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REPLY: Weil's Response

Dr. Burkhoff and colleagues raise concerns about the validity of our recent comparison of the dynamic hemodynamic effects of left ventricular (LV) support using the Impella CP (ICP) versus TandemHeart (TH) systems in swine with a myocardial infarction (1). In an attempt to reconcile the findings with model predictions, we graciously shared data that unfortunately have been misconstrued in an attempt to support alternative explanations for the findings. In response, we have carefully reviewed all of the data and make the following points. First, the published pressure-volume (PV) loops depict actual measurements recorded before and after percutaneous LV support. Second, the PV catheters were recalibrated immediately before all data collection, and the data also reflect averages of at least 400 cardiac cycles rather than from selected PV loops shown in the figures. Third, fluoroscopy was used to initially place each device and repeated to confirm positioning during support. Echocardiography was also used in some animals and confirmed the absence of aortic insufficiency. Appropriate ICP positioning was continuously confirmed without imaging using the differential pressure signal from the device controller. This will alarm the operator in a circumstance in which both the inlet and outlets ports are either above or below the aortic valve. We apologize for not explicitly stating this in the methods but assumed that it would be understood by most people familiar with using the ICP.

Dr. Burkhoff and colleagues and Dr. Møller-Helgestad and colleagues also question the lack of finding a triangular-shaped PV loop during support with the ICP. As demonstrated in the review by Burkhoff et al. review (2), triangulation develops when LV systolic pressure falls below the aortic pressure and the aortic valve remains closed (i.e., during full device support). In our study, animals were not in cardiogenic shock, arterial pressure was maintained, the aortic valve continued to open, and the isovolumic contraction and relaxation periods were short. Thus, we never saw nor expected a triangular PV pattern. Interestingly, our results with the ICP are identical to those of Remmelink et al. (3) in humans with normal arterial pressures following infarction as well as high-risk PCI. They also found no change in total cardiac output or stroke work because ICP support varied between 0 and 2.5 l/min. Their published PV relations did not demonstrate a triangular waveform. Interestingly, although Burkhoff and colleagues question how diastolic properties could change with LV support in our results, Remmelink et al. also demonstrated that a decrease in LV stiffness contributed to the reduction in LV end-diastolic pressure during ICP support.

Dr. Møller-Helgestad queries about how we calculated native cardiac output, which we neglected to detail in the methods. As they suggest, this was the product of heart rate times stroke volume derived from the LV PV catheter. The difference between this value and pulmonary blood flow (i.e., total cardiac output derived via thermodilution) represents the additional forward flow provided by the device. Similar to the human data without shock, our results confirm that reductions in LV diastolic loading occur in the absence of a significant increase in total cardiac output with each device.

At this point, it seems more productive to begin trying to understand why the in vivo behavior of the ICP in swine and humans with normal arterial pressure fails to be predicted by Burkhoff and colleagues' model. A likely explanation is failing to model the dynamic flow behavior of the ICP. As in all axial LV support devices, flow rates are extremely load-dependent. They vary throughout the cardiac cycle and are inversely related to the pressure gradient across the inlet and outlet ports. Thus, instantaneous flow rates are higher in systole than diastole. Because the model assumes that device flow is constant throughout the cardiac cycle, it overestimates the device flow contribution in diastole. The latter may be small when arterial pressure is normal and the aortic-LV diastolic pressure gradient is high. During systole when the aortic valve opens, systolic flow provided through the device may be high but would not become meaningful until the systolic flow rate through the device exceeded the intrinsic cardiac output. Similar considerations are germane to the TH although the relative constancy of left atrial pressure at the inlet may make these variations less pronounced throughout the cardiac cycle. Thus, although we strongly concur with the view that models are useful in helping to predict physiological behavior, they are only as good as their fundamental assumptions. When they do not predict measured results, they need to be revised. In this regard, it seems time to add the complexities of temporal flow variations due to load-dependent changes in device performance into the analyses of Dr. Burkhoff et al. (2) to understand dynamics when cardiac

output is not completely supported by a ventricular assist device.

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Impella Retrieval: Redux

We were pleased to read "First Experience With Successful Percutaneous Retrieval of Retained-Fractured Impella Device" in *JACC: Cardiovascular Interventions* (1). Because temporary mechanical circulatory support devices are used with increasing frequency, we are likely to continue experiencing device-related issues.

We reported on a remarkably similar case 2 months prior: "Impella 5.0 Fracture and Transcatheter Retrieval" (2). The location of device fracture is different, highlighting the potential areas of weakness that should be considered by those implanting these devices. The technique we described for retrieval of the retained device is also used in the current report (1), validating our process as appropriate for this clinical scenario.

With rapidly increasing use of new technology, we are bound to discover new challenges such as reported here. Creative thinking will lead to innovative solutions as highlighted in these papers.

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Please note: Dr. Pal is writing on behalf of Castigliano M. Bhamidipati, DO, PhD, Moses Mathur, MD, MS, Ravi S. Hira, MD, and James M. McCabe, MD. Dr. Pal is a consultant for HeartWare.

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Angiographic Follow-Up in Patients With Coronary Artery Disease

Is There a Window to Be Definitively Closed?

We read with great interest the paper of Shiomi et al. (1) about the impact of angiographic follow-up (AFU) in patients undergoing percutaneous coronary intervention (PCI). The authors have to be congratulated for their effort to clarify a quite controversial theme. The main conclusion is that after a median follow-up of 4.6 years, no benefit in terms of the composite primary outcome (death, stroke, rehospitalization for myocardial infarction [MI] or heart failure) emerged for the AFU group, whereas repeated revascularization rates were higher compared with the clinical/ischemia-driven group.

As already stated in the editorial by Puri et al. (2), many points must be argued to better interpret these results. First of all, the study was not sufficiently powered to show significant differences between the 2 groups. The incidence of target lesion revascularization (TLR) was very low, probably favored by newer-generation drug-eluting stents and a strong compliance to optimal medical therapy as usual in randomized controlled trials (RCTs). In this setting, follow-up (FU) duration and population size were