

The Intra-aortic Balloon Pump in ICCU

The intra-aortic balloon pump (IABP) is the most common and widely available method of mechanical circulatory support. The concept of diastolic augmentation and systolic unloading was introduced by Clauss et al. more than four decades ago and first introduced in clinical practice by Kantrowitz in 1968 for the treatment of cardiogenic shock (1). During balloon inflation, diastolic pressure rises and it is possible to augment systemic perfusion and coronary blood flow in a previously hypoperfused patient. By means of balloon deflation, it is possible to decrease afterload, increase cardiac output, and reduce myocardial O₂ demand. However, the effect of IABP depends on the initial hemodynamic state. Across a wide range of perfusion pressure, coronary blood flow (CBF) remains constant because of interaction between perfusion pressure and vascular tone. If intravascular pressure increases, the vascular tone increases too, and CBF remains constant. In this situation, augmentation of perfusion pressure by IABP would not increase CBF. Moreover, in healthy individuals IABP may even decrease CBF because of a decrease of myocardial O₂ consumption. In situations of extremely low perfusion pressure, coronary arteries reach maximum dilation and the effect of autoregulation is lost. In such a state of hypoperfusion, IABP can really increase myocardial blood supply. A similar situation arises in myocardial ischemia. In the ischemic zone, coronary arteries are maximally dilated because of metabolic agents, and the autoregulation reaction is lost. CBF in this area becomes proportional to perfusion pressure, and IABP can significantly increase CBF in ischemic areas. IABP cannot improve CBF distal to severe fixed coronary stenosis. In patients with critical coronary stenosis, IABP does not augment peak diastolic velocity in the impaired artery before PCI, but it increases it immediately after the procedure (2). So IABP has different effects on CBF: it can decrease it in healthy individuals, increase it in hypoperfusive states, leave it unchanged in cases of severe fixed coronary stenosis, and increase it in the ischemic myocardium after successful PCI. It is not surprising that the results of clinical applications of IABP are contradictory.

The main indication for IABP in CCU is cardiogenic shock (CS). According to the SHOCK Registry, IABP decreases mortality in patients with CS (3). But if we consider the rate of revascularization in various groups we can see that revascularization was performed in nearly 70% of patients with IABP and in only 20% of those in groups without IABP. Among shock patients in the GUSTO-1 trial (4), those in the IABP group had lower mortality, but the use of inotropic drugs, pacemakers, Swan-Ganz catheters, and revascularization was statistically higher in this group. IABP in cases listed in the NRMI register (5) decreased mortality in patients who received thrombolysis but had no effects in patients undergoing

PCI. Even a 6% increase of mortality was evaluated in a recent meta-analysis of non-randomized trials on IABP in STEMI patients with CS and primary PCI (6). So we can conclude that in CS patients IABP often promotes hemodynamic improvement, but it remains uncertain whether it leads to reduced mortality.

In haemodynamically stable patients with severe unstable angina, the main antiischemic effect of IABP is due to a reduction of myocardial O₂ consumption. There have been only two small observational studies in which good symptomatic effects were reported, but of course IABP in such patients can be used only as a supportive measure before revascularization. The use of IABP in STEMI patients with high-risk primary PCI did not influence the risk of death, reinfarction, or heart failure (7).

In conclusion, cardiogenic shock is the main indication for IABP in ICCU, but recommendations for its use are made mainly on the basis of expert consensus.

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