REPLY

We appreciate the suggestions of Dr. Veyrat regarding our study (1) and agree that it would be of interest to trigger the computed tomographic (CT) images at the time point of maximal aortic valve opening as previously calculated with Doppler velocity-time integral. However, some technical and logistic considerations arising from the CT perspective should be taken into account.

The heart rate may be significantly different between the transthoracic echocardiography (TTE) and the CT examination. Frequently, the heart rate increases during contrast agent injection, which is performed immediately before the scan. Therefore, the maximal aortic valve opening cannot be expected to be at the same time point.

Therefore, we chose an "empiric" approach to identify the maximal aortic valve opening. We reconstructed the aortic valve at multiple time points throughout the systole and identified the maximal valve opening based on visibility. Considering a mean duration of the QRS complex of 80 ms (a QRS complex of 120 ms or longer is regarded as bundle branch block), we started the image reconstruction series 50 ms after the beginning of the QRS so as to begin early enough and not miss the valve opening. We would like to emphasize that Figure 1 in our report should illustrate the CT image reconstruction algorithm and not regular cardiac electrophysiology.

In clinical practice, we perform planimetry of the aortic valve area (AVA) mainly in outpatients who are referred to coronary CT-angiography for suspected coronary artery disease and in whom previously unknown aortic valve calcification is present in order to select patients with aortic stenosis and with nonstenotic aortic valve sclerosis. The patients are then re-evaluated with TTE if aortic stenosis is identified. Therefore, a TTE examination before the CT-angiography in each patient would be logistically difficult to organize. Additionally, many patients show valve calcification, but only a minority presents with aortic stenosis. Coronary CT-angiography is currently being increasingly performed in our institution, but the use of CT as an alternative imaging modality, if TTE is inadequate, is extremely rare.

Finally, currently we are using 64-slice CT technology, which has improved planimetry of the AVA in terms of better image quality at higher heart rates and a reduction of the postprocessing time. We perform on average 4 to 8 cardiac CT scans daily, and in our experience, 12% of the cardiac cycle is an appropriate time point for CT image reconstruction in the majority of patients. However, the dynamics of the aortic valve have to be reviewed multiple time points throughout the systole and identified the maximal aortic valve opening. Considering a mean duration of the QRS complex of 80 ms (a QRS complex of 120 ms or longer is regarded as bundle branch block), we started the image reconstruction series 50 ms after the beginning of the QRS so as to begin early enough and not miss the valve opening. We would like to emphasize that Figure 1 in our report should illustrate the CT image reconstruction algorithm and not regular cardiac electrophysiology.

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REFERENCES


Percutaneous Laser Revascularization Does Not Equal Transmyocardial Laser Revascularization

The recent results of a trial of percutaneous laser revascularization (PMR) (1) are noteworthy for their confirmation that this technique does not appear valid; however, these findings cannot be extrapolated to other forms of laser therapy. Although Leon et al. (1) are to be congratulated for performing this study, they have also performed a significant disservice by equating results from a 3- or 4-mm subendocardial laser divot placed under remote control using an unapproved laser device with results documented in thousands of patients with a Federal Drug Administration-approved laser in which channels are placed under direct vision and treat the full thickness of the myocardium as is performed with surgical transmyocardial laser revascularization (TMR). The researchers dismissed the TMR results as being largely placebo because their results with PMR were equivalent with a placebo group. Whereas the placebo effect plays a role in any treatment, to dismiss objective data, including improved perfusion as noted by single-photon emission computed tomography (SPECT) and positron emission tomography (PET) scanning, and improved function as noted by echocardiography and magnetic resonance imaging (MRI) (seen with TMR), is wrong. Such improvements cannot be willed by the patient regardless of the strength of the placebo. The symptomatic benefits seen with TMR have lasted longer in more patients than has ever been seen from any placebo.

Furthermore, to equate surgical TMR with internal mammary artery ligation is unfair at best. In addition to severe angina, all of the TMR patients had angiographic evidence and objective demonstration of malperfusion. It is not known whether the patients who underwent mammary artery ligation even had coronary artery disease.

The primary conclusion that can be drawn from this study is that PMR does not work. To extrapolate that to other laser techniques is akin to stating that, like calcium channel blockers, beta-blockers are ineffectual after a myocardial infarction because both are "blockers."

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