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IABP: The Show Goes on but All that Glitters is not Gold

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Despite remarkable improvements in early diagnosis and treatment of acute myocardial infarction (AMI), cardiogenic shock is still the most common cause of in hospital mortality and morbidity associated with AMI, which occurs in about 7% of these patients [1].

The underlying mechanism in cardiogenic shock is depression of myocardial contractility due to extensive MI, leading to a vicious cycle of reduced cardiac output, low blood pressure, decreased coronary blood flow, and ongoing lowering in contractility and cardiac output. Despite compensatory mechanisms such as peripheral vasoconstriction and redistribution of circulation to the vital organs, this leads to multiple organ failure. The principal concept behind mechanical cardiac assistance in cardiogenic shock is to support the compromised circulation [2].

The intra-aortic balloon pump (IABP) has long been widely recognized as the most promising and effective mechanical haemodynamic support device [3]. The IABP is a catheter-mounted balloon positioned in the descending aorta, usually through a percutaneous femoral approach. Counterpulsation is achieved by rapid inflation in the diastole and deflation in systole of the balloon synchronised to the cardiac cycle.

The counterpulsation exerts its beneficial effects mainly through two synergistic actions: increasing diastolic blood pressure, that improves coronary blood flow, and afterload lowering which reduces left ventricle workload, and thus myocardial oxygen consumption. In patients with impaired left ventricular function and cardiogenic shock, the IABP, through the ischemic reduction and improving cardiac function, theoretically results in a better systemic perfusion and renal function. The final effect is also a reduction of pulmonary congestion and metabolic acidosis. To date, IABP is mainly used in high-risk patients with acute myocardial infarction, especially when complicated by cardiogenic shock.

Further, prophylactic IABP therapy is frequently performed in patients at high risk for hemodynamic instability undergoing elective PCI or coronary artery bypass grafting (CABG).

However, here is a time at which it becomes clear that a situation or process will end, although it does not end immediately. Recent data from American and European Guidelines for the management of ST-elevation myocardial infarction, have strictly reconsidered IABP use for cardiogenic shock downgrading it from a class Ib and Ic ("should be used"), respectively to a class IIa and IIb ("may/can be used") recommendation [4,5].

The most recent and largest study about IABP is the prospective, randomized, open-label, controlled IABP-SHOCK II trial which includes 37 German centers in whom patients with cardiogenic shock complicating, treated with early revascularization and optimal medical therapy, were randomly assigned to IABP group versus control one. Preliminary data from IABP-SHOCK II showed no survival benefit in the short-term follow-up demonstrating no significant differences in terms of 30-days all-cause mortality between the two groups [6]. The newer data in the 6 and 12-months follow-up, clinical outcome and quality of life were investigated. The results confirmed the absence of significant differences in terms of mortality between IABP and control group at 6 and 12 months (48.7% vs 49.2%, p=0.91 and 51.8% vs 51.4%, p=0.91 respectively) as well in terms of reinfarction, requirement for internal cardioverter defibrillator (ICD), stroke and additional revascularization procedures at 12 months follow-up. For survivors, the functional status and the quality of life (QoL) assessment did not differ between the IABP group and control one showing a moderate to good QoL index value, a NYHA class I or II (91% vs 94% p=0.36) and CCS class I or II (98% vs 99%, p=1.00) in both groups. The investigators speculate on possible explanations for the absence of significant short- and long-term benefits and they state that the effect of the IABP on cardiac output is only marginal with an improvement in cardiac output of 0.5 L/min. Furthermore, although previous trials have shown haemodynamic improvements with IABP, the absence of the control group might represent a possible bias on the evaluation of clinical follow-up [7].

These data do not support the routine use of IABP in patients with acute myocardial infarction with or without cardiogenic shock or patients undergoing high-risk elective PCI. In high-risk patients undergoing CABG, prophylactic use of IABP may be considered, although further large scale multi-center trials are needed to confirm previous marginal data. Other indications such as postcardiotomy cardiogenic shock or mechanical complications in acute myocardial infarction, in which IABP could be useful, need to be identified and require further investigations.

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