The effects of mechanical cardiac stabilization on left ventricular performance

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Abstract

Objective: Mechanical cardiac stabilization is beneficial for precise coronary anastomoses on the beating heart. However, the effect of mechanical cardiac stabilization on hemodynamics, left ventricular performance, and the degree of injury to underlying tissue are uncertain.

Methods: Twelve swine (20–30 kg) underwent median sternotomy and a mechanical stabilizing device (United States Surgical, Norwalk, CT) was positioned astride a segment of left anterior descending coronary artery (LAD). Coronary blood flow was measured by Doppler. Sonomicrometry crystals were placed distal to the stabilizer in a region of myocardium subtended by the LAD, and a left ventricular micromanometer was inserted. Regional myocardial function was determined using the preload recruitable stroke work (PRSW) relationship. Data were acquired at three time points: 20 min before (PRE) and after placing the stabilizer (EXPT); and 20 min after removing the stabilizer (POST). Tissue subjacent to the stabilizer was then biopsied. Means ± standard deviation are reported.

Results: The mechanical stabilizer caused a decrease in cardiac output from 4.2 ± 1.5 to 3.6 ± 1.3 l/min (P < 0.05), which returned to baseline values after its removal. Regional myocardial function (percent systolic shortening and Mw and x-intercept of the PRSW relationship) was unchanged. Blood pressure, heart rate, and LAD blood flow remained constant. Histologic findings included a layer of myocyte necrosis less than 1 mm in depth immediately beneath the stabilizer.

Conclusions: These data demonstrate that mechanical stabilization of the LAD may temporarily decrease cardiac output. This is not attributed to impaired contractility or ischemia, but is secondary to direct ventricular compression with reduced stroke volume. Injury to underlying tissue is negligible. © 1998 Elsevier Science B.V. All rights reserved

Keywords: Cardiac stabilization; Ventricular function

1. Introduction

A precise and effective coronary artery anastomosis is the primary objective of surgical revascularization for ischemic heart disease. With the resurgence in popularity of coronary bypass operations on the beating heart, the importance of this principle cannot be underestimated. The constant motion of the beating heart and bleeding from the coronary arteriotomy site hinder precise suture placement in the often tiny coronary vessel. Although bleeding into the operative field can be reduced by temporarily occluding the coronary artery and by continuous saline irrigation or humidified carbon dioxide insufflation, the incessant motion of the beating heart remains the Achilles’ heel of minimally invasive coronary artery bypass. Techniques employed to reduce translational cardiac motion have included specific pharmacologic agents that decrease wall motion through their negative chronotropic, inotropic, and dromotropic properties, and mechanical stabilizers that physically restrict regional wall motion [1]. Insofar as pharmacologic agents are often unpredictable in onset, variable in effect, and sometimes costly, mechanical stabilizers provide a practical and reliable solution to achieve regional cardiac stability for coronary anastomosis on the anterior surface of the heart. The effect of mechanical cardiac stabilization, however, on hemodynamics, left ventricular performance, and the degree of injury to underlying tissue are uncertain. Elucidation of these potentially detrimental sequelae is important, and provide the basis for this report.

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2. Materials and methods

All animals were given humane care in compliance with the Institutional Animal Care and Use Committee at Duke University and the ‘Guide for the Care and Use of Laboratory Animals’ published by the National Institutes of Health (NIH publication no. 85–23, revised 1985).

Twelve male crossbred swine weighing 20–25 kg were sedated with ketamine, 10 mg/kg i.m., induced with thiopental sodium, 10 mg/kg i.v., and orotracheally intubated. Anesthesia was maintained with 1% halothane using an Ohio V5 mechanical ventilator and anesthesia machine (Ohio Medical Products, Madison, WI). Respiratory rate, end-tidal CO₂, fractional inspired O₂ concentration, and inspired and expired halothane concentrations were monitored using a Capnomac infrared spectrophotometer (Datex, Helsinki, Finland). Periodic arterial blood gas specimens were obtained to guide ventilator management. Before surgical preparation, pancuronium bromide, 0.2 mg/kg i.v., lidocaine, 1 mg/kg i.v., and bretulyn tosylate, 5 mg/kg i.v., were administered. The left femoral artery was cannulated with a 9 Fr Fast-Cath (Diag Corp., Minnetonka, MN) through which a 7 Fr pressure transducer (Millar Instruments, Houston, TX) was inserted to obtain arterial blood samples and monitor central aortic blood pressure, respectively.

The heart was exposed through a median sternotomy and suspended in a pericardial cradle. The superior and inferior vena cavae were encircled with umbilical tape. A 5 Fr pressure transducer (Millar Instruments, Houston, TX) was introduced directly into the left ventricle through an apical stab incision. Two epicardial pacing wires were affixed to the right atrium using 5/0 polypropylene suture and connected to an external pacemaker (Medtronic, Minneapolis, MN). After dissecting the fat pad around the pulmonary artery, a 5 MHz Doppler flow probe (Triton Technology, San Diego, CA) was placed around the main pulmonary artery. Pulmonary artery blood flow was monitored with a Doppler flow meter (model 100–1000–05; Triton Technology, Ithaca, NY). A 10 MHz Doppler flow probe was placed around the mid-left anterior descending coronary artery (LAD) to monitor LAD blood flow. Electrocardiography, central aortic blood pressure, left ventricular blood pressure, pulmonary artery blood flow, and LAD blood flow were continuously monitored.

A pair of piezoelectric sonomicrometer crystals (diameter, 1.5–2 mm, Triton Technology) was implanted, approximately 1 cm apart, in the mid myocardium adjacent to the cardiac stabilizer (United States Surgical, Norwalk, CT) in an area subtended by the LAD. At a paced heart rate of 120 beats/min, regional myocardial contraction was determined by measuring the segment length between the two crystals based on the principle of ultrasonic transit-time (Sonomicrometer; Model 120, Triton Technology) [2]. A load-independent measure of regional myocardial function was obtained by continuously recording left ventricular pressure and segment length during transient bicaval occlusions. As previously described, left ventricular pressure-segment length loop analysis was used to calculate the indices of preload recruitable stroke work and percent systolic shortening [3]. The first derivative of left ventricular pressure, \( \frac{dP}{dt} \), was determined as the instantaneous slope of the pressure-time curve. End-diastole was defined as the first positive deflection of left ventricular \( \frac{dP}{dt} \); and, end-systole as the peak negative left ventricular \( \frac{dP}{dt} \). End-diastolic pressure and segment length represented the pressure and length, respectively, that coincided with the defined end-diastole. The time constant of isovolumic relaxation, \( \tau \), was computed assuming a non-zero asymptote of ventricular pressure decay [4]. Left ventricular negative \( \frac{dP}{dt} \) was plotted against ventricular pressure between peak negative \( \frac{dP}{dt} \) and 5 mmHg above end-diastolic pressure to yield \( \tau \) as the negative inverse of the slope [4]. Regional chamber stiffness constants, \( K_P \), were derived by a monoexponential equation using left ventricular pressure-segment length data.

\[
P = A \cdot e^{(K_P \cdot S)}
\]

where \( P \) = left ventricle end-diastolic pressure; \( K_P \) = regional diastolic chamber stiffness constant; \( S \) = end-diastolic segment length; and \( A \) = a curve fitting constant [5].

Electrocardiographic, pressure, and sonomicrometer data were continuously collected and stored at a sample speed of 150 Hz by AT/MCA-Codas (Dataq Instruments, Akron, Ohio).

Time-points for data collection were 20 min before (PRE) and 20 min after placing the mechanical stabilizer (EXPT), and 20 min after removing the stabilizer (POST). The mechanical stabilizer foot-plate was positioned astride a segment of the mid LAD, employing sufficient pressure to reduce cardiac translational motion by approximately 90%. This method of applying the stabilizer foot-plate is consistent with current clinical practice. The surgical preparation with the mechanical stabilizer in place is illustrated in Fig. 1.

At the end of the experiment, animals were euthanized and a transmural biopsy of tissue immediately below the heel of the stabilizer foot-plate was obtained and fixed in formalin. Each specimen was embedded in paraffin, stained with hematoxylin and eosin, and examined under high-power light microscopy.

In a separate series of five animals, a 5 Fr pressure transducer (Millar Instruments, Houston, TX) was placed in the left atrium prior to stabilization of the LAD. Left atrial pressure was measured prior to placement of the stabilizer and then again after 20 min of stabilization.

Data were analyzed by the SAS statistical software program (SAS Institute, Cary, NC). All results are expressed as the mean ± standard deviation. The statistical significance of changes in cardiac output, LAD flow, and load-independent indexes of ventricular function were compared by repeated-measures analysis of variance. Significance was determined at a level of 5% or less.
3. Results

All animals survived the surgical preparation and experimental protocol. The stabilizer was uniformly successful in qualitatively reducing translational cardiac motion. Proper positioning of the stabilizer foot-plate was associated with a decrease in cardiac output from $4.2 \pm 1.5$ to $3.6 \pm 1.3$ l/min ($P < 0.05$), which returned to baseline values ($4.1 \pm 1.7$ l/min) within 20 min of removing the stabilizer (Fig. 2). Blood flow in the LAD decreased from a baseline mean of $15 \pm 10$ ml/min to $11 \pm 4$ ml/min after the stabilizer was positioned. This difference, however, did not achieve statistical significance ($P = 0.07$).

When instantaneous left ventricular pressure was plotted against the regional end-diastolic segment length, pressure-length loops were generated. Plots of stroke work or work loop area versus end-diastolic length yielded linear preload recruitable stroke work relationships. The mean linear correlation coefficients for these relationships was 0.90. There were no statistical differences between the slopes ($M_w$) or the x-intercepts of the PRE, EXPT, and POST groups. Similarly, percent systolic shortening, regional chamber stiffness and the time constant for isovolumic relaxation, $t$, were not affected (Table 1).

Histologic examination of biopsy specimens demonstrated a polymorphonuclear inflammatory cell infiltrate limited to an area just below the stabilizer foot-plate. Intracellular edema, hypereosinophilia, and contraction band formation were also observed, but limited to a depth of less than 1 mm (Fig. 3).

In the series of animals in which left atrial pressure was measured, the mean left atrial pressure increased from $8.6 \pm 2.6$ mmHg to $10.8 \pm 2.6$ mmHg with application of the stabilizer ($P = \text{not significant}$).

4. Discussion

A precise and effective coronary artery anastomosis requires a motionless operative field. This is usually accomplished with the use of cardiopulmonary bypass and cardioplegic arrest. However, recent interest in coronary artery bypass grafting without cardiopulmonary bypass has led to innovations in aiding surgery on the beating heart [6]. Mechanical stabilizers, now widely available, are designed to reduce cardiac translational motion and therefore facilitate meticulous construction of coronary anastomoses. Mechanical stabilizers have been shown to significantly reduce motion at the coronary anastomotic site. Borst and associates were able to maintain durable (15–169 min) reduction in cardiac motion along the X-Y plane by employing a suction-cup based stabilization platform (Utrecht Octopus, Utrecht, Netherlands) [7]. Shennib and colleagues also reported diminished cardiac motion at the left anterior descending coronary artery (LAD) target site using a mechanical stabilization device (Cardiothoracic Systems, Cupertino, CA) similar in principle to the device employed in this study [8]. Although both mechanical stabilizers pro-

![Fig. 1. Diagram illustrating the instrumented heart with stabilizing foot-plate positioned astride the left anterior descending coronary artery. Visible are the pulmonary flow probe, LAD flow probe (f), LV pressure catheter (p) and sonomicrometry crystals (c).](https://example.com/diagram.png)

![Fig. 2. Bar graph displaying a decrease in cardiac output between the PRE group and the EXPT group (stabilizer applied) as well as between the EXPT and POST groups. * $P < 0.05$ vs. PRE, † $P < 0.05$ vs. POST, by two-way analysis of variance for repeated measures and Student’s $t$-test.](https://example.com/bar_graph.png)

<table>
<thead>
<tr>
<th>Regional myocardial function</th>
<th>PRE (mean ± SD)</th>
<th>EXPT (mean ± SD)</th>
<th>POST (mean ± SD)</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$M_w$ (erg cm$^{-3}$ 10$^{-3}$)</td>
<td>60.6 ± 17.0</td>
<td>56.0 ± 15.5</td>
<td>58.7 ± 21.7</td>
<td>NS</td>
</tr>
<tr>
<td>$L_w$ (mm)</td>
<td>9.92 ± 2.9</td>
<td>9.94 ± 2.5</td>
<td>9.94 ± 2.5</td>
<td>NS</td>
</tr>
<tr>
<td>Percent systolic shortening</td>
<td>9.87 ± 4.8</td>
<td>8.72 ± 6.0</td>
<td>9.33 ± 5.9</td>
<td>NS</td>
</tr>
<tr>
<td>$K_p$ (mm$^{-1}$)</td>
<td>0.64 ± 0.04</td>
<td>0.64 ± 0.03</td>
<td>0.64 ± 0.03</td>
<td>NS</td>
</tr>
<tr>
<td>$t$ (ms)</td>
<td>7.87 ± 1.6</td>
<td>8.67 ± 2.1</td>
<td>8.47 ± 2.2</td>
<td>NS</td>
</tr>
</tbody>
</table>

$M_w$, slope; $L_w$, X-intercept; $K_p$, regional chamber stiffness; $t$, time constant describing isovolumic relaxation, NS, not significant, compared using two-way analysis of variance for repeated measures.
vide for a reasonably motionless LAD target site, their effect on quantitative ventricular function is unclear.

In this study, we observed that proper placement of the mechanical cardiac stabilizer caused a transient fall in cardiac output. Possible etiologies of the stabilizer-induced decrease in cardiac output include: (1) left ventricular outflow obstruction; (2) impaired myocardial contractility; and, (3) decreased left ventricular end diastolic volume.

Left ventricular obstruction may occur by direct compression of the outflow tract by the stabilizer foot-plate. Although this can be a potential problem when grafting to the proximal LAD, in this study the stabilizer foot-plate was positioned remotely from the outflow tract at the level of the second or third diagonal branches.

When the stabilizer foot-plate is positioned astride a segment of mid LAD, the device may obstruct blood flow in one or more diagonal branches. This may produce regional ischemia and impaired myocardial contractility. In the present study, however, measurements of myocardial systolic function were unchanged after placement of the stabilizer. These measurements included indices of preload recruitable stroke work that are load insensitive, are highly responsive to the intrinsic performance of non-ischemic myocardium, and are exquisitely sensitive to ischemic injury [9]. In addition, a descriptor of myocardial diastolic function, \( \tau \), did not change with placement of the stabilizer. \( \tau \) represents the fall in ventricular pressure, or wall stress, over time during isovolumic relaxation and reflects active cardiac relaxation. \( \tau \) is insensitive to changes in heart rate and ventricular volume but is prolonged during myocardial ischemia [10]. As measurements were limited to regional function in this study, conclusions regarding indices of preload recruitable stroke work and \( \tau \) cannot be extended to global function. However, the sonomicrometry transducers were deliberately implanted in the region of the left ventricle most likely to be affected by stabilizer-induced ischemia. Insofar as indices of preload recruitable stroke work and \( \tau \) are highly sensitive to changes in regional ventricular function, significant myocardial ischemia was not detected.

The transient fall in cardiac output may be caused by a decrease in left ventricular end diastolic volume. Possible etiologies include: (1) right ventricular outflow tract obstruction; (2) mechanical deformation of the mitral annulus resulting in valvular insufficiency; or, (3) direct left ventricular compression by the stabilizer foot-plate. Obstruction to right ventricular outflow may be caused by direct stabilizer compression of the low pressure main pulmonary artery. Similar in pathophysiology to left ventricular outflow tract obstruction, obstruction to right ventricular outflow may occur when positioning the stabilizer to graft the proximal LAD. In this study, however, the mid LAD was chosen as the target site, far removed from the right ventricular outflow tract. A mild degree of mitral insufficiency after application of the stabilizer was suggested in subsequent studies by an increase in the mean left atrial pressure from 8.6 ± 2.6 mmHg to 10.8 ± 2.6 mmHg (n = 5) (unpublished results). However, this difference was not statistically significant.

In the absence of a right ventricular outflow tract obstruction or significant mitral insufficiency, the transient fall in cardiac output is most likely related to reduced left ventricular end diastolic volume caused by direct left ventricular compression by the stabilizer foot-plate. Although the exact amount of pressure exerted on the ventricular surface by the stabilizer was not rigorously quantified, the minimum pressure required to reduce cardiac motion sufficiently to per-
form a coronary anastomosis was approximated in each experiment. As this approach is consistent with current practice, we predict that similar qualitative changes in cardiac output will occur in the clinical setting. The magnitude of change will primarily depend on heart rate, ventricular loading conditions and myocardial contractility. These results extrapolate easily to other compressive-type stabilizers, such as the CTS system, however it is unclear to what extent the results would correlate with a suction-type stabilizer. Shennib et al., employing a mechanical stabilizer in a canine model, reported no significant change in cardiac output [8]. A possible explanation for this difference is that Shennib et al. computed cardiac output using the thermodilution method, whereas pulse-Doppler methods were employed in the present study. As the error rate associated with thermodilution ranges between 10 and 20% [11], this method may be insensitive to small changes in cardiac output.

Compression of the heart between the stabilizer foot-plate and the pericardial cradle was associated with minor histologic changes in the underlying myocardium. After 20 min of compression, a cellular infiltrate comprised mostly of polymorphonuclear leukocytes was observed in the subjacent myocardium. Moreover, a region of interstitial edema and myocyte necrosis, as evidenced by the contraction bands and hyperesinophilia, extended for approximately 1 mm into the myocardium. These findings, however, had negligible effects on cardiac function. Others have reported no significant myocardial injury in tissue subjacent to the stabilizer [7].

In summary, we found that regional cardiac stabilization, utilizing a mechanical stabilizer, is accompanied by a decrease in cardiac output without a significant impairment of regional systolic or diastolic ventricular performance. The decrease in cardiac output is transient and is not associated with any gross hemodynamic perturbations. Myocardial injury caused by the stabilizer is negligible. These data further support the use of mechanical epicardial stabilization to aid in off-pump cardiopulmonary bypass grafting.

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