Management of Intra-Aortic Balloon Pumps

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Abstract

Intra-aortic balloon pumps (IABPs) continue to be the most widely used cardiac support devices with an annual estimate of 200 000 IABPs placed worldwide. IABPs enhance myocardial function by maximizing oxygen supply and minimizing oxygen demand. The use of IABPs is not without risk, with major vascular injury, ischemia, and infection being the most common complications, especially in high-risk patients. While recent studies have questioned the use of IABPs in patients with cardiogenic shock secondary to myocardial infarction, these studies have limitations making it difficult to formulate definitive conclusions. This review will focus on the mechanisms of counterpulsation, the management of IABPs and the evidence supporting this ventricular support therapy.

Keywords

counterpulsation, Intra-aortic balloon pump, cardiogenic shock, cardiac assist devices, troubleshooting intra-aortic balloon pumps, intra-aortic balloon pumps in the intensive care unit

Introduction

The intra-aortic balloon pump (IABP) is a myocardial assist device that was first introduced into clinical practice in 1968.[1] Since its introduction into medicine, the IABP has become one of the most commonly used myocardial assist devices, with an annual estimate of 200 000 worldwide[2] and 130 000 implanted devices in the United States in 2010. As the technology behind IABPs continues to evolve and the comfort level of practitioners placing these devices increases, it is imperative for clinicians to understand the principles of aortic counterpulsation, indications for using IABPs and management of any potential complications.

Machine Components and Mechanics

The main components of an IABP are the 8 to 9.5 French catheter with a 25- to 50-mL balloon (Figure 1) and the IABP console. The premise of an IABP is the utilization of counterpulsation, which involves the transfer of gas between the console and the balloon according to timing of the cardiac cycle, and will be discussed below. An inner lumen of the catheter is used to monitor the systemic arterial pressure while an outer lumen contains the gas used for balloon inflation. Helium or carbon dioxide can be used for inflation. Helium is commonly used because of its low density (5% of the density of carbon dioxide), which minimizes resistance allowing rapid inflation and deflation. Additionally, helium is metabolically inactive and readily dissolves in blood. The latter decreases the risk of air emboli formation in the event of balloon leakage or rupture. Balloon sizes are based on patient height and when fully expanded should not exceed 80% to 90% of the diameter of the descending aorta (Table 1). Some studies have recommended that inflation volumes be set to 50% to 60% of the patients ideal stroke volume.[3] Optimal inflation volumes are important to IABP augmentation as inflation volumes are directly related to the amount of blood displaced during counterpulsation.

The mechanics of counterpulsation were initially described by Dr Adrian Kantrowitz in 1953. In dog models, Kantrowitz demonstrated an increase in coronary blood flow by timed increases in the aortic diastolic pressure. Following these findings, Clauss et al[4] used an external pump to not only augment the aortic diastolic pressure during diastole but also reduce the systolic pressure and thus unload the left ventricle during systole.[5] Soon, a carbon dioxide–driven IABP that was timed with the electrocardiogram to increase the aortic diastolic

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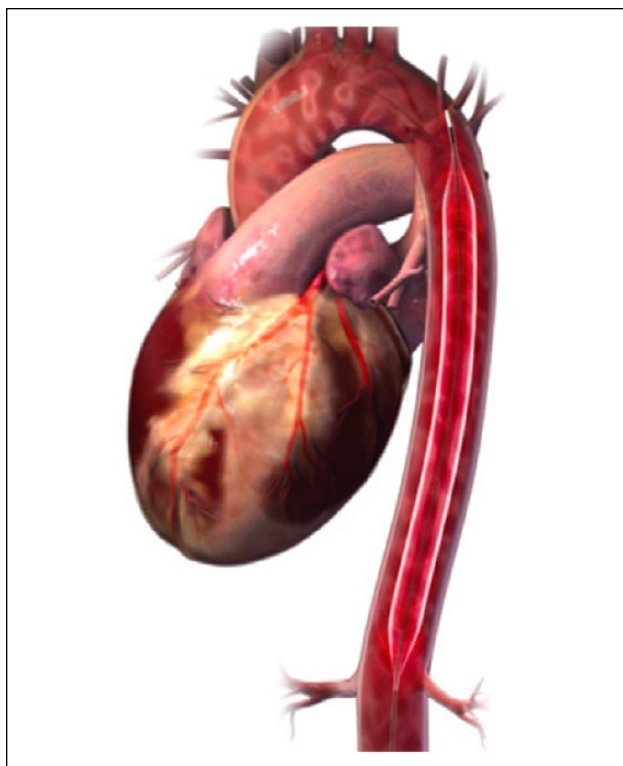
Figure 1. Optimal positioning of an intra-aortic balloon pump. Distal tip of catheter is 2 to 3 cm distal to the left subclavian artery. During inflation, there is no obstruction of the renal arteries. Reprinted with permission from Maquet Cardiovascular LLC.

pressure while also decreasing the left ventricular end-diastolic pressure was trialed[6] and laid the groundwork for the device currently used.

Hemodynamic and Physiological Effects of Arterial Counterpulsation

Counterpulsation is a technique that synchronizes the external pumping of blood with the heart's cycle to assist the circulation and to decrease the work of the heart. IABPs are triggered to inflate on closure of the aortic valve during diastole and deflate immediately before the aortic valve reopens during systole. In other words, counterpulsation pumps when the heart is resting to increase blood flow and oxygen to the heart and stops pumping when the heart is working to decrease the heart's workload and lessen oxygen demand. The IABP augments coronary perfusion pressure (CPP) by increasing aortic diastolic pressure (DBP) and decreasing the left ventricular end-diastolic pressure (LVEDP) in the form of the equation: CPP = DBP − LVEDP. The decrease in oxygen demand is because of a decrease in afterload via the active balloon deflation at the beginning of systole, increasing forward blood flow through a vacuum effect. Figure 2 illustrates an optimal

Table 1. Common Intra-Aortic Balloon Sizes Based on Patient Height.

Patient Height (cm)	Balloon Size (mL)
<152	25-30
152-163	34-40
163-183	40-50
>183	50
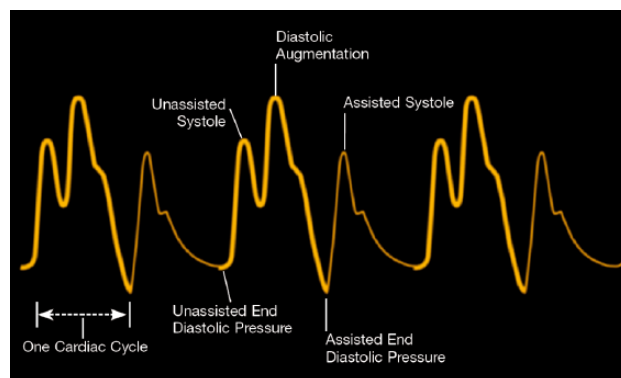
Figure 2. A pressure–time tracing of an intra-aortic balloon pump (IABP) with 1:2 augmentation. This figure illustrates optimal IAB inflation and deflation. Inflation occurs immediately after the aortic valve closes (at the dicrotic notch). A sharp "V" is noted between unassisted systole and diastolic augmentation. Diastolic augmentation should be greater than the unassisted systole. IABP deflation occurs immediately before the aortic valve opens reducing both the assisted end-diastolic pressure and assisted systolic pressure. Assisted systolic pressure should be less than the unassisted systolic pressure. Modified with permission from Maquet Cardiovascular LLC.

IABP augmentation tracing. The magnitude of the augmentation depends on several factors.

Heart Rate

The left ventricle and aortic diastolic filling times are inversely related to heart rate. As the heart rate is increased, diastolic filling times are decreased which minimizes balloon augmentation per unit of time.

Balloon Volume

The volume of gas used to inflate the balloon is directly proportional to the volume of blood displaced. In general, the balloon volume is set to 50% to 60% of the patient's stroke volume.

Aortic Compliance

Decreases in aortic compliance or systemic vascular resistance mitigate the effects of diastolic augmentation.

Preload

Ventricular preload represents the amount of stretch on the myocardium. Based on the Frank-Starling law, in normal myocardium, increased ventricular preload or myocardial stretch during diastole will result in stronger myocardial contractions during the subsequent contraction.[7] Up to a certain degree of preload, this increased stretch will result in increased cardiac output. In cases of cardiogenic shock or decreased myocardial function, excessive preload causes excessive myocardial stretch, increasing myocardial oxygen demand and ultimately decreasing cardiac output. In such cases, IABPs decrease preload up to 25% to 40% as evidenced by decreases in left atrial pressure and LVEDPs.[5,8]

Left Ventricular Performance

Initial studies by Kantrowitz et al[9] demonstrated a 25% to 40% decrease in LVEDP during IABP counterpulsation.[9] More important, this pressure decrement occurs without a decrease in cardiac output.[5] It is known that if ventricular contractility remains constant, there exists a linear relationship between end-systolic pressure and end-systolic volume.[10,11] During IABP augmentation, end-systolic pressure is decreased, corresponding to a similar reduction in left ventricular end-systolic volume. This is because of an increase in left-ventricular stroke volume and ejection fraction.[8,12]

However, the effect of an IABP on cardiac output is controversial with some studies showing only modest increases while early animal studies actually showed an insignificant decrease in cardiac out.[5,13] Since the degree of cardiac output augmentation is caused by a combination of increased CPP, decreased left ventricular preload and decreased myocardial oxygen demand, clinical studies may differ based on presence of inotropic support, degree of myocardial dysfunction, volume status and severity of the underlying myocardial injury. In practice, it is anticipated that the cardiac output increases 20% or 0.5 to 1 L/min with IABP support.[14,15]

Right Ventricular Function

The effects of IABP augmentation on the right ventricle are primarily because of the beneficial effects on left ventricular function and increased myocardial oxygen supply via increased coronary perfusion. Decreasing left atrial pressure and subsequent pulmonary vascular resistance will decrease right ventricle afterload and aid in right ventricle systole. The right coronary artery benefits from the diastolic augmentation similarly to the left coronary artery, which aids in right ventricle oxygen supply. Additionally, the IABP enhances collateral perfusion to ischemic right ventricle territory through the collateralization of right coronary branches from the left coronary circulation. Finally, by assisting the left ventricle, the septal-mediated systolic interaction between the 2 ventricles, which provides as much as 40% of the right ventricle cardiac output, is enhanced.

Aortic Pressures

Optimal synchronization of the IABP increases the arterial pressure during diastole (mean increase of 30 mm Hg in the diastolic pressure) while decreasing the arterial pressure during systole. The latter reduces left ventricular afterload and left ventricular work.[5,8] By unloading the left ventricle, the IABP reduces left ventricular wall tension and thus myocardial oxygen demand. Early studies of cardiogenic shock in dogs demonstrated a decrease in mean systolic pressures up to 26.6% with counterpulsation.[13] The decreases in systolic pressure may be related to both an increase in aortic compliance caused by balloon deflation but also direct stimulation of arterial baroreceptors.[5,16] During inflation of the balloon, the arterial baroreceptors are stimulated, causing a decrease in efferent sympathetic activity and catecholamine release from the adrenal medulla.[16,17].

Myocardial Oxygen Supply and Demand

Studies have demonstrated the effects of IABP on myocardial oxygen supply and myocardial oxygen demand by examining the diastolic pressure time index (DPTI) and the tension time index (TTI), respectively.[5,18] The DPTI is the sum of the diastolic aortic pressure less the left atrial or left ventricular pressure generated within 1 minute and is directly proportional to the amount of myocardial blood flow or oxygen delivery.[5,19] As the DPTI increases, there is a corresponding increase in myocardial blood flow and oxygen supply. During IABP augmentation, the DPTI is increased as the aortic diastolic blood pressure is increased and the left ventricular diastolic pressure is decreased.

The primary determinant of oxygen consumption by the heart is the total amount of tension generated by the myocardium as evidenced by the area under the left ventricular systolic pressure curve.[20] Thus, the TTI is defined by the sum of systolic left ventricular pressure generated within 1 minute and is a surrogate for estimating left ventricular myocardial oxygen demand. During systolic IABP augmentation, the systolic blood pressure and left ventricular afterload are decreased, which in turn decreases the tension time index.[5,18]

Effects of Intra-Aortic Balloon Pump Counterpulsation

Coronary Blood Flow

Coronary blood flow is derived from the CPP divided by the coronary vascular resistance.[5] In coronary artery disease, the coronary vascular resistance remains constant

and thus coronary blood flow is directly proportional to the perfusion pressure. Similarly, during times of ischemic injury to the myocardium, as in cardiogenic shock, vasoparalysis ensues and coronary blood flow will also be directly proportional to the coronary perfusion pressure. Thus, it has generally been assumed that the increase in coronary perfusion pressure by counterpulsation will increase coronary blood flow.[14,21,22]

However, in states of critical coronary artery stenosis, numerous studies have shown that while counterpulsation does improve flow proximal to the area of stenosis, little change in flow occurs distal to the area of obstruction.[23,24] In such situations the benefit of IABP augmentation appears to be secondary to its effects on decreasing myocardial oxygen demand.[25,26] Additionally, the increase in collateral flow observed during counterpulsation does not appear to be sufficient to improve subendocardial perfusion or reduce myocardial infarction size in animal models.[27]

Studies analyzing the effects of counterpulsation on coronary blood flow are conflicting. The heterogeneity in results may be related to the study subjects (animals vs humans), the cardiovascular physiology producing the underlying hemodynamic conditions during the IABP augmentation, and finally the methods used to quantify coronary blood flow.[8] Early animal studies demonstrated that in low flow states when coronary autoregulation was intact, no significant change in coronary blood flow occurred with counterpulsation. However, counterpulsation did increase coronary blood flow in cases where cardiac output was decreased and autoregulation was lost.[18,28]

Cerebral Blood Flow

The effects of IABP augmentation on cerebral blood flow are controversial with early animal studies of cardiogenic shock showing that cerebral blood flow increased with counterpulsation.[29] These findings were corroborated in animal models of ischemic stroke and vasospasm.[30,31] Human studies of cardiogenic shock are conflicting with some studies showing no difference in cerebral blood flow,[32] while others show that cerebral blood flow may be increased.[33] Currently, large clinical studies are lacking and make it difficult to make definitive statements on the effects of counterpulsation on cerebral blood flow.

Renal Blood Flow

Clinically, it is believed that improved cardiac output increases oxygen delivery to the kidneys and minimizes renal ischemic injury during cardiogenic shock. The increased cardiac output may increase renal blood flow up to 25%.[34] Indeed, studies using Doppler ultrasound have shown a significant increase in renal blood flow during IABP augmentation.[35]

However, complications from IABP placement such as arterial dissection, injury to the renal artery, and emboli, juxta-renal balloon position, can decrease renal blood flow and lead to renal injury. In practice, serum creatinine typically decreases following IABP initiation. If serum creatinine increases, juxta-renal balloon position or other aforementioned complications should be investigated.

Hematological Effects

During use of IABP augmentation, the shear forces created by balloon inflation and deflation may lead to a mild thrombocytopenia and hemolytic anemia. Sudden decrease in platelet level or levels <50 000 are unlikely and other causes of thrombocytopenia should be investigated.[34,36,37]

Indications for Intra-Aortic Balloon Pump

In the United States, it is estimated that IABP augmentation is used in approximately 30% of complex cardiovascular procedures.[38] However, there is much debate over the evidence behind many of the clinical settings and indications for which IABP augmentation is used. The Benchmark Registry demonstrates that the most frequent indications for IABP placement are for hemodynamic support during or after cardiac catheterization (20%), cardiogenic shock (19%), weaning from cardiopulmonary bypass (16%), preoperative use in high-risk patients (13%), and refractory unstable angina (12%).[39]

Acute Myocardial Infarction Without Cardiogenic Shock

Early animal models and clinical studies suggested that the use of an IABP may lead increased coronary blood flow and ultimately smaller infarct size and more salvageable myocardium.[40,41] However, subsequent studies have produced inconsistent results.[42] A retrospective study by Mishra et al[43] illustrated that in high-risk patients undergoing percutaneous coronary interventions (PCIs), preprocedure placement of an IABP to support hemodynamics is considered safe and may improve outcomes. Use of IABPs after PCIs for acute myocardial infarction does not appear to have the same benefits.[44,45]

Other studies have failed to corroborate these findings and suggest that there is no significant difference in outcomes for patients treated with IABPs prior to PCI versus PCI alone.[46,47] It should be noted, however, in the "Counterpulsation to Reduce Infarct Size Pre-PCI (CRISP)" trial by Patel et al,[46] 15 patients (8.5%) in the PCI-alone group crossed over for rescue IABP therapy. From this group, 4/15 died within 30 days.[46] The authors

ultimately concluded that rescue IABP therapy should be available for high-risk patients undergoing PCI.[46]

Current ACC/AHA (American College of Cardiology/American Heart Association) guidelines do not comment on the use of IABPs for patients with acute myocardial infarction without cardiogenic shock.[48] Given the lack of definitive evidence, prophylactic use of IABPs in stable patients with acute myocardial infarction is not recommended.

Acute Myocardial Infarction With Cardiogenic Shock

Patients presenting with acute myocardial infarction complicated by cardiogenic shock have mortality rates as high as 30% to 75%.[49] Previous studies and meta-analyses have suggested that the use of IABPs in this particular patient population may improve outcomes.[49,50] Current ACC/AHA guidelines state that the use of IABP may be warranted for patients who do not stabilize on pharmacotherapy (class IIa, level of evidence B).[48]

One of the largest studies addressing this question, the "Intra-aortic Balloon Support for Myocardial Infarction with Cardiogenic Shock" (IABP-Shock-II) Trial, concluded that the use of IABPs for patients with myocardial infarction complicated by cardiogenic shock does not reduce 12 month mortality.[51] However, there are several points to consider when interpreting the results of the IABP-Shock II trial. First, the authors did not include pertinent clinical information in regards to how quickly the patients underwent revascularization, the hemodynamic status of the patients before and after IABP placement, and timing of IABP insertion. With regard to the latter, only a small percentage of patients received IABPs prior to myocardial revascularization. Some may argue that the effects of IABP augmentation may be greater when inserted prior to reperfusion.[2,52]

Other limitations of the trial are related to study design and include a small sample size that may not have been adequately powered to assess for survival, excluding mechanical causes of cardiogenic shock, and the fact that left ventricular assist device use was not controlled. Twice the number of patients in the control group required placement of a left ventricular assist device for additional support (7.5% vs 3.7%), which may have improved the mortality rate in the control group. With regard to the exclusion criteria, some studies have shown that early use of IABPs in patients with postischemic mechanical causes of cardiogenic shock may improve survival.[53] It is possible that the results may have been different had this group not been excluded. Additionally, there was a fairly high crossover rate to the IABP group (10%), which may have affected results. Interestingly, 4 patients from this group crossed over due to the development of postischemic mechanical complications. This study has also been criticized for the high use of catecholamines and the fact that relatively few patients actually had a systolic blood pressure <90 mm Hg prior to randomization.

High-Risk Patients Undergoing Coronary Interventions

The term *coronary intervention* can mean either PCIs or coronary artery bypass grafting (CABG) when analyzing studies examining this topic. High-risk patients are defined as those with pharmacologically uncontrollable angina, patients with severe left ventricular dysfunction, patients with left main coronary disease, patients undergoing multivessel angioplasty, prior history of CABG surgery, and/or age >70 years.[43,54] For patients undergoing PCI, initial single center, nonrandomized studies demonstrated that prophylactic use of IABPs (vs rescue IABP or conservative therapy) in high-risk patients undergoing PCI was associated with fewer complications and lower mortality.[43,55] However, the subsequent randomized controlled Balloon Pump-Assisted Coronary Intervention Study (BCIS-I) essentially found no differences in major adverse cardiac and cerebrovascular events (MACCE) in high-risk patients treated with elective IABP prior to PCI compared with those who underwent PCI without planned IABP support.[56] Long-term all-cause mortality data 51 months later showed that patients treated with elective IABP placement had a 34% (P = .039) reduction in risk of death.[57] Based on these findings, the current ACC/AHA guidelines recommend that elective insertion of an IABP as an adjunct to PCI may be reasonable in high-risk patients (class IIb, level of evidence C).[58]

Acute Right Ventricular Infarction With Shock

For patients with cardiogenic shock secondary to right ventricular failure, immediate hemodynamic improvements due to the augmentation provided by the IABP can be seen.[59] The beneficial effects on RV performance provided by the IABP are because of mechanisms discussed earlier. These findings can likely be extrapolated to include patients with RV failure leading to inability to liberate from cardiopulmonary bypass.

Patients Undergoing Coronary Artery Bypass Graft Surgery

For patients undergoing CABG surgery, the preoperative use of IABPs has been studied, however large-scale randomize controlled trials are lacking. A recent Cochrane review from 2011 that included 6 trials (5 of which were from the same institution and author) suggested that preoperative placement of IABPs may reduce morbidity and mortality in high-risk patients undergoing CABG surgery.[60] Decreased duration on cardiopulmonary bypass as well as

Table 2. Intra-Aortic Balloon Pump Indications and Contraindications.

Indications	Contraindications
Cardiogenic shock	Aortic regurgitation
Acute myocardial infarction	Aortic dissection/aneurysm
Preoperative stabilization for:	Patients with multiple organ failure with no anticipated recovery
Coronary artery bypass graft	
Noncardiac surgery	
Acute mitral regurgitation ventricular septal defect	Aortic stents or grafts
Weaning from cardiopulmonary bypass	Septic shock
Bridge to transplantation	Severe peripheral vascular disease or disease and/or previous arterial stent at potential sites of cannulation
Hemodynamic stability during percutaneous coronary intervention	Patient refusal or do not resuscitate instructions

Figure 3. An anterior–posterior chest radiograph demonstrates a properly positioned intra-aortic balloon pump tip (red arrow) sitting directly inferior to the left subclavian artery take-off and at the level of the carina.
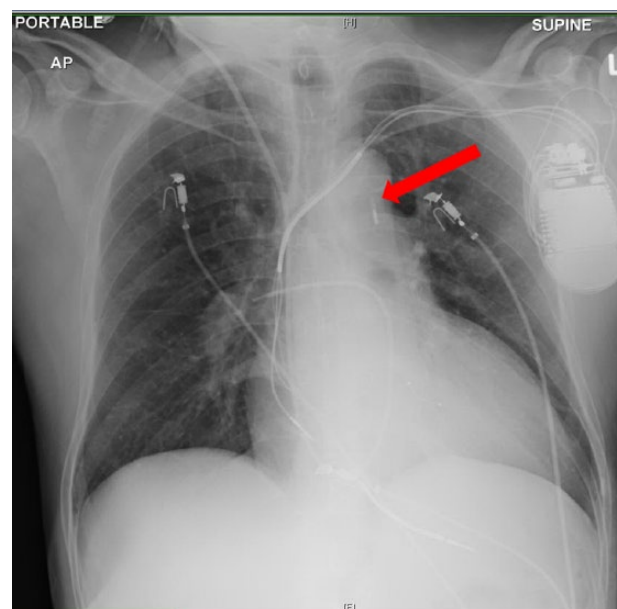
shorter length of stay in the intensive care units has also been described for patients in whom IABPs were placed preoperatively.[61] Other IABP indications and contraindications are listed in Table 2.

Contraindications to Intra-Aortic Balloon Pump

Notably, in patients with aortic regurgitation, IABP inflation during diastole will worsen the degree of regurgitation, increase myocardial wall stress and decrease CPP and DPTI. For patients with known or suspected aortic dissections (ascending or descending), placement of an IABP in the false lumen can lead to further dissection or aortic rupture (Table 2).

Insertion of Intra-Aortic Balloon Pumps

Percutaneous IABP insertion commonly occurs at the femoral artery using a Seldinger, over the wire, technique. Percutaneous insertion through the brachial and radial arteries has also been described.[62,63] When comparing surgical (transthoracic, translumbar, iliac, subclavian, or axillary artery) versus percutaneous approaches, the results are inconclusive with some studies showing improved outcomes with the percutaneous approach while others have suggested decreased adverse events using a surgical approach.[64-67] The limb with the strongest pulse and largest artery should be utilized. Optimal positioning is confirmed with imaging with the balloon tip in the descending

thoracic aorta, 2 to 3 cm distal to the subclavian artery (Figure 1). On chest radiograph (Figure 3), optimal position will be at the level of the carina or between the second and third intercostal spaces.[8,68] Proper positioning can also be ascertained by transesophageal echocardiography imaging. If the IABP is inserted too far, there is risk of subclavian and vertebral artery occlusion. Alternatively, if the IABP is not inserted far enough, mesenteric and renal artery occlusion can occur.

Management of Intra-Aortic Balloon Pumps

Physician and nursing education is a key component of mitigating IABP related complications (Table 3). In general, adjustments will depend on the initial indications for IABP placement. For patients in whom the IABP is placed to augment coronary blood flow, inflation and deflation times will be adjusted in order to optimize the DPTI or myocardial oxygen delivery. However, in patients with impaired left ventricular function where the goal is to minimize ventricular wall stress and left ventricular afterload, the primary focus will be to optimize the timing of deflation. In such cases, the goal is to minimize presystolic pressures.

Console

While the exact console interface will vary depending on the type of IABP, the general components are an internal

Table 3. Points of Care for Patients With Intra-Aortic Balloon Pumps.

Necessary Evaluations	Other Considerations
• Hourly vital signs (from the console and bedside monitor) and urine output • Mean arterial pressure • Peak unassisted systolic pressure • Peak diastolic augmented pressure • Balloon aortic end-diastolic pressure • Documentation of peripheral pulses to rule out ischemia or compartment syndrome • Pain (disproportionate) • Pallor • Pulselessness • Poikiolothermia • Paresthesia • Paralysis • Monitor for signs of balloon rupture • Monitor for signs of bleeding in anticoagulated patients • Inspect catheter site for oozing or infection • Daily laboratory tests • Complete blood count • Basic metabolic panel + arterial lactate • Prothrombin time/partial thromboplastin time/ international normalized ratio • Bed should always be <30°. For intubated patients, ventilator-associated pneumonia protocol can still be performed with reverse Trendelenburg position • Do not use percussion vibration modules when intra-aortic balloon pump is in place. • Adequate patient sedation and analgesia. • Log roll patient when changing bed sheets • Never leave intra-aortic balloon pump in standby mode except for short periods (<3 minutes) while evaluating wean and/or pulling	• Sudden changes in augmented pressure may suggest incorrect inflation and deflation timing • Daily chest radiograph to monitor for migration • Decreased urine output may suggest catheter migration and obstruction of the renal arteries • Decreased peak augmented diastolic pressure suggests distal catheter migration • Loss of left radial pulse suggests proximal catheter migration and obstruction of subclavian artery • Abdominal distention, bloody stools or abdominal hypertension suggests mesenteric ischemia • Blood in helium tubing or intra-aortic balloon failure suggests balloon rupture. If rupture confirmed: • Support hemodynamics • Stop intra-aortic balloon, clamp helium line to prevent gas embolism and prepare to remove balloon • Anemia (hemolysis) and thrombocytopenia are common. Never take blood samples from intra-aortic balloon pump pressure line

computer, monitor and touch pad. The operator must set the desired augmentation ratio. The trigger source and balloon inflation and deflation timing may also be adjusted by the user or the device can set these parameters based on aortic flow calculations on a beat-to-beat basis. The inner lumen of the IABP connects to the fluid transducer that allows pressures to be displayed.

Electrocardiogram (ECG) leads are also directly connected to the console and are used to detect cardiac rhythms as an additional tool in order to optimize balloon inflation and deflation. The console also has multiple alarms to detect leaks within the circuit, blood in the gas line, loss of gas in the outer lumen, and loss of trigger signals.

Intra-Aortic Balloon Pump Triggers

Electrocardiography Trigger. Physiologically, inflation and deflation of the IABP will coincide with the closing and opening of the aortic valve, respectively. The IABP will be triggered to deflate during systole once the peak of the R wave is sensed. IABP inflation will be triggered to occur in the middle of the T wave, which corresponds with diastole. The sensing of the R wave (sensed by height and slope) is dynamic and specifics will vary between manufacturers. During cases of poor ECG quality or arrhythmias, the ECG triggering mode becomes less reliable and other modes may be needed to ensure optimal IABP augmentation.

Table 4. Common Intra-Aortic Balloon Pump Modes of Operation.

Automatic	Semiautomatic	Manual
• Computer sets trigger source	• Operator sets trigger source	• Operator sets trigger source
• Computer sets inflation and deflation times	• Operator sets initial inflation and deflation times. Computer will determine subsequent timing intervals	• Operator sets inflation and deflation times
• Trigger automatically searches for most optimal source if initial trigger is not detected	• Optimal ECG leads: I, II, III, AVR, AVL, AVF, V	• Timing must be adjusted based on underlying heart rate and rhythm
• Optimal ECG leads: I, II, III		• Optimal ECG leads I, II, III, AVR, AVL, AVF, V

Pressure Trigger. The pressure trigger mode is often used in situations of poor ECG quality or inability of the IABP to appropriately sense left ventricular systole and diastole. During the pressure mode, the arterial pressure waveform is used to trigger IABP inflation and deflation. As previously stated, IABP augmentation coincides with opening and closing of the aortic valve. On the pressure waveform, aortic valve closure corresponds with the dicrotic notch while aortic valve opening corresponds to the point immediately before the systolic upstroke.

Internal Trigger (Operator Mode). The internal trigger mode is an asynchronous trigger mode that is usually set to augment at a heart rate of 80 beats/min (range 40-120 beats/min). This mode is only to be used in situations where there is no cardiac output and no ECG, such as cardiac arrest or cardiopulmonary bypass. During the latter, there is literature to suggest that pulsatile flow may improve whole body perfusion.[69,70]

Pacer Triggers. Pacer ventricular (V)/Atrioventricular (AV) trigger mode initiates IABP augmentation based on the ventricular spike. The V pacing and AV pacing modes are set to fixed rates and should not be used for demand pacing. To use the V pacing and AV pacing trigger modes, the patient should have a 100% paced rhythm.

Atrial Fibrillation Trigger. Atrial fibrillation trigger mode is found on some IABP consoles. During this mode, computer will analyze the height and slope of both a positively or negatively deflected QRS complex. The pump will determine the deflation time by sensing an R wave. This mode also rejects pacer spikes and artifact.

Modes of Operation

Modes of operation will vary depending on manufacturer. Common modes include automatic, semiautomatic, and manual (Table 4). In general, during automatic mode, the IABP computer will determine the most optimal trigger source and inflation/deflation timing will automatically be

set. Modern devices use fiber-optic technology (as compared with older models, which rely on fluid transducers) to automatically calibrate and recalibrate balloon inflation and deflation. Most devices can match inflation of the balloon to within 12 milliseconds of the time of aortic valve closure even with severe arrhythmias. Figure 2 demonstrates optimal balloon inflation and deflation pressure tracings.

Suboptimal inflation and deflation times can have significant negative hemodynamic consequences (Figure 4). Table 5 illustrates the waveform characteristics and potential physiological effects from inappropriate balloon pumping timing. Of note, the IABP must be placed in 1:2 or less augmentation in order to compare unassisted to assisted waveforms.

During semiautomatic mode, the operator will select the appropriate trigger and initial inflation/deflation timing. Thereafter, the device will automatically determine timing based on heart rate and rhythm changes. During manual mode, the operator sets both the trigger source and inflation/deflation times and must adjust them for changes in heart rate or rhythm.

Weaning the Patient From Intra-Aortic Balloon Pump Support

As the patient's hemodynamic status improves, weaning the IABP should be considered. Weaning is typically done in a staged fashion (as opposed to abrupt discontinuation) over several hours by gradually decreasing the ratio of augmentation from 1:1 to 1:2 to 1:3, or 1:4. Some devices offer a 1:8 setting; however, this setting likely need not be used as it carries a high risk of thrombosis and adequate weaning information can be gained from 1:3 augmentation. Of note, it is not recommended to remain on settings of 1:3, 1:4, and especially 1:8 for prolonged periods of time (>30 minutes) because of high risk of catheter thrombosis. In fact, it is advisable to systemically anticoagulate patients who require these settings for a sustained (multiple hours to days) weaning trial.

Once in a 1:2 or 1:3 setting for approximately 30 minutes, data pertaining to cardiac function (mixed venous,
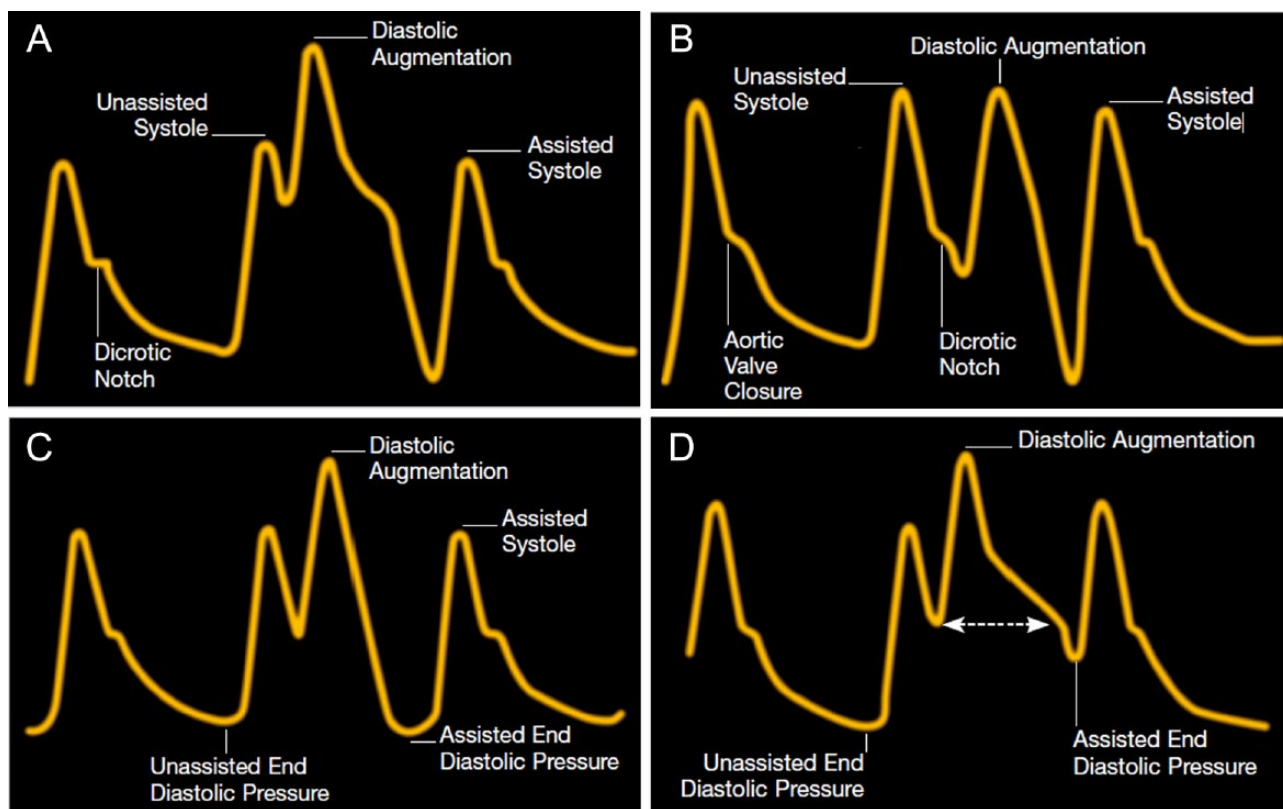
Figure 4. Intra-aortic balloon pump (IABP) timing errors: This figure illustrates the differing timing errors that can occur with inappropriate IABP inflation and deflation. Early inflation, the IABP inflates prior to the aortic valve closure (before the dicrotic notch; A). During late inflation, the IABP inflates after the aortic valve closure (after the dicrotic notch; B). Early deflation, the IABP deflates during diastole (C). Late deflation, the IABP deflates while the aortic valve is opened (during systole; D). Modified with permission from Maquet Cardiovascular LLC.

echocardiography, cardiac indices, pulmonary capillary occlusion pressure, serum lactate, and urine output) should be monitored and compared with pre-weaning values. Inotropic support may need to be added or increased during weaning. Typically, the systolic blood pressure will increase as support is weaned due to lack of afterload reduction; however, this is not a sensitive parameter for weaning an IABP.

If weaning parameter goals are not achieved or the patient demonstrates worsening cardiac function, the IABP can be placed back into a 1:1 augmentation setting and the weaning trial can be repeated when the patient has again shown signs of improvement or perhaps with addition of inotropy. After multiple failed weaning attempts, consideration of another form of ventricular assist device is warranted.

If it is determined that the balloon pump can be safely removed, coagulation status (international normalized ratio, partial thromboplastin time, platelet count) should be within normal or near-normal levels prior to removal. Removal of an IABP can have very deleterious results. Approximately, half of all vascular injuries associated with IABP use occur at the time of placement or removal.[71] Hematomas at the site, pseudoaneurysm formation and retroperitoneal bleed

can all have dire consequences for these patients. IABP insertion site holding pressure manually should be done with a "no peek" technique for approximately 45 minutes. Stabilizing pressure devices may be instituted in some centers after manual compression. During this time, it is prudent to monitor lower extremity pulses to ensure circulation is maintained with the compression. Other details concerning removal are presented in Table 6.

Systemic Anticoagulation

There are no standardized recommendations for anticoagulating patients with IABPs, assuming the IABP is not left in a 1:3 or less augmentation mode for long periods of time.[72] It is likely best practice to individualize antithrombotic therapy on a patient by patient basis.[73] If the patient is considered at risk of thrombosis or if the IABP is planned to be left in weaning mode (1:3 or less augmentation) for greater than approximately 30 minutes, systemic anticoagulation should be considered. As a rule, the IABP should never be left in stand-by mode (no inflations) unless it is being removed. Nonheparinized animal models have demonstrated clot

Table 5. Inappropriate Timing of Intra-Aortic Balloon (IAB) Inflation and Deflation.

	Too Early	Too Late

Inflation	IAB inflates before the aortic valve closes:
	Waveform characteristics
	• IAB inflates prior to dicrotic notch
	• Diastolic augmentation encroaches onto systole
	
	Potential physiological effects
	• Premature closure of the aortic valve
	• Aortic regurgitation
	• Decreased:
	  • Cardiac output
	• Increased:
	  • Left ventricular afterload
	  • Myocardial oxygen demand
	  • Left ventricular end-diastolic pressure
	  • Left ventricular end-diastolic volume
	  • Pulmonary capillary wedge pressure
		IAB inflates late in the diastolic cycle:
		Waveform characteristics
		• IAB inflates after the dicrotic notch
		• Absence of sharp V
		• Suboptimal diastolic augmentation
		Potential physiological effects
		• Suboptimal coronary artery perfusion
		• Decreased diastolic pressure time index

Deflation	IAB deflates before the end of diastole: Waveform characteristics • Deflation of IAB is seen as a sharp drop following diastolic augmentation • Suboptimal diastolic augmentation • Assisted aortic end diastolic pressure may be equal to or less than the unassisted aortic end diastolic pressure • Assisted systolic pressure may rise Potential physiological effects • Suboptimal coronary artery perfusion • Potential for retrograde coronary and carotid blood flow • Suboptimal afterload reduction • Increased myocardial oxygen demand • Decreased diastolic pressure time index	IAB inflates during a portion of the systolic cycle: Waveform characteristics • Assisted aortic end diastolic pressure may be equal to the unassisted aortic end diastolic pressure • Rate of rise of assisted systole is prolonged • Diastolic augmentation may appear widened Potential physiological effects • Afterload reduction is essentially absent • Increased myocardial oxygen consumption because of the left ventricle ejecting against a greater resistance and a prolonged isovolumetric contraction phase • Impeded left ventricular ejection and increased left ventricular afterload

formations when the balloon remains deflated for greater than 20 minutes.[74] The basis for anticoagulation is because of the risk of local clot formation around the balloon in the aorta, which could have dire consequences of systemic embolization. Additionally, systemic anticoagulation may potentially decrease the incidence of limb ischemia because of slower blood flow in limb with the IABP cannula.

Intra-Aortic Balloon Pump Troubleshooting and Complications

Table 7 lists common problems that can occur with an IABP. If the IABP ceases to offer assistance, inotropic and vasopressor agents should be readily available while the troubleshooting takes place. There are multiple complications which can be categorized into 2 main groups: vascular and IABP related (Table 8). Based on data from the Benchmark Registry and other observational studies, the most common complications are vascular injury, infection, and balloon malfunction.[38,39,75,76] Major risk factors for developing IABP related complications are listed in Table 9.[15] With regard to ischemic limb injury, common causes include emboli, dissection, or occlusion during balloon inflation. Patients should routinely be monitored for signs of limb ischemia by pulse palpation as well as end organ ischemia. Adequate patient sedation and analgesia are necessary to minimize the risk of device migration, cannula kinking, or removal of the cannula by the patient.

Sheathless balloon insertion in addition to smaller balloon and catheters sizes have been advocated by some experts as a way to help decrease the incidence of vascular related complications.[77] In patients at significant risk for developing limb ischemia– or vascular-related complications (diabetes mellitus, peripheral vascular

Table 6. Weaning Intra-Aortic Balloon Pump (IABP) Support.

Commonly Used Criteria to Identify Patients Ready for IABP Weaning	Weaning Strategies
Resolution (or resolving) of cardiogenic shock • Little to no pharmacological inotropic support	Weaning (completed over 30 minutes to several hours) • Monitor pulmonary capillary wedge pressure, cardiac indices, SvO_2, arterial lactate, urine output, echocardiography
• Check heart function on transthoracic echocardiography/transesophageal echocardiography • Down trending arterial lactate	Augmentation ratio • 1:1 → 1:2 → 1:3. Once at 1:3, monitor hemodynamics for approximately 30 minutes. (Minimal augmentation settings of 1:4 and 1:8 should be used with caution for prolonged periods without systemic anticoagulation because of high risk of catheter thrombosis)
• Cardiac index >2 L/min/m^2 • SvO_2 >65%	Removal: • Discontinue heparin infusion • Check prothrombin time/partial thromboplastin time/international normalized ratio and platelets (>50 000)
Resolution of vasodilatory shock • Little to no pharmacological pressor support	• Supplies: Suture removal kit, pressure dressing • Turn off balloon pump (balloon should deflate automatically)
• Mean arterial pressure >65 mmHg Heart rate <100 beats/min	• For added safety: Can ensure collapse by manual deflation with a 60-mL syringe • Pull out balloon and allow a couple beats of bleed in order to flush out any clots

Urine output >0.5 mL/kg/h	• Apply pressure for ≥45 minutes ("no peek")
	• Check foot pulses intermittently while holding pressure
	• Apply pressure dressing
	• Monitor for limb ischemia or hematoma for 24 hours
	• Patient should remain flat for 6 hours
	• Consider operative removal for catheters in place for >3-5 days
	• Balloon entrapment
	  ○ Conservative:
	    • Intraluminal streptokinase with heparinized saline flush
	  ○ Surgical removal:
	    • Computed tomography angiogram and vascular surgery consult

disease), the use of a sheathless introducer may decrease the risk of limb ischemia.[78]

Duration of Intra-Aortic Balloon Pump Treatment

One study followed patients with ST elevation myocardial infarctions in whom an IABP was placed for hemodynamic stability after PCI. In this group of patients, IABP use for greater than 2 days resulted in a significant increase in complications (vascular injury, bleeding, gastrointestinal bleeding) as compared with those in whom the IABP was in place for less than 2 days (66% vs 18%; $P < .05$).[79]

However, in studies analyzing the use of IABPs as a bridge to heart transplantation, patients treated with IABPs had few IABP related complications and no difference in mortality when compared with a control group. Long-term survival rates up to 1 year were also similar between the 2 groups. The average number of IABP treated days was 21 with some patients requiring IABP replacement.[80] Currently, there are no specific recommendations concerning optimal duration of IABPs.

Table 7. Management of Common Intra-Aortic Balloon Pump (IABP) Problems.

Common Console Alarms	Management
No trigger	Unable to sense trigger mode • Switch to alternative mode (electrocardiography or pressure) • Reconnect electrocardiogram leads or pressure cable
IABP disconnected	The IABP extension tubing has been disconnected • Reconnect tubing • Refill the IAB with gas • Restart IABP
Rapid gas loss	Leak within the IAB circuit • Check Extension Tubing • Potential hole or leak in IAB • Blood in tubing suggests IAB rupture and IABP should be removed
Check IAB catheter	Kink in IAB catheter or tubing • Relieve any kinks IAB remains within sheath • Check IAB markings; Consider withdrawing sheath Balloon does not completely unfold. • Ensure full augmentation is on. Consider manual inflation and deflation of balloon
Low helium	Critically low levels of helium. Less than 24 IAB fills remaining • Replace cylinder. Make sure yoke is fully tightened
IABP failure	Failure within the IABP console/computer • Rule out electrical malfunction or blood in the condensers
Augmentation below limit set	Alarm limit set too high • Lower alarm limit. Sudden change in hemodynamics: mean arterial pressure, stroke volume, heart rate • Treat accordingly and consider adjusting limit • Search for signs of balloon displacement

Table 8. Complications of Intra-Aortic Balloon Pump (IABP) Treatment.

Vascular	IABP Related
Ischemia • Visceral (bowel, renal) • Extremity • Compartment syndrome • Spinal cord Ischemia • Cerebrovascular accident Arterial injury • Dissection • Aneurysm/pseudoaneurysm • Laceration • Hemorrhage • Hematoma Cardiac tamponade Thrombosis Accidental femoral vein cannulation	Balloon rupture (secondary to calcified plaques) • Gas embolism • Hematological • Hemolysis • Thrombocytopenia Balloon entrapment Infection/sepsis

Table 9. Risk Factors for Intra-Aortic Balloon Pump Complications.

Major	Minor
Peripheral vascular disease	Age >70 years
Diabetes mellitus	Smoking
Female gender	Low cardiac output
Sheathed catheter	Cardiogenic shock
	Obesity
	Inotropic support
	Hypertension
	Increased systemic vascular resistance
	Ankle-brachial pressure index <0.8

Intra-Aortic Balloon Pump With Dysrythmias and Advanced Cardiac Life Support

For patients with tachycardia, augmentation can be hindered by insufficient balloon filling time. If IABP augmentation is necessary, treatment should be aimed at slowing the heart rate. During atrial fibrillation, the IABP should set to trigger in the atrial fibrillation mode (or, secondarily, ECG mode), which will automatically deflate the balloon when an R wave is sensed, despite R-R variability.

During cardiac arrest, the IABP should be placed on the pressure trigger mode and the pressure threshold should be reduced. This will improve the ability of the IABP to synchronize augmentation during chest compressions. ECG trigger mode can also be used if the patient has QRS complexes. If chest compressions are unable to trigger the IABP during pressure or ECG mode is not applicable, then the IABP can be placed on standby but should not be left in this mode for longer than the arrest due to risk of thrombosis. The internal trigger mode can also be used but may lead to asynchronous counterpulsation with return of cardiac function. During defibrillation, it is important to ensure that all staff are also clear from the IABP console or any the connections. The IABP can be continued during defibrillation.

Intra-aortic balloon pumps have been used with success in patients on extracorporeal membrane oxygenation. A recent retrospective analysis of 54 patients with acute heart failure treated with both venoarterial extracorporeal membrane oxygenation and an IABP illustrated a potential synergistic effect as evidenced by improvement in mean arterial pressure, central venous pressure, arterial lactate levels, and mixed venous oxygen saturation.[81]

Limitations of Intra-Aortic Balloon Pumps

Unlike other cardiac assist devices, IABPs indirectly affect left ventricular function by maximizing myocardial oxygen supply and minimizing myocardial oxygen demand.

This is in comparison with a ventricular assist device that acts as a pump to support the heart and promote forward flow. In cases of severe cardiogenic shock where the ejection fraction is <20%, ventricular assist devices may be needed to ensure systemic perfusion.

Summary

Intra-aortic balloon pumps continue to be the most widely used cardiac support devices. Through counterpulsation, IABPs indirectly affect myocardial function by maximizing oxygen supply and minimizing myocardial oxygen demand, thus benefitting many patients in cardiac extremis. While multiple myocardial assist devices exist, large, randomized controlled trials are yet to determine the most optimal device in specific situations. In select patient populations, IABPs may provide the life-sustaining bridge to a more definitive therapy.

References

1. Kantrowitz A, Tjonneland S, Krakauer J, et al. Clinical experience with cardiac assistance by means of intraaortic phase-shift balloon pumping. *Trans Am Soc Artif Intern Organs*. 1968;14:344-348.
2. Kapelios CJ, Terrovitis JV, Nanas JN. Current and future applications of the intra-aortic balloon pump. *Curr Opin Cardiol*. 2014;29:258-265.
3. Booker PD. Intra-aortic balloon pumping in young children. *Paediatr Anaesth*. 1997;7:501-507.
4. Clauss RH, Birtwell WC, Albertal G, et al. Assisted circulation. I. The arterial counterpulsator. *J Thorac Cardiovasc Surg*. 1961;41:447-458.

5. Nanas JN, Moulopoulos SD. Counterpulsation: historical background, technical improvements, hemodynamic and metabolic effects. *Cardiology*. 1994;84:156-167.

6. Moulopoulos SD, Topaz S, Kolff WJ. Diastolic balloon pumping (with carbon dioxide) in the aorta—a mechanical assistance to the failing circulation. *Am Heart J*. 1962;63:669-675.

7. Lappas DG, Powell WM Jr, Daggett WM. Cardiac dysfunction in the perioperative period: pathophysiology, diagnosis, and treatment. *Anesthesiology*. 1977;47:117-137.

8. Trost JC, Hillis LD. Intra-aortic balloon counterpulsation. *Am J Cardiol*. 2006;97:1391-1398.

9. Kantrowitz A, Krakauer J, Zorzi G, et al. Current status of intraaortic balloon pump and initial clinical experience with aortic patch mechanical auxiliary ventricle. *Transplant Proc*. 1971;3:1459-1471.

10. Sagawa K, Suga H, Shoukas AA, Bakalar KM. End-systolic pressure/volume ratio: a new index of ventricular contractility. *Am J Cardiol*. 1977;40:748-753.

11. Suga H, Sagawa K. Instantaneous pressure-volume relationships and their ratio in the excised, supported canine left ventricle. *Circ Res*. 1974;35:117-126.

12. Kapelios CJ, Terrovitis JV, Siskas P, Kontogiannis C, Repasos E, Nanas JN. Counterpulsation: a concept with a remarkable past, an established present and a challenging future. *Int J Cardiol*. 2014;172:318-325.

13. Talpins NL, Kripke DC, Goetz RH. Counterpulsation and intraaortic balloon pumping in cardiogenic shock. Circulatory dynamics. *Arch Surg*. 1968;97:991-999.

14. Scheidt S, Wilner G, Mueller H, et al. Intra-aortic balloon counterpulsation in cardiogenic shock. Report of a co-operative clinical trial. *N Engl J Med*. 1973;288:979-984.

15. de Waha S, Desch S, Eitel I, et al. Reprint of "Intra-aortic balloon counterpulsation—Basic principles and clinical evidence". *Vascul Pharmacol*. 2014;60:52-56.

16. Mullins CB, Sugg WL, Kennelly BM, Jones DC, Mitchell JH. Effect of arterial counterpulsation on left ventricular volume and pressure. *Am J Physiol*. 1971;220:694-698.

17. Normann NA, Kennedy JH. Arterial baroreceptor responses to intraaortic balloon assistance. *J Surg Res*. 1971;11:396-400.

18. Parissis H. Haemodynamic effects of the use of the intraaortic balloon pump. *Hellenic J Cardiol*. 2007;48:346-351.

19. Mueller H, Ayres SM, Conklin EF, et al. The effects of intra-aortic counterpulsation on cardiac performance and metabolism in shock associated with acute myocardial infarction. *J Clin Invest*. 1971;50:1885-1900.

20. Sarnoff SJ, Braunwald E, Welch GH Jr, Case RB, Stainsby WN, Macruz R. Hemodynamic determinants of oxygen consumption of the heart with special reference to the tension-time index. *Am J Physiol*. 1958;192:148-156.

21. Willerson JT, Curry GC, Watson JT, et al. Intraaortic balloon counterpulsation in patients in cardiogenic shock, medically refractory left ventricular failure and/or recurrent ventricular tachycardia. *Am J Med*. 1975;58:183-191.

22. O'Rourke MF, Sammel N, Chang VP. Arterial counterpulsation in severe refractory heart failure complicating acute myocardial infarction. *Br Heart J*. 1979;41:308-316.

23. Yoshitani H, Akasaka T, Kaji S, et al. Effects of intra-aortic balloon counterpulsation on coronary pressure in patients with stenotic coronary arteries. *Am Heart J*. 2007;154: 725-731.

24. Kern MJ, Aguirre F, Bach R, Donohue T, Siegel R, Segal J. Augmentation of coronary blood flow by intra-aortic balloon pumping in patients after coronary angioplasty. *Circulation*. 1993;87:500-511.

25. Williams DO, Korr KS, Gewirtz H, Most AS. The effect of intraaortic balloon counterpulsation on regional myocardial blood flow and oxygen consumption in the presence of coronary artery stenosis in patients with unstable angina. *Circulation*. 1982;66:593-597.

26. Lefemine AA, Low HB, Cohen ML, Lunzer S, Harken DE. Assisted circulation. III. The effect of synchronized arterial counterpulsation on myocardial oxygen consumption and coronary flow. *Am Heart J*. 1962;64:789-795.

27. Muller KD, Lubbecke F, Schaper W, Walter P. Effect of intraaortic balloon counterpulsation (IABP) on myocardial infarct size and collateral flow in an experimental dog model. *Intensive Care Med*. 1982;8:131-137.

28. Powell WJ Jr, Daggett WM, Magro AE, et al. Effects of intra-aortic balloon counterpulsation on cardiac performance, oxygen consumption, and coronary blood flow in dogs. *Circ Res*. 1970;26:753-764.

29. Bhayana JN, Scott SM, Sethi GK, Takaro T. Effects of intraaortic balloon pumping on organ perfusion in cardiogenic shock. *J Surg Res*. 1979;26:108-113.

30. Nussbaum ES, Heros RC, Solien EE, Madison MT, Sebring LA, Latchaw RE. Intra-aortic balloon counterpulsation augments cerebral blood flow in a canine model of subarachnoid hemorrhage-induced cerebral vasospasm. *Neurosurgery*. 1995;36:879-884.

31. Tranmer BI, Peniston C, Iacobacci R, Salerno TA, Hudson AR. Intra-aortic balloon counterpulsation: a treatment for ischaemic stroke? *Neurol Res*. 1989;11:109-113.

32. Cheung AT, Levy WJ, Weiss SJ, Barclay DK, Stecker MM. Relationships between cerebral blood flow velocities and arterial pressures during intra-aortic counterpulsation. *J Cardiothorac Vasc Anesth*. 1998;12:51-57.

33. Pfluecke C, Christoph M, Kolschmann S, et al. Intra-aortic balloon pump (IABP) counterpulsation improves cerebral perfusion in patients with decreased left ventricular function [published online March 7, 2014]. *Perfusion*. doi:10.1177/0267659114525218.

34. Krishna M, Zacharowski K. Principles of intra-aortic balloon pump counterpulsation. *Contin Educ Anaesth Crit Care Pain*. 2009;9:24-28.

35. Sloth E, Sprogoe P, Lindskov C, Horlyck A, Solvig J, Jakobsen C. Intra-aortic balloon pumping increases renal blood flow in patients with low left ventricular ejection fraction. *Perfusion*. 2008;23:223-226.

36. Parissis H, Soo A, Al-Alao B. Intra aortic balloon pump: literature review of risk factors related to complications of the intraaortic balloon pump. *J Cardiothorac Surg*. 2011;6:147.

37. Leinbach RC, Nyilas E, Caulfield JB, Buckley MJ, Austen WG. Evaluation of hematologic effects of intra-aortic balloon assistance in man. *Trans Am Soc Artif Intern Organs*. 1972;18:493-500, 512.

38. Cohen M, Urban P, Christenson JT, et al; Benchmark Registry Collaborators. Intra-aortic balloon counter-pulsation in US

and non-US centres: results of the Benchmark Registry. *Eur Heart J*. 2003;24:1763-1770.

39. Ferguson JJ 3rd, Cohen M, Freedman RJ Jr, et al. The current practice of intra-aortic balloon counterpulsation: results from the Benchmark Registry. *J Am Coll Cardiol*. 2001;38:1456-1462.

40. Ohman EM, George BS, White CJ, et al. Use of aortic counterpulsation to improve sustained coronary artery patency during acute myocardial infarction. Results of a randomized trial. The Randomized IABP Study Group. *Circulation*. 1994;90:792-799.

41. Nanas JN, Nanas SN, Kontoyannis DA, et al. Myocardial salvage by the use of reperfusion and intraaortic balloon pump: experimental study. *Ann Thorac Surg*. 1996;61:629-634.

42. O'Rourke MF, Norris RM, Campbell TJ, Chang VP, Sammel NL. Randomized controlled trial of intraaortic balloon counterpulsation in early myocardial infarction with acute heart failure. *Am J Cardiol*. 1981;47:815-820.

43. Mishra S, Chu WW, Torguson R, et al. Role of prophylactic intra-aortic balloon pump in high-risk patients undergoing percutaneous coronary intervention. *Am J Cardiol*. 2006;98:608-612.

44. van't Hof AW, Liem AL, de Boer MJ, Hoorntje JC, Suryapranata H, Zijlstra F. A randomized comparison of intra-aortic balloon pumping after primary coronary angioplasty in high risk patients with acute myocardial infarction. *Eur Heart J*. 1999;20:659-665.

45. Stone GW, Marsalese D, Brodie BR, et al. A prospective, randomized evaluation of prophylactic intraaortic balloon counterpulsation in high risk patients with acute myocardial infarction treated with primary angioplasty. Second Primary Angioplasty in Myocardial Infarction (PAMI-II) Trial Investigators. *J Am Coll Cardiol*. 1997;29:1459-1467.

46. Patel MR, Smalling RW, Thiele H, et al. Intra-aortic balloon counterpulsation and infarct size in patients with acute anterior myocardial infarction without shock: the CRISP AMI randomized trial. *JAMA*. 2011;306:1329-1337.

47. Mahmoudi M, Hauville C, Gaglia MA Jr, et al. The impact of intra-aortic balloon counter-pulsation on in-hospital mortality in patients presenting with anterior ST-elevation myocardial infarction without cardiogenic shock. *Cardiovasc Revasc Med*. 2012;13:328-330.

48. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines: developed in collaboration with the American College of Emergency Physicians and Society for Cardiovascular Angiography and Interventions. *Catheter Cardiovasc Interv*. 2013;82:E1-E27.

49. Ohman EM, Nanas J, Stomel RJ, et al. Thrombolysis and counterpulsation to improve survival in myocardial infarction complicated by hypotension and suspected cardiogenic shock or heart failure: results of the TACTICS Trial. *J Thromb Thrombolysis*. 2005;19:33-39.

50. Bahekar A, Singh M, Singh S, et al. Cardiovascular outcomes using intra-aortic balloon pump in high-risk acute myocardial infarction with or without cardiogenic shock: a meta-analysis. *J Cardiovasc Pharmacol Ther*. 2012;17:44-56.

51. Thiele H, Zeymer U, Neumann FJ, et al. Intra-aortic balloon counterpulsation in acute myocardial infarction complicated by cardiogenic shock (IABP-SHOCK II): final 12 month results of a randomised, open-label trial. *Lancet*. 2013;382:1638-1645.

52. Pierrakos CN, Bonios MJ, Drakos SG, et al. Mechanical assistance by intra-aortic balloon pump counterpulsation during reperfusion increases coronary blood flow and mitigates the no-reflow phenomenon: an experimental study. *Artif Organs*. 2011;35:867-874.

53. Kettner J, Sramko M, Holek M, Pirk J, Kautzner J. Utility of intra-aortic balloon pump support for ventricular septal rupture and acute mitral regurgitation complicating acute myocardial infarction. *Am J Cardiol*. 2013;112:1709-1713.

54. Hartzler GO, Rutherford BD, McConahay DR, Johnson WL, Giorgi LV. "High-risk" percutaneous transluminal coronary angioplasty. *Am J Cardiol*. 1988;61:33G-37G.

55. Briguori C, Airoldi F, Chieffo A, et al. Elective versus provisional intraaortic balloon pumping in unprotected left main stenting. *Am Heart J*. 2006;152:565-572.

56. Perera D, Stables R, Thomas M, et al; BCIS-1 Investigators. Elective intra-aortic balloon counterpulsation during high-risk percutaneous coronary intervention: a randomized controlled trial. *JAMA*. 2010;304:867-874.

57. Perera D, Stables R, Clayton T, et al; BCIS-1 Investigators. Long-term mortality data from the balloon pump-assisted coronary intervention study (BCIS-1): a randomized, controlled trial of elective balloon counterpulsation during high-risk percutaneous coronary intervention. *Circulation*. 2013;127:207-212.

58. Levine GN, Bates ER, Blankenship JC, et al. 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Society for Cardiovascular Angiography and Interventions. *Circulation*. 2011;124:2574-2609.

59. McNamara MW, Dixon SR, Goldstein JA. Impact of intra-aortic balloon pumping on hypotension and outcomes in acute right ventricular infarction. *Coron Artery Dis*. 2014;25:602-607.

60. Theologou T, Bashir M, Rengarajan A, et al. Preoperative intra aortic balloon pumps in patients undergoing coronary artery bypass grafting. *Cochrane Database Syst Rev*. 2011;(1):CD004472.

61. Christenson JT, Badel P, Simonet F, Schmuziger M. Preoperative intraaortic balloon pump enhances cardiac performance and improves the outcome of redo CABG. *Ann Thorac Surg*. 1997;64:1237-1244.

62. Romagnoli E, De Vita M, Burzotta F, et al. Radial versus femoral approach comparison in percutaneous coronary intervention with intraaortic balloon pump support: the RADIAL PUMP UP registry. *Am Heart J*. 2013;166:1019-1026.

63. Noel BM, Gleeton O, Barbeau GR. Transbrachial insertion of an intra-aortic balloon pump for complex coronary angioplasty. *Catheter Cardiovasc Interv*. 2003;60:36-39.

64. Mueller DK, Stout M, Blakeman BM. Morbidity and mortality of intra-aortic balloon pumps placed through the aortic arch. *Chest*. 1998;114:85-88.

65. Arafa OE, Geiran OR, Svennevig JL. Transthoracic intra-aortic balloon pump in open heart operations: techniques and outcome. *Scand Cardiovasc J*. 2001;35:40-44.

66. Sanfelippo PM, Baker NH, Ewy HG, et al. Experience with intraaortic balloon counterpulsation. *Ann Thorac Surg*. 1986;41:36-41.

67. Iverson LI, Herfindahl G, Ecker RR, et al. Vascular complications of intraaortic balloon counterpulsation. *Am J Surg*. 1987;154:99-103.

68. Vohra HA, Rosin MD. The intra-aortic balloon pump. *Surgery(Oxford)*. 2004;22:1-2.

69. Hosmane SR, Dawson AG. In patients coming to theatre with an intra aortic balloon pump, is it better to turn it off or keep it on while on bypass? *Interact Cardiovasc Thorac Surg*. 2010;11:314-321.

70. Serraino GF, Marsico R, Musolino G, et al. Pulsatile cardiopulmonary bypass with intra-aortic balloon pump improves organ function and reduces endothelial activation. *Circ J*. 2012;76:1121-1129.

71. Manord JD, Garrard CL, Mehra MR, et al. Implications for the vascular surgeon with prolonged (3 to 89 days) intraaortic balloon pump counterpulsation. *J Vasc Surg*. 1997;26: 511-515.

72. Jiang CY, Zhao LL, Wang JA, Mohammod B. Anticoagulation therapy in intra-aortic balloon counterpulsation: does IABP really need anti-coagulation? *J Zhejiang Univ Sci*. 2003;4:607-611.

73. Pucher PH, Cummings IG, Shipolini AR, McCormack DJ. Is heparin needed for patients with an intra-aortic balloon pump? *Interact Cardiovasc Thorac Surg*. 2012;15:136-139.

74. Mueller XM, Tevaearai HT, Hayoz D, von Segesser LK. Thrombogenicity of deflated intraaortic balloon: impact of heparin coating. *Artif Organs*. 1999;23:195-198.

75. Patel JJ, Kopisyansky C, Boston B, Kuretu ML, McBride R, Cohen M. Prospective evaluation of complications associated with percutaneous intraaortic balloon counterpulsation. *Am J Cardiol*. 1995;76:1205-1207.

76. Davidson J, Baumgariner F, Omari B, Milliken J. Intra-aortic balloon pump: indications and complications. *J Natl Med Assoc*. 1998;90:137-140.

77. Nash IS, Lorell BH, Fishman RF, Baim DS, Donahue C, Diver DJ. A new technique for sheathless percutaneous intraaortic balloon catheter insertion. *Cathet Cardiovasc Diagn*. 1991;23:57-60.

78. Erdogan HB, Goksedef D, Erentug V, et al. In which patients should sheathless IABP be used? An analysis of vascular complications in 1211 cases. *J Card Surg*. 2006;21:342-346.

79. Boudoulas KD, Bowen T, Pederzolli A, Pfahl K, Pompili VJ, Mazzaferri EL Jr. Duration of intra-aortic balloon pump use and related complications. *Acute Card Care*. 2014;16:74-77.

80. Gjesdal O, Gude E, Arora S, et al. Intra-aortic balloon counterpulsation as a bridge to heart transplantation does not impair long-term survival. *Eur J Heart Fail*. 2009;11: 709-714.

81. Ma P, Zhang Z, Song T, et al. Combining ECMO with IABP for the treatment of critically ill adult heart failure patients. *Heart Lung Circ*. 2014;23:363-368.