As cardiac catheterization and intervention have been applied to patients with unstable hemodynamic and ischemic conditions, the availability of effective devices for circulatory support has assumed increasing importance. The first such device was the *Intraaortic Balloon Counterpulsation* (IABP), which improved the myocardial oxygen supply/demand ratio and provided limited circulatory support. It remains the most widely used and practical system today, although more complicated devices, such as the left ventricular support device (LVAD) and the cardiopulmonary support (CPS) system, have been introduced to provide more *complete* circulatory support in special circumstances. This chapter reviews the design, function, indications, and technique for use of these important adjunctive devices for circulatory support in the cardiac catheterization laboratory.

**Intraaortic Balloon Counterpulsation and Other Circulatory Assist Devices**

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As cardiac catheterization and intervention have been applied to patients with unstable hemodynamic and ischemic conditions, the availability of effective devices for circulatory support has assumed increasing importance. The first such device was the *Intraaortic Balloon Counterpulsation* (IABP), which improved the myocardial oxygen supply/demand ratio and provided limited circulatory support. It remains the most widely used and practical system today, although more complicated devices, such as the left ventricular support device (LVAD) and the cardiopulmonary support (CPS) system, have been introduced to provide more *complete* circulatory support in special circumstances. This chapter reviews the design, function, indications, and technique for use of these important adjunctive devices for circulatory support in the cardiac catheterization laboratory.

**THE INTRAARTIC BALLOON PUMP**

The well-deserved popularity of the intraaortic balloon is related to its simplicity, ease and rapidity of insertion, ease of operation, long clinical record, and relatively low cost. The concept of using timed inflation of a balloon to generate a positive pressure pulse during diastole (to improve coronary flow), and then withdrawing that volume during systole by balloon deflation (to reduce resistance to systolic ejection, thereby reducing myocardial oxygen requirements), was first conceived by Claus et al., in 1961, and applied clinically by Kantrowitz in 1968.

At first it was used only in patients with cardiogenic shock, but soon it was successfully applied to patients with medically refractory unstable angina. Insertion of the balloon catheter was initially performed surgically, but this has been almost completely supplanted by percutaneous insertion techniques that offer rapid and simple balloon insertion during an emergency.

**Intraaortic Balloon Pump Equipment**

The intraaortic balloon catheter consists of a long (roughly 10 cm) polyurethane bladder that is mounted on a flexible shaft so that it can be positioned in the descending aorta just distal to the origin of the left subclavian artery. This bladder is inflated abruptly with helium immediately after aortic valve closure, causing an increase in aortic diastolic pressure. Inflation is maintained until just before the beginning of systolic ejection (the opening of the aortic valve), when the gas control system rapidly deflates the balloon to produce a sharp fall in systolic aortic pressure and a decrease in the impedance to left ventricular (LV) ejection. The inflation-deflation cycle is triggered by the surface electrocardiogram (ECG), although a fixed cycle can be generated internally for patients in ventricular fibrillation or on cardiopulmonary bypass. The console allows fine adjustment of the timing of balloon inflation and deflation to optimize the hemodynamic effect, as monitored in the arterial pressure waveform.

Although early balloons were single-lumen devices (with just the gas lumen for inflation and deflation), most IABP balloons are now double-lumen catheters. A central lumen allows delivery over a guidewire and then monitoring of the central aortic pressure. In order to minimize damage to the adjacent aortic intima, the inflated balloon diameter should be no more than 90% of the aortic diameter. The 40-cc balloon is generally satisfactory for most adults, and a 30- to 34-cc balloon is reserved for small patients. Pediatric balloons are available in 2.5-, 5.0-, 12.0-, and 20-cc sizes (Table 21.1). Early balloon consoles used carbon dioxide because of its excellent solubility in blood were the balloon ever to leak or rupture. Because the shaft size of balloon catheters has decreased, a gas with a lower molecular weight (i.e., helium) is now used to maintain fast gas shuttle times for crisp inflation and deflation. Because of the low solubility of helium in blood, however, the consoles must have a reliable mechanism to detect and react to balloon leak or rupture, by rapidly withdrawing all helium from the balloon and shutting down the counterpulsation cycle to minimize the risk of serious helium embolization.
Percutaneous Insertion

Unless the IABP is being used because of instability during induction of anesthesia for surgery or difficulty in weaning from cardiopulmonary bypass, the IABP catheter is inserted percutaneously. The large diameter can cause serious vascular complications, and careful preprocedure evaluation is in order.

If feasible, clotting parameters (prothrombin time, partial thromboplastin time, and platelet count) should be checked before balloon insertion. A careful history should be obtained to determine the possible presence of claudication or prior peripheral vascular surgery. Both catheterization and IABP insertion can be performed via femoral artery grafts if special attention is paid to puncture technique (6). Noninvasive evaluation of the peripheral arterial circulation before balloon insertion forms a valuable point of reference for later monitoring of circulatory compromise by the IABP catheter.

Although early single-lumen balloons were inserted directly through a cutdown or large (11F) sheath, currently balloons are inserted percutaneously over a guidewire using either a small (8F to 9F) sheath or sheathless insertion. The technique for correct preparation and puncture of the common femoral artery is described in Chapter 4. If balloon placement is being performed as a stand-alone procedure, the artery is predilated with a 7F or 8F dilator after the wire has been advanced to the level of the diaphragm. Firm pressure is maintained over the puncture site to prevent hematoma as the dilator is removed and the wire is wiped clean. Either the appropriate-size sheath or the balloon catheter itself, if sheathless technique is chosen, is introduced over the wire. When balloon placement is being performed at the conclusion of a catheterization (particularly an intervention performed via an 8F sheath), one of the new, low-profile IABP catheters can be placed via the existing 8F sheath. In this case, it is helpful to place the special balloon guidewire at the level of the diaphragm before the final diagnostic or interventional guiding catheter is removed, thereby obviating the need to renegotiate tortuous iliac vessels.

Before the IABP is handled, powder residue should be washed from the operator's sterile gloves to prevent alteration of the nonthrombogenic properties of the balloon surface. All air is evacuated from the balloon via a large (40- to 50-cc) syringe attached to the one-way valve supplied, so as to maintain the lowest possible profile during introduction. The guidewire lumen is flushed with heparin saline solution, and the balloon is loaded onto the special, stiff 0.021-inch guidewire supplied with the insertion kit. With the guidewire maintained above the carina (bifurcation of the trachea into left and right mainstem bronchi), the balloon should advance to that level with minimal resistance. When the radiopaque tip-marker reaches the carina (generally just distal to the left subclavian artery), the guidewire is removed, the central lumen of the balloon catheter is aspirated and flushed vigorously, and the central lumen is attached to either the coronary manifold or a pressurized flushing device equipped with a pressure transducer (Intraflow, Naimic, Glens Falls, NY) that delivers 3 mL/hr to maintain lumen patency. Special care must be taken to prevent inadvertent injection of air bubbles or thrombi, because the tip of the catheter is only a short distance below the aortic arch. The balloon shaft may be equipped with an outer sleeve, which can be advanced to the hub of the introducer sheath to maintain sterility if subsequent adjustment is required. If a long (23-cm) sheath has been used to negotiate a tortuous iliac artery, it must be partially withdrawn before initiation of counterpulsation, so that the distal end of the sheath does not overlie the distal end of the balloon.

Sheathless Insertion

Although insertion through a sheath is quite easy, most current balloons have a tapered nose to allow them to be inserted directly over a guidewire (i.e., without use of a sheath). Because the balloon shaft is roughly 1.5F (0.5 mm) smaller than the corresponding sheath outer diameter, sheathless insertion results in less femoral arterial trauma and less obstruction to the limb circulation in patients with small or atherosclerotic arteries. Care must be taken to adequately predilate the soft-tissue track and to avoid kinking either the guidewire or the balloon catheter during insertion, and the balloon catheter should not be rotated as it is passed through the soft tissues. If undue resistance is encountered, consideration should be given to reverting to a sheathed insertion.

Care of Central Lumen

Once the balloon is positioned, the central lumen is attached to a transducer with a 3 mL/hr continuous infusion
system pressurized to 300 mm Hg. Air must be purged from the system to prevent inadvertent air embolism, and heparin should be added to the flush solution at a concentration of 10 IU/mL. Power flushing with the continuous infusion system (which runs at 1.5 mL/sec when the valve is pulled open) should be avoided, in favor of syringe aspiration with one syringe and flushing with a second, unless counterpulsation is suspended temporarily. The central lumen should not be used for drawing blood samples. If the central lumen pressure waveform becomes damped and the cause is not related to a loose connection in the system, aspiration of the central lumen should be attempted. If there is marked resistance to aspiration of blood, do not flush. A thrombus may have formed within the lumen, which should then be considered occluded and capped off.

**Initiation of Counterpulsation**

Following connection to the console with the appropriate connector, the system is purged with helium inflation gas. The balloon is filled to half volume and counterpulsation is begun at the 1:2 setting (every other beat) so that preliminary timing adjustments can be made (see later discussion). Fluoroscopy confirms appropriate placement of the balloon proximally, full exit the sheath distally, and uniform expansion without twists or kinks. If it is necessary to adjust the position of the balloon, it may be moved within the sheath but must never be adjusted by advancing the sheath itself, because the latter maneuver may produce arterial damage at the distal end of the introduction sheath as it advances within the artery without the protection of a snug-fitting internal dilator.

Once the half-filled balloon is operating satisfactorily, the balloon should be increased to its full rated volume, and fluoroscopy should again verify that the balloon position is appropriate and that the balloon has assumed a uniform symmetric, cylindrical shape at full inflation. The balloon shaft and sheath (if used) are sewn to the skin, povidone-iodine (Betadine) ointment is applied to the entrance site, a mark is placed across the balloon shaft and the skin to detect any subsequent balloon migration, and a sterile dressing is applied. Five thousand units of aqueous heparin is given intravenously as soon as the balloon is inserted, followed by continuous intravenous heparin.

**Adjusting Counterpulsation Timing**

Maximal benefit depends on proper timing of inflation and deflation (Table 21.2). Timing should be done by inspection of the central aortic pressure tracing through the balloon guidewire lumen, because the change in contour and the timing of the pulse wave as it moves from the central aorta to the periphery can make accurate timing of counterpulsation difficult. Timing is best done with the console set at 1:2 pumping (i.e., counterpulsation of every other beat), so that arterial pressure tracings with and without counterpulsation can be compared (Fig. 21.1).

**FIG. 21.1.**

Appropriately timed counterpulsation of every other beat producing a decrease in both the end-diastolic pressure and the peak systolic pressure.

**Inflation**

While observing a high-fidelity central aortic pressure tracing, slowly move the inflation timing toward later inflation until the dicrotic notch becomes visible. Inflation should then be moved back to a slightly earlier time until the inflation upstroke fuses with the central aortic dicrotic notch to form a “U” (Fig 21.1). Earlier inflation should be avoided, because it would require a portion of LV ejection to be made against the resulting increased central aortic pressure, which can have disastrous consequences in a critically ill patient.

**Deflation**

Deflation should be timed to take place just before the opening of the aortic valve. Starting with deflation that is clearly too early (before the R wave), delay the timing of deflation progressively until the maximum reduction in aortic systolic pressure is observed in the following beat. This is usually accompanied by a parallel 10 to 15 mm Hg decrease in the nadir of central aortic diastolic pressure.
In the presence of atrial fibrillation or marked irregularity of the cardiac rhythm, balloon timing is best adjusted so that deflation occurs on the peak of the R wave to avoid LV ejection against an inflated balloon during occasional short R-R intervals. Atrial pacing may also produce timing difficulties if the console misinterprets the atrial pacing spike as the peak of the R wave. This can be overcome by timing the balloon off of the arterial pressure contour, by choosing a monitor lead that magnifies the difference between the ECG R wave and the atrial pacing spike, or by setting the console to the mode that discriminates between the pacing spike and R wave by sensing both the height and duration of the signal.

Balloon Pressure Wave Form

Although most timing decisions are made by reference to the central aortic pressure waveform, the console also has the capacity to display the pressure waveform in the helium drive system (the balloon waveform) (Fig. 21.2). The balloon waveform ordinarily has a rectangular appearance, onto which a sharp positive overshoot artifact is superimposed just before the peak inflation pressure and a negative deflation artifact at the end of deflation. Loss of these inflation and deflation artifacts produces a waveform with a blunted or somewhat rounded top, which may be seen when the balloon is too large for the aorta, there is a kink in the balloon catheter or connecting tubing, or the balloon is not fully unwrapped.

FIG. 21.2.

Early inflation—This is characterized by absence of the dicrotic notch on the arterial pressure tracing with encroachment of the diastolic augmentation pulse on the prior systole. Late inflation—There is a prominent dicrotic notch, indicating that balloon inflation has occurred well after aortic valve closure. The timing is adjusted until the dicrotic notch just disappears. Early deflation—Timing is adjusted until the aortic end-diastolic pressure is at its lowest value. Late inflation—The balloon remains inflated beyond the end of diastole and into the start of systole, resulting in an increase in resistance to ejection and cardiac work. The timing is adjusted earlier in diastole until the end-diastolic pressure is at its nadir.

Angiography During Counterpulsation

Whenever possible, we like to perform angiography or intervention first, before balloon placement through the same access site. Very unstable patients, however, require counterpulsation during the catheterization procedure. In this case, the balloon catheter is placed first, and cardiac angiography is then performed via either the brachial artery or the opposite femoral artery. If the contralateral femoral approach is used for angiography, a few precautions should be taken to avoid damaging the balloon membrane. The guidewire and catheters should be advanced beyond the level of the balloon with inflation suspended briefly and with the point of the tip of the catheter pointed away from the balloon toward the wall of the aorta (7). Once the catheter is in the ascending aorta, counterpulsation can be resumed. Counterpulsation does not interfere with catheter manipulation past the operating balloon, but the operator should remember to suspend balloon operation temporarily when the next catheter exchange is performed.

Patient Management During Counterpulsation

During counterpulsation, there should be daily evaluation for evidence of sepsis, thrombocytopenia, blood loss, hemolysis, vascular obstruction, thrombus, embolus, or dissection. Mild to moderate thrombocytopenia is expected as a result of platelet destruction, but the platelet count rarely falls below 50,000 to 100,000 per milliliter unless there is some other problem such as heparin-induced thrombocytopenia or disseminated intravascular coagulation. After balloon removal, the platelet count rapidly returns to normal (8).

The level of heparin anticoagulation must be monitored closely, maintaining the partial thromboplastin time at 50 to 70 seconds to prevent serious thrombotic or embolic complications. Evaluation of the circulation to the involved limb should take place once during each 8-hour nursing shift. Dorsalis pedis and posterior tibial pulses should be palpated and graded on a scale of 1+ to 4+ during each shift. The ratio of the Doppler systolic pressure of the calf to that of the upper arm, known as the ankle-brachial index, is a useful technique for evaluating the development of limb
ischemia. An index that is trending downward or is less than 0.5 indicates the presence of serious limb ischemia that may require early balloon removal.

Local sepsis can be minimized by good aseptic insertion technique, daily changes of dressings with application of providone-iodine (Betadine) at the insertion site, careful technique in changing bottles of flush solution, and avoidance of prolonged pumping (beyond 5 days). During insertion prophylactic antibiotics are not given routinely, but they should be administered if there is any compromise in sterile technique. Disseminated sepsis, an uncommon complication, mandates urgent balloon removal.

During counterpulsation, patients must be kept at bed rest. Hip flexion is restricted, and the head of the bed should not be elevated beyond 30°. A limb restraint is attached to the involved leg to maintain appropriate leg position during periods of sleep, confusion, or discomfort. Sedation may be required to achieve this goal.

**Weaning from Counterpulsation and Balloon Removal**

Balloon counterpulsation is a temporary support measure. The balloon is usually removed once the patient's condition has stabilized after the acute insult (usually 24 to 48 hours of support). Before removal of an intravascular balloon, the patient is weaned progressively from support, by decreasing the counterpulsation mode from 1:1, to 1:2, and then to 1:3 counterpulsation. Sufficient time should elapse between each stage to ensure that the patient is tolerating the progressive decrease in the level of hemodynamic support without exhibiting clinical deterioration. In order to reduce the chance of clot formation, pumping should not be reduced below 1:8 until immediately before balloon removal. Heparin should be stopped, and clotting parameters should have an activated clotting time of less than 160 seconds or a partial thromboplastin time of less than 50 seconds.

At this point, the balloon is turned off and a 50-mL syringe and stopcock is attached to the balloon inflation port to create a vacuum. The balloon is withdrawn to but not into the insertion sheath, because the latter maneuver may tear or even embolize a portion of the balloon membrane. After the skin sutures are cut, the sheath and balloon are withdrawn as a single unit. A small spurt of blood should be allowed to escape while the artery is compressed distally to help flush out any adherent thrombus above the site. The proximal vessel is then compressed while distal back-bleeding is allowed. The site is then firmly compressed by hand or with a mechanical compression device for 30 to 60 minutes. Distal limb circulation is checked during and after compression. The patient is kept at bed rest, avoiding hip flexion on the involved side, for the next 24 hours.

**Complications**

The success rate for percutaneous insertion is greater than 90%, but balloon counterpulsation can have serious complications. These have decreased progressively with improvement in balloon profile (usable through 8F and even 7F sheaths) and increased use of the sheathless technique. Still, operators must be aware of and try to prevent complications of the use of this valuable device.

**Limb Ischemia**

The most common IABP complication is local vascular insufficiency that resolves without permanent sequelae once the balloon pump is removed (9). Usually it is apparent within a few hours after insertion and is related to mechanical obstruction at or above the insertion site. The development of local thrombus may produce late ischemia. If the patient's status is too precarious to permit discontinuation of counterpulsation, a new balloon may be placed in the contralateral femoral artery, or cross-femoral grafting can be performed to relieve the ischemia without interrupting IABP support (10).

When balloon French size was much larger (10F and 11F), the rate of significant vascular complications was approximately 10%, with permanent morbidity about 5% and balloon-related mortality about 1% (9,11–25). Currently, limb ischemia requiring IABP removal and that requiring surgical intervention should each be less than 5% (23). An evaluation of the rate of complications in a single institution documented the adverse effects of larger balloons and more prolonged counterpulsation (25) (see later discussion).
To minimize ischemic vascular complications, the balloon pump catheter must be placed in the common femoral artery (see Chapter 4). The profunda and superficial femoral branches of the common femoral artery are too small to allow intraaortic balloon insertion without severe compromise of flow. If the puncture is made too low and the balloon is inserted distal to the common femoral artery, it is likely that evidence of severe obstruction will develop immediately, requiring balloon removal and contralateral insertion or ipsilateral reinsertion at the time of surgical balloon removal and vascular repair.

**Arterial Dissection**

Retrograde dissection (26),(27) may occur as an iatrogenic event at the time of wire advancement. The IABP may then be inserted and may even appear to function normally in the false lumen of the aorta, although severe back pain is often present. There is great danger of aortic rupture. Dissection was much more common when intraaortic balloons were inserted surgically, without the benefit of a guidewire. Echocardiography has been useful both in making the diagnosis and in avoiding the complication (28),(29).

The best defense against this complication is to avoid using stiff guidewires, avoid forcing the guidewire through the vascular system when there is resistance to advancement or pain with advancement, and avoid introducing the guidewire unless there is excellent pulsatile flow through the puncture needle. If there is any doubt concerning intraluminal position of the guidewire or the anatomy, a 5F dilator should be inserted over the guidewire; if there is good backflow of blood, gentle hand injections of contrast should be made to help define the anatomy before balloon insertion.

**Loss of Limb**

This is a rare complication that is usually related to prolonged shock, extensive thrombus formation, or cholesterol embolization (30). The latter is an especially ominous complication because surgical intervention is often ineffective in restoring adequate circulation. It is heralded by bilateral painful, cold, mottled limbs (livedo reticularis) shortly after the intervention. Many patients exhibit increased eosinophils in blood and urine sediment and thrombocytopenia. Rapidly progressive renal failure is common; it is most often permanent (31) and therefore unlike contrast-related nephropathy. Some authorities believe that chronic anticoagulation is contraindicated because further embolization may be promoted by the failure to form an organized thrombus over the eroded plaque (32).

**Cerebrovascular Accident**

Embolic cerebrovascular accident may occur if the balloon has been placed too proximally or if the central lumen of the balloon has been flushed vigorously to correct thrombus-induced damping of the central arterial pressure. As expected, this complication is more common with thoracic balloon placement. Except in extreme emergencies, the central lumen of the intraaortic balloon should not be used as a site for obtaining arterial blood or samples for chemistries. If venous access is difficult, a venous cannula or a side-arm cannula for right-sided heart catheterization can be placed in the internal jugular vein. An arterial cannula in the radial artery is a convenient site for frequent arterial blood samples.

**Sepsis**

If counterpulsation is carried out for less than 3 to 7 days, there is little correlation between the duration of counterpulsation and the development of local or disseminated sepsis. Although there are few studies of longer-term counterpulsation, most show increased local and disseminated sepsis when counterpulsation is carried out for longer than 1 week, suggesting that sepsis is a secondary event that may respond to the same kind of meticulous cleansing techniques applied to total parenteral nutrition catheters (33).

**Balloon Rupture**

This uncommon complication has been reported largely as a result of iatrogenic factors or equipment malfunction. Because helium is so insoluble in blood, helium embolization is a serious event, producing prolonged ischemia or stroke (34). Balloon rupture may produce massive helium embolization if there is failure of the console to recognize
the problem. Hyperbaric oxygen treatment has been applied to maintain tissue viability until helium excretion has taken place (35).

Balloon rupture may occur as a result of heavy calcification of the aorta (36). In addition to helium leakage, it may result in thrombus formation within the balloon that makes percutaneous removal impossible. Usually the balloon has been removed surgically in these instances (37–39), but percutaneous removal of a ruptured entrapped IABP has been accomplished by flushing out the thrombus within the balloon (40).

Risk Factors for Complications

Percutaneous Versus Surgical Insertion

Because of the simplicity and rapidity of insertion, percutaneous insertion has almost completely replaced the direct surgical insertion technique. The newer, low-profile balloons and the increased use of the sheathless percutaneous insertion (41),(42) have contributed to the low complication rate of the percutaneous technique, relegating surgical insertion to those balloons inserted as an emergency in the course of cardiac surgery. If severe peripheral vascular disease precludes the use of a femoral approach during thoracic surgery, thoracic insertion has been applied (43–45), with IABP-related complication rates as low as 4% (46). Although early studies suggested a lower complication rate associated with direct surgical insertion (41),(42), this is no longer the case with current devices and insertion techniques (23).

Sheathed Versus Sheathless Insertion

Sheathless insertion has decreased the IABP complication rate (47),(48). The technique is somewhat more demanding than the standard sheathed insertion technique, requiring a special guidewire with a stiff body and firm pressure over the puncture site during insertion to prevent artery laceration or balloon damage. Sheathless insertion may not be feasible in the presence of marked obesity or dense scar tissue at the puncture site related to prior femoral artery surgery.

Catheter Size

As the catheter size has decreased, there has been a progressive decline in vascular complications. A review of 381 patients between 1977 and 1995 found a vascular complication rate of 30% for surgical insertion, 21% for 12F catheters, 10% for 10.5F catheters, and 8.4% for 9.5F catheters. Additional independent risk factors were duration of counterpulsation beyond 48 hours, peripheral vascular disease, and shock (25). The recent development of 7F and 8F IABP catheters is likely to improve these figures even further.

Thoracic Aorta Insertion

This technique is largely limited to patients with severe peripheral vascular disease who require counterpulsation at the end of their cardiac surgery (43–45). Despite the high risks associated with the technique and with the patient population to which it is applied, the IABP-related complication rate for ascending aorta insertion could be as low as 4% (46). The SupraCor balloon (ABIOMED Cardiovascular, Inc., Danvers, MA) is similar to the existing intraaortic balloon but is designed for placement in the ascending rather than the descending aorta. In an animal study, conventional IABP had no effect on graft flow, whereas the ascending aorta device increased internal mammary bypass flow by 70% and increased venous conduit flow by 50% (49).

Miscellaneous Clinical Factors

The risks of a vascular complication from intraaortic counterpulsation are increased in the presence of vascular disease, female gender, and diabetes. A 1984 report noted that patients with peripheral vascular disease had a vascular complication rate of up to 31% after percutaneous insertion, compared with 16% after surgical insertion. Female gender was an additional risk factor (15% vs. 3.5%), but age, duration of counterpulsation, and indications for insertion were not (18). A single institution retrospective review of 436 IABP patients over a 14-year period in which
the indications for counterpulsation were intraoperative pump failure (42%), unstable angina (24%), preoperative prophylaxis (22%) and preoperative shock (9%) found that only the absence of pedal pulses on admission correlated with an increased incidence of vascular complications (50). Kantrowitz reviewed 733 patients, a large proportion of whom had received a surgically implanted IABP for cardiogenic shock. The vascular complication rate was increased in the presence of diabetes, hypertension, or female gender but was not related to duration of counterpulsation. Bacteremia was positively correlated with the duration of counterpulsation, and local or systemic infection was more common in coronary care unit insertion compared with operating room insertion (26% vs. 12%) (19).

An early analysis of the Beth Israel Hospital experience with 10.5F and 12F percutaneous sheath insertion balloons revealed some degree of limb ischemia in more than 40% of patients, almost one third of whom required balloon removal. Limb ischemia was related to the presence of diabetes, peripheral vascular disease, female gender, and an ankle-brachial pressure index of less than 0.8. There was no association between limb ischemia and age, body surface area, or 10.5F versus 12F balloon size (9). Of the 7,333 patients undergoing percutaneous left-sided heart catheterization procedures at the Beth Israel Hospital between 1980 and 1987, only 1% required operative repair of catheterization-related vascular complications, but the incidence of operative repair after transfemoral intraaortic balloon placement was 11.5%. Most of the patients developing limb ischemia after balloon placement did not require surgical repair, because limb ischemia often resolved with removal of the intraaortic balloon (20), (24).

The increased use of sheathless technique and the development of smaller-profile balloons have been accompanied by a dramatic decrease in the complication rate. A review of 200 consecutive patients undergoing sheathless insertion revealed a major complication rate of only 4.8% and acute limb ischemia requiring surgery in only 4%. At a mean follow-up of 17 months, there was only one false aneurysm and one new case of intermittent claudication (51). Other high-volume catheterization laboratories have duplicated these outstanding results (47).

**Indications and Contraindications**

Although it was designed initially as a support device for patients with cardiogenic shock, current indications for intraaortic balloon pumping have expanded from purely hemodynamic support to the management of refractory ischemia, even when it occurs in the absence of hemodynamic compromise (see Tables 21.3 and 21.4). Balloon pumping may even be instituted prophylactically before high-risk intervention in the setting of severe baseline hemodynamic dysfunction or intervention on a vessel that supplies a large part of the remaining viable myocardium.

**Clinical Results**

A large number of studies have been conducted to examine the hemodynamic effects of counterpulsation and its benefit in various clinical situations. Although the number of such studies is too large to review here, the most important ones are summarized.

**Hemodynamic Effects of Counterpulsation**

The rapid expansion of the balloon in early diastole produces an increase in diastolic pressure and thus coronary perfusion pressure. The abrupt deflation of the balloon at end-diastole removes effective aortic volume, decreasing aortic systolic pressure and thus the resistance to LV ejection (Fig. 21.1). These two effects—a decrease in myocardial oxygen requirements with a concomitant increase in coronary diastolic perfusion pressure—improve the myocardial supply/demand balance. The enhancement in LV emptying improves forward output and decreases left heart filling pressure, although to a lesser extent than the circulatory assist devices discussed later in this chapter.

**Cardiogenic Shock**

The effect of counterpulsation on systemic and coronary circulation is very much dependent on the extent of hemodynamic decompensation before initiation of intraaortic balloon pumping. When instituted relatively early in the course of cardiogenic shock in dogs, counterpulsation produces a 19% to 25% reduction in peak LV wall stress, with an increase in coronary blood flow (73–75) and a reduction in extent and severity of ischemia (76). Improvement in hemodynamics is especially prominent when cardiogenic shock is accompanied by a mechanical
defect such as a ventricular septal defect or acute mitral regurgitation (77). Generally, the institution of counterpulsation in cardiogenic shock produces a marked rise in systemic diastolic pressure and a 5 to 10 mm Hg fall in LV filling pressure, accompanied by a variable effect on systemic pressure and cardiac output (78).

The effect of counterpulsation on coronary hemodynamics in patients with shock is more difficult to characterize. Animal studies have not shown uniform increases in regional or global coronary flow, probably because of failure to control for other variables such as the degree of hypotension, degree of coronary stenosis, or presence of collateral vessels (79). Coronary flow is increased in IABP only when the pressure beyond a coronary stenosis has fallen below the level at which autoregulation can compensate fully (about 40 to 60 mm Hg). When the distal pressure is higher than this level, IABP may not change or may even decrease coronary flow if myocardial oxygen requirements have also been attenuated by an IABP-mediated decrease in LV wall stress. In the clinical setting with the added variable of multivessel disease, different degrees of collateralization, and variation in the severity of coronary stenosis, it is not surprising that the effect of counterpulsation on total coronary flow in patients with shock can be highly variable. In the presence of profound shock, however, both systemic and coronary flows increase significantly after initiation of intraaortic balloon pumping (80). At least in the short term, there is no effect of counterpulsation on renal blood flow or oxygen consumption of patients in shock (81).

Seventy percent of patients in cardiogenic shock studied 14 hours after IABP insertion showed no change or a fall in coronary blood flow with counterpulsation and no significant change in myocardial lactate extraction (74). A comparable group of patients studied 4 to 6 hours after initiation of counterpulsation showed an 18% decrease in LV systolic pressure and a 38% increase in cardiac index. Coronary sinus studies in these patients documented a 34% increase in coronary blood flow and a change from 6% lactate production to 15% extraction—that is, a change toward normal nonischemic myocardial metabolism (82). With multiple ECG leads in a dog model and in humans, counterpulsation produced a marked decrease in the extent and severity of myocardial ischemia (as determined by the sum of ST-segment elevations in multiple ECG chest leads) if it was applied within 3 hours after coronary occlusion (73).

In addition to the suggested benefits on coronary blood flow and ischemia just described, the initiation of counterpulsation in patients with shock has produced a uniform improvement in LV performance. Cardiac index and mean arterial pressure increase significantly, while LV filling pressure decreases substantially (75,78,83–85).

Initial improvement in hemodynamics is usually followed by recurrent deterioration after 3 to 4 days, if definitive revascularization or corrective surgery is performed (86). Although the usual treatment for cardiogenic shock is rapid stabilization followed by percutaneous transluminal coronary angioplasty (PTCA) or surgery, several days of counterpulsation may improve the high mortality rate of cardiogenic shock even if immediate revascularization is not feasible (87).

A retrospective community hospital study of patients with acute infarction accompanied by cardiogenic shock found that the addition of IABP to thrombolysis led to a much-improved hospital survival rate (93% vs. 37%). Although this was a retrospective study, age, systolic pressure, pulmonary capillary wedge pressure, incidence of anterior infarction, and presence of diabetes mellitus did not differ between the two groups (88). A subgroup analysis of 310 patients presenting with shock in the international multicenter Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO) study revealed a wide disparity in the use of the IABP. There was a trend toward lower 30-day (50% vs. 66%) and 1-year (56% vs. 70%) mortality rates . Differences in aggressive diagnostic and therapeutic interventions in the United States compared with the other centers included IABP use (35% vs. 7%), PTCA (26% vs. 8%), and cardiac catheterization (58% vs. 23%), but also lower 30-day (50% vs. 66%) and 1-year (56% vs. 70%) mortality rates (90).

In the absence of early revascularization, the mortality rate of cardiogenic shock is more than 85%. Patients with multiple infarctions or serious comorbidity, those requiring mitral valve replacement or repair for concomitant mitral regurgitation, and those with coronary anatomy that is unfavorable for bypass have the worst prognosis with surgery (84). Nonrandomized studies in which the survival rate for patients with successful revascularization was compared with that for patients in whom no attempt was made or the attempt failed have all shown a much worse survival rate.
in the latter group (older studies, reference 85; current studies, reference 91). A report of 200 consecutive patients presenting with acute infarction and cardiogenic shock found that the hospital survival rate was 67% with a patent infarct-related artery and 25% if the artery was occluded. In-hospital mortality was related to age, lowest recorded cardiac index, and the presence of left main coronary artery disease as well as infarction (54).

Delay in revascularization has a profound deleterious effect on survival. In a nonrandomized study, the in-hospital survival rate was 77% in patients revascularized within 24 hours but only 10% in those undergoing successful revascularization more than 24 hours after the onset of shock (53). The SHOCK Registry was a randomized trial of direct invasive strategy for patients with cardiogenic shock complicating an acute infarction. The mortality rate varied from 84% in patients with acute mitral regurgitation or ventricular rupture to 53% in patients with nonanterior infarction (92).

Counterpulsation has also produced favorable responses in patients with shock due to a nonischemic cause. In a reported series of noncoronary shock patients, most with acute myocarditis, IABP therapy was instituted in ventilated patients for whom pharmacologic support had failed. Three to 4 days of counterpulsation was sufficient to allow adequate recovery of ventricular function in 25% of the patients. The remainder required an LVAD or a biventricular support device for 3 to 79 days. At follow-up (7 to 54 months), all patients had recovered normal ventricular function and were asymptomatic, suggesting that patients with severe noncoronary shock may be supported for up to several weeks before transplantation is considered (93).

The IABP may be especially useful in mitigating the ischemic and hemodynamic abnormalities that accompany a failed angioplasty with abrupt closure. Even when catheter technique rescue is unsuccessful, rapid stabilization with mechanical support followed by prompt surgery can yield a favorable result (61). Of course these events have become unusual in the era of stenting to reverse abrupt closure (see Chapter 23), but counterpulsation should not be forgotten in the rare patient who still requires emergency bypass in the setting of profound ischemia.

Duplex scanning of the common carotid artery in patients during counterpulsation revealed an increase in diastolic flow but a reversal in systolic flow, resulting in no net change in total carotid flow (94).

**Unstable Angina, Acute Myocardial Infarction**

Patients with medically refractory unstable angina make up the large proportion of those treated by IABP. During counterpulsation, they may exhibit only modest decreases in peak systolic pressure and in LV filling pressure, with unchanged cardiac output, LV volumes, LV ejection fraction (LVEF), and regional contraction, as determined by angiographic chordal shortening (95). In patients with anterior wall ischemia, measurements of coronary sinus flow from the ischemic area may improve with counterpulsation, but this is not a uniform response (96).

In patients who were hemodynamically stable, coronary flow velocity was studied using an **intracoronary Doppler wire** (see Chapter 18). There was no increase in mean coronary flow velocity beyond a critical coronary stenosis (97), despite an increase in peak coronary flow velocity (98). This counterpulsation-induced effect on peak coronary flow beyond a critical stenosis was not observed in a carefully controlled study on normotensive dogs (99). In contrast, counterpulsation produced a dramatic increase in the integral of coronary flow velocity, a measure of coronary flow, in hypotensive patients with acute infarction or cardiogenic shock (100). It has been postulated that counterpulsation might improve unstable angina by increasing collateral flow to the ischemic area. In a small series of patients with acute infarction or unstable angina, there was a significant increase in collateral flow in the presence of angiographically documented collaterals but no change in collateral flow when collateral vessels were absent or too small to be seen by angiography (80).

In a dog study of thrombolysis with recombinant tissue plasminogen activator (r-TPA), IABP did not change the mean systemic pressure or coronary blood flow but did decrease the time to reperfusion, suggesting that the time to thrombolysis may be a pressure-dependent variable (101). A Doppler wire study of coronary flow velocity in patients with acute infarction and successful angioplasty revealed no change in mean coronary flow by counterpulsation, but peak velocity of flow increased from 35 to 47 cm/sec (98). A report of patients with anterior infarction and PTCA randomly assigned to either 24 hours of counterpulsation or conventional therapy found reocclusion rates of 2.4% in the IABP group and 17.7% in the conventional therapy group (102). Retrospective review of the Thrombolysis and
Angioplasty in Myocardial Infarction (TAMI) study noted a similar effect on reinfarction or reocclusion (103), and a study that randomly assigned patients with failed angioplasty to either conventional therapy or 48 hours of counterpulsation found a marked improvement in TIMI-3 flow in the counterpulsion group at the 3-week angiography (74% vs. 32% of patients), suggesting that counterpulsation may be a reasonable alternative in rescuing angioplasty to achieve late patency of the infarct-related artery (104).

There have been two large, multcenter, randomized trials of counterpulsion to maintain patency of the infarct-related artery. The first, reported in 1994, confirmed the benefit of this strategy (65). Patients in whom patency was restored within 24 hours after acute infarction were randomly assigned to standard therapy or to 48 hours of counterpulsation. Repeat angiography was performed on day 5. Reocclusion was present in 8% of the IABP patients but in 21% of those receiving standard care, who also were almost twice as likely to suffer death, stroke, or a recurrent ischemic event (65). This benefit was accomplished without any substantial increase in hospital costs (105).

The enthusiasm for counterpulsion to prevent reocclusion after angioplasty has been tempered, however, by the results of a large multcenter study, the Second Primary Angioplasty in Myocardial Infarction (PAMI-II). This prospective, randomized trial studied 1,100 patients within 12 hours after acute infarction. Clinical and angiographic variables were used to stratify patients undergoing primary PTCA into high-risk and low-risk groups. The high-risk patients were randomly assigned to 36 to 48 hours of IABP or standard care. Although IABP produced a decrease in recurrent ischemia (13.3% vs. 19.6%) and unscheduled recatheterization (7.6% vs. 13.3%), it did not reduce the rate of infarct-related artery occlusion (6.7% vs. 5.5%), reinfarction (6.2% vs. 8.0%), or death (4.3 % vs. 3.1%). The authors concluded that prophylactic IABP after primary PTCA in hemodynamically stable high-risk patients with acute myocardial infarction did not decrease the rate of infarct-related artery reocclusion or reinfarction, promote myocardial recovery, or improve overall clinical outcome (106). The reocclusion rate in the control group was extraordinarily low, and the disparity between these two studies may be related to both the clinical characteristics of the patients and the rapidly evolving changes in the techniques of intervention and anticoagulation. The control group reocclusion rate in the more recent study was less than that of the IABP group in the 1994 investigation (5.5% vs. 8%). Counterpulsion may offer little extra benefit for decreasing reocclusion in current intervention practice.

**Prophylactic Intraarticular Balloon Pump Therapy in High-Risk Angioplasty**

The application of new devices, most especially stents and atherectomy, has resulted in a dramatic decrease in the need for prophylactic IABP support. It is prudent to have the IABP console and catheters immediately available and, especially in high-risk interventions, to have placed a 5F dilator in the contralateral femoral artery so that counterpulsion can be instituted with minimal delay. Although prophylactic counterpulsion has been applied especially in high-risk interventions, to have placed a 5F dilator in the contralateral femoral artery so that counterpulsion can be instituted with minimal delay. Although prophylactic counterpulsion has been applied successfully and with a surprisingly low complication rate in this very sick group of patients (62–66,107), in one series high-risk patients did equally well but had fewer vascular complications when counterpulsion was not used (108). The National Registry of Supported Angioplasty reported the results in 801 patients with supported or standby-supported angioplasty. Patients older than 70 years of age and those with left main coronary artery stenosis were at higher risk, and those with LVEF less than 20% did better with prophylactic support. However, those with greater than 50% of myocardium in jeopardy and low LVEF can undergo intervention with a 7.2% in-hospital mortality rate with either prophylactic or standby IABP support (109). With standby CPS, the in-hospital mortality rate for a similar group of patients was 6.0% (110). A nonrandomized comparison of prophylactic IABP with prophylactic CPS did not reveal any differences in myocardial infarction, stroke, emergency bypass surgery, or death, but there was a higher rate of peripheral vascular complications in the CPS group (111). This suggests that the mortality rate is more a function of the baseline characteristics of the patient than of the support device used.

A retrospective analysis of 159 consecutive high-risk patients undergoing rotational atherectomy revealed that the 28 patients with elective preprocedure IABP placement had the same occurrence of slow flow (18% vs. 17%), equal hospital stay, and similar vascular complication rate, compared with controls. However, among the patients developing slow flow, there were no non–Q wave myocardial infarctions in the IABP group, compared with 27% in the control group (112).

**Emergency Coronary Artery Bypass Grafting for Failed Percutaneous Transluminal Coronary Angioplasty**

A retrospective review of emergency coronary artery bypass grafting (CABG) operations for failed PTCA
documented a marked change over a single decade. Of 9,145 patients undergoing PTCA from 1980 through 1990, the group treated from 1980 to 1985 was compared with the subset treated from 1986 through 1990. Although the incidence of emergency CABG within 24 hours after PTCA fell from 3.8% to 2.3%, the mortality rate rose from 4.6% to 7.6%. The patients in the more recent period had a higher incidence of prior PTCA and class III or IV symptoms, but the major determinant of in-hospital mortality after emergency surgery for failed PTCA remained the hemodynamic status at the time of surgery. Patients who required cardiopulmonary resuscitation or were in shock had a mortality rate of 28% (13/46), whereas those with stable hemodynamics at time of emergency CABG had a mortality rate of only 1.4% (3/207). Preoperative IABP use increased from 13% in the first period to 33% in the second. Late survival was excellent, 92% at 2 years and 87% at 5 years

Bridge to Cardiac Transplantation

IABP has been successful at maintaining severely decompensated patients awaiting suitable donor hearts. Although the support is less impressive than that delivered by CPS or the ventricular assist device, the IABP is much more easily maintained over the intermediate term and often is enough to supply the required degree of hemodynamic support (114).

Enhanced External Counterpulsation.

Enhanced external counterpulsation (EECP) has been used to achieve some of the benefits of the IABP with a noninvasive device that produces serial inflation of three sets of cuffs wrapped around the calves, the thighs, and the buttocks, timed to inflate in diastole according to the surface ECG. (Commercial systems are available from Vasomedical Inc., Westbury, NY, and from Cardiomedics Inc., 18872 Bardeen Ave, Irvine, CA 90612.) In a randomized trial comprising 258 patients with acute infarction, application of EECP for 3 hours within 24 hours after presentation resulted in a reduction in mortality in the subgroup of elderly patients (115). When EECP was applied 1 hour each day for 7 weeks to a group of 18 patients with chronic stable angina, exercise treadmill studies revealed improvement in exercise duration, resolution of thallium defects in 12 patients, decreased ischemic area in 4 patients, but no change in systolic blood pressure × pulse double product (116). At 3-year follow-up, 13 of the 18 patients remained free of angina (117). In some patients the EECP has produced dramatic improvement in symptoms and radionuclide stress tests after failure of all other usual modalities of treatment (118). It is presumed that the mechanism for improvement is collateral development, which is dependent on patency of neighboring vessels, there being less improvement in patients with residual three-vessel disease. Although the improvement in the thallium study suggests that there is a decrease in ischemia, the unchanged double product indicates that the improvement is partly related to decreased peripheral vascular resistance and the heart rate response to exercise (119). By means of Doppler techniques to study flow velocity, mean carotid flow velocity was shown to increase by 22% and mean renal artery flow velocity by 19% during sequential external counterpulsation (120).

In the Multicenter Study of Enhanced External Counterpulsation (MUST-EECP), 139 patients were randomly assigned to either hemodynamically inactive counterpulsation or active counterpulsation. In the latter group, total exercise time was increased by almost 1 minute. Angina episodes decreased in the active counterpulsation group only, but hospitalization rates were the same in both groups (121).

Pulmonary Artery Counterpulsation

Rare case reports of pulmonary artery counterpulsation have shown favorable hemodynamic effects on the failing right ventricle, but these patients had profoundly abnormal hemodynamics and most did not survive (122–124). Placement in these cases was in the operating room under direct vision, but percutaneous deployment has been possible in animals (125).

PERCUTANEOUS CARDIOPULMONARY SUPPORT

Although the IABP provides effective circulatory assistance or ischemia relief for the majority of patients, it usually does not increase the cardiac output by more than 1 L/min. This is not adequate to completely support patients with severely compromised pump function. Newer and more potent forms of CPS have been developed for this purpose,
some of which can even support the circulation in the presence of full cardiac arrest. Because these devices markedly decrease cardiac work and myocardial oxygen consumption, they may also be much less effective in reversing myocardial ischemia in the presence of a complete coronary occlusion. The increase in hemodynamic support is accomplished at the cost of much increased complexity and expense for the ventricular assist device and for the CPS device.

**Bard Cardiopulmonary Support System**

**Design**

The Bard percutaneous cardiopulmonary support system (PCPS, C.R. Bard, Billerica, MA) is analogous to the heart-lung machine used during open heart surgery. The basic design is shown in Fig. 21.3. Venous blood is withdrawn from the right atrium and vena cavae, through the Bio-medicus pump, heat exchanger, and membrane oxygenator, after which it is pumped back into the aorta via the percutaneously inserted arterial cannula. The system is battery powered and mounted in a portable cart. Initially the cannulae were inserted surgically, but this has been supplanted by percutaneous femoral artery and vein insertion.

**FIG. 21.3.**

The Bard cardiopulmonary support system (C.R. Bard, Billerica, MA) uses a Bio-medicus pump to pass venous blood through a heat exchanger and membrane oxygenator and into the arterial system. The extra lines are available to provide pressure monitoring, a recirculation loop, and a purging circuit.

**Prophylactic Versus Standby Cardiopulmonary Support**

In contrast to prophylactic use of PCPS, which is defined as the implementation of bypass support before coronary angioplasty, standby-supported angioplasty refers to a planned situation in which both PCPS equipment and personnel are available in the cardiac catheterization laboratory should hemodynamic collapse develop. Most high-risk PTCA's can be performed safely without advance placement of cannulae or initiation of bypass so long as cardiopulmonary bypass support is immediately available. Iliac angiography is performed, followed by placement of 5F sheaths in the artery and vein contralateral to that being used for intervention. Unplanned cardiopulmonary bypass support has been much less effective, in large part because even a short hiatus between cardiac arrest and the institution of hemodynamic support has a major impact on survival. In a high-volume catheterization laboratory that is very experienced in the CPS system, elective coronary interventions have been performed without formal surgical backup but with immediately available standby CPS, which was required in 0.4% of the cases—none of which fell into the high-risk category for PTCA. The importance of rapid initiation of CPS is supported by the lower mortality rate for patients sustaining hemodynamic collapse in the catheterization laboratory (126).

**Contraindications**

The contraindications for PCPS are similar to those for IABP counterpulsation-aortic regurgitation, uncontrolled sepsis, uncontrolled bleeding diathesis, contraindication to anticoagulation, severe peripheral vascular disease, aortic dissection, or severe aortic aneurysm.

**Elective Cardiopulmonary Support Technique**

Because of the large diameter of the cannulae, insertion is preceded by iliofemoral angiography to ascertain feasibility of cannulae placement. Care is taken to ensure that puncture is below the inguinal ligament but in the common femoral artery. Access is achieved in both the femoral artery and the ipsilateral femoral vein using a 0.038 inch J guidewire and 8F long dilator. Anticoagulation is instituted with heparin and followed with regular determinations of activated clotting time. The wire is then replaced with a 0.038-inch heavy-duty guidewire with a
stiff body and a flexible tip. The femoral artery and vein are progressively dilated, first with an 8F, then a 12F and a 14F dilator, followed by the 18F cannulae, which are positioned in the middle right atrium and in the aorta at the bifurcation. The pump uses a disposable system that must be primed, but this can be accomplished simultaneously with catheter insertion. Venous return is via active suction, not gravity, making it essential that patients be well hydrated and that all venous lines remain closed to prevent air embolism. For this reason central venous access should not be attempted while the pump is operating. Cardiopulmonary bypass is initiated at 2.0 L/min and progressively advanced as needed in increments of 0.5 L/min. Pumping can be performed for up to 6 hours before changes in pulmonary and hematologic function require discontinuation. Beyond 6 hours, platelet aggregation, hemolysis, a bleeding diathesis, and increased capillary permeability with plasma loss become major problems. If the cannulae were inserted to perform a high-risk angioplasty, the patient can be weaned over a 15-minute period, with a further 30-minute period of observation prior to removal of the cannulae. Hemostasis is achieved with the use of a mechanical clamp.

**Cardiopulmonary Support Physiology**

In a study of the hemodynamic effects in 14 patients undergoing high risk PTCA, pulmonary artery pressure decreased from 45/23 to 27/14 mm Hg, and the mean pulmonary arterial pressure went from 30 to 18 mm Hg. Aortic systolic pressure decreased from 129 to 106 mm Hg, and mean aortic pressure from 89 to 84 mm Hg, but diastolic pressure and heart rate remained unchanged. Calculated end-systolic wall stress decreased from 122 to 96 dynes-cm² (127). A similar study in 20 patients receiving CPS support found a comparable reduction in afterload and in LV wall stress but no change in LV size or global LV function. Regions supplied by vessels with greater than 50% stenosis deteriorated during CPS, whereas those supplied by normal vessels did not. During balloon inflation under CPS support, two-dimensional echocardiography revealed evidence of regional myocardial dysfunction in the area supplied by the vessel being dilated (128).

**Results**

When PCPS has been placed outside of the catheterization laboratory for cardiac arrest, the long-term survival has been disappointing, despite short-term improvement, because of the delay between the acute event and the institution of PCPS. Patients with severe hemodynamic compromise experience marked hemodynamic improvement in almost all cases, but the long-term survival rate is disappointing (110,129–134). The application of PCPS is most effective when performed in a catheterization laboratory that is already prepared for insertion and technical support (135).

**Supported High-Risk Percutaneous Transluminal Coronary Angioplasty**

The group at Washington Adventist Hospital (Takoma Park, MD) reported on 107 patients with unstable presentation and severely depressed LVEF managed by PCPS-supported intervention. Mean LVEF was 19%, 47% of patients were deemed unsuitable for bypass surgery, and 54% had only one remaining patent artery. Ninety eight percent were successfully dilated with an in-hospital mortality rate of 4.7% and a 21% further mortality rate within 2 years after the procedure. LVEF increased from 21% to 29% in the patients in whom it was measured. Survival free of cardiac symptoms at 1 and 2 years was 83% and 77%, respectively (136). The multicenter registry reported the results in 105 patients with similar anatomic and hemodynamic profiles. The angioplasty success rate was 95%, and the in-hospital mortality rate was 7.6%. Half of the hospital mortality occurred in patients older than 75 years of age who had left main coronary artery disease. If those patients were omitted, the mortality rate fell to 2.6%. Over the next 12 months of follow-up, there were only three additional cardiac-related deaths (137).

**Prophylactic Versus Standby Percutaneous Cardiopulmonary Support**

As interventional catheter techniques have advanced, the necessity for prophylactic support has diminished markedly. A retrospective comparison was made of 389 patients undergoing prophylactic PCPS for a high-risk angioplasty and 180 standby patients for whom preparations were made but PCPS was not initiated before intervention. Only 13 (7.2%) of the standby group sustained a hemodynamic collapse, 12 of whom underwent successful PCPS within 5 minutes. Procedural success was equivalent: 88.7% in the prophylactic group and 84.4% in the standby group. Femoral access complications and the requirement for transfusion were more frequent in the prophylactic group (42%) than in the standby group (12%) (110). Standby PCPS requires immediate access to the technical support required to operate the system but provides a comparable success rate with a much lower morbidity.
Emergency Percutaneous Cardiopulmonary Support for Cardiac Arrest

The national registry experience confirms the importance of early initiation of PCPS. The long-term survival rate was 38% for patients in whom PCPS was operating within 20 minutes after the acute event and only 18% for those requiring more than 20 minutes (133). From a practical standpoint, this means that the device is not likely to be useful for cardiac arrest out of the catheterization laboratory, or even for catheterization laboratory emergencies unless the pump technician and equipment are available immediately (134–137). A subset of patients with hemodynamic collapse due to massive pulmonary embolization may respond well to PCPS, permitting stabilization for emergency embolectomy (138). In at least one case of critical aortic stenosis with cardiac arrest during diagnostic catheterization and unsuccessful cardiopulmonary resuscitation for 45 minutes, institution of peripheral CPS restored cardiac rhythm and blood pressure, with a subsequent successful aortic valve replacement (139).

Complications

Most of the complications are caused by local vascular problems, but there has been a dramatic reduction in the vascular complication rate reported with PCPS, which is now less than 1.5% in elective patients. This improvement has been largely related to changes in anticoagulation protocol and in the duration of support.

Conclusion

In the current interventional era, the full hemodynamic support provided by PCPS is rarely necessary, and the continuous technical back-up required for operation of the CPS device is a major impediment to its widespread use. Furthermore, the failure of the PCPS system to provide increased myocardial perfusion distal to a total occlusion is a serious limitation of the technique: despite the presence of excellent systemic flows and extreme reduction in myocardial oxygen demand, an occluded coronary artery may continue to produce significant myocardial ischemia unless an autoperfusion catheter can be advanced across the occlusion. Therefore, even in the presence of affective PCPS support, acute coronary occlusion after PTCA that cannot be stabilized by catheter technique should be treated with CABG to prevent a large myocardial infarction. The availability of PCPS does not change the requirement for rapid CABG surgery in the event of abrupt and uncorrectable coronary occlusion after PTCA.

Temporary Left Ventricular Assist Devices

Design

The Hemopump (Medtronic Hemodynamics, Minneapolis, Minnesota, 800-678-2500) is a temporary LVAD that uses a single coaxial catheter inserted into the LV cavity by surgical exposure of the femoral artery (21F catheter) or by percutaneous insertion via the femoral artery (14F catheter). A flexible silicone cannula is placed across the aortic valve into the LV cavity. The surgically implanted 21F device can achieve flows of 6 L/min, but the percutaneous 14F catheter can reach only 2 L/min. A rotating turbine fits snugly within the cannula and imparts both a rotational and a longitudinal velocity to the blood. To eliminate the rotational motion of the blood as it exits the catheter, static vanes are mounted downstream from the rapidly rotating turbine pump. Power to the pump is provided by means of a magnetically coupled sheath drive cable, providing a system that is entirely enclosed, with no contact between blood and the drive motor. The pump can be driven at speeds up to 25,000 rpm to provide a pumping rate of up to 3.5 L/min in a continuous, nonpulsatile manner. Forty percent dextrose in water is used to lubricate the 9F sheath that contains the drive cable as well as the pump itself. The control console is compact and easily portable.

Originally designed as an LVAD, the unit has been modified to be used as a right ventricular assist device (RVAD) or biventricular support device (BiVAD).

Percutaneous Insertion Technique

This is similar to the technique required for the PCPS system described, except that only arterial access is necessary for the support system. Single-wall puncture is used, the iliofemoral anatomy is defined, the guidewire is exchanged for a heavy-duty, stiff guidewire, and the femoral artery is progressively dilated to accommodate the 16F sheath. The
pump is advanced to the abdominal aorta, turned on at minimum flow, and then advanced slowly over the guidewire until it enters the left ventricle. Proper placement of the pump is critical. The pump should be 4 cm above the aortic valve sinus to prevent damage to the aortic valve leaflets.

Management During Pumping

Before insertion, arterial blood gas analysis, complete blood count with platelet and differential counts, liver function tests, fibrinogen, fibrin split products, partial thromboplastin time, prothrombin time, and activated clotting time measurements are obtained. A pulmonary arterial catheter and intravenous line should be in place, and a baseline echocardiogram is obtained. Prophylactic antibiotics are administered. Care must be taken to avoid damage to the surface of the cannula to maintain its resistance to thrombus formation. After insertion of the pump, anticoagulation is achieved with the use of intravenous heparin. The pump rotation speed is increased progressively until a satisfactory pumping level has been achieved. A chest x-ray is obtained to monitor cannula position. During the use of the Hemopump, the patient should be monitored closely for signs of complications including coagulation abnormalities, sepsis, arrhythmias, embolization of LV thrombus either systemically or within the 21F pump cannula, decreased peripheral circulation, and cannula migration. A daily chest x-ray should be obtained to check on cannula placement.

Discontinuation of Pumping

A trial of reduced speed pumping is performed every 24 hours with close monitoring of arterial blood gases, cardiac output, heart rate, right-sided heart pressures, and systemic pressures. If this is tolerated well, pumping is reduced gradually every 4 hours. When the patient has been weaned to the lowest pump speed without evidence of hemodynamic deterioration, the pump may be removed.

Indications and Contraindications

This device has been used for cardiac support during high-risk intervention, as a bridge to transplantation, and to treat patients with severe hemodynamic compromise due to acute myocardial infarction, myocarditis, valve disease, or other condition. It is contraindicated in patients with sepsis, bleeding diathesis, aortic dissection or large aortic aneurysm, prosthetic aortic valve, aortic stenosis, aortic insufficiency, or LV or left atrial thrombi. As is true for the PCPS system, severe peripheral vascular disease is a relative contraindication that can sometimes be overcome by antecedent femoral or iliac artery intervention.

Clinical Applications

High-Risk Catheter Intervention

The Hemopump was first used in 1994 for this purpose, and the favorable outcome in two patients encouraged further use (140). The percutaneous Hemopump was used in 32 high-risk patients undergoing catheter intervention. There was good unloading of the left ventricle and maintenance of cardiac output during periods of severe ischemia. In three patients with cardiac arrest, the mean aortic pressure was maintained at 50 mm Hg. However, mortality was high (12.5%), as was procedure-related morbidity. Femoral artery occlusion occurred in two patients, and severe bleeding requiring transfusion was present in four patients (141),(142).

Acute Myocardial Infarction Complicated by Shock

In comparison to the intraaortic balloon, LVAD has shown improvement in regional myocardial blood flow, LV unloading, and infarct salvage in a dog model (143). In patients, initial studies using the 21F surgically inserted device showed an associated mortality rate of 64% while receiving support or immediately after its removal. Although hemodynamics improved substantially within 24 hours (pulmonary capillary wedge pressure decreased from 26 to 16 mm Hg, and cardiac index from 1.6 to 2.4 L/min/m²), the overall survival rate remained only 36% (144). Despite these disappointing long-term results, there have been reports of remarkable recovery from profound
shock with near-fatal stunning after acute infarction with LVAD insertion (145). In an animal study, the addition of IABP to Hemopump LVAD support resulted in an increase in myocardial blood flow to ischemic regions, restoring the flows to normal while the perfusion of peripheral organs remained unaltered (146).

**Bridge to Transplantation**

Although emergency temporary support can be accomplished with the units described earlier, these patients usually require a longer period of hemodynamic support, which is best supplied by surgically implanted LVAD or BiVAD units.