data fit with some clinical series in which initially plications were performed at the commissures leading to early repair failures, and these patients came back with regurgitation early after surgery, even sometimes in the same admission. Therefore, we now perform plications in the middle of the free edge and the middle of the cusp, just an example to demonstrate that our data correlate with what's clinically seen in series.

Dr Schmoker. Or it may be a limitation of finite element analysis as far as where you're seeing those stresses evolving through diastole.

My final question, as you move forward, how reliable do you think this model is going to be for evaluating repair techniques if you use the same root for preintervention, postintervention, and repair techniques as you propose to do? Because the tissue is assessed at room temperature, my concern is that this may affect structural integrity of the matrix materials and cells over the time frame of the acute experiments, which may impair your final analysis

in your valve repair techniques. In other words, I'm looking at a time frame of warm ischemia from harvest to the end of proposed repair. Do you have any plans to look at structural changes, such as with histology?

Dr Al-Atassi. First, we have to produce different models for the different types of AI. Second, in terms of time frame, there are data in the engineering literature, at least, that end-diastolic stresses correlate with material fatigue. So if we establish that correlation, I don't believe we would need a long-lasting root that is exposed to months of evaluations to find out whether our repair is functional. I believe the important thing is if we establish a low-stress model of repair, then we can extrapolate and say that's likely a better repair technique than one that maximizes the stresses on the cusps. Finally, our model is examining macro-level findings, such as dimensions and coaptation, and although ischemic time may affect the root at a cellular and histologic level, it is unlikely to affect the results we are after.

EDITORIAL COMMENTARY

A porcine model for aortic valve insufficiency: If pigs could fly, they would teach surgeons to treat AI

John J. Squiers, BSE,^a William T. Brinkman, MD,^b Mani Arsalan, MD,^{b,c} and J. Michael DiMaio, MD^{a,b}

See related article on pages 656-64.

In this issue of the *Journal*, Al-Atassi and colleagues¹ report their development of an in vitro porcine model for aortic insufficiency (AI) in a left-heart simulator. By incorporating patches into vertical incisions at the sinotubular junction (STJ), they were able to increase STJ diameter and severity of AI while concomitantly decreasing coaptation surface

area, establishing a relationship between aortic root geometry and aortic valve function to elucidate the mechanism of AI.¹ Use of this model will allow surgeons to gain a better understanding of aortic valve function.

The major advantage of this model is its incorporation of the entire valve complex: not just the aortic leaflets but also the STJ, sinus of Valsalva, ventriculoaortic junction, and cusp stress. The ability of Al-Atassi and colleagues¹ to measure coaptation surface area by means of the model, a concept requiring measurements in 3 dimensions rather than the standard 2-dimensional measurement of coaptation zone length, is particularly enlightening. With these successes, Al-Atassi and colleagues¹ have started to define AI in a structural engineering sense, which should enable surgeons to evolve the "art" of aortic valve repair into more of a "science."

From the ^aDepartment of Epidemiology, Baylor Healthcare System, Dallas, Tex; ^bThe Heart Hospital Baylor Plano, Baylor Healthcare System, Plano, Tex; and the ^cDepartment of Cardiac Surgery, Kerckhoff Clinic Heart Center, Bad Nauheim, Germany.

Disclosures: Authors have nothing to disclose with regard to commercial support. Received for publication June 18, 2015; accepted for publication June 23, 2015; available ahead of print July 22, 2015.

Address for reprints: J. Michael DiMaio, MD, Baylor Healthcare System, 6125 Luther Ln, Dallas, TX 75225 (E-mail: jmdimaio@yahoo.com).

J Thorac Cardiovasc Surg 2015;150:664-5

0022-5223/\$36.00

Copyright © 2015 by The American Association for Thoracic Surgery

<http://dx.doi.org/10.1016/j.jtcvs.2015.06.053>

Because the practice of science requires initially the asking of questions and subsequently the answering of questions, we outline here several questions for which this model could potentially provide answers in the future.

First, the model may help to determine evidence-based indications for surgery in AI. In contrast to aortic stenosis, it is currently less well understood when surgical therapy for AI is appropriate. The most recent valvular heart disease guidelines, which contain level of evidence B and C recommendations for AI intervention, may be further strengthened in future versions with more quantitative evidence available to guide surgeons.² Although the model at hand was developed in vitro, there is certainly potential for in vivo exploration with the technique of STJ dilatation established by Al-Atassi and colleagues.¹ Performing this modification in vivo and tracking cardiac remodeling or any number of functional metrics would undoubtedly shed light on the pathophysiology and associated sequelae of AI. Furthermore, testing various timings or indications for subsequent intervention could uncover when surgical aortic valve therapy is ideal. By using the model to gain a mechanical understanding of AI, surgeons may better determine which patients stand to benefit from surgical intervention and when such intervention is optimally performed.

As indications for surgical intervention in AI are crystalized, the advantages and disadvantages of the various therapeutic options will be placed under the microscope. Repair of the aortic valve appears to be the next generation of valve repair, following the revolution in mitral valve therapy built upon the repair technique of Carpentier.³ The question of where and how the aortic valve complex should be rebuilt during repair remains open. Several experts have staked their claims on the frontier of aortic valve repair. El Khoury⁴ addressed the valve directly through a cusp repair technique in tandem with various aortic annulus stabilization methods. Two primary valve-sparing methods, David's aortic root reimplantation technique⁵ and Yacoub's aortic root remodeling procedure,⁶ have become the most frequently implemented worldwide. If and when any of these techniques are superior to the others, however, is still a matter of debate.⁷ The model developed by Al-Atassi and colleagues¹ provides a precisely controlled environment in which these techniques can be studied and compared against one another.

Of course, a truly scientific approach to finding definitive answers for these questions requires quantitative methods of assessment of AI. Echocardiography⁸ and

angiography⁹ provide poor quantifications of AI severity, and echocardiography in particular is plagued by interuser and intertemporal variability in its assessment of valvular regurgitation.¹⁰ With the model developed by Al-Atassi and colleagues,¹ multiple quantitative hemodynamic parameters, including regurgitant volume, regurgitant fraction, and effective regurgitant orifice area, can be readily assessed. The ability of this model to provide such objective data suggests that it may help to provide definitive, preclinical evidence regarding the timing and strategy for aortic valve intervention in the future.

With a better understanding of both *how* and *when* to operate, outcomes for patients with AI are sure to improve. We commend Al-Atassi and colleagues¹ for their unique approach in the development of an in vitro porcine model for AI. In addition to the knowledge published in this issue of the *Journal*, this model shows promise to provide additional answers for surgeons seeking better treatment of aortic regurgitation in the coming years.

The authors thank Leah Gaither for her editorial assistance.

References

1. Al-Atassi T, Toeg HD, Jafar R, Sohmer B, Labrosse M, Boodhwani M. Impact of aortic annular geometry on aortic valve insufficiency: insights from a preclinical, ex vivo, porcine model. *J Thorac Cardiovasc Surg*. 2015;150:656-64.e1.
2. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP III, Guyton RA, et al; American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2014;63:2438-88. Erratum in: *J Am Coll Cardiol*. 2014;63:2489.
3. Carpentier A. Cardiac valve surgery—the “French correction.” *J Thorac Cardiovasc Surg*. 1983;86:323-37.
4. El Khoury G, de Kerchove L. Principles of aortic valve repair. *J Thorac Cardiovasc Surg*. 2013;145(3 Suppl):S26-9.
5. David TE, Feindel CM. An aortic valve-sparing operation for patients with aortic incompetence and aneurysm of the ascending aorta. *J Thorac Cardiovasc Surg*. 1992;103:617-21; discussion 622.
6. Sarsam MA, Yacoub M. Remodeling of the aortic valve annulus. *J Thorac Cardiovasc Surg*. 1993;105:435-8.
7. Tian D, Rahnavardi M, Yan TD. Aortic valve sparing operations in aortic root aneurysms: remodeling or reimplantation? *Ann Cardiothorac Surg*. 2013;2:44-52.
8. Cawley PJ, Hamilton-Craig C, Owens DS, Krieger EV, Strugnell WE, Mitumori L, et al. Prospective comparison of valve regurgitation quantitation by cardiac magnetic resonance imaging and transthoracic echocardiography. *Circ Cardiovasc Imaging*. 2013;6:48-57.
9. Croft CH, Lipscomb K, Mathis K, Firth BG, Nicod P, Tilton G, et al. Limitations of qualitative angiographic grading in aortic or mitral regurgitation. *Am J Cardiol*. 1984;53:1593-8.
10. Ewe SH, Delgado V, van der Geest R, Westenberg JJ, Haack ML, Witkowski TG, et al. Accuracy of three-dimensional versus two-dimensional echocardiography for quantification of aortic regurgitation and validation by three-dimensional three-directional velocity-encoded magnetic resonance imaging. *Am J Cardiol*. 2013;112:560-6.