Getting in sync with intra-aortic balloon pump therapy

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A 68-YEAR-OLD man with severe left main coronary artery disease has just arrived in the CCU from the cardiac catheterization lab. He’s scheduled for coronary artery bypass graft surgery tomorrow morning.

Another patient admitted with ST-segment elevation myocardial infarction (MI) just underwent multiple balloon inflations and intracoronary stent placements for extensive left anterior descending stenoses.

A 79-year-old female patient in the CCU is in profound cardiogenic shock from an extensive anteroseptal MI. Her BP is being maintained with vasopressors, her cardiac output (CO) is 2.1 L/minute (normal range for adults, 4 to 8 L/minute), and her systemic vascular resistance is 2,347 dynes/second/cm⁻⁵ (normal range is 900 to 1,200 dynes/second/cm⁻⁵).

All three of these patients will need an intra-aortic balloon pump (IABP) as part of their management. This article discusses the indications, rationale, function, and potential complications of IABP therapy.

The IABP is a cardiac assist device consisting of a long, cylindrical polyethylene balloon at the end of a flexible catheter. For an average patient, the intra-aortic balloon (IAB) is 20 to 25 cm long. IABs are generally measured by the volume of gas they contain, with sizes ranging from 2.5 mL for infants to 50 mL for large adults. Balloon size generally depends on the patient’s height and aortic diameter. The IAB is inflated with helium, an inert gas with a lower molecular weight than room air. The lighter weight of helium produces faster inflation and deflation times than conventional room air.

The other end of the IAB catheter is attached to a console that contains the mechanism for rapidly inflating and deflating the IAB and computerized circuitry to determine the inflation/deflation timing.

IAB catheter insertion occurs most commonly in the OR during cardiac surgery or in the cardiac catheterization lab during an interventional procedure.¹ The IAB catheter may be inserted at the bedside in the CCU in emergencies. The balloon is usually inserted through the femoral artery via groin site access. Although the balloon is routinely inserted via the femoral artery, it can also be inserted using the brachial or subclavian artery if the femoral approach is contraindicated.

The IAB catheter is advanced until it’s positioned in the descending thoracic aorta 1 to 2 cm below the origin of the left subclavian artery and above the renal artery branches.² Correct location of the balloon is confirmed by fluoroscopy or X-ray. The tip should be visible on X-ray between the second and third intercostal spaces. If the balloon is placed too low, the renal arteries could be obstructed, compromising renal perfusion. On the other hand, a too-high balloon could obstruct the origin of the left subclavian artery or the left carotid artery.

The counterpulsation concept

The IABP is effective because of the unique anatomy of the aortic valve cusps and their relationship to the origin of the two coronary arteries. The aortic valve normally has three cusps: right, left, and posterior. The left main coronary artery originates just above the left aortic cusp; similarly, the right coronary artery originates just above the right aortic cusp.

During left ventricular systole, the aortic valve opens and the aortic cusps are reflected superiorly to obstruct the coronary ostia. During diastole, the aortic valve closes, exposing the openings of the coronary ostia to aortic diastolic pressure. During this diastolic phase, coronary artery perfusion takes place. The coronary arteries...
are the only arteries in the body that are perfused during diastole.

When the aortic valve closes, the end of the aortic root is also effectively sealed. Once diastole begins, the IABP rapidly inflates within the aorta (see *The balloon cycle*). This rapid balloon inflation both seals (or “compartmentalizes”) the proximal aorta and increases the pressure within the proximal compartment consisting of the IAB, aortic arch, coronary arteries, and aortic root. This increase in aortic diastolic pressure increases perfusion to the coronary arteries and improves myocardial oxygen delivery. The increase in aortic pressure as a result of balloon inflation is called *diastolic augmentation*.

At the onset of systole, the IABP rapidly deflates, causing an area of lowered pressure within the aorta. The lowered aortic pressure decreases impedance or resistance to systolic ejection. The decreased afterload effect results in decreased left ventricular workload and decreased myocardial oxygen demand.

IABP therapy is frequently referred to as “counterpulsation” because the balloon inflation occurs during diastole and balloon deflation occurs during systole. IABP counterpulsation increases CO. Patients frequently experience relief from ischemic chest pain or anginal equivalent within minutes of starting IABP therapy.

**Timing of the balloon**

For the IABP to function properly, its inflation-deflation cycle must be synchronized to the appropriate events in the cardiac cycle. The central lumen of the double-lumen IAB catheter allows monitoring of the pressure in the descending aorta during the cardiac cycle. This arterial line provides data to the IABP console. Timing of the IABP is always performed using the arterial waveform as the guide.

The first arterial pressure waveform in *Inflation-deflation timing* demonstrates the classic features of an arterial waveform. In this first waveform, the IABP isn’t active and there’s no balloon inflation. The peak of the waveform represents peak systolic pressure, and the bottom of the waveform reflects the end-diastolic pressure. The dicrotic notch is a significant landmark; it signals the closure of the aortic valve and the beginning of diastole.

The second arterial pressure waveform shows a balloon inflation/deflation cycle. The systolic peak of this waveform is called *unassisted systole* because the previous cardiac cycle (first waveform) wasn’t assisted by the IABP.

The IABP console continuously monitors the patient’s arterial pressure. When it recognizes the dicrotic notch (the onset of diastole), it triggers rapid balloon inflation; pressure within the aortic compartment increases and coronary artery perfusion occurs. The pressure during diastolic augmentation is often considerably higher than both the normal diastolic pressure and even the peak systolic pressures. High diastolic augmentation pressure is one of the indicators of effective IABP function.

Balloon deflation is timed to occur with the onset of systole. When the IABP console identifies the beginning of the patient’s R wave, the balloon is rapidly deflated, lowering the pressure in the proximal aorta. In the second waveform of the illustration, the *assisted aortic end-diastolic pressure* is lower than the previous pressure waveform’s *unassisted aortic end-diastolic pressure*. The lowering of the end-diastolic pressure from balloon deflation is another physiologic indicator of proper IABP function.

Systole that occurs after a balloon deflation is called *assisted systole* because the left ventricle is pumping against lowered aortic resistance. The pressure of the assisted systole is significantly lower than the pressure of the unassisted systole. This pressure difference represents...

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**The balloon cycle**

During diastole, the IAB rapidly inflates (left), increasing perfusion to the coronary arteries. Balloon deflation (right) is timed to occur at the beginning of systole.
the reduction in cardiac workload (afterload) and myocardial oxygen demand associated with counterpulsation.5

In most circumstances, the R wave is the trigger that deflates the IAB. Most IABP consoles have a selectable trigger event for situations where the R wave may not be optimal. A pressure trigger uses the systolic upstroke of the arterial waveform, and may be used when CPR is in progress. An internal trigger setting is used when the patient has no mechanical cardiac cycle, such as in cardiopulmonary bypass or asystole. Other trigger events use the pacing spikes of ventricular or atrioventricular pacemakers.6

Indications and contraindications
IABP therapy is indicated in a variety of clinical situations, including unstable angina, threatening extension of acute MI, intractable ventricular dysrhythmias, and low CO states such as cardiogenic shock.7 In some patients with Stage D heart failure, IABP therapy may be required as a bridge to another mechanical assist device or transplant. It may also be indicated for cardiac support in patients before correction of mechanical defects, such as papillary muscle rupture and ventricular septal defect. Procedure-related uses for the IABP include support for patients undergoing percutaneous coronary intervention.

Because the concept of counterpulsation is based on a competent aortic valve, IABPs can’t be used in patients with severe aortic regurgitation. A thoracic or abdominal aortic aneurysm is another contraindication for IABP therapy because balloon inflation pressure against the weakened aortic wall could lead to rupture.8

A relative contraindication to balloon pump insertion is the presence of severely calcified femoral or aortoiliac disease. The healthcare provider may not be able to insert an IAB catheter into a tortuous or severely atherosclerotic vessel. Vascular studies of the aorta may be helpful and other insertion strategies (such as using the brachial or subclavian artery) may need to be considered. One final consideration before initiating IABP therapy is the inclusion of an end point for therapy. For example, if the patient’s underlying disease isn’t treatable, IABP therapy may not be warranted.

Nursing considerations
When you’re caring for a patient with an IABP, monitor the patient’s vital signs every hour; more frequently if your patient is hemodynamically unstable. Monitor hemodynamic parameters according to your facility’s protocol. Along with hourly vital signs, monitor the patient’s lower extremity neurovascular status, including skin temperature and color, capillary bed refill, pulse quality, motor function, and sensation. Perform regular neurologic assessments.

Modern IABPs can pump effectively during episodes of atrial fibrillation and ventricular tachycardia, but you still need to monitor and assess the IABP’s performance during periods of dysrhythmia. Monitor for balloon malposition or migration. Obtain a prescription for a daily chest X-ray. Loss of radial pulses may mean that the balloon has migrated upward to obstruct the subclavian artery. Patients in cardiogenic shock often have poor urinary output, but absence of urinary output may also signal that the balloon has migrated downward to obstruct the renal arteries.

To reduce the risk of thrombus formation and embolization, avoid blood sampling from the central lumen of the balloon. Thrombus formation may also occur if the balloon is static for a prolonged period, so avoid pausing the balloon for more than 5 minutes.

Avoid complications of immobility by performing frequent skin assessments and turning and repositioning the patient regularly. Prevent pulmonary complications by encouraging coughing and deep breathing, using incentive spirometry, and providing chest physiotherapy.

Teach the patient and family about IABP therapy, answer their questions, and provide emotional support. Explain the need for bed rest and keeping the patient’s access extremity straight and immobile. Instruct the patient to report any changes in neurovascular status such as numbness, temperature change, or pain. Provide written educational materials if available at your institution.

Complications
IABP therapy can offer significant benefit to the patient but does carry risks. Complications from IABP therapy can be divided into three main types: vascular injury, balloon-related problems, and hematologic complications.
Vascular injury includes limb ischemia related to the large diameter of the balloon catheter and patient-associated risk factors such as peripheral arterial disease, advanced age, and diabetes. Limb circulatory compromise can occur in 6% to 25% of patients during IABP therapy. Vigilant neurovascular monitoring is essential, and evidence of circulatory compromise may require removal of the balloon catheter. Other vascular complications include arterial or aortic injury associated with balloon catheter insertion such as aortic dissection, perforation, or femoral artery thrombosis.

Balloon-related complications involve perforation or rupture of the balloon itself, or malposition (which was discussed earlier). During inflation, contact of the balloon with aortic calcifications can potentially erode the balloon and lead to balloon failure. All modern IABP consoles have gas-leak detection capability, and the sudden loss of balloon pressure will trigger an automatic alarm and discontinue the pumping cycle. Although gas embolization is a rare complication, a nonintact balloon represents a source of infection and must be removed or replaced immediately. Another complication of balloon rupture is accumulation of clotted blood within the ruptured balloon, which can make removal difficult. In such cases, a vascular surgical procedure may be needed.

Hematologic complications include thrombocytopenia due to traumatic platelet destruction resulting from the balloon’s pressure against the aortic wall. A drop in platelet count of 50,000 to 100,000 per microliter can be expected; platelet transfusion is rarely needed. Patients requiring IABP therapy routinely receive heparin to prevent thrombus formation on the balloon surface, so appropriate management of the patient’s coagulation status must also be considered.

Weaning and discontinuation
As the patient’s cardiac performance improves, weaning and removal of the IABP should be considered as early as possible. Criteria for weaning include:
- resolution of the signs and symptoms of hypoperfusion secondary to low CO, including angina.
- cardiac index (CI) of 2 L/minute/m² or more, which doesn’t decrease by more than 20%.
- pulmonary capillary wedge pressure that doesn’t increase to more than 20% above preweaning level.
- urine output greater than 0.5 mL/kg/hour.
- heart rate less than 100 beats/minute.
- minimal need for positive inotropes.

Weaning is accomplished by reducing the frequency of balloon-assisted beats from the maintenance ratio of 1:1 to a weaning ratio of 1:2 (every other systole is assisted). Depending on the cardiologist’s judgment, weaning modes of 1:3 or even 1:8 may be initiated if a more gradual wean is needed.

During IABP weaning, monitor your patient’s vital signs, ECG, mental status, urine output, distal perfusion, CO, and CI. Worsening of the patient’s vital signs, hemodynamic parameters, or symptoms such as chest pain may indicate that the patient isn’t ready for weaning. Return the patient to the previous IABP maintenance settings.

The I.V. heparin infusion should be discontinued at least 4 hours before removal. Whenever the pumping cycle is stopped, the IAPB automatically deflates the balloon. The IAB catheter can then be withdrawn from the femoral arteryatraumatically. Direct manual pressure is applied to the arterial puncture site for 30 minutes to obtain hemostasis. Another common method to obtain hemostasis is application of a mechanical compression device.

If your institution doesn’t yet use IABP therapy, it probably will soon. IAB counterpulsation has evolved from a treatment limited to large, top-tiered teaching hospitals to an industry-wide standard of care in the treatment of cardiovascular disease.