Key Words: mitral valve replacement, mitral valve diseases, bioprosthetic valve, RESILIA tissue

Discussion

Presenter: David Heimansohn

Dr Gabriel Aldea (Seattle, Wash). Thank you. I wanted to again congratulate Dr Heimansohn and his colleagues for presenting these data, for allowing me to review the presentation ahead of time, and we all fully agree with efforts to enhance durability of bioprosthetic valves studied here with a resilient platform. The authors demonstrated safety with excellent early mortality—it’s actually 4 times better than predicted—low gradients, excellent New York Heart association class with a follow-up of 4 years. So, I’d like to make several comments and ask several questions of the authors because the study highlights the difficulty and challenges of such studies. To begin with, if we compare it with the COMMENCE aortic trial, the enrollment here was one-eighth of that, which I think has to do with the selection of patients. Most of us will repair most mitral valves for particularly and specifically this reason. The second is that the study is mostly a study of elderly patients, 80% older than 60 years and more than 50% older than 70 years. So presumably, the etiology for mitral valve replacement is significant mitral calcium and stenosis, perhaps with some element of regurgitation that cannot be repaired, and we know that those patients do not do as well long term. In addition, only two-thirds of the patients had follow-up data. A total of 13.4% died in 4 years, again highlighting the complexity of these patients, and therefore only two-thirds of the patients had echocardiographic data.

Finally, the definitions used here, which are the common, standard definitions of an [inaudible], published in 2008, really underestimate the incidence of valve degeneration. And again, a publication by Dr Devere, my colleague, in 2018 in Circulation, with several of the panelists here being coauthors, demonstrated that there are morphologic valve changes that precede physiologic changes, including regurgitation of gradient before we get to the point of structural valve degeneration. So, the questions I had to the authors are—one is patient selection, one is were tricuspid valve repairs excluded, just to make a more pure patient selection? The other is anticoagulation strategy for the patients. The third in their own practice now, given these data—and it’s very young data, intermediate data, which we know the majority of degeneration occurs at 8 or greater years—would they choose porcine pericardial or mechanical valves in their patients? What is the follow-up that is required for all bioprosthetic valves, particularly in these patients—transthoracic echo, computed tomography (CT), because again as the failure that they reviewed noted were talking and focusing on valve calcification—but perhaps we should equally focus on valve thrombosis? And finally, given the very small denominator that is expected to decrease with attrition of some of these patient population, at the end of 10 years, would we have to extrapolate data from aortic valve implantation in the COMMENCE trial to mitral and tricuspid valves? I really thank the association for the opportunity to comment on this important work.

Dr David Heimansohn (Indianapolis, Ind). Thank you, Dr Aldea for taking your time to review this presentation and you asked spot-on questions, some of which have answers and some of which don’t. This study absolutely was limited because of the strict patient selection. They did not allow tricuspid valve repair. And, there was a lot of discussion at the time of the study design and the ongoing study, and near the end, I believe they changed it and allowed it, but that severely limited patient enrollment. The anticoagulation was standard, 3-month anticoagulation after mitral valve repair, and then per the site’s choice, depending on atrial fibrillation and other reasons to undergo anticoagulation. Your valve choice is something we all face every day, and being a busy mitral valve surgeon, as we all are on this presentation, nobody wants mechanical valves. Nobody wants to deal with warfarin. It’s striking to me, even when you push patients and recommend it, they don’t want it.

So, I think that’s where the value of these kind of research studies are and even if you can make an incremental improvement in a valve’s success and durability, I think you’ve gone a long way to helping this population. The other key question, follow-up—in fact, there’s been a lot of discussions with this as well, and I’m involved in other valve studies where I think the science is evolving to 4-dimensional CT scans, certainly in the aortic position—whether it ends up being in the mitral position as well, time will tell. But that’s certainly something that gives us more and different information than the echos do because the echos are more physiologic and flow, and the CT gives you much more anatomic details and can certainly quantitate calcification. And finally, this is going to be carried
out to 10 years. Obviously—who knows—it may be single
digits by the time we get to 10 years because the median age
is 70 years of age. And it probably will have to be correlated
with the aortic study, which they’ve recently decided to
follow those patients younger than age 65 years as a 11-
year substudy, so that may help answer some of the ques-
tions, at least for the tissue processes in this complex tissue
valve world.