Commentary on:
Balloon-Pump Counterpulsation for Management of Severe Cardiac Dysfunction After Aneurysmal Subarachnoid Hemorrhage
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Intra-Aortic Balloon Pump Counterpulsation in Aneurysmal Subarachnoid Hemorrhage

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Intra-aortic balloon pump (IABP) counterpulsation was first developed by a group led by Kantrowitz in the early 1960s (21). The first clinical human use of the device and early clinical research by the same group were performed at Maimonides Medical center in 1967 (12, 28). The IABP consists of 2 parts: an extracorporeal electronically controlled pump containing a solenoid valve, and an intracorporeal Teflon catheter with a flexible polyurethane chamber at one end. This chamber is 10 to 17cm in length and 1 to 2 cm in diameter. The balloon catheter is placed using the Seldinger technique into the femoral artery such that the end of the balloon is located approximately 2 cm distal to the origin of the left subclavian artery. Helium is used to inflate the chamber in a sequence determined by the extracorporeal pump. This pump may be triggered by a manometer within the catheter or by the electrocardiogram. The low density of helium allows for rapid instillation of the gas into the chamber, and a lower risk of embolism should the balloon leak or rupture. The pump is timed in counterpulsation, that is, the pump is deflated during systole and inflated during diastole. Inflation occurs just after the aortic valve closes, timed to the dicrotic notch of the arterial waveform, and displaces blood that would normally remain in the aorta, thereby improving flow to the coronary arteries, the carotid and vertebral arteries, and the peripheral circulation (6, 24). When systole begins, the balloon deflates, thereby reducing afterload and improving cardiac output. Ultimately, IABP counterpulsation decreases cardiac work, improves cardiac output, and heightens tissue perfusion by augmenting blood flow (24).

IABP has traditionally been deployed with concomitant use of systemic anticoagulation using heparin to reduce the risk of thrombus formation on the balloon, resulting in thromboembolism or limb ischemia. A recent meta-analysis by Pucher et al. questioned the necessity of heparinization (25). Pucher et al. identified 3 studies in the literature addressing the question of antiplatelet therapy or anticoagulation in the setting of IABP and concluded that there was no significant difference in limb ischemia but an increased incidence of bleeding complications in heparinized patients undergoing IABP counterpulsation, recommending judicious and selective use of heparin or glycoprotein IIb/IIIa inhibitors. Two of the studies analyzed by Pucher et al. reported IABP times of 46.9 hours and 45.2 hours, respectively (7, 11).

Since its introduction, the IABP has been well studied in the setting of cardiogenic shock, and is currently recommended by the American College of Cardiology—American Heart Association for the treatment of cardiogenic shock refractory to pharmacologic support with class IB evidence (2). Additionally, the safety profile of the device has been well studied, and the Benchmark Counterpulsation Outcomes Registry currently represents the largest registry of patients with IABP implantation and has demonstrated an excellent safety profile (39). The study identified 25,136 patients who underwent IABP implantation for any indication. Mortality was directly attributed to the IABP in 13 patients (<0.1%), and major complications related to the device occurred in 7.2%. Complications included major limb ischemia (0.9%), severe access site bleeding (0.9%), deep vein thrombosis (0.1%), IABP leak (1.2%), and peripheral thrombosis (0.1%). In this series, the IABP was used for an average duration of 2.3 ± 2.5 days.

More recently, some controversy has emerged regarding the efficacy of IABP, and despite the level IB recommendation, a recent

Key words
- Aneurysm
- Balloon pump
- Stunned myocardium
- Subarachnoid hemorrhage
- Takotsubo
- Vasospasm

Abbreviations and Acronyms
CBF: Cerebral blood flow
IABP: Intra-aortic balloon pump
SAH: Subarachnoid hemorrhage

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meta-analysis found few randomized, controlled trials that demonstrate efficacy in the broad patient population of ST-segment elevation myocardial infarction with cardiogenic shock and several observational and cohort studies that only demonstrate efficacy in IABP used as an adjunct to thrombolysis (30). Further investigation is ongoing in the IABP-SHOCK II trial, a 600-patient multicenter prospective randomized trial currently underway (34).

CARDIAC DYSFUNCTION IN SUBARACHNOID HEMORRHAGE (SAH)

Harvey Cushing first described the cardiac sequelae of aneurysmal SAH in a series of cases published in 1903 (8). Cardiac complications of SAH are common, with up to 20% of patients with SAH having elevations in cardiac troponin I, with greater risk for injury in patients with higher Hunt-Hess grade, female sex, large body surface area, left ventricular mass, and higher doses of phenylephrine used (37). Nearly 100% of patients with SAH will have electrocardiogram abnormalities including ST-segment abnormalities, T-wave abnormalities, and QTc prolongation (18). The most severe form, neurogenic stunned myocardium can lead to florid left ventricular dysfunction, heart failure, and in rare cases, the apical ballooning syndrome known as takotsubo cardiomyopathy (17). Although the cause of cardiac dysfunction remains elusive, several theories have support in the literature, including coronary artery spasm causing ischemia, microvascular dysfunction, and catecholamine-mediated neurogenic myocardial stunning (17). In particular, the catecholamine surge associated with the ictus of SAH may underlie the pathophysiology. A genetic polymorphism study by Zaroff et al. analyzing the adrenoceptors of 182 patients with SAH found that certain receptor polymorphisms are associated with increased risk of cardiac injury (40). A case of iatrogenic takotsubo cardiomyopathy after administration of catecholamine for anaphylactic shock supports this theory (15). A recent review of neurogenic stunned myocardium, or neurogenic stress cardiomyopathy by Nguyen and Zaroff nicely summarizes the mechanisms surrounding this phenomenon (20). In addition to the hemorrhagic ictus, patients undergoing triple-H therapy are at risk for cardiac failure. Permissive hypertension improves cerebral blood flow (CBF) at the cost of increased myocardial afterload, whereas hemodilution decreases oxygen delivery to the myocardium, and hypervolemia increases filling pressures, potentially leading to pulmonary edema. Nguyen highlights the current pathophysiology of neurogenic stunned myocardium, including multivessel coronary artery spasm, microvascular dysfunction, and the catecholamine hypothesis.

RATIONALE FOR USE OF CARDIAC SUPPORT IN CEREBRAL VASOSPASM CAUSED BY SAH

In the 1970s and 1980s, the ischemic effects of cerebral vasospasm in SAH were recognized, and hyperdynamic, hypervolemic, and hemodilutional therapies were implemented to counteract delayed ischemic neurologic deficits (13). Coupled with the trend toward early aneurysm surgery, the advent of triple-H therapy has allowed direct, targeted treatment of vasospasm (5, 31). Still, vasospasm occurs in up to 67% of patients with SAH and accounts for 7% to 10% of mortality associated with SAH despite traditional therapy, and is now the highest overall cause of mortality after the initial hemorrhage (10, 14).

IABP has been postulated as an adjunct therapy in heart failure due to SAH since 1972 when Simeone et al. measured CBF in dogs and rhesus monkeys and found a 40% increase in CBF as compared with control subjects (29). This is likely due to the inflation of the balloon during diastole, which displaces blood back toward both the cardiac vasculature as well as the other great vessels. Tranmer et al. used a mechanical perfusion technique in dogs to demonstrate that pulsatile blood flow is critical in maintaining CBF and that the effect of pulsatility was significantly greater in the ischemic brain (35). Tranmer et al. went on to use IABP in this model to demonstrate that IABP also increases CBF in the ischemic brains of dogs, and proposed that IABP may be effective in the treatment of stroke (36). In 1995, Nussbaum et al. first demonstrated improved CBF in an SAH model in dogs (22). By injecting blood into the subarachnoid space, (the 2-hemorrhage model), Nussbaum et al. demonstrated an average 20% increase in CBF during vasospasm.

CLINICAL USE OF IABP

Including the paper by Ducruet et al., review of the literature identifies 9 reports including a total of 25 patients who have undergone IABP in SAH (3, 16, 19, 23, 26, 27, 32, 33). The technique was first described by Apostolides in 1996 in 2 patients in whom congestive heart failure and pulmonary edema developed while they were undergoing triple-H therapy (3). They were treated with 10 and 12 days of IABP with good neurologic outcome. Additional case reports or small series were reported by Nussbaum et al. (23), Rosen et al. (26), Montessuit et al. (19), Sato et al. (27), Taccone et al. (33), and Lazaridis et al. (16). Spann et al. reported on 6 patients with SAH who were prophylactically implanted with IABP immediately after surgery with the goal of measuring the effect of IABP on CBF throughout the postoperative period (32). Details of these series are summarized in **Table 1**, along with data pertinent to IABP placement.

Indications for IABP included prophylactic placement in a patient with inadequate blood pressure response to maximal vasopressor treatment, neurogenic stunned myocardium, and cardiac failure caused by myocardial infarction or ischemia from use of conventional triple-H therapy. In all cases, IABP placement was considered as a last-resort intervention after other conventional interventions had failed.

The effects of IABP were measured by most studies and varied from direct measurement of CBF using Xenon-133 scanning, measurements of systolic blood pressure or mean arterial pressure, and calculation of the left ventricle stroke-work index. In all cases in which the effect of IABP was measured, there were statistically significant improvements in cardiac function and/or CBF. These improvements generally resulted in neurological improvement and systemic clinical improvement.

IABP time ranged from 3 to 12 days, with an average of 5.9 days. It is important to note that the Benchmark Counterpulsation Outcomes Registry average IABP time was 2.3 $\pm$ 2.5 days, significantly shorter in duration. This is likely because patients with primarily cardiac pathology are often placed on IABP as a temporary measure before definitive therapy such as percutaneous coronary intervention or coronary artery bypass graft, whereas patients with cardiac dysfunction in the setting of aneurysmal SAH must be maintained with IABP for the duration of their vasospasm or that of the cardiac dysfunction. This is especially important to note because of the high degree of IABP-related complications in

Table 1. Reports of Intra-Aortic Balloon Pump Use in Subarachnoid Hemorrhage

Study	Number of Patients	Indication for IABP	Cardiac/CBF Measurements	IABP time (average)	Neurologic Exam	IABP Complication	Outcome at Longest Follow-up	Anticoagulation
Apostolides et al., 1996 (3)	2	MI and CS (triple H)	Average LVSWI 33.4 → 60.7	10 days, 12 days (11 days)	+, +	Femoral artery pseudoaneurysm	GOS 3, 2	N/A
Nussbaum et al., 1998 (23)	1	CS (triple H)	LVSWI 52.0 → 89.2 CBF 20.5 → 34.7 (69.3% increase)	5 days	+	None	GOS 3	+
Rosen et al., 2000 (26)	2	CS (triple-H), prophylactic during triple-H	MAP increase 22.9%	3 days, 5 days	=, +	Groin hematoma	GOS 4, 4	+, +
Montessuit et al., 2000 (19)	1	Inadequate response to pressors	MAP increase 20%	4 days	+	None	GOS 4	+
Spann et al., 2001 (32)	6	Prophylactic	mCBF 35.6 → 50.9 mL/100g/minute	4-7 days (5.6 days)	5 +, 1 =	Groin hematoma, IABP displacement	5 'good', 1 died	− x 6
Sato et al., 2001 (27)	2	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Taccone et al., 2009 (33)	2	Stunned myocardium	MAP 98 → 122, 87 → 110	4 days, 5 days	=, =	Sepsis, thromboembolic multiorgan ischemia	GOS 4, 1	N/A, +
Lazaridis et al., 2010 (16)	1	Stunned myocardium	N/A	4 days	+	None	GOS 5	+
Ducruet et al., 2012 (present study)	8	CS (both SAH and triple-H related)	N/A	3-11 days (5.9)		LE ischemia, HIT	mRS 1: 2 mRS 2: 2 mRS 4: 3	− x 7 + x 1
Total/average	25			5.9	11/15 (73%) improved	7/23 (30%)		6/20 (30%)

CBF, cerebral blood flow; CS, cardiogenic shock; GOS, Glasgow Outcome Scale; HIT, heparin-induced thrombocytopenia; IABP, intra-aortic balloon pump; LE, lower extremity; LVSWI, Left Ventricle Stroke Work Index; MAP, mean arterial pressure; mCBF, mean cerebral blood flow; MI, myocardial infarction; mRS, modified Rankin Scale; N/A, not available; SAH, subarachnoid hemorrhage; triple-H, hypervolemic, hyperdynamic, hemodilutional therapy; +, improved; =, unchanged.

the available series (30% or 7 of 23 patients with some type of complication directly related to IABP placement). There were only 2 cases of thromboembolic complications from IABP use, 1 in a patient receiving concomitant heparin anticoagulation and 1 without; 30% of patients were managed with systemic anticoagulation with heparin, and there were no reported uses of antiplatelet agents or glycoprotein IIb/IIIa inhibitors. It is not surprising that although small, the incidence of thromboembolic complications is higher than that reported in the large study of cardiac patients cited previously of 0.9% because of the significantly longer duration of treatment in this group. The meta-analysis by Pucher et al. demonstrated no need for heparinization in 2 studies in which the duration of treatment was around 2 days (25). However, some have argued that the 48-hour point may be a reasonable cutoff to begin anticoagulation (9). There were no hemorrhagic complications of heparinization reported in these series. It is difficult to provide a clear risk-benefit ratio for the use of heparin in the setting of IABP. However, given the high risk of anticoagulation after craniotomy, it may be prudent to withhold anticoagulation in patients who have had aneurysm clipping or in those with unprotected aneurysms, whereas a more liberal use of anticoagulation may be considered in patients with endovascularly treated aneurysms.

In the series of 22 patients with clinical follow-up, there were 2 deaths. The first, in the Spann et al. study, was dead at 3-month follow-up without explanation. This patient had only mild hemiparesis listed as the worst postoperative deficit; the outcome can likely be assumed to be unrelated to IABP use in this case. The second death, in the Taccone et al. study, occurred in a 35-year-old patient, Fisher grade III, Hunt-Hess grade 4, with iatrogenic SAH from a middle cerebral artery dissection during elective embolization of an unruptured middle cerebral artery aneurysm. This patient developed thromboembolism after 5 days of IABP therapy despite adequate anticoagulation during IABP treatment and after IABP removal. Care was withdrawn on postoperative day 17 after developing gram-negative sepsis and thromboembolic ischemic infarcts in the brain, kidneys, and bowels leading to multiple organ failure and coma.

Overall, outcomes were favorable. One must remember that in all cases except the prophylactically treated patients, IABP was used as a last resort in patients with severe cardiac failure or neurologic deterioration refractory to maximal medical and interventional management. Thus, as a rescue modality, IABP has proven successful. Prophylactic use of IABP has not been studied well enough to date to clearly assess its place in triple-H therapy.

DUCRUET PAPER

Ducruet et al. have retrospectively reviewed a prospectively collected database of SAH patients treated at St Joseph's Hospital and Medical Center during a 5-year period and identified 8 patients who underwent placement of an IABP in the setting of SAH. Most of these patients had presented with poor-grade SAH (75% Fisher IV, 75% Hunt-Hess grade IV or V). Transcranial Doppler ultrasound was used for early detection of subclinical vasospasm, and triple-H therapy was instituted based on elevated transcranial Doppler velocities or for neurologic deterioration. All 8 patients demonstrated elevated transcranial Doppler values, and 6 patients developed moderate or severe angiographic spasm. The 6 patients with angiographic spasm underwent 1 more sessions of balloon angioplasty and/or intra-arterial vasodilator infusion.

IABPs were placed in patients who developed severe cardiogenic shock. The authors stated no distinct threshold for placement of IABP. It is inferred from the report that the devices were placed only on a rescue basis for severe cardiogenic shock. Before placement, cardiogenic shock was diagnosed using transthoracic echocardiogram, with a mean left ventricular ejection fraction of 21.2% and a range of 10% to 35%. Although there are certain cases in which IABP is the only remaining solution, it may be helpful if the authors could comment on the specific parameters used when evaluating a patient for placement on IABP. Some patients who have cardiac dysfunction may still benefit from additional angiographic treatment of vasospasm, ongoing inotropic or vasoactive medication, or volume expansion.

The complication rate in this series was high; however, the patients were high-grade on presentation and experienced high morbidity associated with vasospasm and triple-H therapy. Only 1 complication in the series was directly attributable to IABP placement. One patient developed leg ischemia after removal of the IABP and was treated successfully with heparinization and limb warming. Another patient, who required IABP counterpulsation for 7 days, was treated with intravenous heparin and developed heparin-induced thrombocytopenia.

Seven of 8 patients had clinical follow-up, 4 had either no disability or mild disability (mRS 1 or 2), and 3 remained with severe disability (mRS 4). All patients were alive at latest follow-up. Although the overall clinical outcomes do speak to the efficacy of the intervention, it would be helpful to understand physiological measures such as improvement in mean arterial pressure, CBF, or neurologic status after IABP placement.

Without a defined threshold for use of IABP or physiological and clinical effects after IABP placement, it is difficult to assess the efficacy of IABP in this series or to determine which population may benefit most from IABP placement. Taken with the existing literature, however, this series adds a significant number of patients to a growing body of evidence that IABP can be used safely in the setting of cardiac failure refractory to medical management in cerebral vasospasm in SAH.

Overall, Ducruet et al. present the largest series to date of patients in whom IABP was used to treat heart failure in the setting of aneurysmal SAH. They are to be commended for furthering our understanding of this technique and its application to patients in whom traditional treatment has failed.

NEW HORIZONS

Building on the work of Tranmer et al. in the stroke population, a new aortic dual-balloon catheter for partial occlusion of the aorta in patients with stroke is being developed (NeuroFlo, CoAxia, Maple Grove, Minnesota, USA). This device partially occludes the abdominal aorta, allowing preferential perfusion of the upper body and cerebral vasculature in ischemic stroke. The device has been approved for study in stroke patients not eligible for intra-arterial or intravenous thrombolysis, and preliminary results of an interim analysis are promising (38). There have been 2 reports of use of the NeuroFlo device in treatment of cerebral vasospasm for SAH, both with successful results (1, 4). In the Appelboom et al. case study, the balloon was placed using ultrasound guidance alone, as a bedside procedure in the intensive care unit. Safety and efficacy data are yet to be seen both in the stroke and the SAH populations.

CONCLUSIONS

IABP placement for cardiogenic shock in the setting of aneurysmal SAH has been reported in several case studies and small series in the neurosurgical literature. Complications related to the technique have been low, especially given the severity of illness in the cases reported. Ducruet et al. have added a significant amount of experience and have demonstrated safety and good long-term clinical outcomes in their series. The precise indications for the use of this technique have yet to be defined, and further study should focus on identifying patients who would most benefit from use of this device. Similar devices that aim to increase CBF, such as the NeuroFlo device, also show promising theoretical benefits, but the experience of these devices in SAH remains limited.

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