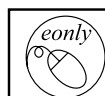# Evolving Technology

Ventricular constraint in dilated cardiomyopathy: A new, compliant textile mesh exerts prophylactic and therapeutic properties

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Background: Dilated cardiomyopathy is associated with a progressive decrease in cardiac function, leading to end-stage heart failure. We aimed to stop this process by mechanically constraining the heart with a new, compliant textile mesh.

Methods: In 16 male Munich minipigs (50 ± 7 kg), dilated cardiomyopathy with congestive heart failure was induced through 4 weeks of rapid ventricular pacing (220 beats/min). In the early-mesh group (n = 8), a polyvinylidene fluoride mesh was positioned around both ventricles before pacing was started. In the other group (n = 8), experimental dilated cardiomyopathy through rapid pacing was induced (no mesh). After mesh grafting, rapid pacing was continued (late mesh).

Results: Rapid pacing in the no-mesh group (control group) significantly decreased both systolic (cardiac output, peak systolic pressure, and the derivative of pressure increase [dP/dt_{max}]) and diastolic (minimum rate of pressure rise [dP/dt_{min}] and left ventricular end-diastolic pressure) variables, whereas these variables remained almost unchanged in the early-mesh group. In the late-mesh group the passive-elastic constraint not only prevented further deterioration but even exerted reverse remodeling to some extent (dP/dt_{max} and left ventricular end-diastolic pressure, $P < .05$).

Conclusions: Ventricular constraint with the new mesh seems to be a prophylactic and therapeutic option in cardiac insufficiency caused by ventricular dilation. This passive-elastic cardioplasty induced reverse remodeling of dilated hearts and significantly improved diastolic and systolic ventricular function.

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C ardiac failure is one of the most common diseases in the Western world. The number of patients with myocardial insufficiency has tripled in the past 20 years and is estimated to amount to 1%.[1]

This increase is caused by improved conservative therapeutic options, as well as increasing numbers of elderly persons. Both heart transplantation and left ventricular (LV) assist devices are promising therapeutic options. However, a variety of complications and contraindications exist.[2] Except for problems with biologic compatibility and immense costs, long waiting lists underline the problem with suited heart donors. On the other hand, LV assist devices present a large technical effort. Hence one could conclude that these therapeutic tools will not be able to supply the growing number of patients adequately under economic, numeric, and medical aspects.

ET

Among alternative attempts for long-term cardiac support, the latissimus dorsi muscle was wrapped around the insufficient cardiac muscle as a cardiomyoplasty.[3,4] This procedure has already been applied in more than 1000 patients, yet it has not become a clinical standard. Although the question of whether dynamic or adynamic cardiomyoplasty would be superior remained unanswered,[5,6] further ventricular dilation could be avoided because of the "girdling" effect.[5] Postoperatively, the patient status was improved by one to two New York Heart Association classes.[7]

The girdling effect seems to limit the diastolic volume and thus improves systolic function. In consequence, application of a passive external device could represent a new direction in the therapy of myocardial insufficiency.[5]

Not surprisingly, static support devices have been developed.[8,9] New synthetic fibers were designed and other techniques to manufacture better-suited mesh grafts were developed to further improve such cardiac binding.

Preclinical studies have shown that constraining devices can provide end-diastolic support by (1) reducing mechanical wall stress, (2) improving function, and (3) reversing cardiac remodeling.[10,11] Thus far, those devices have been implanted worldwide in more than 130 patients with dilated cardiomyopathy (DCM) with or without concomitant cardiac surgery, improving the patient's functional status.[11] Therefore, passive ventricular constraints are suggested as a very promising tool for the treatment of heart failure and DCM.[12]

This study investigated the applicability of a new, elastic, textile mesh made of polyvinylidene fluoride fibers[13] as a cardiac constraint. We determined in pigs whether a passive-elastic cardioplasty improves ventricular function after established heart failure and whether it prevents the development of tachycardia-induced ventricular dilatation.

Experiments were performed on a total of 21 pigs. In a prophylactic group, mesh grafting was performed before the onset of 4 weeks of rapid pacing. In a therapeutic group, heart failure was induced through 4 weeks of rapid pacing without the mesh, such that this group served as controls. Then the mesh was implanted, and rapid pacing was continued for another 4 weeks. The hemodynamic effects of rapid pacing and of the mesh were monitored throughout the individual 4-week protocols.

Materials and Methods

The study was performed on anesthetized male Munich minipigs (49.5 ± 7.0 kg of body weight). The animals were randomly assigned to one of two groups. Of a total of 21 pigs, 5 died because of ventricular fibrillation, pneumothorax, or massive bleeding. The study was performed according to the "Guidelines for the Care and Use of Laboratory Animals" (National Institutes of Health, 1985), and the study was approved by the bioethical committee of the district of Duesseldorf, Germany.

Mesh

Thus far, a polyethylene terephthalate was fabricated into a multifilament compliant mesh knit (CorCap; Acorn, St Paul, Minn) for permanent implantation. In the present study, a mesh was chosen that was made of polyvinylidene fluoride (PVDF) fibers (FEG Co, Aachen, Germany). PVDF as a polymer has been commonly used in the clinic for more than 10 years.[13] PVDF sutures provided greater breaking strengths in tendons than in those repaired with polypropylene sutures,[14] seemed to be more biostable than polypropylene sutures in the long term,[13,15] demonstrated minimal tissue response in vivo, and can be sterilized with β- or γ-radiation and therefore can reduce dependence on ethylene oxides and chlorofluorohydrocarbons.[16] On the other hand, histologic analysis of explants found no inflammatory cells in the tissue around either PVDF or polypropylene sutures.[16]

PVDF mesh is already in use for abdominal hernia repair, where it proved to be advantageous because of improved biostability, lowered bending stiffness, and minimum tissue response,[17] and as scaffolds for tissue regeneration in the esophagus.[18]

The CorCap mesh is described to be compliant. The PVDF mesh used in this study is compliant as well, but because of its weaving technique, it also had elastic properties, making it a passive-elastic epicardial constraint. Yet we cannot provide actual mechanical data.

Anesthesia

Premedication consisted of ketamine (0.2 mg/kg administered intramuscularly), azaperone (0.08 mg/kg administered intramuscularly), and atropine (0.02 mg/kg administered intramuscularly). After 15 minutes, thiopental (3 mg/kg administered intravenously), pancuronium bromide (0.1 mg/kg), and fentanyl dihydrogencitrate (5 μg/kg) were administered. Anesthesia was maintained with enflurane, and N_2O_2 was established.

General Surgery

A left lateral thoracotomy was performed in the early-mesh and no-mesh hearts of the late-mesh group. Lidocaine (2%; 5 mL) was infused to prevent arrhythmia. After resection of the thymus gland, the pericardium was opened to expose the anterior wall of the heart. Heparin (1000-2000 IE) was given according to the body weight. Through the LV anterior wall, a Konigsberg catheter was implanted to assess the intraventricular pressure (LV pressure; P4.0X6-S-CK; Konigsberg Instruments, Pasadena, Calif). Then a bipolar pacing electrode (KY 66 II; Osypka Co, Rheinfelden, Germany) was placed into the right ventricle and was secured to the right ventricular wall. An electrocardiographic electrode located on the LV pressure catheter was fixed at the left atrium. For measuring cardiac output (CO), a flow probe was placed around the pulmonary artery (Triton ART2 Flow Probe, TRI-200-307-K/L-Y50CM-CSTY-KB; Konigsberg Instruments). All connecting wires were placed subcutaneously through the back of the animal, and the pacemaker electrode was connected with the 1-cavity pacemaker (Pace 101H, Osypka Co).

In contrast to the early-mesh group, the mesh in the late-mesh group was implanted after sternotomy at the time of reoperation. The graft was positioned around both ventricles and secured with sutures on the atrioventricular level, leaving the apex uncovered (Figure 1). The anterior part of the mesh was closed with a running
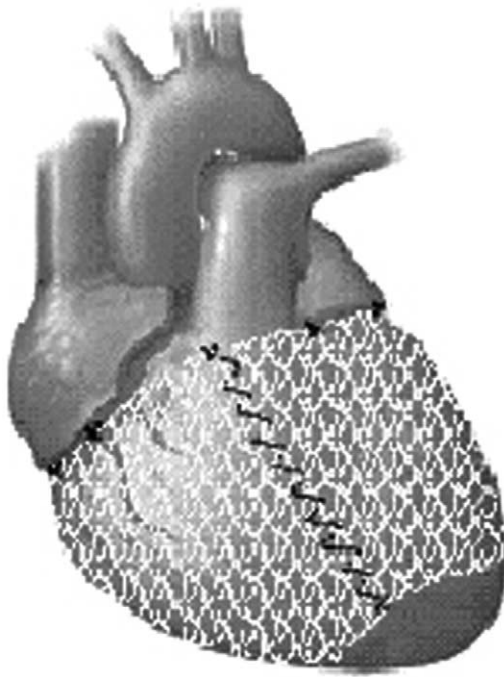
Figure 1. Schematic of the mesh after implantation. Neither the apex nor the auricles were covered by the mesh. The basal portion of the mesh was secured on the atrioventricular level.

suture. Particular attention was paid to the mesh tightness, which was chosen not only to increase the end-diastolic pressure in the early-mesh group but to also reduce the end-diastolic pressure by a maximum of 5 mm Hg without affecting the peak systolic pressure in the late-mesh group. In all animals, the pericardium was resected, and the thorax was closed in accordance with local

custom. Antibiotic prophylaxis was given to all animals with lincomycin (2 · 20 mg/kg administered intravenously).

Experimental Protocol
The animals were randomly assigned to either the early-mesh or the late-mesh group.

Early-mesh group (n = 8). For induction of DCM, 24-day rapid pacing was started after an average of 4.1 days of recovery after surgical intervention (Figure 2). This prophylactic protocol was performed to demonstrate the effects of a textile mesh (ie, of a passive-elastic epicardial constraint) on the development of DCM.

Late-mesh group (n = 8). In the first part of the protocol, rapid pacing of hearts without mesh was started 5.0 days after surgical intervention (no-mesh group). DCM was induced through rapid pacing and was considered established when CO and the derivative of pressure increase (dP/dt_{max}) had decreased to 60% of baseline values (on average, 22.1 days). Hence these no-mesh animals served as control animals for the early-mesh group. After median sternotomy, the mesh was implanted as above (late-mesh group). A 21.5-day rapid pacing period was initiated after 3.7 days of recovery after reoperation (Figure 3). This therapeutic protocol was thought to demonstrate the effects of further rapid pacing on a heart with established DCM that was supported with a passive epicardial constraint.

In both parts of the protocol, rapid pacing was started at 140 beats/min. After 2 to 3 days, the pacing rate was increased to a maximum of 220 beats/min. After completion of either protocol, the mesh and the myocardium were examined both macroscopically and histologically.

Data Acquisition and Statistics
The following variables were assessed: CO and LV pressure. From the LV pressure signal, peak systolic pressure, end-diastolic pressure, maximum pressure increase and decrease, and heart rate (HR) were derived.
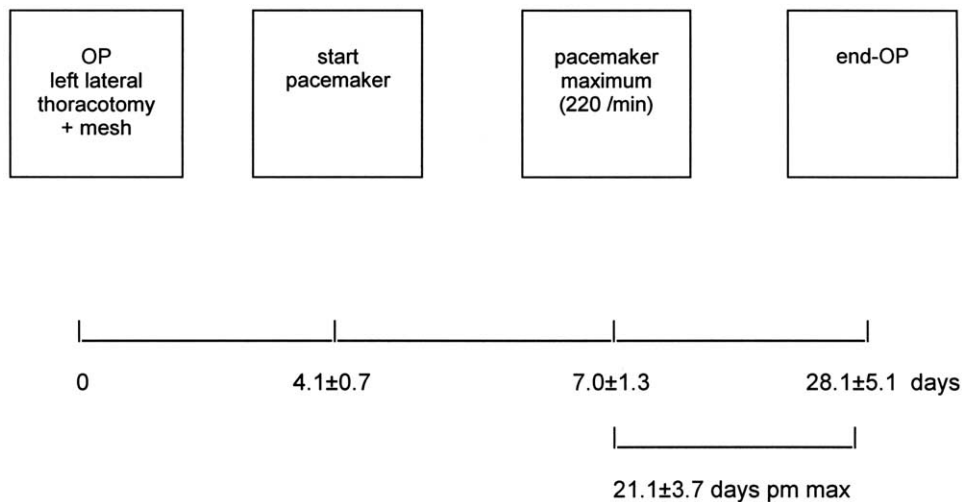

Figure 2. Experimental protocol. In these animals mesh grafting was performed already during the first operation (early-mesh group). Rapid pacing was initiated after hemodynamic stabilization. *OP,* Operation; *pm,* pacemaker.
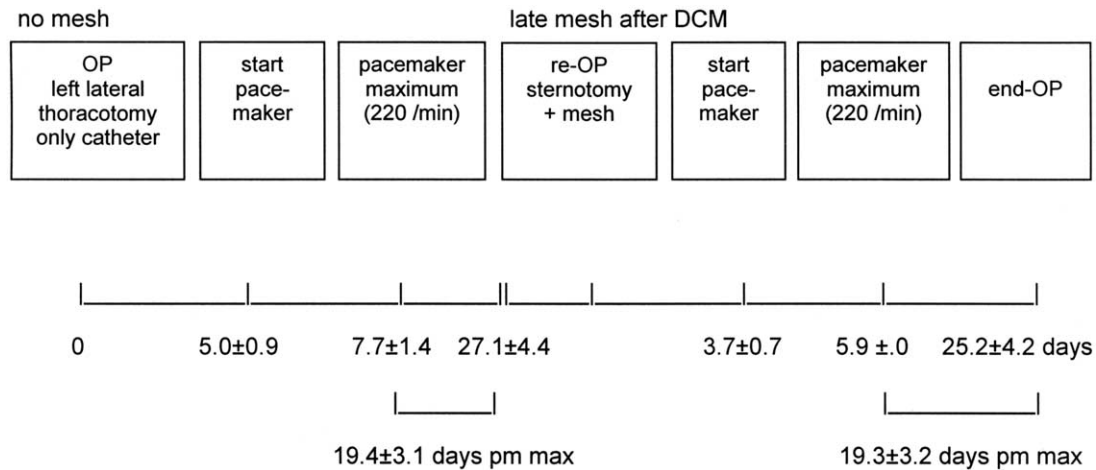
Figure 3. Experimental protocol. The hearts of these animals were paced after the first operation until heart failure was established (no-mesh group). The mesh was positioned during a reoperation (late-mesh group). Thereafter, rapid pacing was continued. *OP*, **Operation;** *pm*, **pacemaker.**

These variables were stored at 7 points in time: intraoperatively, at the start of pacemaker use, at maximum pacing rate, after 25% of maximum stimulation, after 50% of maximum stimulation (T50%), after 75% of maximum stimulation (T75%), and at the day of end operation.

Statistical analysis was performed with a 2-way analysis of variance (SPSS 11.5 for Windows; SPSS Inc, Chicago, Ill). If significant differences in mean values were detected, individual mean values were compared by Fisher least-significant-difference post-hoc tests.

Results

Comparison of the Early-Mesh Group With the No-Mesh Group

Systolic variables, such as CO, LV peak pressure (LVP_{max}), and the derivative of pressure increase (dP/dt_{max}) were significantly higher in the no-mesh group compared with in the early-mesh group (Table 1). HR and diastolic variables, such as early (dP/dt_{min}) and late (LVP_{ed}) relaxation, were not significantly different.

Because of the given pacing rate, HR did not differ between groups during the entire protocol. Systolic measures (CO, LVP_{max}, and dP/dt_{max}) in the early-mesh hearts only slightly decreased or were maintained during the protocol, whereas the no-mesh hearts deteriorated more drastically ($P < .05$): CO by only -1.4 ± 0.2 L/min versus -4.1 ± 0.3 L/min (mean $\pm$ standard error of the mean; Figure E1, *A*); LVP_{max} by $+9 \pm 1$ mm Hg versus -39 ± 3 mm Hg (Figure E1, *B*); and dP/dt_{max} by $+288 \pm 30$ mm Hg/s versus -1350 ± 122 mm Hg/s (Figure E1, *C*).

Early diastolic relaxation deteriorated significantly in the groups with and without support over time: in the early-mesh group from 1976 ± 155 mm Hg/s to 1488 ± 144 mm Hg/s and in the no-mesh group from 1778 ± 202 to 1026 ± 110 mm Hg/s. At the end of the protocol, the differences between the groups were significant (Figure E2, *A*).

The LV end-diastolic pressures at the onset of the protocol were not significantly different. Despite the increased LV end-diastolic pressures in the supported hearts compared with the no-mesh hearts (11 ± 3 vs 5 ± 2 mm Hg), LV end-diastolic pressure remained unchanged during pacing (11 ± 3 vs 10 ± 2 mm Hg) but significantly increased in the no-mesh hearts (5 ± 2 vs 24 ± 2 mm Hg), also reflecting ventricular insufficiency (Figures E2, *A*, and E3, *A*).

It is remembered that rapid pacing in the no-mesh group (control group) lasted only about 22 days, whereas it lasted about 24 days in the early-mesh group. Thus, the deterioration in the control animals will very likely be underestimated.

TABLE 1. Baseline values of the early-mesh group and the no-mesh group

	Early-mesh group	No-mesh group	*P* value
Heart rate (1/min)	91 ± 5	103 ± 8	NS
Cardiac output (L/min)	6.4 ± 0.4	7.8 ± 0.6	.012
LVP_{max} (mm Hg)	88 ± 6	110 ± 7	.048
dP/dt_{max} (mm Hg/s)	1763 ± 189	2625 ± 149	.005
dP/dt_{min} (mm Hg/s)	1976 ± 155	1778 ± 202	NS
LVP_{ed} (mm Hg)	11 ± 3	5 ± 2	NS

The systolic variables of the hearts with mesh were significantly smaller compared with those of the no-mesh hearts, whereas the diastolic measures exhibited no significant differences. *NS*, Not significant; LVP_{max}, left ventricular peak pressure; dP/dt_{max}, derivative of pressure increase; dP/dt_{min}, derivative of pressure decrease; LVP_{ed}, left ventricular end-diastolic pressure.

Comparison of the No-Mesh Group With the Late-Mesh Group

Spontaneous HR did not significantly differ between the first operation and reoperation (Table 2). CO at reoperation was reduced by 11% compared with baseline CO at the first operation, whereas LVP$_{max}$ was moderately increased by 6%. However, the contractile state and both diastolic measures significantly demonstrated cardiac insufficiency: dP/dt$_{max}$ was decreased by 28%, dP/dt$_{min}$ was decreased by 29%, and LV end-diastolic pressure was increased by 183%.

Macroscopic intraoperative findings showed severe dilation and massive loss of contractile function in all 8 hearts.

Because the same pacing rate was chosen in both protocols, HR did not differ between groups. CO at the end of the protocol in the no-mesh hearts was decreased by −4.1 L/min, and that in the late-mesh hearts was decreased by only 0.9 L/min (Figure E4, A). Likewise, both other systolic measures (LVP$_{max}$ and dP/dt$_{max}$) in the no-mesh hearts decreased significantly during the protocol (30% and 50%; Figure E4, B and C). After reoperation and mesh grafting, LVP$_{max}$ was maintained (−3%), and dP/dt$_{max}$ even increased (+36%) in these hearts.

During rapid pacing, early diastolic relaxation was decreased in the no-mesh hearts (−42%, $P < .05$), whereas it increased in the late-mesh hearts (+20%; Figure E3, A). Similarly, LV end-diastolic pressure had increased during pacing by 380% ($P < .05$) in the no-mesh hearts, and this measure of late relaxation was decreased in the late-mesh hearts by 50% ($P < .05$; Figure E3, B).

Histology

In accordance with the literature, only minimal tissue response and no inflammatory cells in the tissue around the mesh were found. In addition, adhesion to the surrounding tissue was moderate, such that the mesh could be removed from the epicardium.

Discussion

Myocardial insufficiency is frequently the consequence of ventricular dilation. Therapeutic options are aimed to stop or even reverse this process.[19] In the past, the ventricular size was surgically reduced. However, recurrence of ventricular dilation and myocardial failure has led to the abandonment of this option.[20,21]

Among other techniques, pacemaker-stimulated skeletal muscle was used to wrap the ventricle.[22,23] Use of the latissimus dorsi muscle as a dynamic cardiomyoplasty improved the clinical status, which correlated with an obvious reverse remodeling of the ventricular myocardium.[5,24,25] To this day, it is not clear whether this improvement was solely due to preventing further ventricular dilation (ie, the girdling effect).[5]

TABLE 2. Baseline values of the no-mesh group and the late-mesh group

	No-mesh group	Late-mesh group	P value
Heart rate (1/min)	103 ± 8	106 ± 7	NS
Cardiac output (L/min)	7.8 ± 0.6	7.0 ± 0.4	NS
LVP$_{max}$ (mm Hg)	110 ± 7	117 ± 9	NS
dP/dt$_{max}$ (mm Hg/s)	2625 ± 149	1886 ± 180	.011
dP/dt$_{min}$ (mm Hg/s)	1778 ± 202	1260 ± 281	.038
LVP$_{ed}$ (mm Hg)	5 ± 2	15 ± 2	.002

The contractile state (dP/dt$_{max}$) and both early (dP/dt$_{min}$) and late (LVP$_{ed}$) relaxation exhibited the deleterious consequences of almost 4 weeks of rapid pacing. *NS,* Not significant; *LVP$_{max}$,* left ventricular peak pressure; *dP/dt$_{max}$,* derivative of pressure increase; *dP/dt$_{min}$,* derivative of pressure decrease; *LVP$_{ed}$,* left ventricular end-diastolic pressure.

If so, cardiac function could obviously also be improved by using a prosthetic membrane (ie, a static cardioplasty).[8,9] In those studies on dogs, the ventricular size in supported hearts was markedly reduced compared with that in unsupported hearts. As a functional consequence, ejection fraction in the supported animals remained better preserved after 4 weeks of rapid pacing (approximately 30%) than that seen in control animals (approximately 18%).[9] In the latter study, an adynamic cardiomyoplasty (ie, unstimulated skeletal muscle) also reduced cardiac enlargement and functional deterioration after rapid pacing. This treatment appeared to be more effective, perhaps because of the adaptive capabilities of the skeletal muscle wrap compared with the static myoplasty. However, cardiac binding is a less difficult and less invasive procedure, which might be useful as an adjunct to prevent or delay progressive ventricular dilatation in heart failure.

In addition, the efficacy of a passive constraint to reverse chronic chamber remodeling and adrenergic downregulation was tested in the failing canine heart, in which ischemic DCM was induced by repeated microembolizations. Reverse remodeling with reduced systolic wall stress and improved adrenergic signaling was achieved by passive external support. Hence this approach might prove useful in the treatment of chronic heart failure.[26]

Similarly, the midterm results of patients with symptomatic heart failure suggest that ventricular constraint with static cardioplasty might be useful for preventing further cardiac dilation.[27] Not only was ventricular remodeling prevented in these patients, but also a reverse remodeling was observed. The same authors later used a basal ventricular constraint to also treat mitral regurgitation without entering the heart.[28]

Because other clinical studies were unable to demonstrate improved systolic function after dynamic cardiomyoplasty,[5,29] textile meshes were developed with not only static but also dynamic properties. Konertz and coworkers[30]

were the first to use a polyethylene terephthallate mesh that was, because of its higher distensibility, in contrast to the almost rigid meshes used so far.[8,9] This cardiac support device helped improve diastolic ventricular function but not systolic function.

The results could possibly be even more promising if wrapping of the support devices could be better standardized. In one study a composite membrane was wrapped around both ventricles plus the atria plus the venae cavae.[31] In contrast, we wrapped the textile mesh only around both ventricles, leaving the apex free. Although the tightness of the mesh was adjusted to its effects on LV end-diastolic pressure, the end-diastolic volume would have been more appropriate for standardizing the wrapping procedure. In consequence, we have used the conductance catheter and assessed pressure-volume loops in the meantime.[32]

In the first part of our study, we investigated the prophylactic effects of a passive-elastic cardioplasty provided by an elastic mesh made of PVDF. This polymer is an established biomaterial that was only recently suggested for construction of surgical meshes.[17]

In fact, this new cardioplasty helped significantly reduce systolic deterioration compared with that seen in unsupported hearts. These beneficial effects might in part be due to the mesh's improved elastic properties that were already described to be important.[8,27,33] Furthermore, adhesions between the mesh and the epicardial layer will affect such elastic mesh properties.[19] In our two experimental groups, we found only moderate adhesion, which we ascribe to the properties of the PVDF mesh that reportedly exerts only minor tissue reactions.[13,17]

The preservation of diastolic function in the supported hearts in this study is in concert with that seen in former studies on the beneficial prophylactic effects of passive epicardial constraint on diastolic function.[30]

In the second part of our study, the influence of the passive-elastic constraint on preexisting cardiac failure was investigated. Cardiac failure was achieved by rapid ventricular pacing hearts without the mesh. Tachycardia-induced heart failure is the most commonly used experimental heart failure model.[34] Ventricular pacing at 220 to 240 beats/min results in profound low-output, biventricular, edematous failure in 2 to 3 weeks.[35] To avoid myocardial recovery after this procedure, we paced the hearts even longer in the present study, namely for about 4 weeks. At the time of reoperation, severe myocardial dilation with pleura effusion and decreased systolic and diastolic ventricular function was found in all animals of the late-mesh group. Independent of our hypothesis, irrespective of whether cardiac failure after rapid pacing is reversible, LV dysfunction was already clearly reduced after mesh grafting. Because the mesh graft also decreased the end-diastolic pressure, we suggest that the passive-elastic cardioplasty reduced the end-diastolic volume and in so doing improved systolic function in terms of CO, LV peak pressure, and dP/dt_{max}.

In addition, systolic and diastolic function during restarted rapid pacing was maintained or even better compared with that seen in unsupported hearts. These findings are in contrast with those of other studies. In dogs only the diastolic, but not the systolic, function was improved.[9] In turn, solely dynamic cardiomyoplasty was postulated to improve both systolic and diastolic function, presenting a promising new therapy for DCM.[36]

In summary, the textile cardioplasty used in this study successfully prevented deterioration of both systolic and diastolic myocardial function in tachycardia-induced heart failure (ie, it exerted a prophylactic potency). Furthermore, our passive-elastic mesh improved the consequences of rapid pacing on hearts with experimentally induced DCM. Thus, the mesh graft also exerted a therapeutic potency. The beneficial effects of the textile cardioplasty are attributed to the passive-elastic properties of the mesh and to its reportedly minor tissue reactions.

If larger clinical studies further provide positive results and if surgical intervention for positioning the mesh could become less invasive, the passive-elastic cardioplasty might become a promising alternative in the therapy of DCM, in particular because of its surgical and economic advantages.

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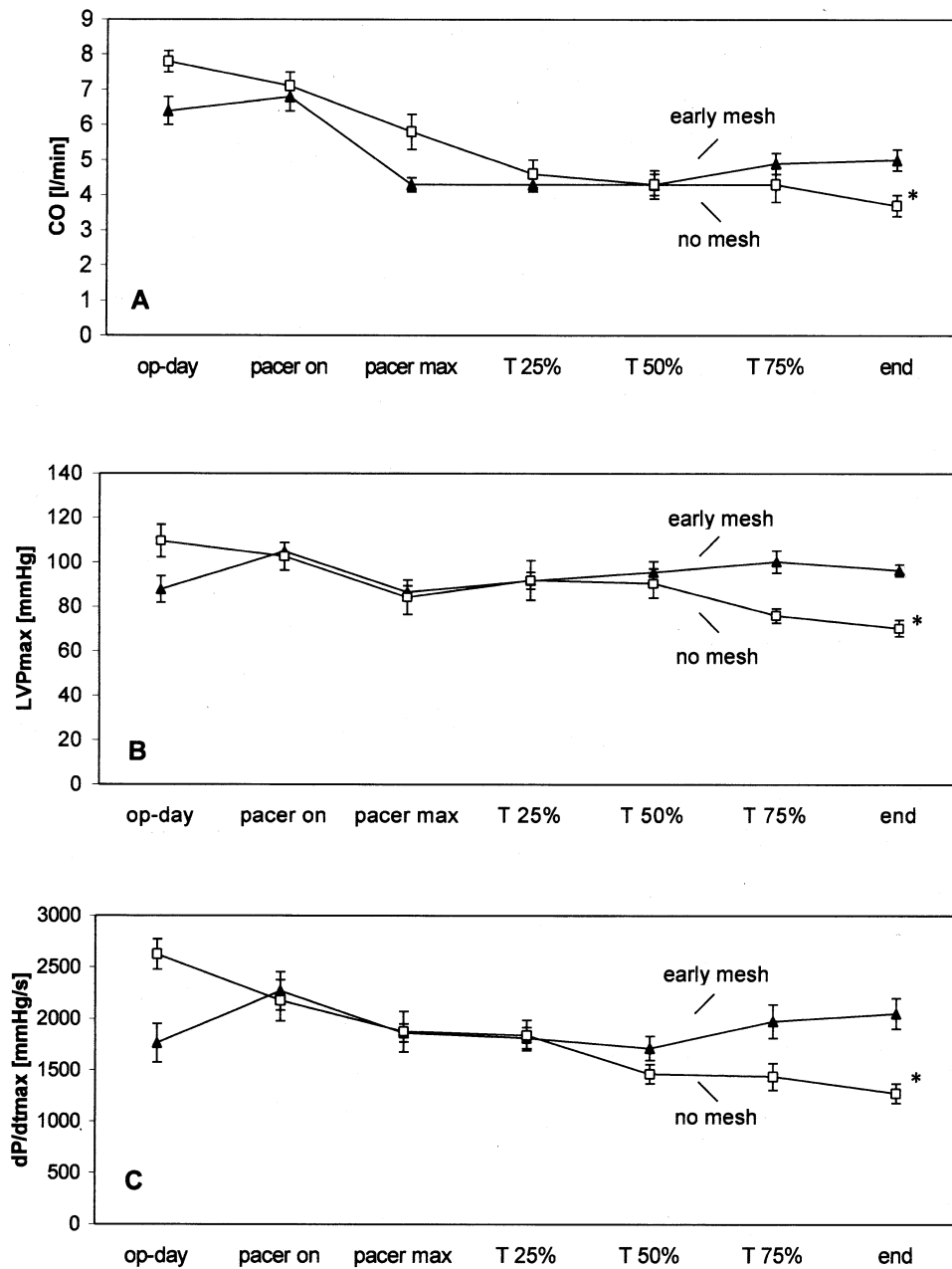
Figure E1. Systolic variables of no-mesh hearts and early-mesh hearts. Although the no-mesh hearts started with slightly reduced baseline values, the mesh reduced deterioration of systolic function during pacing. A, Cardiac output *(CO)*; B, peak left ventricular pressure *(LVP$_{max}$)*; C, maximal left ventricular pressure rise *(dP/dt$_{max}$)*. *P < .05 vs baseline; *T 25%, T 50%, T 75%*, 25%, 50%, and 75% duration of rapid pacing.
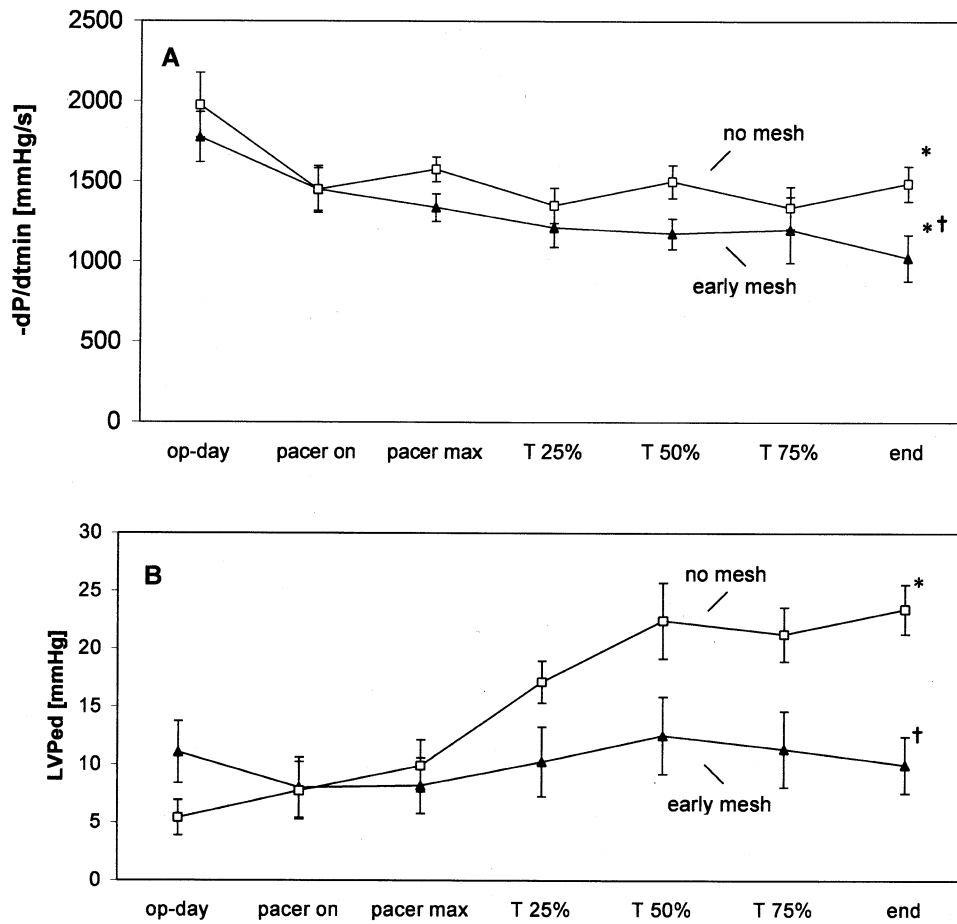
Figure E2. Diastolic variables of no-mesh hearts and early-mesh hearts. A, dP/dt$_{min}$ in the no-mesh hearts and the early-mesh hearts deteriorated significantly over time. However, the decrease in the early-mesh hearts was relatively smaller, such that the difference between the two groups became significant at the end of the protocols. B, The end-diastolic pressure *(LVP$_{ed}$)* significantly increased in the no-mesh hearts. At the end of the protocol, LVP$_{ed}$ in these hearts was significantly higher than in the early-mesh hearts. *$P < .05$ vs baseline; †$P < .05$ vs no mesh; *T 25%, T 50%, T 75%,* 25%, 50%, and 75% duration of rapid pacing.
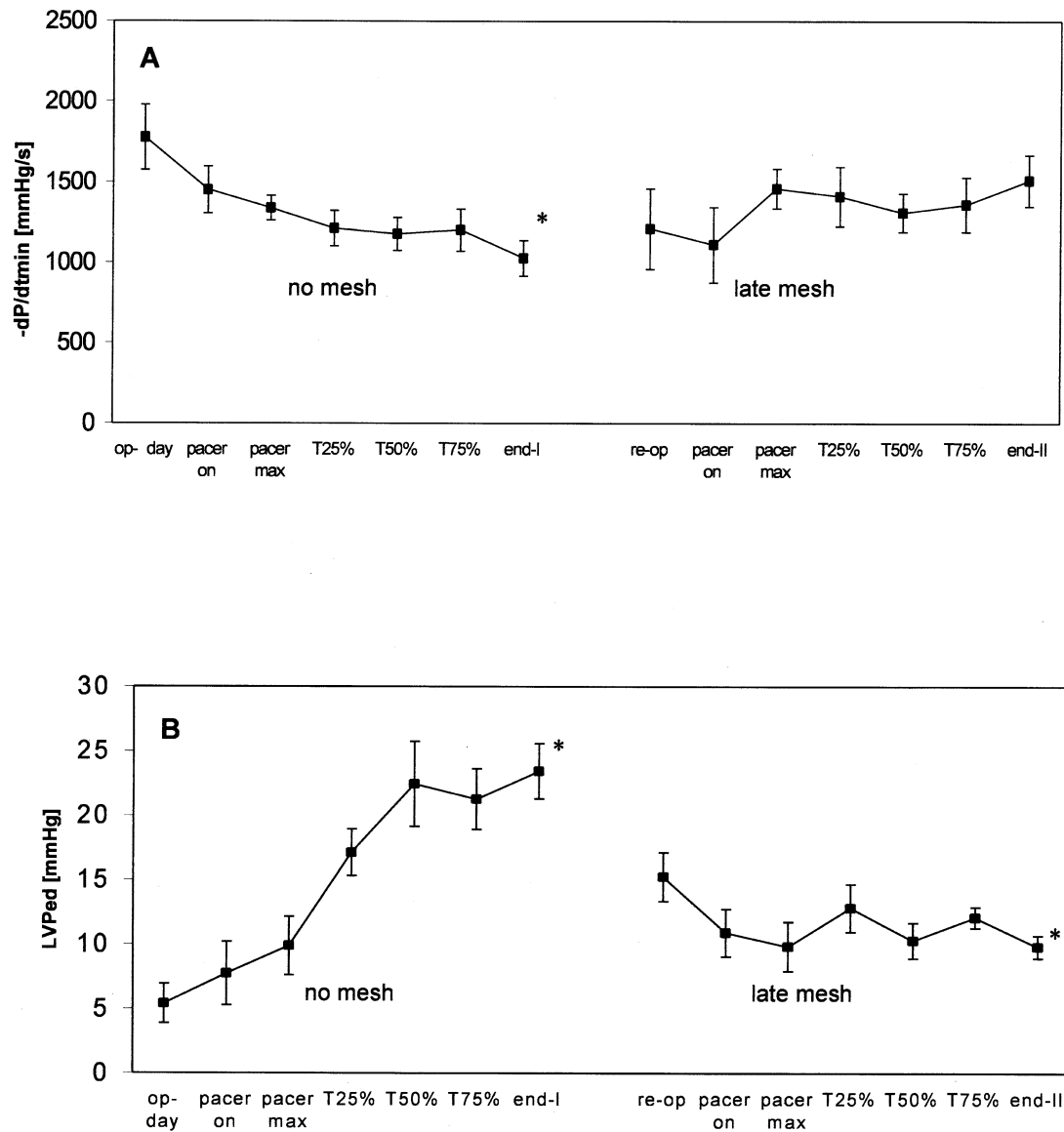
Figure E3. Diastolic variables of no-mesh hearts *(left)* and late-mesh hearts *(right)*. Diastolic function was significantly decreased after about 4 weeks of rapid pacing in the no-mesh hearts. After mesh grafting and after additional 4 weeks of pacing, early relaxation (A, dP/dt_{min}) had the tendency to improve, while the end-diastolic pressure (B, LVP_{ed}) even significantly decreased. *$P < .05$ vs baseline; *T25%, T50%, T75%,* 25%, 50% and 75% duration of rapid pacing.
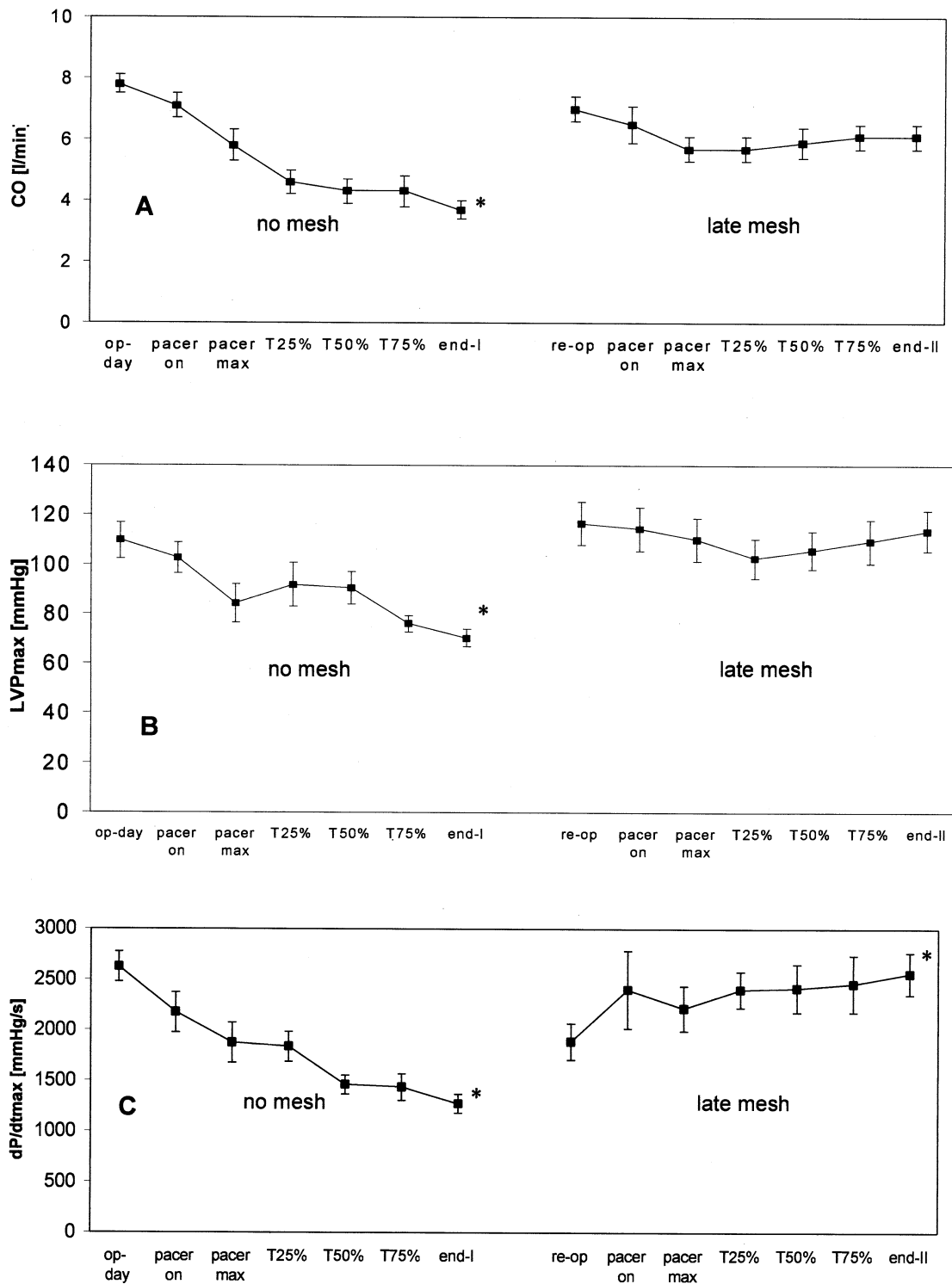
Figure E4. Systolic variables of no-mesh hearts (left) and late-mesh hearts (right). Systolic function was significantly decreased after about 4 weeks of rapid pacing in the no-mesh hearts. Mesh grafts in these failing hearts already improved the systolic function in terms of cardiac output (A, CO), peak LV pressure (B, LVP$_{max}$), and contractile state (C, dP/dt$_{max}$). The meshes furthermore prevented deterioration of CO and LVP$_{max}$. DP/dt$_{max}$ significantly improved during additional 4 weeks of rapid pacing. *P < .05 vs baseline; T25%, T50%, T75%, 25%, 50% and 75% duration of rapid pacing.