# Effect of Ventricular Size and Patch Stiffness in Surgical Anterior Ventricular Restoration: A Finite Element Model Study

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*Background.* **Surgical anterior ventricular restoration (SAVER) has been proposed for dilated ischemic cardiomyopathy with an akinetic distal anterior left ventricular wall. We tested the hypothesis that SAVER increases stroke volume, reduces mean myofiber stress and achieves optimal results without a patch.**

*Methods.* **A finite element model of the left ventricle (LV) with an akinetic but contractile anteroapical LV wall segment was used. Separate versions of the model with normal and dilated LV sizes were developed and used to simulate the SAVER operation with and without a patch of varying stiffness from 10 to 100 kilopascals.**

*Results.* **The SAVER operation reduced myofiber stress in the akinetic infarct and infarct borderzone, but caused a reduction in the Starling relationship. In all cases, stroke volume decreased while ejection fraction increased after SAVER. The SAVER operation was more beneficial in dilated ventricles, and the reduction in stroke volume after SAVER without patch was minimal. The effect of patch stiffness was mixed as stiffer material causes a greater reduction in stress yet has the greatest negative effect on stroke volume.**

*Conclusions.* **These simulations support the use of SAVER in dilated hearts without a patch.**

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Surgical anterior ventricular restoration (SAVER) has been proposed as therapy for dilated ischemic cardiomyopathy with an akinetic distal anterior left ventricular (LV) wall [1]. Patch aneurysmorraphy, originally used to treat the LV with dyskinetic anterior wall [2], has increasingly been used to treat the LV with akinetic anterior wall [3, 4]. Given the increased incidence of dilated ischemic cardiomyopathy [5], interest in SAVER is growing. A working group has been established (RESTORE) [1], and surgical remodeling is a treatment arm of the multicenter National Institutes of Health funded Surgical Treatment of Ischemic Heart Failure (STICH) trial [6].

The effect of SAVER on LV function is difficult to anticipate. Early descriptions of SAVER exclude akinetic segments behind a Dacron patch [1] and fold the remaining myocardium over the Dacron in a "pants-over-vest" closure [1]. Currently, SAVER also refers to procedures omitting the patch and the double-layer closure. SAVER, therefore, represents a related group of operations performed on the LV with akinetic anterior LV wall.

We have previously simulated the LV with akinetic anterior LV wall using a mathematical (finite element [FE]) model [7]. The FE method is by far the most widely used mathematical tool in mechanical engineering design and analysis. Based on an anteroapical myocardial infarction (MI) in sheep that was reperfused after 1 hour, that simulation showed that unless passive infarct stiffness is greater than 285 times normal, contracting myocytes are required to prevent infarct wall dyskinesia [7]. Therefore, it is not merely possible for myocytes to survive in akinesis: contracting myocytes are obligatory. Of note, the determination of akinetic anterior wall contractile function is critically important to the mechanical analysis of the SAVER operation since exclusion of a weakly contracting anterior wall segment will clearly have a different effect than exclusion of an inert segment.

We used our FE model of the LV with an akinetic but contractile anterior LV wall [7] to simulate the SAVER operation. Separate versions of the model with normal and dilated LV sizes were developed and used to simulate the SAVER operation with and without patch of varying stiffness. We hypothesized that SAVER increases stroke volume (SV), reduces mean myofiber stress and that optimal results are achieved without a patch.

## Material and Methods

The three-dimensional (3-D) FE method of Costa [8] for large elastic deformations of ventricular myocardium

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**Abbreviations and Acronyms**

| | |
|---|---|
| SAVER | = surgical anterior ventricular restoration |
| LV | = left ventricular |
| FE | = finite element |
| SV | = stroke volume |
| 3-D | = three-dimensional |
| 2-D | = two-dimensional |
| $C$ | = diastolic stiffness parameter |
| $T_{max}$ | = the isometric tension achieved at the longest sarcomere length and maximum peak intracellular calcium concentration |
| $P_{ES}$ | = end-systolic pressure |
| $V_{ES}$ | = end-systolic volume |
| $P_{ED}$ | = end-diastolic pressure |
| $V_{ED}$ | = end-diastolic volume |
| $E_{ES}$ | = end-systolic elastance |
| $\beta_{0ES}$, and $\beta_{1, ES}$ | = stiffness parameters of the LV end-systolic elastance |
| $\beta_{0ED}$, $\beta_{1, ED}$, and $\beta_{2, ED}$ | = stiffness parameters of the LV diastolic compliance |
| $E_A$ | = arterial elastance |

was used, together with the mathematical descriptions for normal diastolic and systolic myocardial material properties (stress-strain relations) of Guccione [9].

### *Sheep Reperfused Infarct and Echocardiography*

A single sheep from a group previously reported by Bowen [10] underwent anteroapical ischemia and reperfusion after 1 hour. Subdiaphragmatic two-dimensional (2-D) long-axis echocardiographs were obtained through a sterile midline laparotomy (1.8 to 4.2 MHz probe, SONOS 5500; Agilent Technologies, Andover, MA) 12 weeks postinfarction [10], and videotaped [10]. The animal model displayed significant LV remodeling at 12 weeks (infarct thickness 5.1 ± 0.3 mm; LV volume at end-systole 33 ± 6 mL) [10].

Echocardiographs at early diastolic filling (Fig 1), end-diastole, and end-systole were selected, digitized and analyzed (Findtags; Medical Imaging Lab, Johns Hopkins University, Baltimore, MD). Guided by the video echocardiogram, epicardial and endocardial contours were hand-traced, and the border between akinetic and kinetic regions identified.

### *Finite Element Mesh*

LEFT VENTRICLE WITH AKINETIC INFARCT, NORMAL SIZE. Thirty-two cardiac contour points were used to construct a 2-D mesh of the LV in prolate spheroidal coordinates (Fig 2, A). Using a focal length of 25.0 mm, the FE software (Continuity; Cardiac Mechanics Research Group, University of California, San Diego, CA) interpolated a 3-D model composed of 16 elements. The FE mesh was subdivided into eight elements circumferentially and

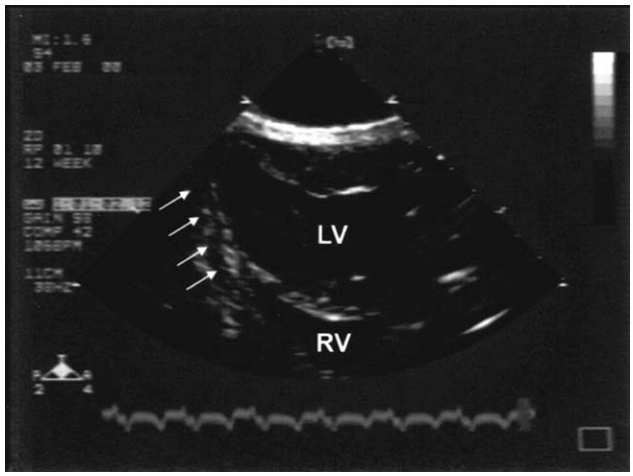


*Fig 1. Transesophageal echocardiogram of an ischemic ovine heart with akinetic infarct* (arrows) *at early diastole.* (LV = *left ventricle;* RV = *right ventricle.*)

three elements transmurally producing the 192-element model of the end-diastolic LV shown (Fig 2, B). This subdivision allowed regional, nonaxisymmetric variation of the ventricular wall.

LEFT VENTRICLE WITH AKINETIC INFARCT, DILATED. To evaluate SAVER on a markedly dilated LV representative of clinical candidates, the model was scaled to a focal length of 30.06 mm and wall thickness reduced by 33% (Fig 3, A). The resulting LV has identical myocyte mass and distribution, but 75% increased chamber volume at early diastole.

### *Finite Element Boundary Conditions*

Boundary conditions were implemented, and local muscle fiber orientation was assumed to vary as previously described [11, 12]. Assumptions about the effect of residual stress on sarcomere length have been reported. [12–14]

### *Material Properties*

DIASTOLIC MATERIAL PROPERTIES. Diastolic and systolic material properties have been presented [9, 15]. A sharp boundary was assumed between the infarcted and noninfarcted regions. The noninfarcted region was assigned normal diastolic stiffness ($C$ = 0.876 kPa) and systolic material properties ($T_{max}$ = 200 kPa). $T_{max}$, the isometric tension achieved at the longest sarcomere length and maximum peak intracellular calcium concentration, in the noninfarcted region was chosen so that the preoperative ejection fraction (EF) approximated that in the sheep model [10].

Infarct material properties were determined as described by Dang [7]. Reduction of the infarct ability to develop active stress was accomplished by scaling $T_{max}$ by a "percentage of contracting myocytes." This approach (as opposed to decreasing peak intracellular calcium concentration [16]) preserves the shape of the active stress and sarcomere length relationship. In the normal-sized model, the infarct was assigned 13.5% contracting myocytes ($T_{max}$ = 27.14 kPa). In the scaled model, the