Intra-aortic balloon pumping in infants and children

L. GEORGE VEASY, M.D., ROBERT C. BLALOCK, B.A., JEFFREY L. ORTH, AND MARK M. BOUCEK, M.D.

ABSTRACT  From November 1981 to November 1982, intra-aortic balloon pumping (IABP) was used after surgery in eight patients who were from 6 weeks to 6 years old and who weighed from 4.2 to 16.2 kg. In seven patients, specially constructed intra-aortic balloons with 2.5 and 5.0 ml volumes mounted on No. 5F catheters were used. In the largest and oldest patient, a two-chamber 10 ml balloon was used. The pumping module used was the Dataspot System 82. Effective diastolic augmentation of arterial pressure was accomplished in seven of the eight patients and suprasystolic diastolic augmentation was accomplished in four. The two youngest and smallest patients are the only long-term survivors. There were two short-term survivors who died 5 and 10 days after successful IABP. In only one patient was there no appreciable effect of IABP. Miniaturization of the equipment has permitted IABP to be used effectively in pediatric patients. 

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INTRA-AORTIC BALLOON PUMPING (IABP) has become a standard mode of treatment for perioperative low cardiac output syndrome and cardiogenic shock after myocardial infarction in adults.1-3 However, IABP has been used little in children. The only report of the use of IABP in children suggests that it is not successful in those who are under 5 years of age.4 Our experience with both in vitro and in vivo testing of small balloon catheters in small animals with three different pumping modules convinced us that this valuable method of circulatory support could be used in children.5 6 This report describes our initial experience in eight patients who underwent IABP after open heart surgery.

Materials and methods

All eight patients had previously undergone open heart surgery for correction of the lesions listed in table 1. All balloon catheters were introduced by cutdown through the common femoral artery with a side-arm Dacron graft. Introduction of the balloon catheters was accomplished in the operating room in two patients and in the intensive care unit in six patients. All the catheters were connected to the Dataspot System 82 pumping module modified with a pediatric volume-limiting chamber. The catheter position was checked by chest x-ray to confirm that the tip of the catheter was adjacent to the origin of the left subclavian artery.

The balloon catheters were inserted only after maximum use of pharmacologic support had failed to improve the patients’ low cardiac output syndrome (table 1).

A two-chambered 10 ml balloon was used in the first and largest patient. In all others either a 2.5 or 5.0 ml volume balloon was used. These balloons were mounted on a No. 5F catheter and were specially manufactured by Dataspot Corporation. The 2.5 ml balloon has an 8.0 mm diameter and is 10.5 cm in length while the 5.0 ml balloon has a diameter of 10.0 mm and is 13.0 cm long (figure 1).

The electrocardiograph was used for triggering the inflation and deflation of the balloon and adjustments were made for inflation and deflation time to permit optimal alteration of the patient’s pulse contour (usually from the right radial artery).

The balloon size and duration and outcome of IABP in each patient are shown in table 2.

Patient histories

Patient No. 1. R. C. was a 6-year-old white boy with Holt-Oram syndrome who underwent open heart repair of multiple ventricular septal defects and repair of previous pulmonary artery banding. A left ventriculotomy was required to position the large patch necessary to cover his ventricular septal defects. Inotropic support was needed during the first 4 postoperative days. He then appeared to be on his way to recovery. However, on his tenth postoperative day he developed pulmonary edema as a result of acute left heart failure. He failed to respond to inotropic drugs and 100% O2. A two-chambered 10 ml IAB was inserted into his thoracic aorta through a Dacron sleeve sutured to his common femoral artery. IABP effected suprasystolic level augmentation of his diastolic pressure with a 14 mm Hg drop in end-diastolic pressure. The initial heart rate was 130 beats/min, which slowed to 105 beats/min after 4 hr of IABP. Thermodilution cardiac output data and mixed venous samples were not available. Peripheral perfusion improved and urine output increased from 0.6 ml/kg/hr for the 3 hr preceding IABP to 8.0 ml/kg/hr for the first 2 hr after initiation of IABP. After 3 hr of IABP, his PaO2 increased from 58 to 117 mm Hg. The following morning the patient’s chest film showed clearing of pulmonary edema. IABP was maintained on every other beat for 3 days and then every third beat for 2 more days. The morning
VEASY et al.

TABLE 1
Patient characteristics

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (M)</td>
<td>6.1</td>
<td>16.2</td>
<td>Muscular VSDs; pulmonary artery band</td>
</tr>
<tr>
<td>2 (F)</td>
<td>1.3</td>
<td>9.6</td>
<td>Congenital mitral valve deformity</td>
</tr>
<tr>
<td>3 (M)</td>
<td>0.5</td>
<td>5.2</td>
<td>Atrioventricular defect</td>
</tr>
<tr>
<td>4 (F)</td>
<td>1.2</td>
<td>7.9</td>
<td>Atrioventricular defect</td>
</tr>
<tr>
<td>5 (M)</td>
<td>0.1</td>
<td>4.2</td>
<td>Congenital mitral valve deformity; atrial septal defect</td>
</tr>
<tr>
<td>6 (F)</td>
<td>0.7</td>
<td>5.8</td>
<td>d-TGA; VSD</td>
</tr>
<tr>
<td>7 (F)</td>
<td>0.8</td>
<td>5.3</td>
<td>Atrioventricular defect</td>
</tr>
<tr>
<td>8 (M)</td>
<td>0.5</td>
<td>6.1</td>
<td>d-TGA; VSD; pulmonary artery band</td>
</tr>
</tbody>
</table>

The ages of the patients in whom IABP was used ranged from 6 weeks to 6 years; weights ranged from 4.2 to 16.2 kg. The lesions requiring open heart repair are listed.

VSD = ventricular septal defect; d-TGA = d-transposition of great arteries.

after the discontinuation of IABP, he unexpectedly developed ventricular fibrillation and could not be resuscitated. At post-mortem examination the patch covering his ventricular septal defects measured 4.0 x 3.5 cm. There were hemorrhagic areas around the patch extending into the papillary muscle and free wall of the ventricle. Microscopic examination showed multiple areas of fibrosis of both ventricles. There was no evidence of thrombus formation or of aortic injury from the prolonged period of IABP.

Patient No. 2. C. C. was a 16-month-old, 9.6 kg white girl who underwent prosthetic replacement of a congenitally deformed mitral valve that was causing severe mitral stenosis. A 17 mm Bjork-Shiley valve was inserted, but because of the small mitral annulus, part of the valve was anchored in the left atrium. This resulted in abnormal medial orientation of the valve orifice. The patient had been on cardiopulmonary bypass for a total of 55 min and when she could not be weaned from it after an additional 20 min of support, a 2.5 ml IAB was inserted into the thoracic aorta through a Dacron sleeve attached to the left common femoral artery. After approximately ½ hr of IABP, and while on 0.2 µg/kg/min iv isoproterenol and 0.2 µg/kg/min iv epinephrine, her blood pressure stabilized (90 mm Hg peak systolic) and she was taken from the operating room to the intensive care unit. While pressure recordings were not made, the monitoring scope displayed definite augmentation of diastolic pressures, although not at suprasystolic levels. Approximately 30 min after her return to the intensive care unit, her blood pressure suddenly dropped to zero and she became asystolic. Vigorous resuscitative efforts were to no avail. While the cause of death could not be defined since autopsy permission was denied, the clinical course suggested malformation of the prosthesis.

Patient No. 3. J. N. was a 6-month-old, 5.2 kg white boy who underwent repair of a complete atrioventricular defect. After the 50 min repair on cardiopulmonary bypass, he continued to have low cardiac output in spite of vigorous pharmacologic support (10.0 µg/kg/min dopamine, 2.0 µg/kg/min nitroprusside, and 0.12 µg/kg/min epinephrine). Approximately 6 hr after his return to the intensive care unit, a 2.5 ml IAB was inserted into the thoracic aorta by a cutdown on the right common femoral artery with the use of a Dacron sleeve. Effective diastolic augmentation of arterial pressure was obtained with IABP at a rate of 168 beats/min. Better diastolic augmentation was noted, however, when IABP was timed for every other beat. At a rate of 140 beats/min, however, there was no difference between the augmentation accomplished with pumping at every other beat compared with at every beat. The patient’s clinical status showed progressive improvement with a steady rise in blood pressure, improved peripheral perfusion, and an increase in the mixed venous Po2 from 26 to 44 mm Hg after 4 hr of IABP. There was striking improvement in urine output from 0.3 ml/kg/hr in the 2 hr before to 9.0 ml/kg/hr in the second hour after IABP was started. By the following morning he had improved to the point that the rate of IABP could be decreased to every third beat; IABP was discontinued after 15 hr. This patient was our first long-term survivor. He left the hospital on his twenty-fourth postoperative day and is now doing well 10 months after surgery.

Patient No. 4. C. C. was a 14-month-old, 7.9 kg white boy who underwent open heart repair of a complete atrioventricular defect that included absence of the normal right superior vena cava and a persistent left-sided superior vena cava. The latter was redirected from the left atrium to the right atrium with a pericardial baffle. Although his septal defects were completely repaired, he was left with significant mitral regurgitation. After a rather stormy 8 day postoperative course with continued deterioration, a decision was made for a trial with IABP. As in the previously described patients, a 2.5 ml IAB was inserted while the patient was in the intensive care unit. Effective diastolic augmentation of arterial pressure was obtained at a rate of 140 beats/min. IABP was maintained for the next 4 days, during which time he was maintained on a dopamine intravenous drip.

FIGURE 1. Small balloons of 2.5 and 5.0 ml volume mounted on No. 5F catheters were used in all but the oldest (6 years old) patient.
TABLE 2

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>Balloon size (ml)</th>
<th>Augmentation</th>
<th>Duration</th>
<th>Survival outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6.1</td>
<td>16.2</td>
<td>10.0</td>
<td>+</td>
<td>5 days</td>
<td>Short-term death</td>
</tr>
<tr>
<td>2</td>
<td>1.3</td>
<td>9.6</td>
<td>2.5</td>
<td>+</td>
<td>2 hr</td>
<td>Death</td>
</tr>
<tr>
<td>3</td>
<td>0.5</td>
<td>5.2</td>
<td>2.5</td>
<td>++</td>
<td>15 hr</td>
<td>Long-term death</td>
</tr>
<tr>
<td>4</td>
<td>1.2</td>
<td>7.9</td>
<td>2.5</td>
<td>++</td>
<td>4 days</td>
<td>Short-term death</td>
</tr>
<tr>
<td>5</td>
<td>0.1</td>
<td>4.2</td>
<td>2.5</td>
<td>++</td>
<td>30 hr</td>
<td>Long-term death</td>
</tr>
<tr>
<td>6</td>
<td>0.7</td>
<td>5.8</td>
<td>2.5</td>
<td>0</td>
<td>0</td>
<td>Death</td>
</tr>
<tr>
<td>7</td>
<td>0.8</td>
<td>5.3</td>
<td>2.5</td>
<td>+</td>
<td>8 hr</td>
<td>Death</td>
</tr>
<tr>
<td>8</td>
<td>0.5</td>
<td>6.1</td>
<td>5.0</td>
<td>+</td>
<td>6 hr</td>
<td>Death</td>
</tr>
</tbody>
</table>

+++ = suprasystolic augmentation; + = effective, but not suprasystolic; 0 = no effect.

\*Two chamber.

at 5.0 to 10.0 \( \mu \text{g/kg/min} \). Thermodilution cardiac output was not measured but mixed venous \( \text{PO}_2 \) values before IABP were in the upper 20s and low 30s and rose to the low and middle 40s during the first 24 hr after IABP. He was taken back to surgery 10 days later for prosthetic (17 mm Shiley-Ionescu) replacement of his mitral valve. He did well until approximately 6 hr after surgery when his serum potassium level began rising rapidly. He suffered cardiac arrest 8 hr after surgery in spite of efforts to control the hyperkalemia. Postmortem examination revealed a small total mitral and tricuspid anulus size with the prosthetic mitral valve obstructing the tricuspid side of the canal. There was no evidence of aortic injury that may have resulted from IABP.

Patient No. 5. A. W. was the youngest patient in our series. He was in congestive failure at birth as a result of mitral regurgitation secondary to a congenitally deformed mitral valve with virtual absence of the posterior leaflet. There was an associated atrial septal defect. At 6 weeks of age and at a weight of 4.2 kg his mitral valve was replaced with a 19 mm Bjork-Shiley valve and his atrial septal defect was closed by direct suturing. After surgery he required vigorous pharmacologic support (16 \( \mu \text{g/kg/min} \) dopamine, 0.10 \( \mu \text{g/kg/min} \) isoproterenol, 0.6 \( \mu \text{g/kg/min} \) phenolamine, and 2 \( \mu \text{g/kg/min} \) nitroprusside) to maintain arterial pressures and a scanty urine output. Thermodilution cardiac index was only 1.3 and mixed venous \( \text{PO}_2 \) was only 19 mm Hg. IABP was started with a 2.5 ml balloon. It effected an immediate improvement in his blood pressure with suprasystolic diastolic augmentation (figure 2). The satisfactory diastolic augmentation was obtained even at rates that went as high as 176 beats/min. After 4 hr of IABP, his thermodilution cardiac index had increased to 2.0 and his mixed venous \( \text{PO}_2 \) had risen to 28 mm Hg. After 12 hr his cardiac index rose to 3.2 and his mixed venous \( \text{PO}_2 \) to 34 mm Hg. During the night his pressures continued to improve (figure 3). After 30 hr his condition was stable and the IAB was removed without event. He left the hospital on his thirteenth postoperative day and, although he continues to take digoxin and furosemide, he is still alive 8 months after IABP.

Patient No. 6. A. W. was an 8-month-old white girl who weighed 5.8 kg and underwent a Mustard procedure for d-transposition of the great arteries and repair of a muscular ventricular septal defect. Three hours after surgery, she experienced a sudden drop in cardiac output and was bleeding excessively through her chest tubes. While an exploratory thoracotomy was being done in the operating room a 2.5 ml IABP was introduced via the left common femoral artery. Balloon position was checked by palpation of the thoracic aorta. When IABP was started, no alteration of the right radial pulse was observed. The 2.5 ml balloon was then replaced with a 5.0 ml IAB catheter, but again no effect of IABP could be detected. In spite of administration of 0.2 \( \mu \text{g/kg/min} \) iv epinephrine and 0.3 \( \mu \text{g/kg/min} \) iv isoproterenol, the condition of the patient continued to deteriorate and she died in the operating room 30 min later. An autopsy was not performed. We are unable to explain the failure of IABP in this patient.

Patient No. 7. C. C. was a 9-month-old, 5.3 kg white girl who underwent repair of an atrioventricular defect. After surgery her pressures continued to drop in spite of the administration of intravenous dopamine given at a rate of 15 \( \mu \text{g/kg/min} \) IABP was instituted with a 2.5 ml balloon catheter inserted in the usual manner. Excellent diastolic augmentation of arterial pressure was obtained at a rate of 150 beats/min. The patient did show some clinical improvement initially, but her improved status could not be maintained. She died 10 hr after surgery, 8 hr after IABP had been initiated. Thermodilution cardiac output values were not obtained and mixed venous \( \text{PO}_2 \) values were in the high 30s and low 40s even before IABP was instituted and were essentially unchanged by IABP. Autopsy revealed a small mitral valve orifice and a mildly hypoplastic left ventricle. No thrombi or aortic injury was found.

Patient No. 8. D. H. was a 6-month-old, 6.1 kg white boy who underwent a Senning procedure for d-transposition of the great arteries and repair of a ventricular septal defect and pulmonary artery band. The patient had had a previous coarctation repair. Considerable difficulty was experienced in weaning the patient from cardiopulmonary bypass, and he was placed on IABP 1 hr after return to the intensive care unit. In spite of effective diastolic augmentation with a 5.0 ml IAB at a rate of 150 beats/min and the administration of intravenous dopamine (12 \( \mu \text{g/kg/min} \) and isoproterenol (0.15 \( \mu \text{g/kg/min} \)), the patient failed to improve and died 6 hr after IABP was instituted. Autopsy revealed a hypoplastic right ventricle with marked subaortic obstruction that appeared mild on an angiogram. There was no evidence of aortic injury from IABP.

Results

Only two patients (Nos. 3 and 5) in this group of eight who had IABP were long-term survivors (6+ months). It is noteworthy, however, that these two
patients were the youngest (6 weeks and 6 months) and also the smallest of the group (4.2 and 5.2 kg). Two additional patients (Nos. 1 and 4) were short-term survivors (5 and 10 days) after IABP. In our opinion survival in these four patients would not have been possible without IABP.

Autopsy in patient 1 showed residual pathologic findings that readily explained his ventricular fibrillation and also made long-term survival unlikely.

The postmortem examination of patient 4 revealed obstruction of the tricuspid orifice from a prosthetic mitral valve, the result of a small total atroventricular canal. His death followed reoperation 10 days after the successful use of IABP.

Of the four patients who died in the immediate postoperative period (patients 2, 6, 7, and 8) three had satisfactory alteration of the pulse contour with IABP. In one of these three, suprasystolic augmentation of diastolic pressure was accomplished. With IABP all three experienced an initial clinical improvement with improved peripheral perfusion, increased urine output, and improved blood gas determinations.

The only patient in whom IABP was used to wean from cardiopulmonary bypass is included in this group of four early postoperative deaths. Patient 2 was successfully removed from cardiopulmonary bypass and taken to the intensive care unit where satisfactory diastolic augmentation was noted. The patient had begun to produce urine and seemed well on her way to recovery when she developed sudden cessation of all cardiac activity. The parents denied permission for autopsy but we believe that her sudden demise was related to malfunction of her prosthetic mitral valve.

Postmortem examinations were performed on patients 7 and 8. The autopsy findings in patient 7 suggested that survival would have been unlikely because of a small mitral anulus and a mildly hypoplastic left ventricular chamber. Severe subaortic stenosis and a hypoplastic right ventricular chamber were present in patient 8 and made survival impossible.

In a single patient (No. 6), IABP with a 2.5 and 5.0 ml balloon did not result in any recognizable alteration of the pulse contour or deter a rapidly deteriorating course. The failure of IABP in this case remains unexplained.

The alterations of the pulse contours with IABP in these eight patients are summarized in table 3. In patient 6 no effect was noted with IABP and in patient 2 recordings were not made, but effective augmentation was observed on the monitoring scope. Satisfactory alteration of the arterial pulse contour by IABP was accomplished in seven patients with presystolic dips of between 4 and 14 mm Hg and decreases in peak systolic pressures of from 1 to 6 mm Hg. Diastolic augment-

![FIGURE 3. Improved arterial pulse with increased diastolic augmentation continuing through the night in a 6-week-old, 4.2 kg patient (No. 5). Each frame shows arterial pulse augmented with IABP followed by nonaugmented pulse without IABP support. With IABP, note the suprasystolic diastolic augmentation, the lowered end-diastolic pressure (presystolic dip), and the lowered systolic pressure after the augmented beat.](image)

<table>
<thead>
<tr>
<th>TABLE 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Effect of IABP on blood pressure</strong></td>
</tr>
<tr>
<td>Patient No.</td>
</tr>
<tr>
<td>---</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2*</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td>5</td>
</tr>
<tr>
<td>6</td>
</tr>
<tr>
<td>7</td>
</tr>
<tr>
<td>8</td>
</tr>
</tbody>
</table>

*In patient 2, effective IABP was observed but not recorded. NR = not recorded.
tation of pressure exceeded peak systolic pressure in four of the patients (table 3).

Balloon catheters were removed when the patients responded and no longer needed the circulatory assistance (patients 3, 4, and 5), when they were of no value (patient 6), or when death seemed imminent or had occurred (patients 1, 2, 7, and 8). Removal was never necessary because of ischemia of the lower extremity. In the four patients who underwent autopsy, there was no evidence of aortic injury, thrombi, or emboli. Heparinization was used only in patients 1 and 4.

Discussion

The circulatory assistance from IABP is achieved by displacing intra-arterial blood volume. This displacement occurs with pneumatic inflation of the balloon immediately after closure of the aortic valve. The balloon is totally deflated just before the onset of ventricular systole. The displacement of the blood volume results in a drop in aortic end-diastolic pressure, which in turn decreases left ventricular outflow impedance. Ideally, peak arterial pressure is shifted into diastole, resulting in increased tissue perfusion that includes the coronary circulation. The increased supply of oxygen to the myocardium and the reduced demand for left ventricular work results in an increase in the myocardial oxygen supply/demand ratio. Right ventricular function has also been shown to improve with IABP in adults suffering from cardiogenic shock.

The concept of IABP was first tested in a mock circulation model in 1961 by Dr. S. D. Moulopoulos, a cardiologist from Athens who was working in the laboratory of Dr. W. J. Kolff at the Cleveland Clinic. The first clinical application of circulatory assistance was in 1968 by Kantrowitz et al. Although the procedure was slow to gain acceptance, during the middle and late 1970s it became established as a standard mode of treatment for cardiogenic shock and perioperative low cardiac output syndrome in adults. In spite of its current universal acceptance for use in adults, IABP has been used little in children. There is only one report in the literature describing the use of IABP in children. This report suggested that it could be of value in children over 5 years of age but could not be used in those less than 5 years old, primarily because the commercially available balloons and catheters were too large. In addition, it was felt that aortic elasticity in the young child prohibited effective diastolic augmentation. The larger balloons were considered to be responsible for renal failure and in two of the 14 patients severe lower extremity ischemia required removal of the balloon catheter.

In 1977 our group began work to develop equipment that would provide the electronics and pneumatics necessary for the higher rates and smaller displacement volumes that would be necessary in IABP in infants and children. This work was completed in the laboratories of the Division of Artificial Organs, University of Utah, which is headed by Dr. W. J. Kolff, who in 1961 headed the laboratory in Cleveland where IABP was initially conceived and tested. The in vitro and in vivo testing of small balloons (0.75 to 5.0 ml) in small cats and dogs convinced us that IABP could be used effectively in infants and children. Our initial experience, although limited, strongly supports this contention.

Effective augmentation of diastolic pressure was accomplished in seven of the eight patients and suprasystolic diastolic augmentation was accomplished in four. In only one patient was IABP observed to be ineffective.

In addition, there was a noteworthy lack of complications. While the involved lower extremity often felt cooler than its counterpart, there was never enough vascular compromise to require removal of the IAB. The common femoral artery was used in all cases. The No. 5F catheter did not significantly compromise arterial flow even in the youngest and smallest patients (4.2 and 5.2 kg). We could not ascribe any compromise of renal function to IABP. On the contrary, urine output characteristically dramatically increased after the institution of IABP. In the four patients who underwent autopsy, we could detect no evidence of aortic wall injury. Only two of the patients underwent heparinization during IABP. There was no evidence of thrombus formation or embolization either clinically or at autopsy in any of the patients. None of the eight had any local or systemic infection.

Contrary to previous clinical experience, we were pleased to find that balloon inflation and deflation could match heart rates faster than 130 beats/min. In patient 4 IABP was effective at rates as high as 176 beats/min. It was our general impression, however, that IABP was more effective pumping every other beat at rates exceeding 150 beats/min. Continuous monitoring of the patients’ pulse contours and repeated adjustment of inflation and deflation timing were necessary to maintain optimal alteration of the arterial pulse contour with IABP.

At the time IABP was instituted all patients were receiving inotropic support, which was continued until the patient became stable and could be weaned from pharmacologic support. The use of inotropic drugs with α-adrenergic agonist effects may decrease the compliance of the systemic arterial bed of the younger
patient and may enable more effective IABP. It is our belief that IABP should be considered an adjunct and not a replacement for pharmacologic support of the patient.

Catheter selection was done quite empirically. Generally, we attempted to use the largest balloon catheter that we felt could be accommodated by the patient. Aortic diameter was estimated and the balloon catheter was placed in its transparent cover on the surface of the patient to evaluate whether the balloon would extend beyond the twelfth thoracic vertebral to avoid compromise of the celiac and renal vessels. For the smaller patients, this posed little problem since there was only a choice of 2.5 ml (8 mm diameter and 10.5 cm length) and a 5 ml (10 mm diameter and 13 cm length) balloon catheters. We attempted to select a balloon size that would be 40% to 60% of the estimated stroke volume.

While it is unlikely that IABP will ever be used as frequently in children as it is in adults, the availability of appropriately sized equipment should enable IABP to become an accepted and valuable mode of circulatory support in pediatric patients. Our limited clinical experience has not proved IABP to be a panacea for postoperative low cardiac output syndrome, but it has demonstrated quite conclusively that it can be successfully used, even in infants.

If the patient does not have a lethal lesion and the possibility of the presence of a significant residual anatomic defect has been excluded, IABP should be considered for use in infants and children whenever pharmacologically refractive low cardiac output syndrome is present. Specific indications for use of IABP could include cardiac index dropping below 2.0, mixed venous Po2 dropping below 20 mm Hg, and urine output dropping below 1 ml/kg/hr. It is important to realize that IABP can only augment cardiac output and should not be delayed until augmentation would have minimal or no effect.

Additional experience will be needed to define specific indications, proper time for intervention, and factors that can predict successful or unsuccessful outcome. We hope that our initial experience with IABP will encourage other groups to use this mode of circulatory assist in children.

References
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