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ANESTHESIA MANAGEMENT FOR INTRA-AORTIC BALLOON PUMP INSERTION : A CASE REPORT

Kasmarandi AT[1], Lestari MI[2], Irwanto FH[2]

[1]Resident, Departement of Anesthesiology and Intensive Care, Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia, [2]Staff lecturer, Department of Anesthesiology and Intensive Care, Muhammad Hoesin Hospital, Palembang, Indonesia,

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ABSTRACT

Introduction: Intra-aortic balloon pump (IABP) is the most widely used mechanical circulatory support device in patients with hemodynamic instability, including cardiogenic shock and acute myocardial ischemia. Anesthesia management for IABP insertion demands a highly integrated approach, balancing rapid procedural requirements with myocardial protection and hemodynamic stability. This report presents a case of anesthesia management in a critically ill patient undergoing IABP insertion concurrent with coronary artery bypass grafting (CABG).

Case: A 64-year-old male with cardiogenic shock, three-vessel coronary artery disease, and reduced ejection fraction (32%) underwent CABG with concurrent IABP insertion under general anesthesia. Induction utilized propofol, midazolam, fentanyl, and rocuronium. Intraoperative management included invasive hemodynamic monitoring, goal-directed fluid therapy, and ventricular arrhythmia prevention. The patient required inotropic support with dobutamine, milrinone, and epinephrine postoperatively, with IABP providing hemodynamic augmentation.

Discussion: Key anesthesia considerations include airway management with hemodynamic-sparing induction agents, invasive monitoring, real-time transesophageal echocardiography guidance, IABP timing and management, prevention of perioperative ventricular arrhythmias, and rationale for the multimodal inotropic/vasopressor strategy used in this patient.

Conclusion: Successful anesthesia management for IABP insertion requires meticulous hemodynamic optimization, appropriate induction agent selection, real-time imaging guidance, and vigilant perioperative monitoring. The integration of established IABP management principles with individualized anesthetic care is essential for favorable patient outcomes.

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1. Introduction

The intra-aortic balloon pump (IABP) represents the most commonly employed device for mechanical circulatory support in cardiac surgery and critical care settings. Positioned in the descending thoracic aorta just distal to the left subclavian artery, the IABP inflates during early diastole to augment coronary perfusion pressure and deflates immediately before systole to reduce left ventricular afterload, thereby improving the myocardial oxygen supply-to-demand ratio and cardiac output.[1,2]

IABP is indicated preoperatively in cardiogenic shock, acute myocardial ischemia, and high-risk coronary angiography; intraoperatively to facilitate weaning from cardiopulmonary bypass in severely compromised ventricular function; and postoperatively for refractory low cardiac output states or myocardial ischemia unresponsive to surgical or interventional solutions.[1,3]

Anesthesia management for IABP insertion is uniquely challenging given that candidates are often hemodynamically unstable, with limited physiological reserve and a narrow tolerance for intraoperative perturbation. The anesthetic approach must simultaneously ensure procedural success, maintain hemodynamic stability, facilitate real-time monitoring, and prevent complications including ventricular arrhythmias and vascular injury.[4,5]

This case report describes the perioperative anesthesia management of a 64-year-old male with cardiogenic shock and three-vessel coronary artery disease who underwent CABG with concurrent IABP insertion, with particular focus on induction strategy, hemodynamic monitoring, IABP management principles, and ventricular arrhythmia prevention.

2. Case Report

A 64-year-old male (date of birth: 16 August 1961) presented to the Cardiovascular ICU (CVICU) with a diagnosis of: continuous invasive mechanical ventilation secondary to respiratory failure, cardiogenic shock, post-coronary artery bypass grafting (CABG) for three-vessel coronary artery disease (CAD3VD), congestive heart failure with severely reduced ejection fraction (EF 32%), post-IABP insertion, and hypertension. The procedure was performed by cardiologist.

General anesthesia was induced with propofol 70 mg, midazolam 5 mg, fentanyl 400 mcg, and rocuronium 40 mg. Endotracheal intubation was performed using an ETT size 7.5 at a depth of 18 cm. Total operative duration was four hours, without an intraoperative shock phase.

Fluid balance: crystalloid 1,500 mL, colloid 1,000 mL, packed red blood cells 800 mL, and fresh frozen plasma 300 mL were administered. Estimated blood loss was 2,050 mL and urine output was 400 mL. Surgical procedures performed included CABG x2 (LIMA-LAD and SVG-OM grafts), three DC shocks at 15 J and one at 20 J. Aortic cross-clamp time was 28 minutes and cardiopulmonary bypass duration was 47 minutes.

Postoperatively, the patient remained sedated with non-assessable GCS, though pupils were 3 mm bilaterally and reactive to light. Hemodynamics were maintained with an intra-aortic balloon pump (IABP) and continuous inotropic support comprising dobutamine (5 mcg/kg/min), milrinone (0.375 mcg/kg/min), and epinephrine (0.07 mcg/kg/min), achieving a blood pressure of 136/59 mmHg and a heart rate of 90 bpm. Mechanical ventilation was provided via synchronized intermittent mandatory ventilation (SIMV) mode (RR 16/min, PS 10 cmH_2O, PEEP 5 cmH_2O, FiO_2 50%), generating expired tidal volumes of 408–523 mL (preset tidal volume 400 mL) and an oxygen saturation of 100%. Additional post-procedural invasive monitoring and access included a nasogastric tube and an indwelling urinary catheter, with an initial postoperative urine output of 200 mL. Postoperative medical management included intravenous antimicrobial coverage

(cefuroxime 1.5 g q8h), stress ulcer prophylaxis (omeprazole 40 mg q24h), analgesia and sedation (intravenous paracetamol 1 g q8h alongside continuous infusions of morphine and dexmedetomidine), and respiratory support (enteral N-acetylcysteine 200 mg q8h and nebulized budesonide q8h).

3. Discussion

3.1 IABP: Physiological Principles and Clinical Rationale

The IABP consists of a polyethylene balloon (25–50 mL, helium-filled) positioned in the descending aorta distal to the left subclavian artery. Counterpulsation—inflation during early diastole and deflation immediately before systole—produces hemodynamic effects that are particularly favorable in the context of cardiogenic shock and post-CABG ventricular dysfunction.[1,6]

Key hemodynamic consequences include: reduction of aortic systolic pressure, augmentation of diastolic pressure (enhancing coronary perfusion pressure), reduction of left ventricular end-diastolic pressure and volume, decreased wall tension, reduced afterload, and net improvement in cardiac output.[1,7]

In this patient with EF 32%, three-vessel disease, and cardiogenic shock, IABP insertion provided critical mechanical support for post-CABG ventricular recovery. The magnitude of IABP benefit depends on balloon inflation volume, heart rate (inversely related to filling time), and systemic vascular resistance.[6]

Contraindications to IABP in this case were systematically excluded: there was no evidence of aortic regurgitation, aortic dissection, or severe peripheral vascular disease. Moderate-to-severe aortic regurgitation represents an absolute contraindication because diastolic inflation worsens regurgitant volume and paradoxically increases both preload and afterload.[1,7]

3.2 IABP Insertion Technique

IABP insertion was performed percutaneously via the femoral artery using the modified Seldinger technique, which is the standard approach. The balloon was positioned with its tip just distal to the left subclavian artery, confirmed by transesophageal echocardiography (TEE) and intraoperative fluoroscopy. In patients undergoing concurrent cardiac surgery, intraoperative TEE provides real-time confirmation of guidewire and balloon catheter position without radiographic dependence.[1,7,8]

Key insertion steps as outlined by Chikwe et al. include: arterial puncture at mid-inguinal ligament level, guidewire advancement under fluoroscopic or echocardiographic guidance, sequential dilation, balloon measurement to position the tip at the third rib space, and immediate connection to the manometry line and IABP console.[7]

For this patient, the sheathless technique is preferred to minimize femoral artery lumen compromise and reduce the risk of distal limb ischemia—the most common IABP complication, occurring in >10% of cases. Regular assessment of distal pulses by palpation or Doppler is mandatory throughout the support period.[7]

3.3 IABP Timing and Management

Optimal IABP function depends on precise timing of balloon inflation and deflation relative to the cardiac cycle. Two triggering modes are available: ECG triggering (inflation at the peak of the T-wave, deflation on or just before the R-wave) and pressure triggering (inflation at the dicrotic notch, deflation just before the aortic upstroke). The pressure triggering mode is preferred when

monopolar electrocautery or poor ECG signal quality compromises ECG triggering—a relevant consideration during CABG surgery.[7]

IABP augmentation efficacy is confirmed by the characteristic 1:2 trace: diastolic augmentation pressure (C) should exceed the patient's systolic pressure, IABP end-diastolic pressure (A) should be lower than native aortic end-diastolic pressure, and assisted systolic pressure (B) should be lower than unassisted systolic pressure. Suboptimal traces indicate timing errors (early/late inflation or deflation) requiring console adjustment.[7]

Intraoperative DC cardioversion was required three times at 15 J and once at 20 J. IABP functioning should not be compromised by defibrillation; bipolar cautery is preferred over monopolar when using ECG triggering. If arrhythmias prevent reliable IABP triggering, pressure mode or manual triggering should be employed.[7]

The risk of thrombus formation on the balloon increases significantly if the IABP is left at a 1:3 augmentation ratio for more than one hour, or if the balloon is deflated. Thrombocytopenia is an additional known complication due to mechanical platelet destruction; daily platelet counts should be monitored with consideration of IABP removal if counts fall below 60×10^9/L.[7]

3.4 Airway Management and Induction of Anesthesia

Patients presenting for IABP insertion frequently have severely compromised physiological reserve, making airway management and induction of anesthesia particularly high-risk. In this patient, modified rapid sequence induction (RSI) was employed with propofol 70 mg, midazolam 5 mg, fentanyl 400 mcg, and rocuronium 40 mg, followed by endotracheal intubation with ETT 7.5 at 18 cm depth.[2,5]

Selection of induction agents in this context requires careful consideration. Propofol, while providing reliable hypnosis, carries risk of significant vasodilation and reduction of preload—particularly hazardous in cardiogenic shock. The relatively low dose used (70 mg) reflects deliberate titration to minimize hemodynamic depression. Etomidate, with its superior cardiovascular stability profile, is frequently recommended as an alternative induction agent for hemodynamically unstable patients; however, its adrenocortical suppressive effects remain a limitation in septic or critically ill patients.[3,4]

Fentanyl at 400 mcg provided opioid-based analgesia and sympatholysis, blunting the hemodynamic response to laryngoscopy and surgical stimulation without direct myocardial depression. Midazolam provided anxiolysis and amnesia. The combination of short-acting intravenous agents allows rapid dose adjustment—critical during IABP insertion when hemodynamic conditions may fluctuate abruptly during airway instrumentation, vascular access, and device activation.[3]

Preoperative checklist for difficult airway prediction is essential; loss of airway in the context of hemodynamic instability can precipitate catastrophic hypoxia and circulatory collapse before the benefits of IABP counterpulsation are realized.[3,5]

3.5 Hemodynamic Monitoring, Vascular Access, and Rationale for Combined Inotropic-Vasopressor Support

Continuous invasive arterial pressure monitoring is indispensable during IABP management: it provides both real-time blood pressure measurement and the pressure waveform required for IABP pressure triggering. The radial artery is the preferred site for invasive monitoring, providing reliable beat-to-beat measurement without compromising the femoral access site required for

IABP insertion. An arterial line at a non-femoral site also allows independent monitoring of IABP augmentation pressure versus native systolic pressure.[3]

Central venous access enables administration of vasoactive agents (dobutamine, milrinone, epinephrine in this case), volume resuscitation, and measurement of central venous pressure as a surrogate for preload. Dynamic preload indices such as stroke volume variation (SVV) and pulse pressure variation (PPV), derived from the arterial waveform under controlled mechanical ventilation, are preferred over static pressure measurements for guiding fluid therapy in IABP patients with mechanical ventilation.[3]

Rationale for combining milrinone with epinephrine on a background of IABP support: milrinone, a phosphodiesterase-3 inhibitor, acts as an inodilator—it augments contractility while simultaneously lowering systemic vascular resistance (SVR) through direct arterial and venous vasodilation. When used alone in a patient already receiving afterload reduction from IABP counterpulsation, this combination poses a meaningful risk of additive vasodilation and systemic hypotension, since both interventions act to reduce SVR and afterload through independent mechanisms. Epinephrine was therefore added at a low dose (0.07 mcg/kg/min) primarily for its alpha-adrenergic vasopressor effect, offsetting milrinone-induced vasoplegia while contributing further inotropic support at a dose range that is predominantly beta-adrenergic. This pairing reflects a deliberate strategy to decouple inotropic and vasopressor requirements: milrinone and IABP address forward flow and afterload reduction, while epinephrine maintains an adequate SVR and mean arterial pressure (MAP) to sustain coronary and end-organ perfusion pressure. In this patient, the postoperative hemodynamic profile—BP 136/59 mmHg (MAP approximately 85 mmHg) with HR 90 bpm on triple support—suggests this balance was achieved without evident hypotension, consistent with an effective interplay between afterload reduction (IABP and milrinone) and vasopressor support (epinephrine).

3.6 Intraoperative Fluid Management

Fluid management during IABP-assisted CABG surgery must account for the competing hemodynamic goals of the procedure. Excessive volume loading in a patient with EF 32% risks pulmonary congestion and worsening diastolic dysfunction; conversely, inadequate preload impairs cardiac output and compromises coronary perfusion pressure.[1,3]

Total intraoperative fluid administration comprised 1,500 mL crystalloid, 1,000 mL colloid, 800 mL PRBCs, and 300 mL FFP—a balanced replacement strategy against blood loss of 2,050 mL. The substantial blood loss reflects the combined operative complexity of CABG with IABP and the coagulopathic risk of cardiopulmonary bypass. Heparin anticoagulation for CPB (target ACT $\geqslant$ 400–480 seconds), with subsequent protamine reversal, was essential to prevent clot formation on the bypass circuit and IABP balloon while managing bleeding risk.[3,7]

Systemic heparinization is not an absolute requirement for IABP support alone; however, it reduces the risk of thrombus formation on the balloon, particularly if the IABP is left deflated or at a 1:3 ratio for extended periods.[7]

3.7 Real-Time Echocardiographic Guidance

Transesophageal echocardiography (TEE) provided continuous real-time assessment of ventricular function, volume status, and IABP catheter position during the procedure. TEE is superior to pressure-based measurements for assessing left ventricular preload and performance, and it

offers visualization of the thoracic aorta for guidewire and balloon catheter confirmation without radiographic exposure.[3,7,8]

A specific advantage of TEE during CABG-IABP procedures is the detection of systolic anterior motion (SAM) of the mitral valve following IABP activation—a phenomenon that can be exacerbated by IABP-induced afterload reduction in patients with hypertrophic obstructive physiology. When SAM is detected, IABP augmentation ratio adjustment (e.g., reducing to 1:3) allows timely hemodynamic optimization.[2]

3.8 Ventricular Arrhythmia Prevention

The requirement for DC cardioversion (3 × 15 J and 1 × 20 J) during this procedure underscores the arrhythmia burden inherent to ischemic cardiomyopathy with EF 32% under cardiopulmonary bypass. Preoperative electrolyte optimization is the cornerstone of arrhythmia prevention: serum potassium 4.0–5.0 mEq/L prevents early afterdepolarizations; magnesium 2.0–2.5 mg/dL suppresses catecholamine-mediated arrhythmias and stabilizes calcium channels; and calcium within the normal range prevents action potential prolongation.[12,13]

Defibrillator availability with pre-verified pad placement (optimized relative to the sterile field), team simulation for rapid response, and multimodal prophylaxis—including rate control with beta-blockers and broad-spectrum suppression with amiodarone infusion—constitute the contemporary approach. Prophylactic lidocaine use has declined due to toxicity concerns. Biphasic defibrillation at 150–200 J with escalation protocols ensures cardioversion efficacy compatible with IABP timing.[14,15]

3.9 IABP Complications: Recognition and Management

The most common IABP complication is distal limb ischemia, occurring in >10% of cases, primarily due to femoral artery lumen compromise by the IABP or sheath. Sheathless insertion reduces this risk. Regular distal pulse assessment by palpation or Doppler is mandatory throughout support. Development of limb ischemia mandates consideration of IABP removal or contralateral arterial insertion.[7]

Thrombocytopenia from mechanical platelet destruction is an additional concern requiring daily platelet count monitoring. IABP-associated sepsis, though rare, carries high mortality and requires device removal upon positive blood cultures. Balloon rupture (evidenced by blood in the helium tubing) requires immediate deflation and removal, with the patient placed in Trendelenburg position to minimize cerebral air embolism risk; helium is an inert gas absorbed rapidly.[7]

Aortic complications including dissection and iliac artery rupture, while uncommon, are catastrophic and represent absolute contraindications to continued IABP support. IABP migration with inadvertent obstruction of the left subclavian or left common carotid artery may occur if the patient flexes the hip or assumes an upright position; regular radiographic position verification is essential.[7]

3.10 IABP Weaning

Weaning criteria include reduction of pharmacological inotropic support to moderate levels, hemodynamic stability, and patient ability to tolerate supine positioning. The standard weaning protocol involves reduction of inflation ratio from 1:1 to 1:2 for six hours, followed by further reduction to 1:3 if cardiac index $\geqslant$2.0 L/min/m², SvO_2 $\geqslant$65%, absence of angina or ST changes, stable urine

output, and minimal rise in PAWP are maintained. Heparin is discontinued and coagulation verified (platelets >90 × 10^9/L, APTT <50s) before device removal. Extended periods at 1:3 ratio (>1 hour) increase thrombus risk on the balloon and should be avoided.[7]

4. Conclusion

Anesthesia management for IABP insertion in the context of cardiogenic shock and concurrent CABG requires a highly integrated, individualized approach. Key principles include: hemodynamic-sparing induction with carefully titrated agents, invasive hemodynamic monitoring with real-time TEE guidance, optimal IABP timing using ECG or pressure triggering, a coherent inotrope/vasopressor strategy that accounts for the afterload-reducing effect of IABP itself, strict electrolyte management and defibrillator readiness for arrhythmia prevention, and vigilant monitoring for IABP-related complications including limb ischemia, thrombocytopenia, and balloon malposition.

In this patient, the combination of multimodal inotropic support, IABP-mediated afterload reduction and coronary perfusion augmentation, and mechanical ventilatory support achieved hemodynamic stabilization after complex CABG surgery with severely impaired ventricular function. Systematic integration of IABP management principles with individualized anesthetic strategy is essential to optimize outcomes in this critically ill population.

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