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Use of Intra-Aortic Balloon Pump in Cardiogenic Shock Associated with Advanced Heart Failure: An Outdated Strategy?

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The use of an intra-aortic balloon pump (IABP) was first described in the 1960s.^{1,2} It is a short-term circulatory assist device that uses helium gas for inflation of a balloon positioned in the descending aorta during diastole and active deflation during systole.¹ The most evident hemodynamic effects are increased coronary perfusion, reduced left ventricular afterload, and an increase in cardiac output by 0.5 to 1 L/min.^{1,3} Due to its greater availability, lower cost, easy implantation, and low complication rates, IABP quickly became the most used percutaneous device in cardiogenic shock.² Nevertheless, in spite of the advantages described and the extensive clinical experience, there are still some controversies in relation to its use.

The randomized IABP-SHOCK II Trial evaluated the use of IABP versus clinical treatment in patients with post-acute myocardial infarction (AMI) cardiogenic shock.⁴ In this study, the use of IABP did not reduce the primary endpoint of 30-day mortality or the relevant secondary outcomes,⁴ and, from then on, the use of IABP in post-AMI cardiogenic shock started to be discouraged by guidelines, with class III, level of evidence B for routine use (not recommended).^{5,6} It is, however, noteworthy that other devices that provide greater hemodynamic support compared to IABP have also not shown a benefit in increasing survival in post-AMI cardiogenic shock.⁷

We know that most of the evidence on cardiogenic shock comes from studies in patients in the context of acute coronary syndromes.² Nevertheless, cardiogenic shock has more recently been recognized as a heterogeneous clinical syndrome, with a broad spectrum of clinical phenotypes and different stages of organic dysfunction.^{2,8,9} Accordingly, we can differentiate, for example, post-AMI cardiogenic shock from shock due to chronic heart failure (HF) decompensation. In the first case, an abrupt reduction occurs in cardiac output with rapid onset, generally in patients without previous ventricular dysfunction. In the

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second, a gradual reduction occurs in cardiac output in patients who already have ventricular dysfunction, which is often severe. Therefore, an incremental support of 0.5 to 1 L/min may be insufficient to stabilize a patient with post-AMI cardiogenic shock; however, it may be sufficient to stabilize a patient with chronic HF, who has already adapted to hemodynamic conditions with borderline cardiac output. From the pathophysiological point of view, we know that the inflammatory component is predominant in post-AMI cardiogenic shock, whereas, in chronic HF, peripheral vasoconstriction is predominant.¹⁰ This difference corroborates the benefit of the effect of the reduced ventricular afterload of IABP in cardiogenic shock associated with decompensated chronic HF.

In patients with advanced HF, the outcome of reduced mortality is unlikely to be achieved with temporary ventricular assist devices (VAD). Therefore, the main objective is stabilization until definitive treatment, especially heart transplantation or implantation of longterm VAD. Accordingly, a series of studies has demonstrated the feasibility of using IABP as a bridge to transplantation or long-term VAD implantation.^{2,3,11} In a single-center, observational, retrospective study, Fried et al. evaluated 132 patients with cardiogenic shock associated with chronic HF who received aortic counterpulsation therapy.3 The 30-day survival was 84.1%, of which 70.4% underwent long-term VAD implantation, 8.2% underwent heart transplantation, and 21.4% were discharged without need for escalation of device support.3 In another prospective observational study, conducted in Brazil, metabolic and hemodynamic variables were evaluated before and after IABP implantation in 223 patients.¹¹ After institution of aortic counterpulsation therapy, there was a reduction in serum lactate (32.9 versus 17.1 mg/dL, p < 0.01); increased central venous saturation (50.6% versus 66%, p < 0.01), and reduced vasopressor use (36.2% versus 25.5%, p = 0.0036). In a recent case series from the Heart Institute of the University of São Paulo, 90% of patients were transplanted under priority status, and 50% of them were using IABP.¹² Similarly, in the United States, after the change in the organ allocation policy that prioritizes patients using short-term VAD, the use of IABP as a bridge to transplantation significantly increased, from 7% to 24.9%.11 As a result, in patients using IABP, there was a decrease in waiting time and an increase in the probability of receiving a heart transplant.13

Although initially described in the 1960s, the use of IABP has recently been revisited in other scenarios and in different forms of use. 14-17 One of them is the use of IABP as an initial strategy for decompression of

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left heart chambers after the institution of peripheral venoarterial extracorporeal membrane oxygenation. In a meta-analysis, decompression strategies were related to greater success in weaning from extracorporeal membrane oxygenation, and the device that was most used for this purpose was IABP.¹6 Techniques for implanting IABP via the subclavian or axillary artery have also sparked interest, enabling mobilization out of bed and preventing physical deconditioning and frailty.²¹¹5

In conclusion, we believe that studies conducted in the context of acute coronary syndromes are inappropriate for evaluating the use of IABP in advanced HF. Furthermore, a series of studies has demonstrated its efficacy in this profile. In the current scenario, IABP should not be seen as an outdated strategy in advanced HF, but rather as a contemporary one with an impact on improved clinical and hemodynamic parameters, ventricular decompression associated with the use of peripheral venoarterial extracorporeal membrane oxygenation, and as a bridge to transplantation or long-term VAD.

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