium.¹⁴ Avoiding the complex trauma associated with the use of assisted circulation with or without cardioplegia,¹⁵ aspiration of the left ventricular wall under lidocaine protection is a harmless, fully reversible procedure that does not place sustained stress on the aspirated segment.¹⁶ The area to be plicated can be increased or reduced, whereas its topography and shape can be repeatedly varied until optimal ventricular function has been achieved.

The technique has its limitations. Intervention on the mitral valve, if also needed, would require separate access through the atrium. The technique of transverse suturing for coapting the longitudinal splints is less than ideal, and can be replaced by using clips to approximate the splints at both their protruding ends. We recognize also that cardiac output calculated from sonometric data gives at best a rough estimate of the true situation. Finally, should our technique be attempted clinically, we would advise performing the intervention in a setting in which mechanical assistance is available to offer a safe fallback.

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