Reply to the Editor:

We appreciate Ji and Undar's kind appraisal of our study on the benefits of pulsatile cardioplegia in failing hearts, because their input to the importance of pulsatile cardiopulmonary bypass is well recognized.¹ They propose that energy equivalent pressure (EEP) and surplus hemodynamic energy (SHE) should be reported when studying the effect of pulsatile flow, because the difference between EEP and mean arterial pressure (MAP) conveys the extra energy generated by each pulsatile wave. EEP and MAP are identical and the difference is zero when the pressure wave is perfectly harmonic.

Our study did not report those values,² because EEP and MAP were not significantly different for the waveforms used. A recent study by Huo and Kassab³ quantitatively demonstrated this point computationally, based on a full analysis of steady state versus pulsatile flow in the entire coronary arterial tree based on measured anatomical data and experimentally to validate the model. The inlet pressure waveform measured from a porcine model was input into an isolated heart to verify the model predictions and showed excellent agreement between the model and the experiment. Mean flow of both pulsatile and steady-state experiments and mathematical model were not statistically different and were consistent with normal heart microsphere measurements.² The EEP, MAP, and SHE were 75.38 mm Hg, 73.54 mm Hg and 1.84 mm Hg, respectively, providing only a 2.4% SHE/EEP ratio.

These calculations and this discussion reflect why our study did not focus on the EEP and SHE parameters. Rather, we believe that a different mechanism is at play because changes occurred only in failing hearts, because no significant changes were found between pulsatile and nonpulsatile cardioplegic flow existed in normal hearts. More importantly, vascular resistance rose in failing hearts in both the beating and cardioplegic states (with and without pulsation) such that factors responsible for initiating such pulsatility effect, such as altered number of vessels, lumen diameter, and vascular length, must be understood as ventricular geometry becomes expanded and spherical. This pulsatility dependency observation implies that dilated heart failure may have impaired coronary flow reserve capacity by limiting coronary autoregulation. This implication reinforces the

need to ensure increased perfusion pressure during myocardial protection strategies with the beating and pulsatile or nonpulsatile cardioplegia delivery.

Here, a method of pulsatile cardioplegia delivery was introduced to demonstrate the capacity to overcome this limitation of nonpulsatile cardioplegia in failing hearts. Our findings underscore the value of coronary pulsation, either by compression of vessels by the beating heart or by internal stretching by pulsatile cardioplegic flow. Our findings support the work of Undar relating to the significance of pulsatile flow in the use of cardioplegia for cardiac protection. Furthermore, our observations reinforce the importance of studying models that reflect clinical entities, such as heart failure, because the observed enhancement of subendocardial muscle perfusion would have gone undetected in studies of normal hearts.

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Note: The following *Reply to the Editor* refers to a letter to the editor published in a previous issue of the *Journal*: Kulik A, Rubens FD, Ruel M. Intraoperative indocyanine green angiography: Ready for prime time? *J Thorac Cardiovasc Surg* 2007;133:592-3.

Reply to the Editor:

We thank Dr. Kulik and colleagues for their interest in our prospective study comparing transit-time ultrasound flow (TTF) and indo-

cyanine green angiography (ICG).¹ We appreciate the opportunity to elaborate on the three issues they raise:

1. Methods to improve visualization with ICG angiography
2. Appropriate TTF criteria for graft assessment
3. Cost Effectiveness

Regarding the first issue, indocyanine green angiography is a method of fluorescent dye-contrast angiography that uses near infrared laser as the energy source. We have previously demonstrated poorer visualization of thick pedicled arterial grafts with this technique due to limited penetration of the laser into thicker tissues.² To improve visualization, we ensure that there is no pedicle covering the area of the anastomosis itself and we routinely expose the native vessel for approximately 1 cm beyond the anastomosis. We do not skeletonize the entire graft. Our evaluation of patency is based on both anatomic visualization of the anastomosis itself and the opacification characteristics (ie, TIMI flow) of the native circulation. Also, further experience with the technique has led us to perform selective angiograms directly into the grafts which allows for improved visualization of the distal anastomosis verses injections into the central venous line which can be confounded by native flow.²

Regarding the second issue, whereas criteria for pulsatility index (PI) and diastolic flow fraction (DFF) are standard, there is little agreement on cut-off values that distinguish between normal and abnormal mean flow values. Although most surgeons would agree that a flow of less than 5 ml/min is definitely abnormal and will often prompt revision, flows between 5 to 40 ml/min could potentially represent abnormal grafts. There is no clear consensus regarding cutoff values that determine graft problems and little prospective data on the subject. The study by Di Giammarco and colleagues, who recommend a 15 ml/min mean flow cutoff, was retrospective in design.³ Because patients who had follow-up angiograms were generally symptomatic, their study design could not accurately assess the false positive rate. The study by Walpoth and colleagues, who recommend a 20ml/min mean flow cutoff, had no angiographic controls to determine sensitivity or specificity.⁴ In our study, surgeons were permitted to intervene on any graft they deemed to be poorly functioning, even if it