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Development of an Off Bypass Mitral Valve Repair

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ABSTRACT

Background: The Bow Tie Repair (BTR), a single edge-to-edge suture opposing the anterior and posterior leaflets of the mitral valve (MV), has led to satisfactory reduction of mitral regurgitation (MR) with few re-operations and excellent hemodynamic results. The simplicity of the repair lends itself to minimally invasive approaches. A MV grasper has been developed that will coapt both leaflets and fasten the structures with a graduated spiral screw.

Methods: Eleven explanted adult human MVs were mounted in a mock circulatory loop created for simulating a variety of hemodynamic conditions. The MV grasper was used to place a screw in each valve, which was then continuously run for 300,000 to 1,000,000 cycles with a fixed transvalvular pressure gradient. At the completion of these studies, the valves were stressed to a maximal transvalvular gradient for ten minutes. In seven cases, MR was induced and subsequently repaired using the MV screw. In vivo, the MV screw was tested in nine male canines. Through a subcostal incision, the MV grasper entered the left ventricle, approximated the mitral leaflets and deployed the MV screw under direct visualization via an atriotomy. Follow-up transthoracic echocardiograms were done at postoperative week 1, 6, and 12 to identify screw migration, MV regurgitation/stenosis or clot formation. Dogs were sacrificed up to postoperative week 12 to allow gross and histologic assessment.

Results: In vitro, no MV screw detached from the valve leaflets or migrated during the durability testing period of 6.8 million cycles, including periods of stress load testing up to 350 mm Hg. The percent regurgitant flow used to assess MR statistically decreased with the placement of the screw from 72 ± 7% to 34 ± 17%; $p = 0.0025$. In vivo, seven dogs whose valves were examined within the first 48 hours revealed leaflet coaptation with an intact MV screw and no evidence of MR. Two dogs, followed for a prolonged period, had serial postoperative echocardiograms demonstrating consistent coaptation, no screw migration, no clot, and no regurgitation or stenosis. In the animal sacrificed at 12 weeks, the MV screw was integrated into the tissue of both leaflets.

Conclusions: The MV screw has provided durable leaflet coaptation and has reduced regurgitation in human MVs. Initial data on the MV screw's biocompatibility and interactions with living valve tissue is promising. Our early success supports further efforts towards the maturation of this prototype into off bypass mitral valve repair technology.

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INTRODUCTION

The importance of the mitral valve's papillary architecture to left ventricular function has shifted the desirable surgical approach to MR from replacement to repair [Bonchek 1984, Christakis 1985, Sand 1987, Rankin 1988]. However, the variability of the subvalvular apparatus and the numerous pathologies of mitral valve regurgitation often complicate appropriate repair technique selection. The BTR, a single edge-to-edge suture opposing the anterior and posterior leaflets of the MV, has led to satisfactory reduction of MR with few re-operations and excellent hemodynamic results [Fucci 1995, Maisano 1998, Umana 1998]. Unlike many repairs that address valvular disease at the annular level in hopes that the valve leaflets will coapt, the BTR treats subvalvular pathology by fastening the leaflets [Maisano 1998, Umana 1998].

The simplicity of the BTR lends itself to minimally invasive approaches, including avoidance of cardiopulmonary bypass and sternotomy. A MV grasper has been developed

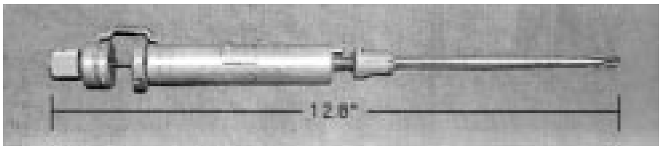

Figure 1. Mitral Valve Grasper

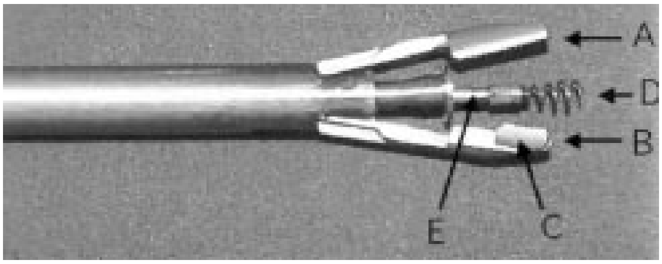

Figure 2. Grasping Mechanism and Mitral Valve Screw
A and B = jaws; C = adjustable jaw; D = Mitral Valve Screw; E = holding stem

that will approximate both leaflets of the mitral valve and coapt the structures with a fastener, the Mitral Valve Screw. For proof of concept, we created a mock circulatory loop using explanted human mitral valves and studied the ability of the MV grasper to hold, manipulate, and coapt valve leaflets and of the screw to withstand various loading conditions and durability testing while reducing mitral regurgitation. Next, we implanted the MV screw into nine dogs for various durations to assess in vivo function.

MATERIALS AND METHODS

The MV grasper is a stainless steel tool (see Figure 1) that performs the BTR by deploying a 0.25 inch long stainless steel spiral screw (see Figure 2) that coapts the leaflets of the MV. The two parts of the MV grasper system are the grasping mechanism and the screw. The grasping mechanism is a simple but precise mechanical prototype that has three functions (see Figure 2): 1) MV grasper can hold and release the MV leaflets by opening Jaws A and B; 2) Once the leaflets are grasped, they are moved back and forth over each other with an adjustable jaw (C) to minimize MR prior to screw deployment; 3) The screw (D) is advanced by clockwise rotation and released by counterclockwise rotation of a holding stem (E). The screw is made of 0.015-inch 316-gauge stainless steel wire. The largest diameter rung of this corkscrew (0.125 inches) has a sharpened point that pierces the grasped leaflets. The subsequent decreasing rung diameters cause both leaflets to be drawn tighter and closer together over a wide surface area creating coaptation (see Figure 3). The front end of the device is the conceptual part of this prototype; the back half is simply a mechanical adjunct. The MV grasper was created in specific dimensions in anticipation of a minimally invasive transthoracic application in a canine model.

A mock circulatory loop was used to assess the efficacy and durability of the BTR with the MV screw in human MVs. The mock loop was constructed using a single reservoir of adjustable height, a human mitral valve, a TCI (Thermocardio Systems Inc, Woburn, MA) Heartmate IP left ventricular assist device (LVAD) as a left ventricle, and a mechanical aortic valve (see Figure 4). These various components were connected using Bentley polyvinyl chloride tubing (Baxter Healthcare Corp, Irvine, CA). Left atrial pressure (LAP), (preload), is altered by adjusting the height of the inflow reservoir, while adjusting the maximum height of the outflow tubing alters aortic pressure (afterload). By adjusting preload and afterload, as well as the rate and ejection duration of the LVAD, a wide range of hemodynamic conditions can be simulated. Hemodynamics are monitored using high fidelity pressure transducers (Millar Instruments; Houston, TX) on both sides of the MV as well as an ultrasonic flow probe (Transonics Flow

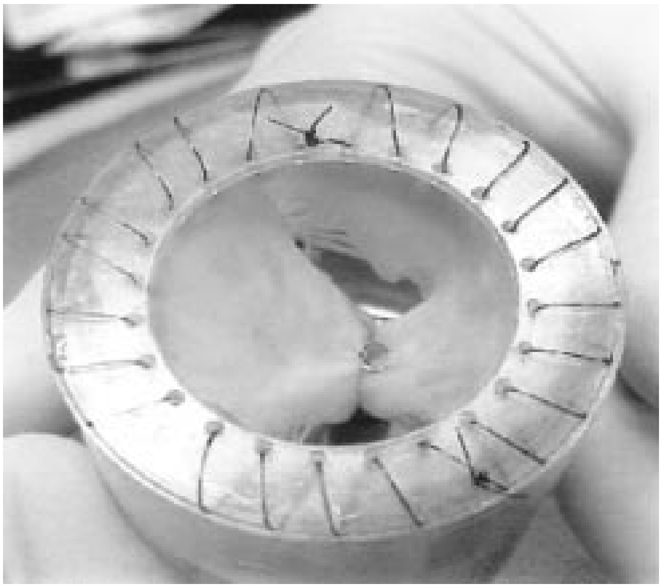

Figure 3. Coaptation by Mitral Valve Screw

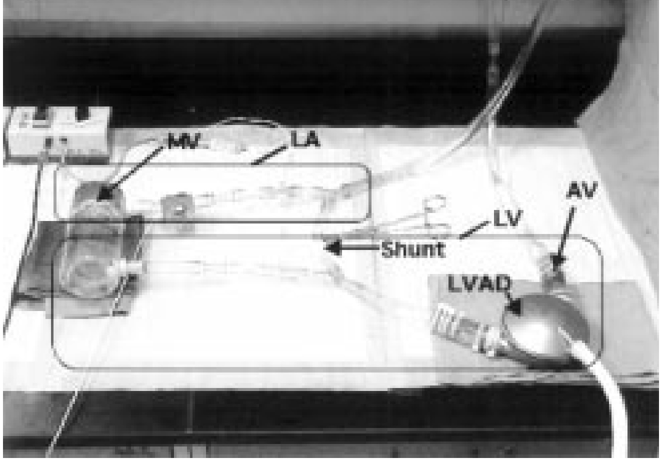

Figure 4. Mock Circulatory Loop
AV = aortic valve; LA = left atria; LV = left ventricle; LVAD = left ventricular assist device; MV = mitral valve

Figure 5. Suspension of Human Mitral Valve

Figure 6. Canine Model: In-vivo screw placement

Instruments, Ithaca, NY) on the inflow tubing. Hemodynamic parameters that can be measured or calculated using this design include LAP, left ventricular pressure (LVP), transvalvular pressure gradient, total flow, transvalvular resistance, and percent regurgitant flow. The percent regurgitant flow is calculated by the ratio of the integrals of the reverse flow over the total flow. Transvalvular pressure gradient is the maximum difference between instantaneous LAP and LVP. The largest transvalvular gradient created across a valve is the stress transvalvular pressure gradient. This stress gradient is created by increasing the afterload of the LVAD, which increases the LVP the LVAD produces to eject its volume. Since no valves exist between the LVAD and the MV, this rise in LVP is transmitted to the MV resulting in an increased (stress) transvalvular pressure gradient.

Eleven consecutive mitral valves were explanted from the hearts of cardiac transplant recipients at the time of transplantation with the approval of the Columbia-Presbyterian Institutional Review Board. The valves were suspended in a Plexiglas chamber with the annulus of the valve sewn to one ring and the papillary muscles anchored to another (see Figure 5 ◎). All eleven valves were initially placed in the mock loop to achieve maximum coaptation and competency. Mitral valve regurgitation was created in seven valves to mimic one of two main pathophysiologies of ischemic MR: posterior chordal shortening or posterior chordal lengthening/rupture [Sarris 1988, Llaneras 1993, Llaneras 1994]. A model of posterior chordial shortening was created in three valves by pulling the posterior papillary muscle down away from the annulus so the leaflets did not coapt. In four valves, a model for posterior lengthening/rupture was created by loosening or releasing the posterior papillary muscle to create prolapse. The MV grasper was used to place a screw in all of the valves noting regurgitant and total flow pre and post repair. At this point, with the mock loop at 100 cycles/min, all the valves were run at a fixed maximum transvalvular pressure gradient to assess the durability of the repair. At the end of each durability test, different loading conditions were placed on the MV via increasing transvalvular pressure gradients that were sustained for 10 minutes.

The MV screw was then tested in an in vivo model using nine male mongrel canines (weight 33 ± 6 kg). Once anesthetized, a sixth intercostal left thoracotomy was performed as well as a small left subcostal incision just below the xiphoid from which the apex of the heart could be identified and pursestrings placed. The dogs were placed on cardiopulmonary bypass so that the MVG and its manipulation of the mitral valve could be observed under direct vision. The remainder of the procedure was done on a warm beating heart with cardiopulmonary bypass times less than 30 minutes after ascending the learning curve. Via the subcostal incision, the apex of the left ventricle was incised through which the MV grasper entered to grasp the mitral leaflets and deploy the screw. A left atriotomy allowed direct visualization (see Figure 6 ◎). Once adequate coaptation of the leaflets was achieved, the atrium and ventricle were closed and the dog was separated from cardiopulmonary bypass. Follow up studies of these dogs were done by transthoracic echocardiograms within the first postoperative week and then at postoperative week six and 12 to identify screw migration, MV regurgitation/stenosis or clot formation. Hematocrits, blood smears for cellular morphology, lactate dehydrogenase (LDH), and urine for hemoglobin and hemosiderin were checked on postoperative days six and 90 to assess hemolysis. Dogs were sacrificed up to postoperative week 12 to allow gross and histologic assessment. These experiments were all performed under the standard operating procedures of the Experimental Surgery Section at Columbia University, and the protocol was reviewed and approved by the Institu-

Table 1. Endurance and Stress Testing of Mitral Valve Screw

Model	# Of Cycles Ran	Transvalvular Pressure Gradient*	Stress Transvalvular Pressure Gradient†
Human Valve I	300,000	90 mm Hg	20 mm Hg
Human Valve II	300,000	115 mm Hg	180 mm Hg
Human Valve III	300,000	120 mm Hg	200 mm Hg
Human Valve IV	300,000	120 mm Hg	250 mm Hg
Human Valve V	300,000	120 mm Hg	350 mm Hg
Human Valve VI	300,000	120 mm Hg	250 mm Hg
Human Valve VII	1,000,000	120 mm Hg	230 mm Hg
Human Valve VIII	1,000,000	120 mm Hg	250 mm Hg
Human Valve IX	1,000,000	120 mm Hg	200 mm Hg
Human Valve X	1,000,000	120 mm Hg	200 mm Hg
Human Valve XI	1,000,000	120 mm Hg	200 mm Hg

*Transvalvular Pressure Gradient = maximum difference between instantaneous left atrial and ventricular pressures.

†Stress Transvalvular Pressure Gradient = maximum difference between instantaneous left atrial pressure and an increased ventricular pressure

tional Animal Care and Use Committee of Columbia University.

All hemodynamic data were collected using the Maclab/8e (AD Instruments, Mountain View, CA) 12-bit analog-to-digital conversion board at a sampling frequency of 200 Hz using Chart v3.6 software (AD Instruments). All results are reported as the mean ± standard deviation. Continuous variables were analyzed in a paired manner using the paired Student's t-test. A p-value of less than 0.05 was considered statistically significant. Data analysis and the calculation of the percent regurgitant flow was performed using Igor Pro v3.13 (Wavemetrics, Lake Oswego, OR), Microsoft Excel 98 (Redmond, WA), and SAS v6.12 (SAS Institute, Cary, NC).

RESULTS

In vitro, no MV screw detached from the valves' leaflets or migrated during the durability testing period of over 6.5 million cycles, including periods of stress load testing (see Table 1, Movie 1). The mitral screw was able to pierce

Table 2. Assessment of Percent Regurgitant Flow

Ischemic MVψ Pathology	% Regurgitant Flow Baseline	% Regurgitant Flow with Pathology	% Regurgitant Flow with MV Screw Repair
Chordal Shortening	66.1	74.5	63.5
Chordal Shortening	26.8	71.7	34.8
Chordal Shortening	39.7	84.9	26.5
Chordal Rupture	28.0	65.2	45.6
Chordal Rupture	52.3	63.1	39.5
Chordal Rupture	18.8	68.7	17.0
Chordal Rupture	12.7	73.2	13.7
Mean ± Std Deviation	34.9 ± 19.0	71.6 ± 7.2ψ	34.4 ± 17.3Ω

ψMV = mitral valve; p=0.0027 vs baseline; Ω p=0.9 vs baseline, p=0.0025 vs pathology

the leaflets of all the human valves regardless of fibrosis and achieve coaptation without damaging the native valve apparatus. Both chordal shortening and chordal lengthening models resulted in significant MR as seen by the significant increase in percent regurgitant flow from baseline (see Table 2). Assessing the area of malcoaptation visually, seven of the MV screws were placed at the midpoint, and four were placed in a paracommissural location. The percent regurgitant flow statistically decreased with the placement of the screw in both types of ischemic MV pathophysiologies tested (Table 2). Coaptation of the leaflets with the MV screw did not change total flow through the mitral valve (1.1 ± 0.3 to 1.2 ± 0.4 L/min; p = 0.7) or the resistance of the mitral valve (12.9 ± 11.9 to 10.2 ± 2.8 mm Hg·min /L; p = 0.6).

In vivo, three animals were sacrificed in the operating room after successful MV screw placement with one valve demonstrating a tear caused by the grasping mechanism. Four additional animals whose valves were examined within the first 48 hours revealed leaflet coaptation with an intact MV screw and no evidence of MR. Two dogs were followed for a prolonged period and had serial postoperative transthoracic echocardiograms demonstrating that the screws remained in place and had not caused regurgitation or stenosis. Consistent coaptation of the MV leaflets was present with the MV screw which was clearly visualized on

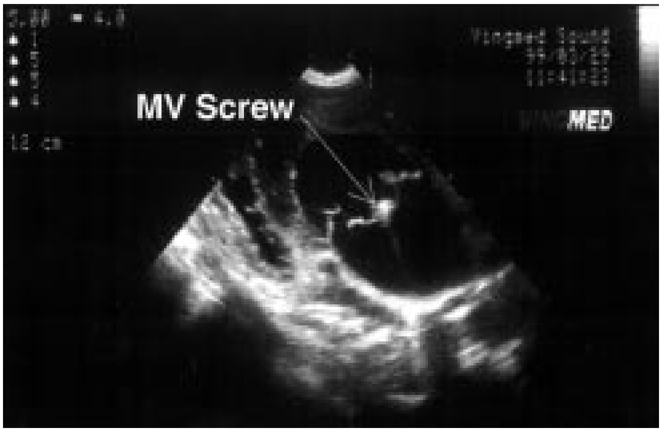

Figure 7. Three month postoperative echocardiogram of canine mitral valve with screw

Figure 8. Post-mortem view of endothelialized screw

echocardiogram with minimal artifact (see Figure 7). No clot was seen in any of the echocardiograms. No blood smears or lab results (all urine negative for hemoglobin and all LDH < 40). indicated hemolysis. In the animal sacrificed at 12 weeks, coaptation of the MV leaflets could still be demonstrated over an area of 22 mm^2 and the MV screw was endothelialized (see Figure 8).

DISCUSSION

Malcoaptation of the leaflets, the unifying pathology for mitral valve regurgitation, is only indirectly addressed by most repair techniques. Attempts to decrease annular size, transplant chordae, or resect portions of the leaflets hope to create an architecture in which the leaflets will once again coapt. The BTR actively corrects the common pathology of leaflet malcoaptation by fastening the leaflets together ensuring a fixed area of coaptation without disturbing the subvalvular or annular architecture. This allows for preserved if not improved left ventricular function [Maisano 1998, Umana 1998] and for exercise induced annular dilatation [Umana 1997]. The BTR has resulted in excellent postoperative functional status and an overall freedom from re-operation of 95% at six years with impressive initial reports of its use alone in patients with ischemia [Maisano 1998, Umana 1998] or dilated cardiomyopathies [Batista 1990, Starling 1997].

The BTR's advantage of addressing subvalvular architecture coupled with its simplicity and efficiency makes the technique ideal for the conceptual focus of a minimally invasive approach to mitral valve surgery. The fastener of our system like the BTR suture was designed to guarantee the central concept of leaflet coaptation over a specified and fixed area. The screw's graduated size draws the leaflets into apposition leading to eventual fibrosis, a necessary requirement for a permanent repair. The MV screw repair has proven to be durable as evidenced by its testing in human valve tissue for over 6.5 million cycles with transvalvular pressure gradients up to 350 mm Hg as well as by its testing in an in vivo model for up to three months, both resulting in no screw migration or tissue damage. This durability is not surprising since the mathematically modeled and calculated stresses on the closed MV demonstrate that the net force on the area of leaflet coaptation is minimal [Arts 1983]. The forces applied to the valve by the left ventricle at the area of coaptation are not only parallel to the annulus but the force on each leaflet is symmetrical and opposite to the force on the opposing leaflet. Therefore, ventricular contraction seems to push the area of coaptation together with only a minimal force pushing the leaflets apart or towards the atria. Diastole may cause a more significant stress on the area of coaptation by pulling the leaflets apart [Arts 1983] The material and design of the screw also proved to be biocompatible since the screw did not cause thrombosis or hemolysis and did become integrated into the mitral valve tissues.

The screw accomplished its primary directive of coaptation, which was seen in our canine model as well as with the human mitral valves. This effect was lasting as seen by autopsy of the dogs and over the 6.5 million cycles the human valves endured in the mock loop. This coaptation decreased ischemic mitral regurgitation as modeled in the mock loop by chordal shortening and chordal rupture. Mitral stenosis was not evidenced in the human valves, which showed no significant increase in resistance or decrease in flow with MV screw placement. In the canine model, echocardiograms and/or LAPs during the operation and at three months also did not indicate mitral stenosis.

Although, we have had success with the MV grasper and its ability to coapt leaflets, we have seen and can anticipate further problems with the MV grasper when severely calcific or fibrotic valves are encountered. The ability of the MV grasper to hold these types of leaflets equally, or even at all, may be limited. Unequal grasping can also lead to deployment of the MV screw into just one leaflet, thus producing no coaptation. The grasping of scarred valves may also prove challenging. Therefore, as the MV grasper concept moves forward, the grasping mechanism will need refinement while maintaining the mechanism's catheter compatible size of 7 mm diameter (see Figure 2).

The MV screw has proven to be durable and capable of leaflet coaptation and of reducing regurgitation in human mitral valves. Initial data from in vivo studies on the MV screw's biocompatibility, transthoracic application, and interactions with living valve tissue is promising. The success of this initial proof of concept supports further efforts toward the maturation of this prototype into a novel, simple, and cost-effective catheter-based system to perform mitral valve repair.

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REVIEW AND COMMENTARY

1. Editorial Board Member TL41 writes:

This is an original, potentially useful and practical addition to the available techniques. It is well written and takes advantage of the graphics capabilities of Web publishing.

The durability of the technique, however, has not been very rigorously tested; one million cycles are equivalent to about one week and the in vivo experience is a maximum of three months.

2. Editorial Board Member NC124 writes:

I believe that the title should be changed to something regarding a new approach to MV repair (minimally invasive) due to the fact that even the operation might be done only via the apex of the LV, with echo guidance. These experiments were done under CPB. They should try to do some experiments off the pump and then expose the options.

3. Editorial Board Member S389 writes:

The paper was called off bypass MV repair, but it seems that the dogs were placed on CPB.

Authors' Response by David L.S. Morales, MD to TL41, NC124 and S389:

As noted by the comments, our reported series is but the first step in the development of this off bypass technique of mitral valve repair. However, our series did serve as a successful proof of concept for this technique, opening the door for further investigations. Our next series of experiments with our canine model will be done off bypass under transesophageal echocardiogram (TEE) guidance. In our reported series, we used echocardiography in three dogs to assess the MV grasper's echogenicity while in the left ventricle. The artifact created by the MV grasper did not prohibit the identification of the mitral valve or the location of the screw once it had been deployed. These findings are encouraging, and we look forward to reporting the results of our upcoming series.

In regards to durability testing, the next series of dogs will be survived for six months with echocardiography done every six weeks after the first two months. In terms of in vitro testing, the mock loop is best at creating different hemodynamic situations but is limited by the LVAD in terms of the speed at which it can cycle. We plan to run a series of experiments using a valve tester in which the FDA required 400 million cycles (approximately equivalent to 10 patient years) can be run within a reasonable time.

4. Editorial Board Member NC124 writes:

What are the actual risks of embolization if done off the pump? Could it be done via an atrial trans-septal approach?

Authors' Response by David L.S. Morales, MD:

Embolization of the screw is unlikely in any situation unless the screw is deployed when there is no tissue in the grasper. However, there is a safety mechanism in the system that does not allow screw deployment from the holding stem unless the screw is embedded in tissue. Once in valvular tissue, the screw appears to become quickly incorporated into the tissue. This coupled with the screw's helical design makes dislodgment highly unlikely. We have had no cases of screw embolization or migration with any of our work thus far.

We have not explored atrial trans-septal access to the MV because without a cardiotomy the MV leaflets can only be grasped from a ventricular approach. Atrial access would necessitate an instrument transversing the valve to grasp the leaflets, making accurate assessment of MV regurgitation or its reduction by a repair more difficult.

5. Editorial Board Member LO23 writes:

An interesting new approach to possible percutaneous mitral valve repair. An analysis between the group with midpoint application as opposed to paracommisural application would be interesting and relevant.

Authors' Response by David L.S. Morales, MD:

The decision to place the screw in a regurgitant MV in the paracommisural versus midpoint location is based on the surgeon's evaluation of where mal-coaptation is occurring. The screw is then placed in the midpoint of this area to provide coaptation regardless of location on the valve. Investigation into whether a paracommisural (asymmetric) BTR can achieve the reduction in MV regurgitation that a midpoint (symmetrical) BTR achieves could be completed in the mock loop. This comparison of repair location has already been started in our series of human valves reported. Preliminary data shows that both repairs result in equivalent reduction of regurgitation.