

Case Report

Trans-Aortic Counterpulsation: A Viable Alternative?

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Abstract: Transthoracic intra-aortic balloon pump (IABP) insertion has been a relatively rare and uncommon procedure. However, it is an established beneficial option in patients with severe peripheral vascular disease (PVD) accompanied with bi-lateral femoral arterial occlusion. There are several viable alternatives to trans-aortic IABP insertion, including trans-axillary or in abdominal aorta (requiring a laparotomy). Cardiac surgery has the advantage of an open sternum, facilitating effortless direct intra-aortic balloon (IAB) insertion into the aorta. The IAB can be inserted either through a 9-mm graft or directly into the ascending aorta. During cardiac surgery, direct insertion into the ascending aorta with the balloon tip lying distally in the abdominal aorta is facilitated with an open sternum. The base of the balloon lies ~2 cm below the left subclavian and can be confirmed

through a trans-esophageal echocardiogram (TEE). Elimination of a graft insertion saves the team from time-consuming maneuvers and additional hemorrhagic complications. In our experience, postoperative vasoplegic syndrome coupled with myocardial edema contributed to patient instability and was treated with vasopressin and transthoracic IAB insertion. The CS 100 (Datascope Corp., Mahwah, NJ) console allowed the ability to time the balloon accurately. This case report details our experience with one such patient and establishes trans-aortic counter-pulsation as a safe and viable option in patients with severe PVD, where percutaneous insertion is precluded or has failed. **Keywords:** transthoracic, peripheral vascular disease, intra-aortic balloon, counter-pulsation, vasoplegic syndrome. *JECT. 2007;39:91–95*

Transthoracic insertion of a direct intra-aortic balloon (IAB) was first described by Melvin and Goldman in 1982 (1). Despite being contraindicated by vendors in the market for intra-aortic balloon pump (IABP), direct insertion of the IAB is sometimes the only option in patients suffering from severely debilitating peripheral vascular disease (PVD). Availability of the IABP as a device for left ventricular (LV) assistance is critical in cardiac surgery. This device is generally precluded in patients with severe PVD because of unavailability of arterial access and/or after failure of insertion. Although other arteries (e.g.,

axillary artery, subclavian artery) can be considered for arterial access, chances are that the vascular disease process is widespread.

There are two primary methods (2) of direct IAB insertion: through a Dacron graft anastomosed end to side to the aorta or directly with concentric pledgeted purse string sutures. We used the latter method in this case study.

CASE REPORT

A 67-year-old man was admitted to the cardiology clinic for angina. Evidence of penicillin allergy and hypertension was discovered, with no other significant cardiac risk factors. The patient revealed an active smoking and alcohol abuse pattern. There was a history of severe peripheral vascular disease with rigorous bilateral femoral claudication. The patient was unable to walk for >5 minutes with-

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out experiencing debilitating calf pain. On admittance, he suffered a non-ST elevation myocardial infarct (MI). Additionally, the patient had suffered from pericarditis in 2003 and was treated with non-steroidal anti-inflammatory drugs (NSAIDS). Blood work was ordered, and he was started on integralin and heparin infusion. Angiography facilitated through the radial artery revealed a grade 2 LV, 90% stenosis in the left anterior diagonal (LAD) and right coronary artery (RCA), and 30% stenosis in the circumflex artery. The subject suffered transient bouts of hypotension in the cardiac catheterization laboratory. The cardiologist was unable to insert an IAB because of confirmed severe PVD. The patient was transferred to the operating room (OR) suite for emergent aortocoronary bypass (ACB) surgery. In the OR, the perfusionist noted body surface area (BSA) of 1.82 m^2 , with top flows (at a cardiac index of 2.4) of 4.36 L/min . All laboratory results were normal, with a hematocrit (HCT) of 0.40 and a creatinine of 88 mmol/L . Partial thromboplastin time (PTT) was 88 seconds, which was attributed to recent heparin infusion. The incumbent was also on Plavix, nitro, and acetylsalicylic acid (ASA). It was decided to use 2 million units (200 mL) of aprotinin in the prime, followed by a full Hammersmith protocol. The HL-20 heart lung machine (HLM; Maquet-Dynamed, Lund, Sweden) was set up with a Jostra oxygenator in combination with a Rotaflo centrifugal head and a closed system consisting of Bio-line tubing (Maquet AG, Hirrlingen, Germany) coated with heparin/albumen. The pump was primed with 800 mL of Ringer lactate, 500 mL of pentaspan, 50 mL of sodium bicarbonate, 5000 U of heparin, and 200 mL (5 million units) of aprotinin, with a total prime volume of 1500 mL. The heparin dose was determined using a heparin dose response (HDR) cartridge placed in the Hepcon HMS (Heparin Management System) Plus (Medtronic, Minneapolis, MN). Thirty-five thousand units of heparin Leo was administered before initiating bypass at an activated clotting time (ACT) of 672 and a heparin level of 400 U/kg. Before initiating CPB, 300 mL of prime was taken off using the arterial line and an equivalent amount from the venous line through retrograde autologous priming (RAP), reducing the prime volume to 950 mL. The RAP technique on the arterial line was not the classical rapid method but was performed in a slow and concerted manner, draining the fluid into an empty drain bag connected to the manifold using the patient's mean arterial pressure (MAP) as the driving force.

The pump run was uneventful, the patient was drifted to 34°C , re-warming was started at 18 minutes of cardiopulmonary bypass (CPB), and three doses of cardioplegia were administered. The total aortic cross-clamp time was 32 minutes, and the CPB time was 45 minutes. Left internal mammary artery (LIMA) was grafted to the LAD, and a reversed saphenous vein graft (RSVG) was grafted to

the posterior inter-ventricular (PIV) branch. The heart was re-perfused for 11 minutes after cross-clamp release, and defibrillation was performed to reverse ventricular fibrillation.

A loading dose of 2 mg of milrinone was administered, and a nor-epinephrine (N/E) infusion was started and titrated to effect. The patient was weaned off the pump slowly over a period of 5–7 minutes. The patient came off pump with a good MAP but a sluggish heart rhythm. The fluid balance at the end of the procedure left the patient positive by 750 mL. The pulmonary artery (PA) pressures were noticed to be high (55/30) on coming off pump and increased to 60/35 mmHg over 5 minutes. After protamine administration, manipulation of the chest to insert sternal wires was seen to generate a hypotensive crisis. Subsequent TEE revealed a sluggish left ventricular contraction and an overall downtrend in heart function. Dobutamine was initiated along with an infusion of N/E. On sternal closure, the patient's MAP vacillated in the low 80s, and the PA pressure trended upward. The anesthesiologist was able to stabilize pressures by increasing the inotropic infusion.

On transfer from the OR table to the transfer bed, the patient experienced additional systemic hypotension coupled with pulmonary hypertension. The decision was made to re-open the chest and examine the grafts. Palpation and ultrasound of both the grafts after chest reopening showed good patency.

Three liters of crystalloid and 500 mL of pentaspan were infused to stabilize the patient before going on CPB for the second time, along with a unit of blood.

The HLM was re-set up and primed while the patient was being prepared. RAP was once again performed, taking away 500 mL.

Emergency CPB was reinitiated after bolusing 35,000 units of heparin in the Swan Ganz and 15,000 units of heparin in the pump prime. Because a fair amount of volume was administered to stabilize pressures, the starting HCT this time was only 0.28.

On CPB, the hematocrit had fallen to 0.20, and a significant amount of volume was observed in the pump circuit. A decision was made to maintain a higher hematocrit. To achieve this objective, 2 units of packed cells were administered, and 1400 mL of volume was taken off the CPB circuit using hemoconcentration.

The cardiac surgeon was of the opinion that an additional RSVG placed on the LAD distal to the mammary artery may help coronary flow. Previously, an attempt was made to perform the anastomosis off-pump, but the patient was unable to tolerate lifting of the heart. The anastomosis was done without cross-clamp on a beating heart in a CPB time of 50 minutes. The initial ACT was 788 seconds, with a heparin level of 250 U/kg. Five minutes into the pump run, ACT was re-checked and came back at

198 seconds, with a heparin level of 400 U/kg. The ACT was confirmed with another Hemochron junior signature plus device (ITC, Edison, NJ) from an adjacent OR and was found to be in the low 200s. Another 10,000 units of heparin were administered. The next ACT came back at 354 seconds. Another 10,000 units of heparin were given, and the ACT peaked at 444 seconds. At no time were clots seen in the chest or in the circuit. This acceleration in heparin consumption is discussed later.

The post-pump fluid balance, after the second pump run, was negative by 750 mL. Pulmonary hypertension persisted to a lesser degree.

At this time, the right radial line ceased to function (Figure 1), and a cut down was performed to expose the left radial artery, which was found to be inadequate to measure blood pressure. The patient was weaned using milrinone, N/E, and dobutamine.

Intraoperative TEE revealed a worsened anterior wall and apical function compared to pre-CPB images. Lactate and blood sugar had spiked. ST elevation was noticed. Vasopressin was added to the inotropes already running.

Insertion of sternal wires for the second time re-initiated the bouts of hypotension previously experienced and a rise in PA pressures. At this time, a decision was made to insert the IAB through the transthoracic route. A 40 mL/8 Fr IAB was inserted directly (Figures 2 and 3) into the ascending aorta, with the balloon positioned 2 cm below the origin of the left subclavian artery. The helium drive line was connected to a CS 100-IABP console (Data-scope Corp.). IAB augmentation was initiated at 1:1 using an electrocardiogram (ECG) trigger slaved from the anesthesia monitor (Figure 4). A suspect radial artery line would have made timing from the radial artery a difficult endeavor. Thankfully, the CS 100 was able to time the balloon accurately in the "auto" mode. An immediate effect was seen on patient hemodynamics, with the MAP

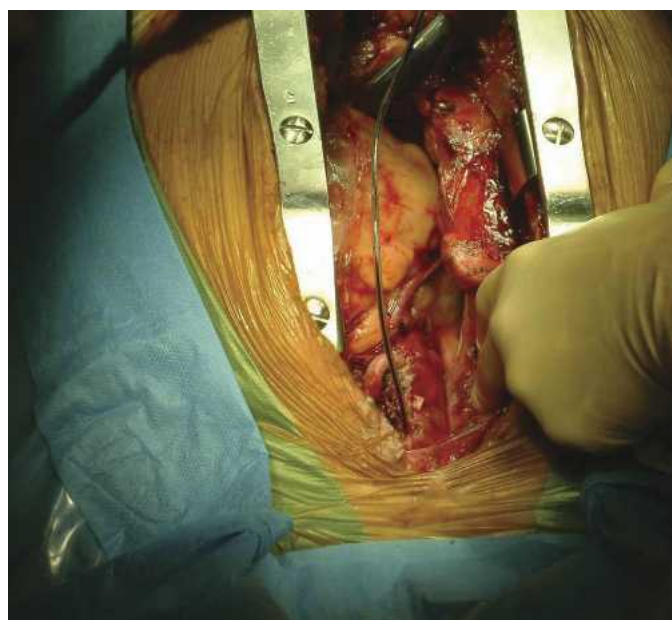


Figure 2. IABP: intra-thoracic view.



Figure 3. IABP: external view.



Figure 1. Damp radial arterial waveform.

settling at a stable 100. MAP was improved, and the pulmonary hypertension resolved to a great degree over the next half hour. TEE confirmed a more vigorous heart function in both the left and right sides. Subsequently, PA pressures, while being unstable earlier, now trended down into the low 40s. The cardiac output was high, with the index closer to 3 L/min/m². Urine output improved, and ST elevation settled, returning to baseline. Surprisingly, the patient did not appear coagulopathic. Despite this, 5 units of platelets with 4 units of fresh frozen plasma were infused as a preventative measure.

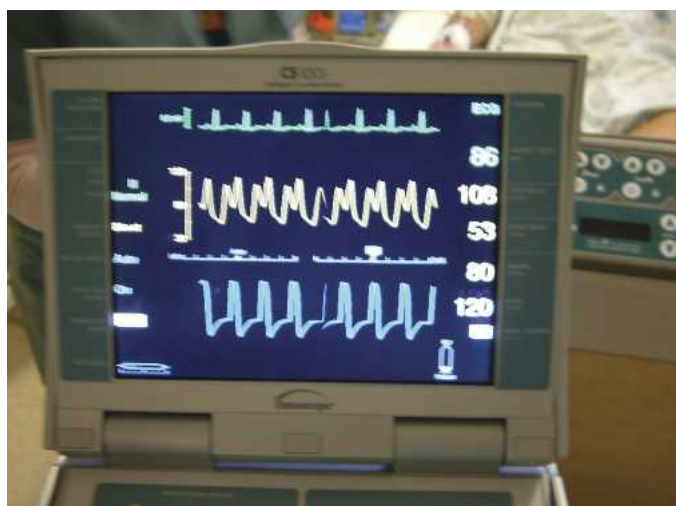


Figure 4. IABP waveform with TIABP.



Figure 5. Axillary artery waveform.

On transfer to the intensive care unit (ICU), an axillary artery was placed to monitor invasive blood pressure (Figure 5).

On the fourth postoperative day, the native heart function was satisfactory without IABP augmentation, and the IAB was removed. Murky fluid was noted on opening the mediastinal cavity, and 300 mL of fluid was suctioned out of the right pleural cavity. The mediastinum was irrigated with betadine/saline solution and closed after insertion of two pleural, one mediastinal, and one Feeit chest tubes.

The patient was extubated on postoperative day 5, was responding to commands, and seemed oriented.

DISCUSSION

Datascope Corp. policy does not advise or encourage transthoracic insertion of the IAB, their reasoning being

that the technique is not Food and Drug Administration (FDA) approved for IAB therapy. All the contents of the Datascope instructions for use (IFU) manual need to be FDA approved. Despite this fact, transthoracic IABP (TIABP) insertion has been attempted several times, and there are several studies showcasing this technique.

Fischer et al. (3) compared the effects of counterpulsation in the ascending aorta and the descending aorta in sheep with induced cardiac failure. They found that the increase in coronary perfusion and afterload reduction was comparable.

In another retrospective study of a 100 patients, Hazelrigg et al. (4) reported an 81% survival (4). Evaluation of complications detailed balloon rupture in 6.2% (5/81), cerebrovascular accidents in 2.5% (2/81), transient ischemic attacks in 1.2% (1/81), IABP arteriotomy site bleeder in 3.7% (3/81), and mediastinitis in 3.7% (3/81). It was noticed that, despite being a high-risk group, the complications from TIABP insertion were well below the expected rate. Balloon rupture and mediastinal bleeding seemed to increase with transthoracic insertion, whereas neurologic events and mediastinal infections did not (4).

Another article as early as 1980 cited improved survival in 28 patients requiring TIABP coupled with delayed sternal closure of 302 patients in which IAB was inserted femorally (5).

A Scandinavian study mentioned 58.3% TIABP mortality as opposed to 46.1% trans-femoral IABP insertion (6). Another study showcased 81% survival in 39 patients over a period of 3 years, with no deaths directly related to TIABP (7).

A hyper-dynamic state after CPB for the second time on bypass suggested an inflammatory reaction akin to vasoplegic syndrome. The hypotension and high-dose vasoconstrictor (particularly vasopressin) use confirms this fact, although the diagnosis was unrecognized until arrival in the ICU.

Vasoplegic syndrome is characterized by hypotension, tachycardia, normal or elevated cardiac output, and low systemic vascular resistance (SVR) unresolved by fluid administration alone and requiring high-dose vasoconstrictor therapy (8). This syndrome in general has an unclear etiology, although it can be attributed to inflammatory response activated specifically by CPB, surgical trauma, blood transfusion, heparin, pre-operative angiotensin converting enzyme (ACE) inhibitors, milrinone, and hypothermia (9). The salient features of vasoplegic syndrome are similar to those observed in septic shock mediated by physiologic alterations caused by cytokines and tumor necrosis factor- α . The sudden accelerated heparin consumption could also have been caused by vasoplegic syndrome and/or antithrombin-III (AT-III) deficiency/consumption.

The discovery of NO as a mediator in vasoplegia after

CPB has encouraged the role of inhibitory drugs like methylene blue at a dosage of 1.5–3 mg/kg (10). In a randomized study, methylene blue reportedly increased MAP and SVR after administration, lowering postoperative lactate levels and vasopressor use in patients treated with ACE inhibitors before surgery (11).

In conclusion, TIABP insertion is a viable technique in patients where trans-femoral insertion is precluded or fails because of severe PVD. We found the direct insertion technique superior because it eliminated the need of an additional Dacron graft and reduced patient trauma during insertion and removal.

In retrospect, we could have used methylene blue (3 mg/kg) to assist us in the hemodynamics. Methylene blue is known to competitively inhibit NO at the heme moiety of guanylate cyclase, thus reducing cyclic guanosine monophosphate (cGMP) production. This in turn would increase both SVR and MAP (11).

An interesting point to note is that the CS 100 Datascope IABP console was able to time the balloon accurately and reflect precise MAP values despite the balloon being inserted retrogradely from the aortic root down into the descending aorta. The console transducer was able to accurately reproduce MAP values by calculating the delay from the tip of the IAB catheter to the balloon transducer and displaying descending aortic pressure. This delay can be seen by going into the pump options menu of the IABP console and is displayed in milliseconds.

Patients with poor left ventricular ejection fraction (LVEF) tend to suffer from severe myocardial edema, especially after CPB combined with concomitant fluid resuscitation therapy. Sternal closure is poorly tolerated in these patients, and direct ascending aortic IAB insertion, delayed sternal closure, and vasopressin therapy would in our opinion tide over the hazardous postoperative phase characterized by hypotension, pulmonary hypertension, and possible vasoplegia. All of these could contribute to early morbidity and mortality. Postoperatively (14 days), in the cardiovascular (CV) surgery floor, the neurologist

did note post surgery delirium, cerebrovascular accident (CVA) with expressive aphasia, or a post-cardiac surgery confusional state.

The CVA could have been caused by perioperative events. These symptoms disappeared over time, and no early/late mortality or permanent morbidities were observed other than those expected for a patient suffering from long-term severe PVD.

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