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Comprehensive Review

The Intra-aortic Balloon Pump: A Focused Review of Physiology, Transport Logistics, Mechanics, and Complications

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ABSTRACT

Critical care transport medicine (CCTM) teams are playing an increasing role in the care of patients in cardiogenic shock requiring mechanical circulatory support devices. Hence, it is important that CCTM providers are familiar with the pathophysiology of cardiogenic shock, the role of mechanical circulatory support, and the management of these devices in the transport environment. The intra-aortic balloon pump is a widely used and accessible cardiac support device capable of increasing cardiac output and reducing work on the left ventricle through diastolic augmentation and counterpulsation. This article reviews essential CCTM-based considerations for patients supported by intra-aortic balloon pump, including indications for placement, mechanics and physiology, potential issues during transport, and associated complications.

Introduction

Cardiogenic shock (CS) is a state of hypoperfusion secondary to cardiac failure.[1,2] CS represents a clinical syndrome, comprising 2 primary categories: acute myocardial infarction (AMI)-CS or acute decompensated heart failure (ADHF)-CS. These 2 subcategories carry distinct clinical and hemodynamic characteristics.[3,4] Prevalence of CS is increasing, with 8% patients of ST-elevation myocardial infarction developing CS.[5] The rising prevalence of CS has resulted in increasing utilization of mechanical circulatory support (MCS). Although other mechanical circulatory support devices have been developed, the intra-aortic balloon pump (IABP) remains the most commonly used.[6] It has specific advantages in its ease of insertion and is an attractive option in hospitals with limited resources.

Patients supported by an IABP may require transport by critical care transport medicine (CCTM) teams to facilities with more advanced intervention or further care options. The successful interfacility transport of patients supported by IABP has been described, although in limited detail and scope.[7–9] To our knowledge, there are no professional society guidelines specifically describing the transport of patients with an IABP. This review focuses on the essential CCTM-based considerations for patients supported by IABP, including indications for placement, mechanics and physiology, and associated complications.

Abbreviations: ADHF, acute decompensated heart failure; AMI, acute myocardial infarction; CCTM, critical care transport medicine; CO, cardiac output; CPP, coronary perfusion pressure; CS, cardiogenic shock; ESPVR, end-systolic pressure-volume relationship; IABP, intra-aortic balloon pump; MCS, mechanical circulatory support; PV, pressure-volume.

Keywords: cardiogenic shock; heart failure; mechanical circulatory support.

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SCAI Stage	Physical Exam		Biochemical Markers		Hemodynamic Parameters	
	Typically includes	May include	Typically includes	May include	Typically includes	May include
A At Risk	Normal JVP Lungs sounds clear Warm and well perfused Strong distal pulses Normal mentation	Clear lung sounds	Normal lactate	Normal renal function (or at baseline) Normal labs	Normotensive (SBP > 100 or normal baseline for patient)	If hemodynamics assessed: Cardiac index ≥ 2.5 CVP ≤ 10 PCWP ≤15 mmHg Pulmonary artery saturation ≥ 65%
B Beginning CS	Elevated JVP Warm and well perfused Strong distal pulses Normal mentation	Rales in lung fields	Normal lactate	Minimal acute renal function impairment Elevated BNP	SBP < 90 OR MAP < 60 OR > 30 mmHg drop from baseline Pulse ≥ 100	N/A
C Classic CS	Volume overload	Looks unwell Feeling of impending doom Cold and clammy Extensive rales Ashen, mottled, dusky or cool extremities Delayed capillary refill Urine output <30 ml/hr	Lactate ≥ 2 mmol/L	Creatinine increase by 1.5x of baseline (or 0.3 mg/dL) or >50% drop in GFR Increased LFTs Elevated BNP	If hemodynamics assessed (strongly recommended) Cardiac Index < 2.2 PCWP > 15 mmHg	N/A
D Deteriorating Doom	Any of stage C and worsening (or not improving) signs and symptoms of hypoperfusion despite initial therapy	N/A	Any of stage C and lactate rising and persistently > 2 mmol/L	Deteriorating renal function Worsening LFTs Rising BNP	Any of stage C and requiring escalating doses or increasing numbers of pressors or addition of a mechanical circulatory support device to maintain perfusion	N/A
E Extremis	Typically unconscious	Near pulselessness Cardiac collapse Multiple defibrillations	Lactate ≥ 8 mmol/L	CPR (A-modifier) Severe acidosis pH < 7.2 Base deficit > 10 mmEq/L	Profound hypotension despite maximal hyemodynamic support	Need for bolus doses of vasopressors

Figure 1.
SCAI classification of cardiogenic shock incorporating the 2022 revisions.[11,12] BNP, brain natriuretic peptide; CPR, cardiopulmonary resuscitation; CVP, central venous pressure; JVP, jugular venous pressure; LFT, liver function test; MAP, mean arterial pressure; PCWP, pulmonary capillary wedge pressure; SBP, systolic blood pressure.

Pathophysiology of CS

Definition of CS

Major clinical trials have used variable criteria to identify patients with CS, leaving clinicians without a standardized definition (Figure 1). However, most recently, the Shock Academic Research Consortium group, comprising multidisciplinary expert panels, suggested both a practical and a clinical trial definition of CS.[10] From a clinical practice perspective, their suggested definition for CS is as follows: "Cardiac disorder that results in both clinical and biochemical evidence of sustained tissue hypoperfusion," whereas the research-focused definition suggests "Cardiac disorder that results in a systolic blood pressure (SBP) <90 mm Hg for ≥30 minutes (or the need for vasopressors, inotropes, or MCS to maintain systolic blood pressure of ≥90 mm Hg) with evidence of hypoperfusion."[10]

Similarly, a multidisciplinary group was convened by the Society for Cardiovascular Angiography & Interventions (SCAI) to establish a novel CS classification system. The SCAI criteria is a letter-based (A through E) system using a combination of physical examination, clinical factors, biochemical markers, and hemodynamic parameters to classify patients into separate categories (Figure 1).[11] There are many advantages to the SCAI criteria, including applicability to previous trials and retrospective data, facilitating further concerted research efforts.[11] These criteria allow clinicians to make rapid, bedside assessments as patients migrate between categories of severity rather than relying on more resource-intensive data. An update to the original SCAI criteria was published in 2022, with modifications aimed at clarifying differentiating factors separating categories, and rephrasing the physical examination, and biochemical and hemodynamic parameters to include subcategories.[11,12] Multiple validation studies have used the SCAI criteria for CS, which support the correlation between mortality and increasing SCAI grade.[12] Critical care transport teams may use SCAI grade in communication with referring or receiving facility providers and assessment of patient deterioration and potential interventions. Overall, the definition of CS remains heterogeneous, whether through landmark clinical trials or in bedside and clinical practice.[10,13–15]

Pressure-volume loops and CS

The pressure-volume (PV) loop (Figure 2) serves as a model demonstrating both normal cardiac physiology and the effects of maladaptive mechanisms in CS. There are 4 distinct stages of the cardiac cycle demonstrated on the PV loop: (1) ventricular filling, (2) isovolumetric contraction, (3) ejection, and (4) isovolumetric relaxation.

Correlating to distinct phase changes in the cardiac cycle, at point A, end-diastolic filling has occurred. Extrapolation of this portion of the cycle tracing demonstrates the end-diastolic PV relationship. This nonlinear relationship describes innate structural properties of the left ventricle (LV), which are affected by pathologies such as hypertrophy, myocyte remodeling, fibrosis, and ischemia.[16] For example, the slope change (diastolic pressure/diastolic volume) reflects stiffness of the LV, and the inverse of this equation reflects compliance (diastolic volume/diastolic pressure).

The vertical line from A to B represents isovolumetric contraction, with subsequent aortic valve (AV) opening secondary to the gradient between the intraventricular pressure and aortic diastolic pressure. From point B to point C, blood is ejected out of the LV into the systemic circulation. When linearly extrapolated relative to the volume axis intersection (V_0), point C in the cardiac cycle represents end-systolic pressure-volume relationship (ESPVR). ESPVR is the maximum pressure generated for a given ventricular volume, corresponding to contractility. The relationship between the slope of this line (end-systolic elastance E_{es}) and the volume axis intersection (V_0) is reflected as follows: end-systolic pressure (P_{es}) = $E_{es} \times (V_{es} - V_0)$, and demonstrates ESPVR.[16] E_a depicts the afterload. The E_a line starts on the volume axis at the end-diastolic volume and intersects the ESPVR at the ventricular end-systolic PV point of the PV loop. As contractility increases, the slope of this line is translated leftward and/or upward (Figure 2). At point C, isovolumetric relaxation occurs, which permits mitral valve opening (point D) and the subsequent initiation of the upcoming cardiac cycle.

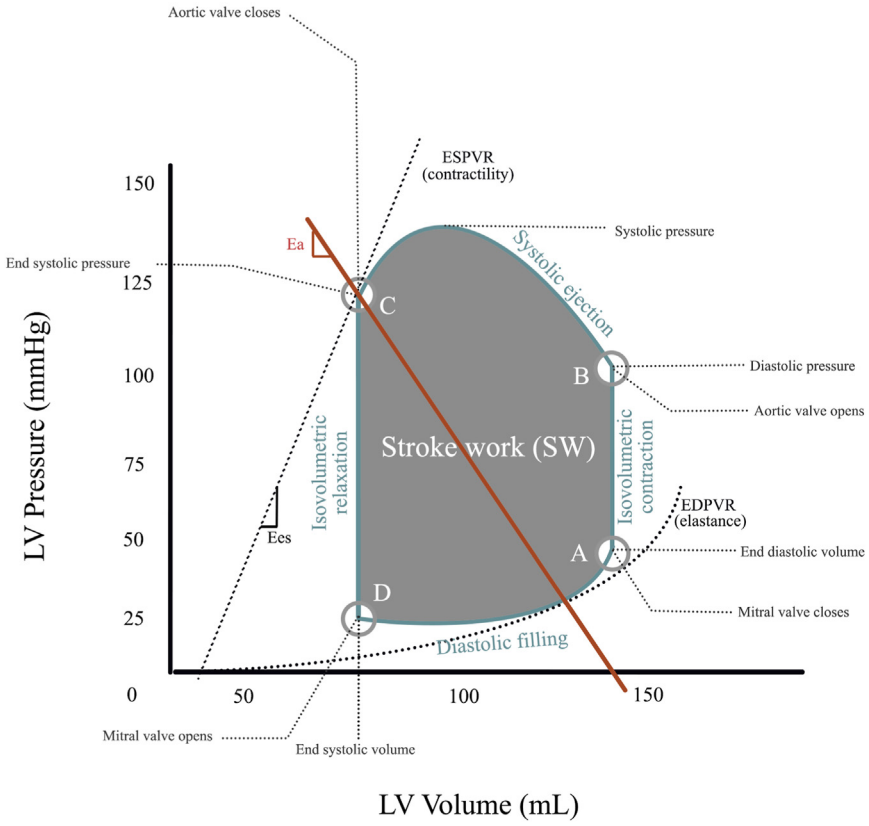


Figure 2.
Pressure-volume loop in normal cardiac physiology. The PV loop demonstrates normal cardiac physiology and the relationships between volume and pressure. Diastolic filling of the ventricle (D to A) is followed by early systole and isovolumetric contraction (A to B), during which the pressure generated by the ventricle results in opening of the semilunar valves, closure of the atrioventricular valves, and forward ejection of blood. Systolic ejection (B to C) is followed by ventricular isovolumetric relaxation (C to D) during which ventricular pressures drop below the diastolic aortic and pulmonary pressure leading to semilunar valve closure and the beginning of diastole. EDPVR, end-diastolic pressure-volume relationship; ESPVR, end-systolic pressure-volume relationship; LV, left ventricle.

The maladaptive, self-perpetuating cycle of CS

Cardiogenic shock generally results from impaired myocardial contractility, leading to a maladaptive spiral of reduced cardiac output (CO), systemic hypoperfusion, and coronary ischemia. Propagation of coronary ischemia further reduces contractility. This self-perpetuating cycle activates multiple neurohumoral pathways resulting in a vasoconstrictive and volume overload state, which ultimately progresses to multiorgan failure and death.[17,18]

The PV loop can aid in describing the hemodynamic changes in the setting of CS. The effects of acute LV dysfunction are shown in Figure 3. The changes in the PV loop include downward and rightward shift of ESPVR reflecting reduced contractility, with a mild increase in end-diastolic PV relationship as filling pressures are elevated. The PV loop area, corresponding to stroke work (ventricular work required for ejection of stroke volume), is reduced.

As demonstrated in the PV loop, increased left-sided filling pressures result in pulmonary congestion, hypoxic vasoconstriction, increased right ventricular afterload, and elevated central venous pressure. In response to this hypoperfusion, endogenous catecholamines are released as a compensatory mechanism. This neurohumoral activation results in an increase in contractility and heart rate, increasing myocardial oxygen demand, further taxing an overstretched cardiac system.[2,19] Finally, to support a perfusing arterial pressure, venous and arteriolar vascular beds constrict, leading to centralization of blood volume, further elevating filling pressures.[19]

IABP impact on cardiac function

The well-described maladaptive physiologic mechanisms implicated in CS serve as potential opportunities for targeted interventions, such as MCS devices. These devices, when applied to the right patient, have the ability to counteract some of these maladaptive processes by offloading the LV, reducing afterload, and augmenting coronary perfusion pressure (CPP). However, the response to MCS device

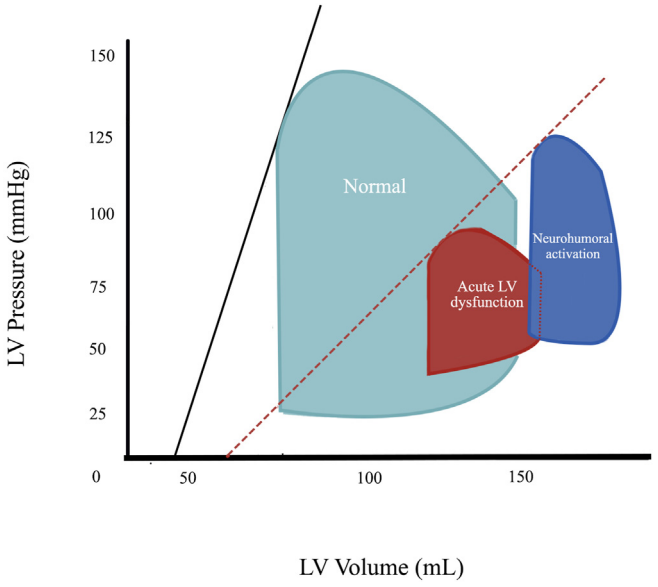


Figure 3.
Neurohumoral activation and LV dysfunction impact the PV loop. The physiologic adaptations found in cardiogenic shock with acute left ventricular dysfunction are demonstrated by changes in the PV loop. With a downward and rightward shift in the acute LV dysfunction loop, filling pressures (LV volume) are significantly elevated, with lower overall generated systolic pressures, and reduced contractility as portrayed by the shift in ESPVR. Neurohumoral activation releases endogenous catecholamines resulting in increased myocardial oxygen demands, centralization of blood volume leading to pulmonary congestion, and further elevation of filling pressures. LV, left ventricle; PV, pressure-volume.

insertion varies by CS subpopulation, whether AMI-CS or ADHF-CS given the distinct compensatory pathophysiology and hemodynamics.[20–22] There is significant active research interest in studying the impact of the IABP and MCS on patients with ADHF-CS, with some studies suggesting benefit in this subpopulation. Given the response variability in individual patients, there may be responders and nonresponders to IABP therapy.[20–22]

The IABP impacts CO and coronary perfusion. Immediately before systole (QRS-T interval), the balloon is rapidly deflated. This rapid deflation results in a decrease in afterload and increase in LV stroke volume, in addition to decreased peak systemic blood pressure, earlier opening of the AV, and a shorter LV isometric contraction phase (Figure 4).[23] In diastole (T-P interval), the balloon inflates, temporarily increasing afterload and ultimately increasing the CPP and systemic diastolic blood pressure.[23] CPP is the difference between aortic end-diastolic pressure and left ventricular end-diastolic pressure (LVEDP). Therefore, the increase in CPP is mediated by an increase in aortic pressure with a concomitant decrease in LVEDP.[23] However, it is vital to note that this increase in CPP does not translate necessarily into increased coronary blood flow (CBF) because CBF is also impacted by coronary vascular resistance.[23,24] In addition to increasing CPP, the IABP provides a modest increase in CO of 0.5-1 L/min, increases myocardial oxygen delivery, and decreases myocardial oxygen demand.[23–26]

IABP mechanics

Studies first demonstrated the utility of the IABP and counterpulsation in 1958, and the device was used clinically just 1 decade later. The IABP facilitates diastolic augmentation to increase CBF and LV systolic offloading to decrease LV work.[27] The IABP consists of a console, which is responsible for delivery of a volume of gas, catheter, and balloon (Figure 5).[23–25]

The intra-aortic balloon has a uniform construction, although there are variations in membrane length (22.0-27.5 cm), inflated diameter (15-18 mm), and balloon volume (30-50 mL).[24] The balloon is rapidly inflated and deflated by injection and retrieval of a gas, typically helium or carbon dioxide.[24] Helium, which is inert, is the preferred agent, because its lower viscosity reduces resistance and increases laminar flow, allowing for more rapid gas entry and retrieval.[24] Optimal balloon sizing requires that no more than 90% of the aortic diameter is obstructed when the balloon inflates, although in clinical practice, the aortic diameter is infrequently measured before insertion.[24] Improper sizing carries risks as oversized balloons increase the risk of vascular complications, whereas an undersized balloon will offer suboptimal cardiac augmentation.[24] In the majority of cases, the 40-mL balloon is appropriate and should be used in patients 162-182 cm tall.[24,25] Use of a 50-mL balloon should be considered in patients who are >182 cm.[25]

IABP console

The console serves as the control unit for the device and is responsible for inflating and deflating the balloon in a coordinated manner. It consists of a monitor unit, control unit, gas source, and valve unit (Figure 5).[23–25] The monitoring unit receives the data from the patient (ie, electrocardiogram [ECG] and infrared blood pressure monitor), and the control unit uses this information to inject and retrieve the gas.[23,24] The monitoring unit displays the systemic and augmented blood pressure and allows for monitoring of the device's function. The control unit allows for modulation of the balloon's ratio of supported beats. The number of supported beats is inversely proportional to the support that the balloon is providing, that is, if the balloon is in a 1:1 mode, each beat is being supported, whereas in 1:2 mode, only every second beat is supported.[28] The monitoring unit also permits observation of the device's functionality. A normal tracing of a 1:1 IABP mode can be seen in Figures 6 and 7.[28] The user will be able to observe an incorrectly triggering IABP by looking at the device's monitor tracing or the waveform of the patient's arterial line. The control unit will read the data from the monitoring unit and inject gas into the balloon during the T-P interval of the ECG and will deflate during the QRS-T interval when the ECG trigger mode is used.[14,23–26,29]

IABP insertion technique

The IABP is most commonly implanted through the femoral artery; however, alternative sites include the brachial, axillary, subclavian, or iliac arteries.[23–25,28,30] Longer-term IABP implants may also be placed in the operating room, using surgical cutdown. The IABP is typically placed under fluoroscopic guidance but can be inserted at bedside as well. Proper placement should be confirmed by chest radiograph before use. A radiopaque metallic marker on the distal tip of the IABP is used to assess placement and should be ~2 cm superior to the carina, which approximates the area of the takeoff of the left subclavian artery in most adults. An alternative landmark that in clinical practice is frequently used is the aortic knob, with the IABP tip located 1-2 cm

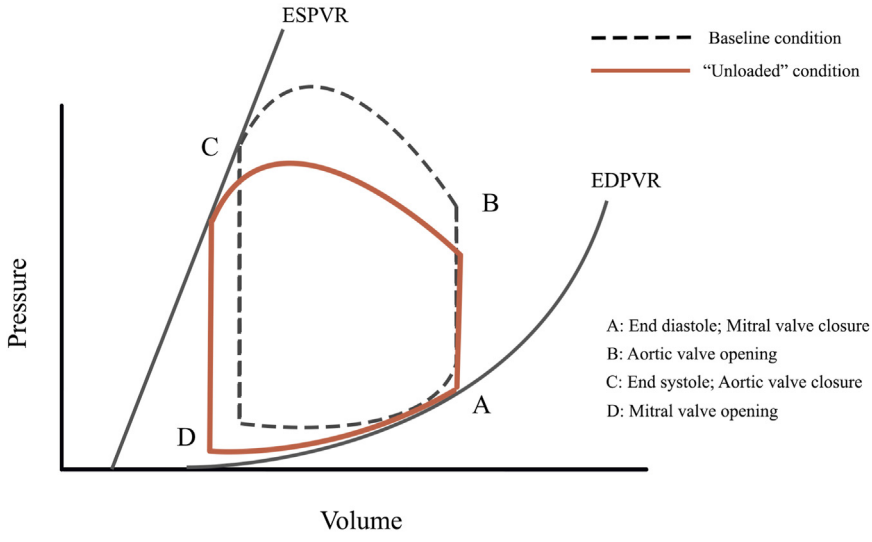


Figure 4.
IABP impact on the PV loop. The IABP has multiple favorable effects on cardiac function. While inflated, the IAB displaces intra-aortic blood during diastole, propagating forward movement of blood into systemic circulation and ideally, increasing coronary blood flow. Owing to the reduction of end-diastolic intra-aortic blood volume, the pressure required of the LV to initiate aortic valve opening is reduced (afterload reduction, seen at point B), leading to reduced LV work and myocardial oxygen demand. Before systole, the balloon then deflates, which also contributes to reduced LV afterload and aortic pressures due to negative pressure mechanics. The time spent in isovolumetric contraction is reduced, leading to reduced myocardial demand. SV increases (EDV, point A; ESV, point C), resulting in modest increases in CO. EDV, end-diastolic volume; IAB, intra-aortic balloon; IABP, intra-aortic balloon pump; LV, left ventricle.

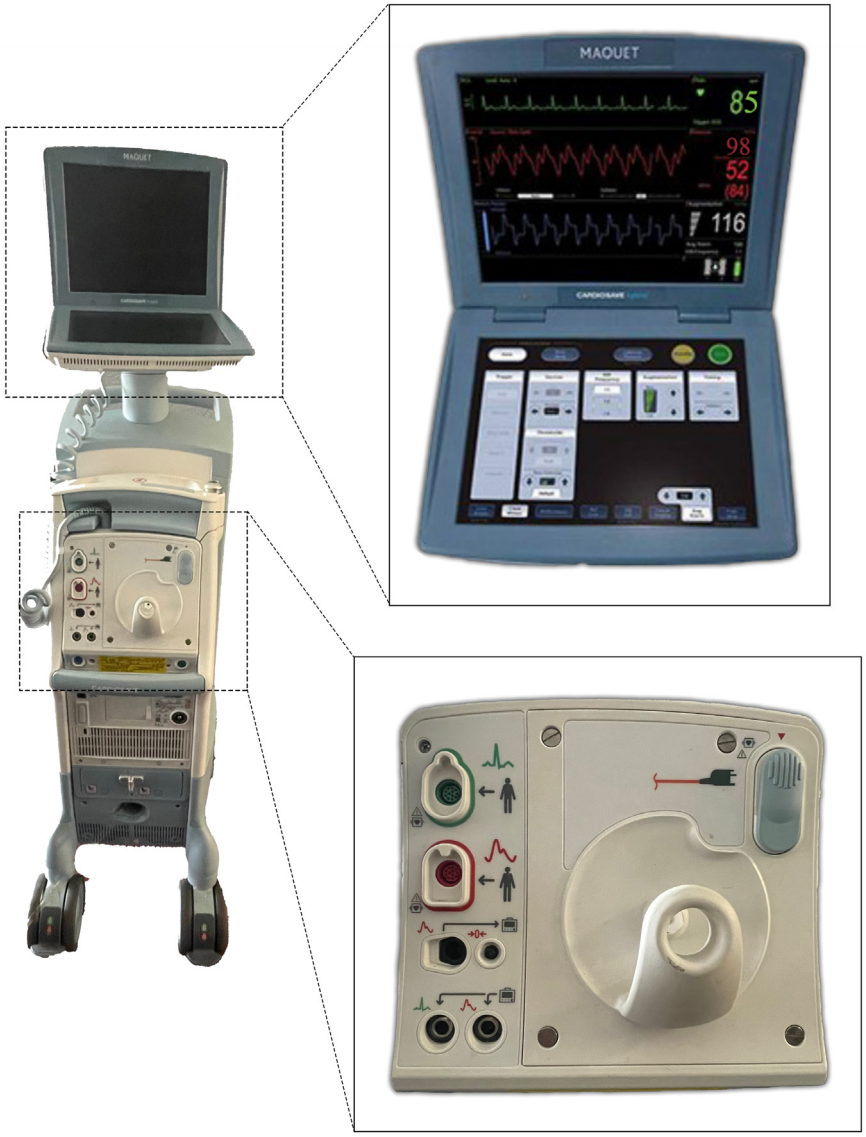


Figure 5.
IABP console configuration. The IABP console consists of a foldable screen, where settings may be entered and adjusted, waveforms are represented, and alarms display; a connection panel, for fiberoptic (top right port), ECG (green, top left), pressure/arterial line (second from top left, red), and helium extender tubing (donut port, central) cables; 2 lithium ion batteries (pictured below front-facing vents); and a helium tank system (not pictured, located on the lower back-side of the console). ECG, electrocardiogram; IABP, intra-aortic balloon pump.

distal to the aortic knob.[23–25,28] Of note, some operators may place the IABP using a 9F sidearm sheath, which allows for pressure transduction of the sidearm, serving as a backup in the event of failure of the primary pressure measuring system.

Once the balloon's location has been confirmed, the gas lumen of the balloon must be checked to ensure the absence of blood, which would suggest balloon rupture. Once completed, the console is set and therapy initiated.[25]

Evidence for IABP utility in the literature

Despite being the most commonly used device for CS, the IABP lacks reproducible evidence supporting improvement in patient-centered outcomes, specifically reduction in mortality.[14,23,24,29] However, the use of the IABP has been shown to impact physiologic parameters in a statistically significant manner: an increase in cardiac index, decrease in pulmonary capillary wedge pressure, and decrease in multiorgan dysfunction have been observed with its use.[29] Initial studies between the 1970s and 1990s reported a mortality benefit in patients with CS; however, subsequent trials have shown consistently negative results.[23] IABP-SHOCK, and the follow-up study IABP-SHOCK II, found no differences in mortality, hemodynamics, or vasopressor requirements in patients with AMI and/or CS who were medically managed with or without augmentation via an IABP.[23,24,29] Lack of benefit in this patient population has been substantiated by numerous trials and meta-analyses.[23,29] However, there are data that suggest potential utility of the IABP in patient populations without AMI-CS, specifically, in patients affected by ADHF.[22,31–33] Patients in CS secondary to ADHF are characterized by distinct hemodynamic profiles in comparison with patients with AMI-CS. For example, 1 retrospective study compared hemodynamic responses to IABP in patients with and without AMI-CS. Before IABP insertion, the ADHF-CS group had higher baseline pulmonary artery pressures than the AMI-CS group, and after IABP insertion, they showed a nearly 5-fold greater augmentation in CO, whereas patients with AMI-CS experienced almost no improvement in CO.[20,32] The IABP may also serve as a bridge to advanced heart failure therapies in patients with advanced heart failure.[22,31,33] There is a need for larger randomized controlled trials evaluating the potential benefit of IABP in patients with ADHF-CS. Irrespective of the primary indication, in many locales, the IABP remains the most frequently inserted MCS device, and CCTM crews will frequently transfer these patients.

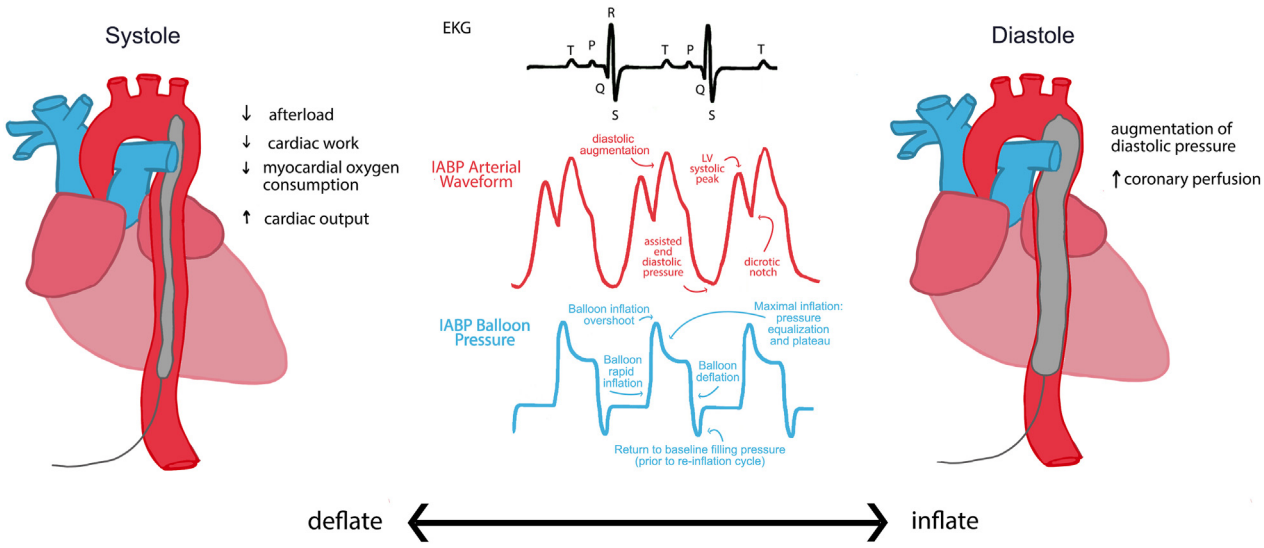


Figure 6.
Pictorial representation of IABP in systole and diastole with ECG tracing, arterial augmented waveforms and corresponding IABP tracings. The IABP functions to inflate during diastole and deflate during systole. This can be accomplished by timing relative to ECG or pressure waveforms, pictured above, to accurately inflate during the appropriate portion of the cardiac cycle. The IAB waveform, pictured in blue, is timed to correlate vertically with diastole in the arterial and ECG tracings. ECG, electrocardiogram; IAB, intra-aortic balloon; IABP, intra-aortic balloon pump.

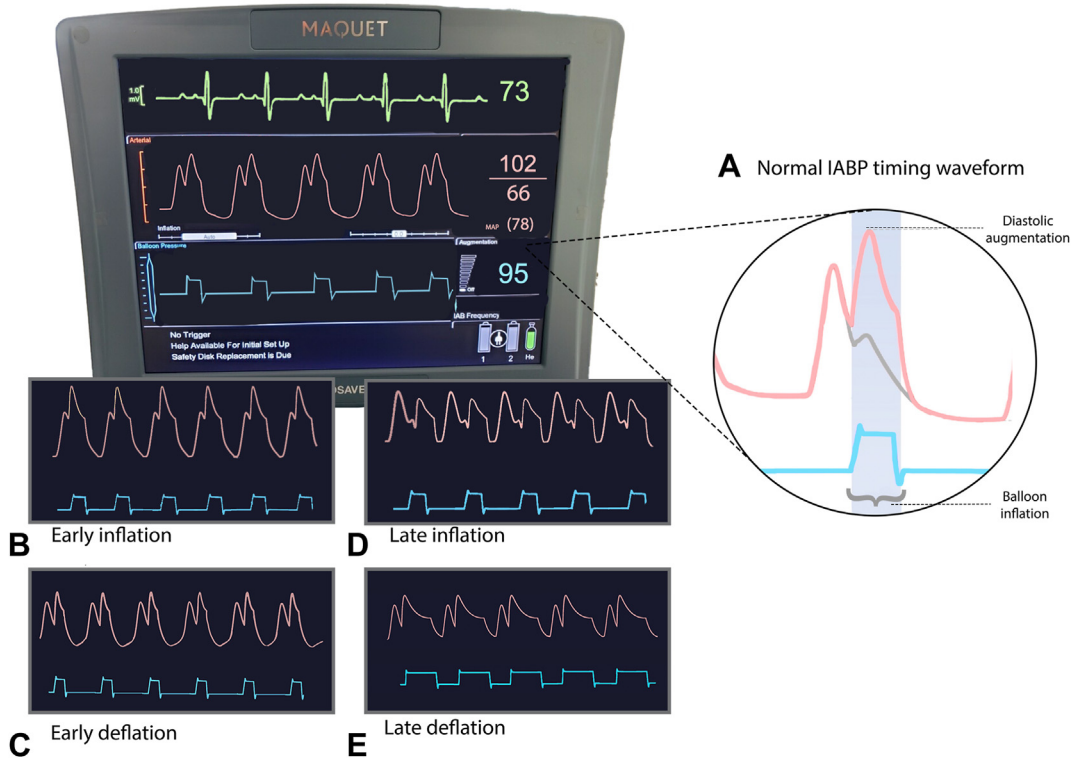


Figure 7.
Normal and abnormal IABP waveforms and their impact on cardiac physiology. IABP monitor interface demonstrating waveform changes in the setting of (**A**) correct inflation timing, (**B**) early inflation, (**C**) early deflation, (**D**) late inflation, and (**E**) late deflation. The arterial waveform in normal IABP inflation timing results in augmentation of diastole correlating with balloon inflation. The balloon pressure waveform illustrates rapid inflation of the balloon, followed by the plateau at maximal inflation, and subsequently rapid deflation before returning to baseline in preparation for the next cycle. In early inflation (**B**), afterload increases, and the aortic valve undergoes premature closure, which increases the end-systolic volume and reduces CO. In early deflation (**C**), there is reduced overall impact and efficacy of the IABP on cardiac function; there is minimal diastolic augmentation and a lack of reduction in myocardial oxygen demand and LV afterload. In late inflation (**D**), there is significantly reduced diastolic augmentation given the delayed timing; there still may be a small impact on improved coronary perfusion, although it is suboptimal compared with the potential of an adequately timed device. Finally, in late deflation (**E**), there is an increase, rather than decrease, in LV afterload, which translates into increased myocardial oxygen demand. CO, cardiac output; IABP, intra-aortic balloon pressure.

IABP triggering

ECG trigger

The IABP is meant to inflate during the closing of the AV and deflate during its opening. The ECG trigger option is the preferred and generally the most reliable. In this mode, the R wave on the patient's ECG corresponds with the opening of the AV and onset of systole; this triggers deflation of the IABP. The IABP then senses the middle of the T wave on the patient's ECG, corresponding with diastole, and triggers inflation.

Arrhythmias can impair the IABP's ability to trigger inflation, as can low ECG voltage, improper electrode placement, and electrical interference with the ECG signal. It is important to ensure proper electrode placement and that the lead with the most prominent R wave is selected and fixing underlying causes of arrhythmia (ie, administer appropriate anti-arrhythmics, defibrillate, and correct electrolytes). If the issue persists, the IABP can be set to a different trigger option.

Atrial fibrillation trigger

If the patient is in atrial fibrillation, an atrial fibrillation trigger mode can often be used, which triggers balloon deflation at each R wave, despite variability between the complexes.

Pressure trigger

Often used when ECG quality is poor, the arterial waveform can also trigger inflation and deflation of the IABP. In this mode, the dicrotic notch in the arterial waveform triggers balloon inflation, and the point just before systolic upstroke (corresponding to the opening of the AV) will trigger deflation. This mode can also be used during cardiopulmonary resuscitation because the arterial waveform should be generated by effective chest compressions.

Internal trigger

The IABP can be set to an intrinsic/asynchronous rate without physiologic triggering. This mode should only be used in situations where CO and ECG tracings are absent, as in the case of asystole or in a patient supported on cardiopulmonary bypass.

Pacer trigger

Pacing spikes (if present) can also be used to trigger IABP inflation. The IABP is triggered to inflate by the V pacing spikes. This mode can be safely used if the patient is 100% pacer dependent.

Automode

Automode is a feature that is capable of automatically discerning and choosing the most appropriate ECG lead and trigger and sets inflation and deflation timing. This mode also is able to respond and select the appropriate trigger in arrhythmias (eg, atrial fibrillation). The simplicity and safety of automode makes this generally the best operation mode choice for transport providers, minimizing potential for error. In comparison, semi-automode requires that the provider establishes timing, then the device console automatically adjusts subsequent timing to the patient's heart rate and rhythm, and that the provider selects the lead and trigger sources.

Diagnosing and troubleshooting IABP complications

IABP failure alarm

This alarm suggests an intrinsic failure within the IABP console or computer. Consider reinitiating the console or computer device or changing devices if necessary after troubleshooting.

IABP disconnected alarm

This alarm suggests disconnection of the IABP extension tubing. Ensure IABP extension tubing is connected and refilled with gas.

Rapid gas loss alarm

This alarm suggests a leak within the IABP circuit or a hole or leak in the balloon. Because the IABP is a closed system, a leak may cause the balloon's waveform to begin to fall below the baseline of zero. Some manufacturers may have a "pump rupture" or "autofill failure" alarm instead. Verify connections are without leaks. Examine the catheter for blood or coffee ground-like material. If this is observed, the integrity of the balloon has been compromised and likely needs to be removed. Stop the pump and prepare for removal and replacement on arrival to the receiving center. CCTM providers should be prepared to increase support for patients who lose the function of their IABP en route.

Check IABP catheter alarm

This alarm suggests a kink in the IABP tubing or catheter. If a sheathed IABP was placed, it is possible that the IABP is partially retained in the sheath, hampering inflation of the balloon.

This alarm could also sound if the IABP has not fully unfolded in the aorta. The balloon pressure waveform may have a rounded peak rather than a flat plateau. Check the IABP tubing and catheter, removing any kinks. If a sheathed IABP was placed, check the markings on the IABP to determine whether displacement has occurred. Transport providers should not manipulate the IABP insertion depth and, if displaced, should notify the receiving facility providers.

Low helium alarm

This alarm suggests the helium supply is running low. Replace the helium cylinder. Ensure there are backup cylinders available to avoid prolonged pauses of the IABP between cylinder changes. Some manufacturers may have an "autofill failure" alarm instead. Certain IABP models provide for rapid helium refilling when the pump console is separated from the cart, as in most transport scenarios; helium may be rapidly refilled within minutes if the console is reconnected to a same-model cart.

Augmentation below limit set alarm

This alarm suggests either augmentation is set too high or there has been a change in the patient's hemodynamic status. Assess the patient for decreasing cardiac function or worsening vascular resistance and add inotropes or vasoactive agents as needed. Assess for improving cardiac function and consider decreasing or removing IABP if appropriate. Consider decreasing the alarm limit if other clinical data suggest the patient is adequately supported.

No trigger alarm

This alarm suggests that the IABP is unable to sense the trigger mode that has been selected. There are various trigger modes that may be useful in different circumstances, detailed in the earlier section. Troubleshooting will be based on the triggering mode being used and may require changing to a different mode.

Normal and abnormal IABP waveforms

Timing errors

Correct timing of the inflation and deflation cycles of the IABP is critical. If the balloon is not adequately timed to inflate and deflate in sync with the patient's cardiac cycle, it can impair an already dysfunctional LV. Timing is assessed in the 1:2 mode so that pressure differences between augmented and nonaugmented beats can be observed and used for diagnostics. A correctly timed IABP should result in an arterial line waveform that shows inflation occurring at the dicrotic notch and forming a sharp V shape. The slope of the augmented diastolic pressure should be parallel to, and reach a peak higher than, the previous systolic upstroke. The assisted end-diastolic blood pressure (EDBP) should be lower than the previous unassisted EDBP by 15-20 mm Hg. Finally, the peak of assisted SBP that follows IABP inflation should be ~5 mm Hg lower than the previous unassisted SBP.

Errors with timing, causing either early or late inflation or deflation, can be detected by variance on the arterial waveforms.

Early inflation. Early inflation will cause the IABP to inflate before the AV has closed. This has the potential to cause the AV to close prematurely, because of increased afterload, leading to impaired LV emptying and possibly aortic regurgitation. Incomplete LV emptying will lead to an increase in LV end-diastolic volume, LVEDP, and pulmonary capillary wedge pressure, which will increase myocardial oxygen demand. Early inflation will lead to diastolic augmentation occurring prior to the dicrotic notch with loss of the desired "V" shape. The diastolic augmentation may encroach onto the unassisted systolic peak to such a degree that the 2 peaks become difficult to differentiate. Early inflation can be corrected by adjusting the timing so that the IABP inflates at the dicrotic notch; however, the best solution is to use automode to optimize timing rather than manually manipulating this variable in critically ill patients.

Late inflation. Late inflation of the IABP occurs well after closure of the AV. This has the potential to cause inadequate perfusion of the coronary arteries due to suboptimal diastolic augmentation. The arterial waveform will show IABP inflation occurring well after the dicrotic notch, again resulting in loss of the optimal V shape. The augmented diastolic blood pressure may be equal to or lower than the unassisted SBP. This can be corrected by adjusting the timing so the IABP inflates at the dicrotic notch; however, similar to the suggestion for remedying early inflation, automode should be used rather than transport providers manually altering variables on the console.

Early deflation. Early deflation of IABP during the diastolic phase ends before isovolumetric contraction occurs. This decreases the efficacy of the IABP to reduce afterload, reducing the boost in CPP and obviating the potential reduction in myocardial oxygen demand. Early deflation may lead to retrograde coronary and carotid blood flow due to the vacuum-like effect in mid-diastole. The arterial waveform will show a sharp drop in pressure soon after the peak of diastolic augmentation. The assisted SBP may be equal to or higher than the unassisted SBP and the assisted EDBP may be equal to or less than the unassisted EDBP. Early deflation can be corrected by prolonging the IABP inflation time, although automode typically accounts for and corrects this error.

Late deflation. Deflation should end near the end of diastole, during isovolumetric contraction; if IABP deflation is prolonged, it may still be in the process of deflating after the AV has opened. In this scenario, rather than reducing afterload, the resistance from a still-inflated balloon during systole will increase afterload, interfering with LV ejection, and prolonging isovolumetric contraction. These effects will increase myocardial oxygen demand. The characteristic arterial waveform of late deflation will show an assisted end-diastolic pressure that is equal to or greater than the unassisted end-diastolic pressure (rather than appropriately decreased) and a widened augmented diastolic peak. If late deflation occurs, the IABP inflation time should be shortened, although automode typically accounts for and corrects this error. There are several alarms common to various IABP brands. It is important for the provider to be able to recognize the potential causes of these alarms and to address them.

Cardiac arrest. In the setting of cardiac arrest, IABP support may be continued. The ECG trigger becomes dysfunctional and cannot be used as the primary timing source; in shockable rhythms, there is no discernible organized rhythm. Interestingly, in situations where the patient is in pulseless electrical activity, the IABP will continue to sense the organized rhythm; however, during compressions, there will be significant artifact. The pressure trigger, rather than ECG trigger, is the preferred sensing method in this scenario. In the transport setting with a small number of providers, this can be accomplished by simply disconnecting the ECG leads from the pump console. The patient's underlying cardiac rhythm should still be monitored through the primary cardiac monitor using electrodes or pads. Defibrillation can be safely performed with the IABP device functioning.

Tachydysrhythmias may also impact IABP performance. Small prospective and some retrospective studies have evaluated the impact of tachycardia on IABP efficacy and function, suggesting reduced performance indices at heart rates greater than 110 bpm; however, there are some IABP models that are capable of matching 1:1 rates up to 220 bpm.[34–36] An opportunity for troubleshooting with reduced IABP efficacy is to change the ratio from 1:1 to 1:2. The primary treatment for tachydysrhythmias is to treat the underlying cause.

Transport considerations

When CCTM teams receive a transport request from a cardiac catheterization laboratory or other critical care area, we recommend that the transport team routinely inquire about the use of MCS devices. Patients supported by IABP will require additional preparation to ensure safe transport, including additional specialized equipment, appropriately trained personnel, and any necessary adaptations for the transport method (eg, a floor securement device for rotor-wing transport).

Equipment assessment. To ensure adequate equipment operations and safety, the following should be performed:

1. Ensure the pump battery is fully charged.
   a. Current commercial devices support battery life of 2 to 2.25 hours.[37] A backup battery should be available in case of equipment failure or unforeseen transport delay.
   b. The pump should be connected to AC power or an inverter whenever possible to preserve battery life.
   c. The mechanic should ensure the vehicle's electrical system can accommodate pump power requirements along with other necessary electric systems so the system will not be overloaded in transport.

2. Check the helium tank level.
   a. Before departure, the helium tank should be filled. For air transport, the balloon will empty and fill with some variation in conjunction with altitude changes.
3. Secure the pump console onto the transport vehicle mount to comply with local and national safety standards.
a. Commercial devices include portable consoles to meet the physical constraints of medical transport. These should be installed according to manufacturer recommendations and comply with ground and aviation safety standards.
   b. All personnel should be familiar with proper device installation within the transport vehicle.
   c. During patient transport, the controller screen should be visible to the medical team to monitor function and view alarms, which may not be audible with ambient background noise.
4. Include adapters for connection to devices from local facilities.
   a. The transport service should communicate with local cardiac catheterization facilities regarding devices and compatibility.
   b. The most common commercial devices such as Arrow IABP (Teleflex) and Maquet/Datascope IABP (Getinge) include adapter extension catheters to connect the balloon to different consoles.
   c. Bring any catheter extension tubing from the transferring facility to the receiving facility.
5. Be prepared for infrequently encountered but severe complications with backup equipment.
   a. Include a 60-mL syringe with a 3-way stopcock for manual rapid balloon inflation and deflation of 40-50 mL of air through the helium extender tubing, in case of complete console dysfunction and failure. The goal here is no longer hemodynamic support but rather the prevention of thrombosis.

Patient assessment. In addition to the standard history and examination before transport of any critically ill patient, assessment of a patient with an intra-aortic balloon pump should include evaluation of the device, functioning of the device, and consideration of potential complications. Placement of the device is radiographically confirmed after the procedure. Standard intra-aortic balloons have a radiopaque tip that should appear ~1-2 centimeters distal to the aortic knob (Figure 8). If there are any concerns about placement or migration of the device on assessment, a repeat x-ray and/or evaluation by the referring physician should be requested by the CCTM team before transport.

The most common complications of IABPs include limb ischemia and hemorrhage.[38] Therefore, a thorough neurovascular exam must be conducted on all patients before transport and again at the receiving facility. Distal pulses should be assessed, with Doppler if necessary, to monitor for limb ischemia. Loss of the left radial pulse is concerning for limb ischemia due to either thrombosis or proximal migration of balloon to occlude the left subclavian artery. The insertion site should be examined for ecchymosis, hematoma, or bleeding. If feasible, leaving the insertion site visible and easily accessible will facilitate rapid site assessment and IABP repositioning throughout transport if needed. When transferring a patient with an IABP in place, do not raise the head of the bed greater than 30° because this may strain or kink the femoral catheter and cause bleeding.

To maintain and maximize static positioning of the device, there are several practical techniques that may be considered, such as use of foley anchors for device securement, or knee immobilizers to reduce the potential for patient movement kinking or otherwise impacting device and line positioning.

Before transferring the patient's device to the transport pump console, note settings, and timing. If the patient's device is set at an augmentation ratio of 1:1, adjust to 1:2 to analyze timing and augmentation, and then return to original settings. A standard checklist for patient assessment and documentation serves to ensure essential measures, and evaluations are recorded and performed. Examples of patient assessment checklists and IABP device evaluation forms for transport are provided in Supplemental Figures S1 and S2, respectively.

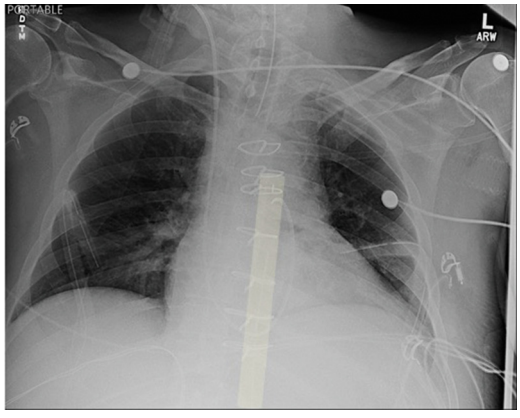


Figure 8.
Chest x-ray demonstrating correct positioning of the IAB. The IAB is highlighted by the radiopaque marker at the distal point of the balloon (superior aspect of the yellow-highlighted portion). There is also a radiopaque midline marker within the balloon itself, which is faintly visible. Additional support devices pictured include bilateral chest tubes, an endotracheal tube, and a right internal jugular approach Swan-Ganz catheter. IAB, intra-aortic balloon.

Altitude changes. For proper counterpulsation therapy and to prevent overdistention, the IABP must be adjusted to compensate for changes in atmospheric pressure. This is an essential consideration in the context of aeromedical transport of patients in rotary-wing or fixed-wing aircraft.

Some IABPs will perform this compensation automatically. For example, in the autofill mode, the Maquet CS300 system (Getinge) will automatically purge and fill the balloon when the atmospheric pressure decreases by 25 mm Hg (occurring approximately every 1000 feet [304.8 m] of ascent) or when the atmospheric pressure increases by 50 mm Hg (occurring approximately every 2000 feet [609.6 m] of descent). If the device fails to automatically adjust, pressing "pump status on" will cause the pump to automatically adjust to altitude. Rapid ascent without appropriate adjustment will cause "high baseline" or "high fill pressure" alarms, terminating counterpulsation. Rapid descent may cause "helium loss" or "gas loss" alarms.[39] CCTM providers should be aware of the compensatory capabilities of the IABP system used by their agency.

Logistical considerations. Patients supported by MCS, such as an IABP, challenge the spatial constraints of CCTM, with the most restrictive conditions being the helicopter environment. However, lighter and more compact modern equipment have made interfacility air transport of patients with IABP relatively common. Figure 9 depicts a potential cabin configuration of an EC145 helicopter with a patient supported by an IABP in which the flight physician or paramedic sits at the patient's head while a flight nurse sits to the patient's left side with the IABP console. Any configuration should ensure accessibility to infusion pumps, the IABP console, cables/connections into the IABP, patient monitors, and a ventilator, if the patient is supported by either invasive or noninvasive positive pressure ventilation, because patients experiencing CS often require in-flight adjustments of both medical and mechanical support.

Conclusion

CS is characterized, in general, by impaired ventricular contractility leading to hypoperfusion, which then promotes further myocardial insult and dysfunction, ultimately culminating in systemic end organ damage. MCS devices, including the IABP, can be used to

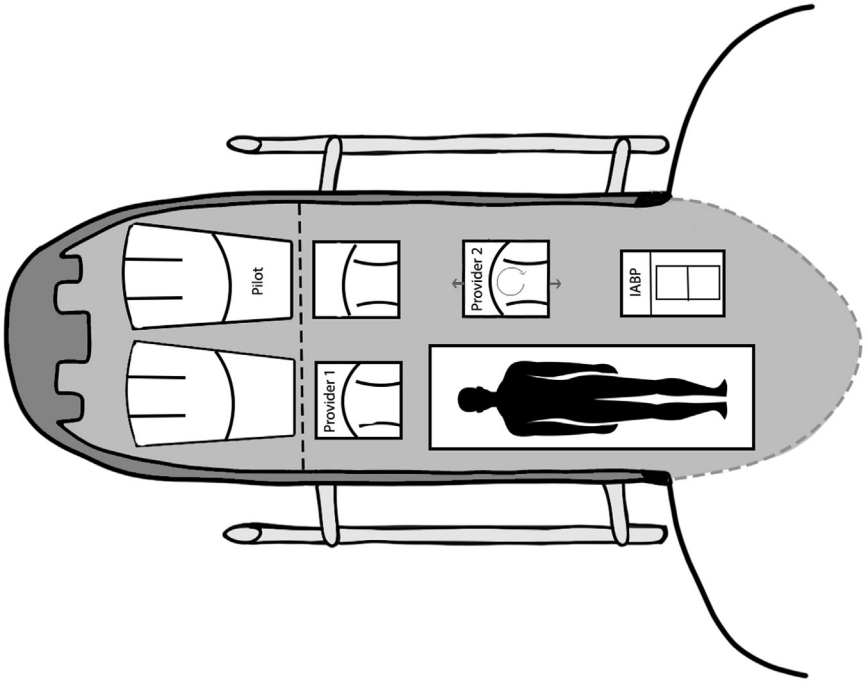
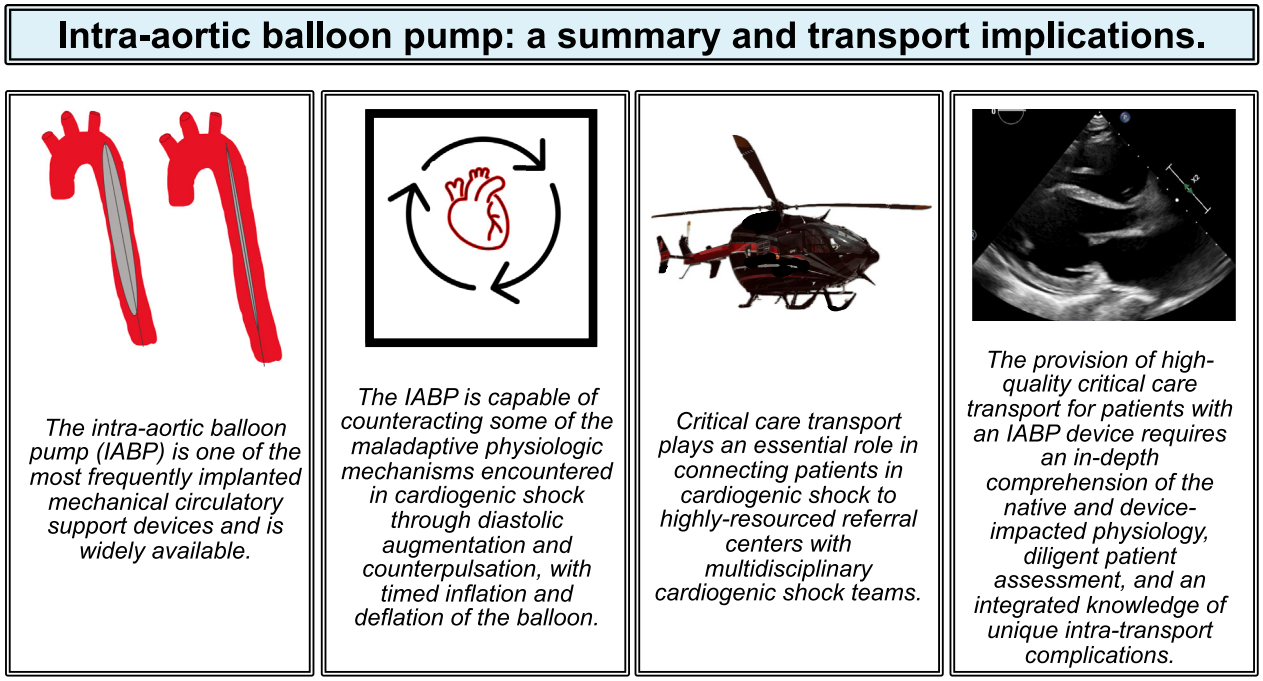


Figure 9.
Top-down view of the configuration of an EC145 helicopter with a patient supported by an IABP. Pictured is the proposed, commonly encountered configuration for rotor-wing transport of a patient with IABP support. Dotted lines represent closed doors, with clamshell doors depicted in the open position.

supplement perfusion and avoid irreversible end organ damage in this population. The IABP is a percutaneously inserted counterpulsation pump positioned in the descending aorta that serves to improve coronary perfusion, reduce afterload, and augment CO by approximately 0.5-1 L/min. As IABPs are increasingly encountered in the transport environment, knowledge of the underlying physiologic changes, mechanics, and troubleshooting procedures for the IABP are essential (Central Illustration). The initial assessment should include a diligent physical examination, which serves to ensure device securement, assessment for device complications, and inspection of device settings. Careful monitoring for complications, such as inflation and deflation timing errors, balloon rupture, and helium gas supply errors is an essential component of care and requires specific training.



Central Illustration.
The intra-aortic balloon pump is a frequently encountered mechanical support device. Critical care providers must be expertly trained in caring for patients supported by the IABP, given the essential role of transport providers in connecting patients to highly resourced specialty centers for cardiogenic shock. IABP, intra-aortic balloon pressure.

The transport of patients supported by IABP devices has been successfully and safely accomplished with well-trained providers and systems. The provision of high-quality critical care transport in patients with IABP devices requires diligence and in-depth comprehension of the native and device-impacted physiology and intratransport complications.

Declaration of competing interest

The authors do not have any conflicts of interest to disclose.

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Ethics statement and patient consent

The research presented here adheres to the relevant ethical guidelines.

Supplementary material

To access the supplementary material accompanying this article, visit the online version of the *Journal of the Society for Cardiovascular Angiography & Interventions* at 10.1016/j.jscai.2024.101337.

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