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REVIEW

Is there still a role for the intra-aortic balloon pump in the management of cardiogenic shock following acute coronary syndrome?

Le ballon de contre pulsion intra-aortique a-t-il encore un intérêt dans la prise en charge du choc cardiogénique ?

Guillaume Leurent[a,*], **Vincent Auffret**[a], **Camille Pichard**[a], **Marc Laine**[b], **Laurent Bonello**[b]

[a] *Université de Rennes 1, Department of Cardiology, CHU de Rennes, Inserm, UMR 1099 LTSI, 35000 Rennes, France*

[b] *Aix-Marseille Université, AP—HM, Mediterranean Association for Research and Studies in Cardiology (MARS Cardio), Centre for CardioVascular and Nutrition research (C2VN), Inserm 1263, INRA 1260, Cardiology Department, Hôpital Nord, 13000 Marseille, France*

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Summary The intra-aortic balloon pump has been widely used in the management of cardiogenic shock. Reducing cardiac afterload and myocardial oxygen consumption, and improving coronary blood flow, this safe and simple mechanical circulatory support has been considered the cornerstone of cardiogenic shock management for decades. However, because it failed to provide any clinical benefit in recent randomized trials, the latest guidelines discourage its routine use in this clinical setting. Moreover, new percutaneous circulatory supports providing greater haemodynamic improvement have recently been developed. Thus, intra-aortic balloon pump use has declined considerably in this clinical setting. However, the device does retain a minor role in cardiogenic shock management — mainly in the setting of mechanical complication of acute coronary syndrome, and for left ventricular unloading in patients treated with extracorporeal life support.

Abbreviations: CS, cardiogenic shock; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; LV, left ventricular; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction.

* Corresponding author. Service de Cardiologie, CHU de Rennes, 2, rue Henri-Le-Guilloux, 35033 Rennes cedex, France.
E-mail address: guillaume.leurent@chu-rennes.fr (G. Leurent).

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MOTS CLÉS
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Décharge ventriculaire gauche ;
Flux coronaire

Résumé La contre pulsion par ballonnet intra-aortique (CPBIA) a été largement utilisée dans la prise en charge du choc cardiogénique (CC). Parce qu'elle réduit la post-charge, améliore le débit coronaire et diminue les besoins myocardiques en oxygène, cette assistance mécanique circulatoire simple d'utilisation et sûre a longtemps été considérée comme la pierre angulaire de la gestion du CC pendant des décennies. Cependant, face à l'absence de bénéfice clinique associé à son utilisation dans de récents essais randomisés, les dernières recommandations ne préconisent plus son utilisation systématique dans ce contexte. De plus, des supports circulatoires alternatifs plus élaborés se sont récemment développés. Pour ces raisons, les implantations de CPBIA ont fortement chuté dans cette indication. Cependant, elle garde un rôle modeste à jouer dans la gestion du CC, notamment en cas de complication mécanique du syndrome coronaire aigu, ou à visée de décharge du ventricule gauche des patients traités par circulation extracorporelle de type ECLS.

Presentation of the device

The intra-aortic balloon pump (IABP) was first described 50 years ago [1] in the management of cardiogenic shock (CS) [2]. The device is made of a balloon, which is inserted through the femoral artery (7—8 French sheath) and placed in the descending aorta, between the subclavian artery and the renal arteries, under X-ray guidance. The balloon is linked to a mobile management console, which drives the helium inflation and deflation of the balloon in synchrony with the cardiac cycle, triggered by the electrocardiogram signal or the systemic arterial waveform. Although the ''automatic'' mode is widely used, several variables can be modified (''semiautomatic'' or ''manual'' modes) — mostly the volume and duration of the inflation, and the trigger for the inflation of the balloon, according to the haemodynamic setting. Lastly, the balloon inflation/deflation rate can be settled (1 out of 1, 2 or 3 cardiac cycles). The main contraindications for the device are severe peripheral arterial disease precluding insertion of the balloon, more-than-moderate aortic regurgitation and severe dynamic left ventricular (LV) outflow tract obstruction.

Inflation occurs immediately after aortic valve closure at the onset of diastole. As a result of this diastolic inflation, the IABP increases the diastolic pressure upstream of the balloon, especially in the coronary arteries, to levels above that of systolic pressure (Fig. 1). In physiological conditions, autoregulation of the vasomotor tone in the coronary arteries and arterioles allows optimization of the coronary blood flow. Therefore, the coronary blood flow becomes directly dependent on perfusion pressure. However, in the setting of CS this autoregulation is ineffective, especially when the mean aortic pressure drops below 60 mmHg [3]. Moreover, coronary vascular tone autoregulation is also ineffective in the setting of myocardial infarction [4,5]. As myocardial perfusion occurs during diastole, coronary artery blood flow is supposed to be improved by the IABP, which should be beneficial during CS secondary to an acute coronary syndrome. In addition to this first benefit of IABP, it is also associated with a reduction of LV afterload. As balloon deflation occurs immediately before aortic valve opening, at the onset of systole, it reduces aortic volume by 40—50 mL. Consequently, the device reduces LV systolic and end-diastolic pressures and volumes, central venous and pulmonary wedge pressures and myocardial oxygen consumption [6]. Finally, the device increases cardiac output slightly, by approximately 0.5 L/min [2,7]. Given these combined haemodynamic properties, IABP should, in theory, be beneficial in the management of CS.

Nonetheless, the physiopathology of CS is complex [8], with varying definitions according to different trials and registries [9]. Apart from the systemic inflammatory response syndrome, it combines systolic and diastolic dysfunctions, resulting in pulmonary congestion, a decrease in cardiac output and end-organ malperfusion. Because of the adaptive tachycardia, the myocardial oxygen demand increases, thus creating a vicious circle. The hypoxaemia and increased cardiac work worsen the ischaemic initial trigger in the setting of CS secondary to an acute coronary syndrome [10]. Thus, the use of an IABP in this particular setting seemed attractive, to stop this vicious circle through its LV unloading/anti-ischaemic properties combined with a slight improvement in cardiac output. There was every reason to believe that this device would improve the poor prognosis of patients with CS. Despite animal experiments showing that IABP is effective at reducing the no-reflow phenomenon and improving myocardial salvage [4], the clinical benefits of the device in humans are still a matter of debate.

Data from registries and non-IABP-specific randomized trials

Despite the theoretical benefits, data from registries are inconsistent, with variable results according to the type of revascularization. Observational data from the fibrinolysis era strongly advocated for wide utilization of the IABP. Among 310 patients with CS included in the Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries (GUSTO-1) trial, the 1-year all-cause mortality was 57% in the IABP group versus 67% in the ''no IABP'' or ''late IABP implantation'' group ($P = 0.04$), despite a significantly higher haemorrhagic risk in the IABP group [11]. However, this study was observational, as the trial did not randomize patients between IABP and medical therapy. Similarly,

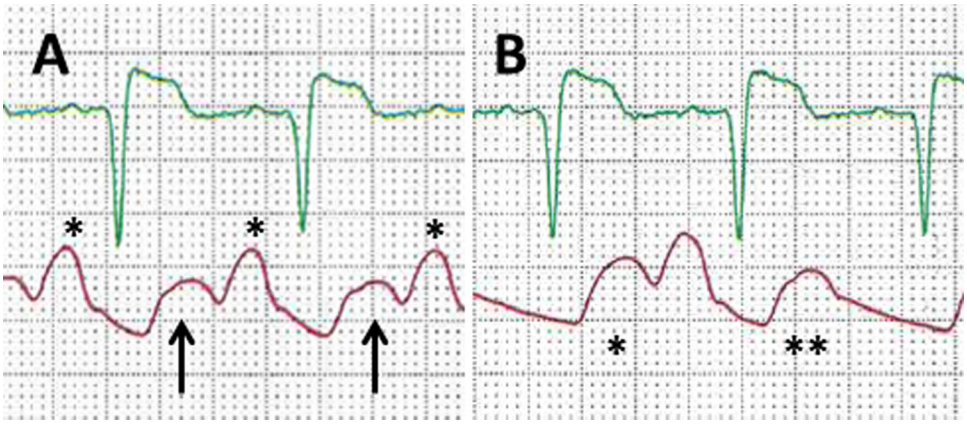

Figure 1. Modification of arterial pressure by the intra-aortic balloon pump, settled on a 1/1 mode (panel A) or 1/2 mode (panel B). A. The diastolic inflation of the balloon increases the diastolic pressure; as a result, the assisted diastolic pressure (*) becomes greater than the assisted systolic pressure (arrow). B. Because of the reduction in left ventricular pressures, the assisted systolic pressure peak (**) is lower than the unassisted systolic pressure peak (*).

in the SHould we emergently revascularize Occluded Coronaries for cardiogenic shocK? (SHOCK) registry, intrahospital mortality was significantly lower in IABP-treated patients (50% versus 72%; $P < 0.0001$), but this benefit was mainly the result of the revascularization of these patients [12]. Last, an analysis from the National Registry of Myocardial Infarction (NRMI) of about 23,180 patients with ST-segment elevation myocardial infarction (STEMI)-related CS showed that an IABP was used in 31% of patients, and was associated with a mortality reduction in patients treated by fibrinolysis, but not in patients treated by primary angioplasty [13].

More recent registries, with modern management of myocardial infarction, including systematic and prompt reperfusion by primary angioplasty, did not report any benefits of the IABP. Thus, in the Euro Heart Survey on percutaneous coronary intervention (PCI) [14], 25% of the 653 patients with CS were treated with an IABP. In-hospital mortality rates with and without an IABP were 56.9% and 36.1%, respectively, and the use of an IABP was not associated with improved survival (odds ratio: 1.47, 95% confidence interval: 0.97—2.21; $P = 0.07$) according to the multivariable analysis.

Data from randomized studies

For 40 years, the only trials assessing IABP on top of optimal management of ischaemic CS (including systematic reperfusion) randomized small populations and displayed divergent results [7,15]. In 2012, the publication of the Intra-Aortic Balloon Pump in Cardiogenic Shock II (IABP-SHOCK II) trial [16] changed this situation significantly. In this multicentre trial, 600 patients with ischaemic CS (including 69% with STEMI) were randomized prospectively to receive or not to receive an IABP on top of optimal medical therapy, including reperfusion therapy. The definition of CS was the combination of systolic blood pressure ≤ 90 mmHg for > 30 minutes (or infusion of catecholamine needed to maintain systolic pressure > 90 mmHg) with clinical signs of pulmonary congestion and impairment of end-organ perfusion (altered mental status or cold clammy skin and extremities or oliguria or serum lactate > 2.0 mmol/L). The main exclusion criteria were resuscitation > 30 minutes, onset of CS > 12 hours before screening and mechanical causes of CS.

The trial was neutral for its primary endpoint (all-cause mortality at 30 days): 39.7% in the IABP group versus 41.3% in the control group ($P = 0.69$). All secondary endpoints (e.g. in-hospital reinfarction or stent thrombosis, serum lactate concentration, renal function or length of stay in the intensive care unit) were also similar between groups. However, the safety endpoints were reassuring, with no significant difference between the two groups with respect to the rates of stroke, bleeding, sepsis or peripheral ischaemic complications requiring intervention in the hospital. Meta-analysis of the published randomized trials assessing IABPs in ischaemic CS, albeit largely driven by IABP-SHOCK II, reached similar conclusions [17,18].

Moreover, these negative results were confirmed by the 1-year [19] and long-term follow-up [20] of the IABP-SHOCK II trial: relative risk of mortality at 6.2 years was 66.3% in the IABP group versus 67.0% in the control group ($P = 0.98$), without any significant differences in recurrent myocardial infarction, stroke, repeat revascularization, rehospitalization for cardiac reasons or survivors' quality of life.

Because the IABP-SHOCK II trial is one of the most important in the field of CS, its main limitations should be underlined:

- it was an open-label trial, with the usual bias of an unblinded study;
- a substantial and asymmetrical number of crossovers were noted, as 30 patients (10%) from the control group subsequently underwent insertion of an IABP (even if the per protocol analysis was consistent with the intent-to-treat analysis, it is likely that the patients in whom crossover occurred were those in the worst condition, i.e. at highest risk of events);
- 270 patients (45%) had resuscitation before randomization, and 226 patients (37%) were treated by therapeutic hypothermia.

Authors did not report the cause of death in the trial, but cerebral damage is the main cause of death after cardiac arrest, without any potential benefit from an IABP in this setting. Moreover, it is known that haemodynamic compromise

after cardiac arrest is mainly caused by functional hypovolaemia (because of a deep vasoplegia in the setting of systemic inflammatory response syndrome) [21]. Therefore, extensive filling is generally a more useful therapy than mechanical haemodynamic support in patients with CS after a cardiac arrest. Unfortunately, invasive monitoring was not requested in the trial, and data on filling pressure were not reported. This post cardiac arrest setting, present in a large proportion of patients who were randomized, may explain the neutral results of the IABP-SHOCK II trial.

Contemporary data

Following publication of the IABP-SHOCK II trial, guidelines downgraded IABP recommendation in the management of CS to Class 3, suggesting that its routine use may be harmful [22,23]. However, it is still widely implanted in the setting of CS. In 2015, in the multicentre prospective French CP-GARO registry, 62% out of 175 IABPs were implanted for CS [24]. In the USA, the absolute number of IABPs implanted in patients with CS increased from 3149 in 2004 to 4225 in 2014, despite the guidelines and the fact that other mechanical supports were promoted [25].

The IABP-SHOCK II trial showed the futility of IABPs in the setting of ischaemic CS without mechanical complication. However, it seems that many cases of CS do not fit within this framework, depending on the inclusion criteria of the study: 27% of the 219 cases of CS included in the CARDSHOCK registry did not match the IABP-SHOCK II criteria [26]. Moreover, the recent FRENSHOCK registry prospectively included 772 patients with CS: only 36% of cases were ischaemic in nature, and 8% had mechanical complications. Thus, 516 patients (66% of the total population) would have been excluded from the trial [27]. As a matter of fact, the rate of ischaemic CS has decreased significantly over the last decade, from 65% in 2005 to 45% in 2014, according to the USA Nationwide Inpatient Sample databases [28].

Data on IABPs in non-ischaemic CS are scarce. An analysis of 489 consecutive patients treated with an IABP for CS showed that the CS aetiology had no effect on mortality [29]. Also, another recent IABP registry of 193 patients (including 59 [30.6%] with non-ischaemic CS) reported no difference in the 30-day mortality rate between the ischaemic and non-ischaemic CS groups (34.3% vs. 40.7%; $P = 0.39$) [30]. However, to date, there is no recommendation or robust data supporting the wide use of IABP in the setting of non-ischaemic CS.

Finally, analysis of IABP-related trials and registries is reassuring regarding the safety of the device: the rate of direct IABP-related complications (mostly peripheral ischaemia) was 4% in both the CP-GARO registry [24] and the IABP-SHOCK II trial [16], with a median duration of support of 41 hours and 3 days, respectively. Other potential complications of the balloon, such as renal impairment, are very difficult to assess, as they can also be caused by the CS itself. Accordingly, there were no differences in renal function, peripheral ischaemia, bleedings or ischaemic or haemorrhagic strokes between the two patient groups in the IABP-SHOCK II trial, demonstrating the safety of the device [16].

Residual potential applications of the IABP

Mechanical complications

As mentioned above, patients with mechanical complications (mostly ventricular septal defect or papillary muscle rupture) were excluded from the IABP-SHOCK II trial. Moreover, the occurrence of mechanical complications after randomization was the only condition for crossover of patients from the control group to the IABP group [16]. Mechanical complications have become rare since the STEMI systematic revascularization strategy era. However, they are still associated with high mortality rates, and only correction (surgical or percutaneous) allows survival. However, surgery may carry an extremely high-risk, especially in the acute setting, because of the friable nature of the infarcted myocardium. Thus, postponing the surgical correction for a few days (or weeks) is sometimes proposed. The cornerstone of medical management of mechanical complications is afterload reduction, to increase effective LV stroke volume by reducing left-to-right shunting or mitral regurgitant volume. In this setting, an IABP may be considered routine care, even in patients who remain haemodynamically stable [31]. According to the European guidelines, an IABP should be considered in patients with CS arising from mechanical complications [23].

In the STS database regarding surgical repair of ventricular septal defect after myocardial infarction, 65% of the 2876 patients were supported preoperatively with an IABP, and another 8% had a balloon placed during surgery [32]. In a retrospective single-centre registry regarding surgery for papillary muscle rupture after myocardial infarction, 21 of 48 patients (44%) received an IABP [33]. In those two reports, preoperative IABP was significantly associated with higher intrahospital mortality, but it is likely that the most severe patients were preferably implanted, with an obvious selection bias. In another retrospective analysis of 81 mechanical complications after myocardial infarction, IABP support reduced 30-day mortality in patients with CS (61% vs. 100%; $P = 0.04$), but not in patients without CS (20% vs. 27%; $P = 0.7$) [34].

It must be acknowledged that data on the usefulness of IABP in mechanical complications are scarce and are mainly derived from retrospective studies. On the other hand, several appealing reports advocate for a strategy of full mechanical support (e.g. an Impella® device [Abiomed, Danvers, MA, USA] or extracorporeal membrane oxygenation [ECMO]) to allow for planned or delayed surgery. In a single-centre retrospective analysis, veno-arterial ECMO support in postinfarction ventricular septal rupture complicated by CS was feasible, and was associated with a lower rate of multiorgan failure. However, this approach was strongly limited by bleeding complications [35].

LV unloading during ECMO

Over the last decade, the use of veno-arterial ECMO for CS has increased significantly [25]. Despite the absence of randomized data on its benefits, many position papers or guidelines have selected this circulatory assistance as the cornerstone of management for the most severe patients [22,36]. However, because of the retrograde flow in the

aorta towards the left ventricle, ECMO greatly increases LV afterload and, subsequently, pulmonary wedge pressure, especially in low residual LV ejection fraction. In order to avoid pulmonary oedema, which is associated with the worst prognosis, several options are proposed for LV unloading [37]: indirect venting through an IABP; or direct venting by central configuration of ECMO, an Impella® device or atrial septostomy. To date, there are no randomized data about the most efficient strategy for LV unloading. However, because of their ease of use, inexpensive nature and bedside availability, IABPs are widely used in this setting [38,39]. Furthermore, it has been suggested that the pseudopulsatile blood flow created by the balloon might improve regional microcirculation [40]. A prospective single-centre observational study extensively analysed the haemodynamic effect of an IABP on top of veno-arterial ECMO: while microcirculation variables were not affected by pumping, the balloon significantly decreased pulmonary wedge pressure and decreased LV end-systolic and end-diastolic diameters. Interestingly, the biggest benefit was reported in patients with an initial pulmonary wedge pressure > 15 mmHg [41].

However, in a retrospective analysis of 799 patients, 302 of whom received combined ECMO and IABP treatment, and 227 of whom received ECMO support only, IABP implantation did not improve patient outcomes (14-day mortality rate: 47.7% vs. 48.5% for patients treated by ECMO only), despite a significant increase in systolic blood pressure with IABP implantation (107.6 vs. 99.5 mmHg) [42].

High-risk PCI

The use of mechanical circulatory support increases in the setting of high-risk PCI [43]. As its elective insertion was associated with a significant reduction in long-term mortality in a randomized trial [44], IABP is sometimes used before complex PCI. However, given that it does not concern patients with CS, this topic will not be discussed further.

Preshock

It is now admitted that, in clinical practice, there is a spectrum of presentation for CS, from preshock to refractory severe shock. The ''refractory'' shock (so-called ''crash and burn'') is the worst presentation. However, a significant number of patients first present a state of ''mild'' or ''pre'' shock, i.e. arterial hypotension without tissue hypoperfusion [45]. In this situation, an IABP, with its limited yet beneficial haemodynamic properties, may have the potential to reduce clinical deterioration. However, this has not been investigated, as 89% of the IABP-SHOCK II patients received catecholamine at randomization, and the median serum lactate concentration was 4.1 mmol/L, underlying the severity of the CS of those patients. One wonders whether an IABP would be more clinically relevant as first-line haemodynamic support in patients in preshock, before catecholamine use. Indeed, these drugs, which increase myocardial oxygen consumption and have an arrhythmogenic effect, may actually worsen the prognosis of a patient with CS [46].

A recent publication provided a risk score to accurately identify patients at highest risk of developing CS at the acute phase of STEMI [47]. Akin to patients with preshock, these patients may derive benefits from the increase in their myocardial blood supply, along with the slight haemodynamic improvement allowed by an IABP, rather than the real haemodynamic support provided by more elaborate devices, which have significant complication rates [48]. The so-called ''ORBI Risk Score'' may help to include patients with preshock in prospective randomized trials, investigating the hypothesis that IABPs may prevent adverse outcomes.

Likewise, in the registry mentioned previously of 193 patients with CS treated by an IABP, 30-day mortality was 24% when the IABP was implanted within 1 hour of CS onset versus 49% when implanted at least 1 hour after recognition of CS ($P = 0.001$). Thus, time to IABP implantation predicted mortality, according to the multivariable analysis, suggesting that early use may be associated with a survival benefit [30].

Furthermore, the Counterpulsation Reduces Infarct Size Pre-PCI for Acute Myocardial Infarction (CRISP-AMI) trial failed to show any reduction in final infarct size in patients with anterior wall STEMI without CS [49]. However, a small substudy of this trial focused on patients with ''large'' ischaemic area (summed ST deviation ≥ 15 mm) and poor ST deviation resolution despite optimal revascularization. In this subgroup ($n = 36$), the 6-month mortality rate was 24% in control group versus 0% in the IABP group ($P = 0.046$) [50].

Regardless of the clinical setting, the respective place of the IABP in relation to new percutaneous circulatory supports, providing greater haemodynamic improvement, but associated with higher complication rates, should be addressed [22,51].

Conclusions

Despite their theoretical benefits, data from randomized studies do not support the routine use of IABPs in the setting of CS. However, they are still widely used in clinical practice when no other haemodynamic support is available. Indeed, the clinical settings of many cases of CS were not investigated in clinical trials. Furthermore, potential IABP benefits in patients who are less severely haemodynamically compromised should be evaluated in randomized controlled trials.

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