Discussion

Dr. Lenox D. Baker (Norfolk, Virginia): I would like to thank Dr. Kron for sending me a copy of his manuscript prior to this meeting to review it. I think the group from Charlottesville certainly needs to be commended on attacking an extremely sick group of patients. These people not only have poor left ventricular function, but many of them have mitral insufficiency with mechanical problems related to it, along with the arrythmia problems that are the primary focus of the study.

I think that Drs. McGuire and Horsley yesterday pointed out that the effects of some of the primary physicians' attitudes toward certain treatments, especially in ulcer disease, have affected the types of patients we are seeing now. I think that this concept also applies to ischemic coronary disease. I think that the CASS study, along with the promulgation of HMOs and gatekeepers are giving cardiac surgeons a much sicker group of patients to handle now than we saw 5 years ago. I think this type of patient with these postinfarction mitral insufficiencies and tachyarrhythmias is going to become more common.

Down in Norfolk, we do not have the advantage of the excellent electrophysiological work of Drs. DiMarco, Echt, and others, but we have had a smaller number of these patients still come to surgery. The question always comes up, how best to handle them. We have gone ahead and, under the example of Drs. Kron, Hammond, and others, done extensive scar resections, including four mitral valve replacements, and have been satisfied with the early results with these patients. We think that they have been improved under this type of aggressive approach rather than trying to leave untreated disease behind. The technique of mitral valve replacement through the ventriculotomy is actually much easier than trying to do this through an atriotomy, and we have been comfortable with that.

The questions I have for Dr. Kron are: In areas where we do not have sophisticated electrophysiology, and also especially in these acute patients when we do not have time for it, is is reasonable to go ahead and resect all scar, including mitral valve replacements? We do not worry about the risk of mitral valve replacement in these people as much as the long-term risk of not treating significant mitral insufficiency or, worse, having to come back and replace the valve at a later time.

One further question: If you do have to go back in a patient on whom you have not resected the mitral valve, do you go back in through the ventricle or do you go back in through a standard atriotomy?

Again, I would like to compliment Dr. Kron for excellent management of this group of patients.

Dr. John W. Hammon (Nashville, Tennessee): I also rise to congratulate Drs. Nolan and Kron and their coauthors on a very innovative approach to treatment. This, I think, needs to be emphasized as the sickest group of patients in the arrhythmia group, those patients who have extensive enough scarring to involve the papillary muscle apparatus of the mitral valve.

I think that the controversy in this group of patients is based on opposing thoughts that a surgeon might have when viewing a patient with a scarred papillary muscle that may not be causing any mechanical mitral insufficiency or only mild mitral insufficiency and following the usual adage "if it's not broken don't fix it."

Dr. Nolan and Kron's results showed that in their three patients scarred papillary muscles that were not "causing any problems" later caused difficulties and were difficult to treat at that time. In fact, if a patient has an extensive ventricular resection, multiple coronary bypasses, and mapping for an indefinite period in the future, he is a very poor candidate for any further ablative or reconstructive cardiac surgery. Thus, all that needs to be done, I think, ought to be done at the one opportunity the surgeon has to do it right.

I would like to ask the authors what their feelings are now, based on their past experience. Would they replace the mitral valve in the patient with extensive papillary muscle scarring even if the site of arrhythmia cannot be localized to this area? This remains a most difficult problem for all of us.

Thank you very much for allowing me to discuss the paper.

Dr. Irving L. Kron (Closing discussion): The majority of deaths in our series have been due to arrhythmias on the papillary muscle. It took us a long time to tumble to the fact that one really had to be aggressive for this group. These are people we would prefer not to operate on. We know it ahead of time. They usually have posterior infarctions, and, while one can predict very good results in nice straightforward aneurysms, the result in these patients is basically that the arrhythmia can be poorly handled if it occurs after operation.

We have noted when we used the cryoablation technique that everything works just fine for about 48 hours, and, as the tissue edema goes down, the arrhythmias may recur. We have gone to a much more aggressive approach, since these people die if their arrhythmias are left untreated.

I would like to answer the questions. Dr. Baker first asked about extended endocardial resection and mitral valve replacement in those patients without the use of intraoperative mapping. We would obviously prefer Dr. Baker to refer those patients to our institution, but when the situation is acute, there is no doubt that the patient would die in the next few hours without surgical therapy. The results in this type of case, I think, are good, with just complete endocardial resection and valve replacement needed in an acute situation.

The problem with this technique, unfortunately, is that you will probably miss about 20 to 25% of the arrhythmias. We have noticed this with our technique of sequential resection. What we will do is to perform the resection where it was initially mapped. We then attempt to restart the arrhythmia, and we can about 20% of the time restart the arrhythmia, even after we had taken out everything we thought we needed to. Undoubtedly Dr. DiMarco will point out that there may be something more to excise on the septum, and there usually is. Therefore, we feel that mapping is important in these patients. We agree with Dr. Hammon's earlier comments that this is still an experimental procedure, and we hope that we continue to learn what techniques are best to use.

What about the concept of what to do in a patient who needs a later mitral valve replacement who has had a previous ventriculotomy? It is very simple, surprisingly enough, to go back through the ventricle. In other words, this is basically purely thinned-out scar that we have already closed, and we have had the opportunity to do this on two occasions. This is much simpler than dissecting the entire heart, and we would recommend it for those of you unfortunate enough to go back in on one of these patients.

Dr. Hammon asked a question that I am not sure I have a very good answer to. He asked, when does one resect papillary muscle when the site of the arrhythmia can clearly be localized on the septum or another site? We are going to hedge just a little bit here because we have only had three cases. It is my bias—and not my scientific belief, but bias—that a patient who has severely scarred papillary muscles and presents relatively acutely with an arrhythmia within 6 to 8 weeks of myocardial infarction probably should have papillary muscle resection and valve replacement at that time. I think that is the safest short-term solution.

The patient who presents many months after myocardial infarction with arrhythmias localized to another site probably can be managed safely by leaving the papillary muscles alone and performing endocardial resection where needed. We are not absolutely certain, and we may have to come back to you later with further answers for this.

Thank you again for the privilege of the floor.