

Arterial elastance reduction with intra aortic ballon counterpulsation is associated with hemodynamic stabilization in patients with acute decompensated heart failure and low output state.

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Background

Ventricular–arterial (V–A) decoupling decreases myocardial efficiency and is exacerbated by tachycardia and by increased total peripheral resistances (TPR) that worsen static arterial elastance (Ea); unlike aortic pressure by itself, this measure is independent of the function of the ventricle. Therefore, it is an index which describes arterial properties.

Intra aortic balloon pump (IABP) has been an essential tool to support hemodynamics in patients with cardiogenic shock following acute myocardial infarction. However, recent randomized control studies and meta-analyses do not show survival benefit of IABP use in this setting.

In contrast, studies evaluating the efficacy of IABP support in patients with acute decompensated heart failure (ADHF) are lacking, although small retrospective studies show that prolonged IABP supports elicit improved hemodynamics and peripheral organ function. We thus investigated whether Ea, a variable which can be obtained non-invasively, can predict hemodynamic stabilization with IABP therapy in patients affected by ADHF deteriorated in low output state.

Methods and Results

17 patients with ADHF deteriorated in low output state treated with IABP, after clinical worsening within 4 hours from the beginning of inotropic therapy, were enrolled in this prospective study. Patients were considered to have hemodynamic stabilization after implantation of IABP with at least 5/8 of the following: heart rate <130 and >60 bpm; mean arterial pressure >65 mm Hg; SVO₂ >60%; PaO₂ >60 mmHg; lactates decrease =25%; wedge pressure <18 or E/E' <14; diuresis >0.5 mL/kg/h; epinephrine dose max 0.12 µg/kg/min without upgrade of other inotropes/vasopressors.

Ea was calculated as mean arterial pressure (MAP)/stroke volume (SV). Left ventricle cardiac power index (CPI) (W/m²) was calculated as MAP x cardiac index/451. Data obtained at baseline and at 12-hour from IABP implantation were analyzed by within-patient repeated-measures analysis performed throughout the Wilcoxon signed-rank test.

IABP was associated with a decrease of the Ea median value (1.64, IQR 1.26-1.83, min 1.13 - max 4.11, vs. 1.28 IQR 1.04-1.51, min 0.64 - max 1.99 mmHg-1; p=0.04). We did not observe a significant change in HR (median 80, IQR 70-90 vs 80, IQR 73-94 bpm; p=0.809), whereas a parallel increase in CPI was detected (median 0.29, IQR 0.22-0.38 vs 0.40, IQR 0.37-0.51 W/m²; p=0.02).

Conclusions

IABP implantation decreased Ea in patients with ADHF deteriorated in low output state: the hemodynamic benefits were associated with an improved systemic perfusion.

As HR did not change after IABP, we can infer that the improvement of Ea is a consequence of reduction of TPR elicited by counterpulsation. Since Ea is a major determinant of V–A coupling, its reduction may contribute to improving cardiovascular efficiency in ADHF deteriorated in low output state.