Letter to the editor regarding the results of the retrospective study "Predictors of intra-aortic balloon pump hemodynamic failure in non-acute myocardial infarction cardiogenic shock" published in the *American Heart Journal*

We read with interest the results of the retrospective study by Hsu et al[1] in which they reported that in a cohort of 74 patients undergoing intra-aortic balloon pump (IABP) insertion for cardiogenic shock (CS) complicating acute decompensated heart failure not due to acute myocardial infarction, a risk score using baseline left ventricle cardiac power index ≤ 0.28 W/m^2 and ischemic history predicted 28-day adverse events with excellent discrimination.

We do agree with the authors' observation that to characterize clinical and hemodynamic profile of patients undergoing IABP implantation is paramount to better define which group may have the benefit of IABP support and which will not. The authors conclude that, in this latter case, a closer observation and an earlier consideration of more advanced hemodynamic support are certainly pivotal.

As far as it concerns monitoring, pulmonary artery catheter provides information regarding fluid status and right heart filling pressures: filling pressures are a necessary but often overlooked component of pulmonary artery catheter measurements even though it is well known that these parameters are associated with clinical outcomes.

We have the following 3 observations to make and would seek the authors' reply regarding them:

1. The patients were included if they "…underwent IABP placement for treatment of acute CS, which was defined as a systolic blood pressure $\leq$ 90 mm Hg for at least 30 minutes with evidence of poor end-organ perfusion or need for inotropic support," but according to the baseline characteristics reported, not every patient was treated with inotropic support. What was the rationale to directly implant IABP before a trial with vasoactive agents?
2. The authors do not mention which inotropes/vasoconstrictors were used, at what dosage, and for how long before IABP implantation: could high-dose inotropes/vasoconstrictors have had an additional detrimental effect on patients who had adverse outcome? In fact, these latter patients had significantly higher systemic vascular resistances and lower CI; so clearly, their ventriculoarterial coupling was impaired. We reckon that, in this case, increasing the dose of inotropes is not of benefit, and quite often, we make this choice because we are led in our clinical practice by a fixation on raising SBP in response to organ hypoperfusion. In these cases, perhaps, a careful use of vasodilators could improve the recruitment of microcirculation and can promote decongestion.
3. Finally, we would like to stress that treatment goals in CS complicating acute decompensated heart failure should focus not only on improving cardiac function but also on reducing filling pressures, which can only reliably be achieved through invasive measurement: it would have been very relevant if the authors could have provided information pertaining to the trend of central venous pressure, pulmonary capillary wedge pressure, and mean pulmonary arterial pressure by adverse outcome status. It is important that clinicians appreciate and understand the physiologic meaning of these measurements and take them into account when treating patients who have CS.

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Reference

1. Hsu S, MD Kambhampati S, Sciortino CM, et al. Predictors of intra-aortic balloon pump hemodynamic failure in non-acute myocardial infarction cardiogenic shock. Am Heart J 2018;199:181-91.

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