The Blalock-Taussig Anastomosis in Infants Younger Than 1 Week of Age

L. HENRY EDMUNDS, JR., M.D., LARRY W. STEPHENSON, M.D., AND JAMES P. GADZIK, M.D.

SUMMARY Thirty-one newborns 7 days of age or younger had a Blalock-Taussig subclavian–pulmonary arterial anastomosis for hypoxia and acidosis secondary to cyanotic congenital heart disease. Seven infants (23%) died within 30 days of operation, four of shunt thrombosis and three of sepsis. Six newborns died 1–44 months after operation, two of respiratory insufficiency and sepsis, two at reoperation and two of unknown causes. Eighteen (58%) were alive an average of 38 months after operation.

The lowest preoperative arterial Pao, increased from a mean of 21 torr to 34 torr after the shunt was opened. Postoperatively 13 infants had one or more arterial Pao, of 25 torr or less, but only nine were also acidotic. The shunt was patent in three of these infants and thrombosed in six (19%). The mean diameter of thrombosed shunts (2.3 mm) was significantly less than that of patent shunts (2.9 mm). Seventy-eight percent of successful shunts remained open 4 years or longer and none closed abruptly after hospital discharge.

In newborns, early mortality of the Blalock-Taussig anastomosis is nearly four times that for infants 1 week to 2 years of age. However, the use of prostaglandin E, to dilate the ductus arteriosus should reduce early mortality caused by shunt thrombosis or stenosis. The presence of a partially patent ductus in newborns makes clinical determination of shunt patency difficult. Although the results of the Blalock-Taussig anastomosis in cyanotic newborns are unsatisfactory, other procedures have not produced better results. Long-term advantages of the Blalock-Taussig anastomosis make it useful when the subclavian artery is 2.5 mm in diameter or greater.

MANY NEWBORNS with cyanotic congenital heart disease are dependent on the ductus arteriosus for all or almost all of their pulmonary blood flow. Most of these infants have lesions that cannot be corrected in the neonatal period,1–7 and therefore require a systemic–pulmonary arterial shunt for survival. In older infants and children, the Blalock-Taussig subclavian–pulmonary arterial anastomosis is preferred and has many advantages over alternative shunts.8–11 However, in newborns, the question of which shunt is best is not settled. Newborns younger than 1 week of age have unique hemodynamic and anatomic considerations that make shunt construction more difficult than in infants only a few weeks older. Early mortality of systemic–pulmonary arterial shunts in newborns is more than twice that of older infants.4

In this report we analyze the early and late results of the Blalock-Taussig anastomosis in 31 newborns ages 7 days or younger.

Methods

Between August 1971 and August 1979 31 consecutive newborns 7 days of age or younger had a Blalock-Taussig anastomosis and no other procedure for severely cyanotic congenital heart disease. Twenty-four patients (77%) were admitted within 1 day of birth, and 25 had operation within 3 days of birth. All infants had severe cyanosis and hypoxia, and all infants had cardiac catheterization before opera-
tion. Only six infants were treated after prostaglandin E₁ (PGE₁) was available.

Six of the 31 patients had associated anomalies. Two were premature (2.0 and 2.1 kg) and had additional anomalies (skeletal deformities, hypocalcemia, dysmorphism, severe cleft lip and palate). Three others had renal and genitourinary anomalies that were associated with hypoglycemia in two and chromosomal and skeletal anomalies in one. The sixth infant had an abdominal parasitic Siamese twin and omphalocoele, duplication of the colon, and severe bilateral hydronephrosis.

The patients weighed 2-4.4 kg (mean ± SD 3.1 ± 0.5 kg). The lowest measured arterial Pao₂ (without PGE₁ and with variable inspired oxygen concentration) preoperatively was 15-30 torr (21.2 ± 3.6 torr). Only one infant had cardiac arrest or received catecholamines preoperatively. Five infants received infusions of PGE₁. Twenty-four required sodium bicarbonate preoperatively to correct acidosis. Of the seven patients who did not receive sodium bicarbonate, four received infusions of PGE₁. Twenty-three infants required emergency operation immediately after cardiac catheterization.

Operation was carried out under ketamine, nitrous oxide or halothane anesthesia, usually with a muscle relaxant. Arterial (umbilical or radial) and central venous catheters were inserted before operation. The lateral chest opposite the side of the aortic arch was entered in the fourth interspace. The azygous or hemiazygous vein was ligated and divided. The subclavian, proximal carotid, innominate and ipsilateral pulmonary arteries were dissected out, and the subclavian artery was brought down through the loop of the recurrent laryngeal nerve. The subclavian artery was transected as proximal as possible, usually at the level of the first branch. The anastomosis was made using magnification with a running posterior row of 7–0 or 8–0 suture. The anterior row was made with interrupted sutures in 22 infants and continuous sutures in nine.

All infants have been followed through September 1979.

Results

Eighteen patients (58%) remain alive; seven died within 30 days of operation and six died 1–44 months after operation. Anatomic diagnoses and outcomes are presented in table 1. Nine of 13 patients who died had autopsies.

The anastomosis was made on the left side in seven newborns. One of these infants first had a right thoracotomy followed immediately by left thoracotomy and a successful anastomosis when a rightsided ductus and right aortic arch were encountered. The diameter of the anastomosis ranged from 2–4 mm (2.8 ± 0.5 mm). Operative time ranged from 90–240 minutes (average ± SD 139 ± 30 minutes). Nineteen newborns received sodium bicarbonate during operation, five received PGE₁ and two received cate-

<p>| TABLE 1. Anatomic Diagnosis and Survival Results |
|---------------------------------|------------------|----------------|-----------------|-------|-------|</p>
<table>
<thead>
<tr>
<th>Pulmonary atresia</th>
<th>Total</th>
<th>No or minor complications</th>
<th>Survival after major complications*</th>
<th>Early deaths</th>
<th>Late deaths</th>
<th>Survivors</th>
</tr>
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<tbody>
<tr>
<td>Ventricular septal defect</td>
<td>7</td>
<td>2</td>
<td>3†</td>
<td>2†</td>
<td>5</td>
<td></td>
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<tr>
<td>Transposition of great arteries</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
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</tr>
<tr>
<td>IVS and hypoplastic right ventricle</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Single ventricle</td>
<td>3</td>
<td>2</td>
<td>1†</td>
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<td>2</td>
<td></td>
</tr>
<tr>
<td>Asplenia syndrome</td>
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<td></td>
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<td>2</td>
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</tr>
<tr>
<td>Polysplenia syndrome</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td>1†</td>
<td></td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>2</td>
<td>1†</td>
<td>1</td>
<td></td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Transposition of great arteries</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Hypoplastic right ventricle</td>
<td>2</td>
<td>2</td>
<td></td>
<td></td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Atrioventricular canal</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Ebstein's anomaly</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Total</td>
<td>31</td>
<td>16</td>
<td>6</td>
<td>7</td>
<td>6</td>
<td>18</td>
</tr>
</tbody>
</table>

*Including two patients with patent shunts that were suspected of being closed.
†Associated congenital anomalies.
Abbreviation: IVS = intact ventricular septum.
Cholamines. The subclavian artery was transected at the level of the first branch in 13 of 16 patients for whom data are available. A thrill was present after opening the shunt in all except the two premature infants. The lowest arterial PaO₂ measured in the operating room after opening the shunt (FIO₂ = 1.0) ranged from 15–50 torr (33.7 ± 8.4 torr) in 26 infants who did not receive PGE₁, and 33–59 torr (45 ± 11.3 torr) in five infants who did receive PGE₁.

Sixteen infants (52%) had no complications or only minor complications, such as atelectasis, temporary feeding problems or minor wound separation. Seven of these newborns were extubated the day of operation, eight the next day and one 3 days postoperatively. Ten received sodium bicarbonate postoperatively, but only one received catecholamines. PGE₁ was decreased rapidly postoperatively in the three who received it. A continuous shunt murmur was heard by a cardiologist or two independent observers 1–5 days after operation. Infants were discharged from the intensive care unit 2–4 days after operation and from the hospital 7–19 days (mean ± SD 11.3 ± 4.6 days) postoperatively. Only one infant was recatheterized during the same hospitalization to clarify the anatomic diagnosis (fig. 1).

Complications or death occurred in 15 patients (47%). Nine infants had shunt related problems, seven had respiratory problems (two premature infants had both), and one had heart failure. Seven infants died within 30 days of operation (23%) and two died after prolonged respiratory support.

Early after operation the shunt was closed or suspected of being closed in nine newborns. In three with acidosis and postoperative arterial PaO₂ levels as low as 15, 20 and 24 torr, the shunt was proved open by operation in one and by repeat cardiac catheterization in two. No additional procedures were carried out in the two full-term infants who survived. A stenotic shunt was revised in the premature infant, and 9 days later a second Blalock-Taussig anastomosis was performed on the left. None of the 22 patients without shunt-related problems developed acidosis after operation, but four had at least one arterial Po₂ as low as 25 torr postoperatively.

The shunt closed in six infants; five were recognized within 1 days of operation and the sixth, in another premature infant, was not recognized until the thirteenth day after surgery. The lowest arterial PaO₂ ranged from 12–22 torr (table 2), and all required NaHCO₃ for acidosis. Reoperation was carried out in five of the six infants with occluded shunts. The shunt was revised in one and found patent in another who later died and had an occluded shunt at autopsy. Three infants had second shunts, one a second Blalock-Taussig anastomosis; one a Potts, and the one a Waterston. Four of the six patients with an occluded shunt died. Occlusion of the shunt was confirmed at autopsy in three infants and by cardiac catheterization before a Waterston anastomosis in another. An occluded right Blalock-Taussig anastomosis was proved at cardiac catheterization 6 years later in the infant who had a second Blalock-Taussig anastomosis, and was not proved in the premature infant who had a Potts anastomosis.

Seven newborns developed major respiratory complications. None had phrenic nerve paralysis. Three infants with severe associated congenital anomalies and patent shunts died 13, 20 and 23 days after operation of overwhelming sepsis and pneumonia. Two infants were never extubated, had tracheostomies, required prolonged respiratory support and died of sepsis 90 and 100 days after operation. One of these had severe associated congenital anomalies. One infant recovered from atelectasis and pneumonia. A premature infant, who had a presumed occluded Blalock-Taussig shunt and a Potts anastomosis, developed repeated episodes of aspiration pneumonia but recovered after a long hospitalization.

One newborn, with single ventricle and pulmonary and tricuspid atresia, developed heart failure that was successfully controlled by digoxin, diuretics and 7 days of respiratory support.

In summary, 16 of the 31 infants had successful shunts and no or only minor complications. Closure of the shunt was suspected in three more infants; two recovered uneventfully after the shunt was proved open. The shunt occluded in six infants and was the immediate cause of death in four. Three other infants, all with severe associated anomalies, died of sepsis within 30 days of operation. Two survived shunt occlusion. Twenty-four survived 30 days or longer, but two died late from associated anomalies, respiratory problems and sepsis.

Follow-Up

Four of the 22 hospital survivors died later, two unexpectedly and two after second operations. One in-

**Figure 1.** A right Blalock-Taussig anastomosis made at age 1 day. The angiogram was performed 1 day after operation.
TABLE 2. Shunt Patency

<table>
<thead>
<tr>
<th>Shunt patent</th>
<th>Diameter of anastomosis (mm) n</th>
<th>Lowest postoperative paO₂ (torr) n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shunt patent</td>
<td>25 (81%) 2.9 ± 0.6</td>
<td>29 ± 5*</td>
</tr>
<tr>
<td>No shunt complications</td>
<td>22</td>
<td>2.9 ± 0.6</td>
</tr>
<tr>
<td>Suspected closed</td>
<td>3</td>
<td>2.5 ± 0.5</td>
</tr>
<tr>
<td>Shunt closed</td>
<td>6 (19%) 2.3 ± 0.4</td>
<td>16 ± 4</td>
</tr>
<tr>
<td>Early death</td>
<td>4</td>
<td>2.3 ± 0.3</td>
</tr>
<tr>
<td>Successful second shunt</td>
<td>2</td>
<td>2.5</td>
</tr>
</tbody>
</table>

*p < 0.001: shunt patent (n = 25) vs shunt closed (n = 6).
†p < 0.05: shunt patent (n = 25) vs shunt closed (n = 6).
‡p < 0.001: no shunt complications (n = 22) vs shunt closed (n = 6).
§NS: Suspected closed (n = 3) vs shunt closed (n = 6).

A patient who had pulmonary atresia, intact ventricular septum and hypoplastic right ventricle died at home at age 7 weeks, 3 weeks after a successful pulmonary valvotomy. Both the shunt and pulmonary valve were open at autopsy. A second infant with the same diagnosis died after patch expansion of the right ventricular outflow tract and pulmonary annulus. The shunt was patent at autopsy. A third child with severe pulmonary stenosis, complete atrophicventricular canal and an underdeveloped right ventricle died of progressive right-heart failure at age 44 months after ligation of a patent shunt and open cardiac repair with a Rastelli conduit. The fourth infant with asplenia and single ventricle died unexpectedly before her first birthday and did not have an autopsy.

Eighteen patients (58%) are alive 1 month to 8 years after operation (38 ± 34 months). Three have had successful definitive corrective operations at age 5–6 years. Two others, ages 6 years, are eligible for corrective operations. Five infants less than 30 months of age are awaiting definitive repair of their anatomic deformities when they are older. A decision regarding future open or closed cardiac surgery has been deferred in four infants younger than 1 year of age; three have tricuspid atresia and the fourth, Epstein’s anomaly with pulmonary insufficiency. No decision regarding future surgery has been made in two other infants with single ventricle. Two children have uncorrectable lesions; one, age 32 months with pulmonary atresia, single ventricle and other cardiac anomalies, has had multiple cerebrovascular accidents. The other, with pulmonary atresia, intact ventricular septum and hypoplastic right ventricle, developed mild pulmonary hypertension that precluded a Glenn anastomosis or Fontan procedure. A polytetrafluoroethylene (PTFE) graft was placed between the aorta and right pulmonary artery at age 53 months to control polycythemia and hypoxia.

The fate of the anastomosis is known in 20 of the 21 patients who survived their initial hospitalization with a patent Blalock-Taussig shunt and in the five infants who died of sepsis. One premature infant who survived had a successful Potts anastomosis after a presumed occluded Blalock-Taussig shunt. Four infants died of shunt thrombosis. The fate of the shunt is unknown in one infant who died late.

Of the five infants who died of sepsis, the shunt was proved open at autopsy in three, by catheterization in one, and by clinical examination in the fifth, who died at 100 days of age. The shunt was proved open by autopsy in three infants who died after hospital discharge. Three patients required second shunts at ages 16, 26 and 53 months for stenosis after hospital discharge. Three patients required second shunts at ages 16, 26 and 53 months for stenosis of the original anastomosis (fig. 2A). A patent shunt was ligated at 44–72 months (mean 61 months) in four patients who had corrective operations (fig. 2B). Twelve anastomoses remain open 1–79 months postoperatively (28 ± 33 months). Four shunts have lasted more than 6 years. Figure 3 presents the actuarial curve for shunt patency of hospital survivors.

Discussion

The cyanotic newborn who requires a systemic–pulmonary arterial anastomosis to replace the closing ductus presents unique therapeutic problems not encountered in older infants and children. Most of these infants have complex cardiac lesions that are not amenable to intracardiac repair. Some infants have associated noncardiac anomalies that are also life-threatening. Cyanotic newborns may rapidly develop severe hypoxia and acidosis and often require rapid diagnosis and treatment. Aside from the closing ductus, other neonatal hemodynamic changes, such as closure of the foramen ovale, elevation of systemic vascular resistance and reduction of pulmonary vascular resistance, may adversely and unpredictably affect the circulation, particularly when associated with complex cardiac anomalies. Elevation of pulmonary vascular resistance due to persistent fetal pulmonary arterial architecture in the newborn reduces both pressure difference and shunt flow between the systemic and pulmonary arterial circulations. Low flow through tiny structures encourages thrombosis; however, large shunts may permit too much pulmonary blood flow after pulmonary vascular resistance falls. Also, tiny, sometimes friable structures are involved in construction of the shunt and technical imperfections are poorly tolerated. These reasons make construction of satisfactory systemic–pulmonary arterial shunts more difficult in newborns than in infants only 7–10 days older.

The introduction of prostaglandin E, greatly improves the outlook for cyanotic newborns who depend on the ductus arteriosus for pulmonary blood flow. While the ductus normally constricts within a few hours after birth, anatomic closure does not occur for approximately 1 week. PGE, effectively dilates the ductus arteriosus during the first week after birth, but may fail in infants 10 days or older. With PGE, severe hypoxia and acidosis can be controlled.
preoperatively during evaluation and cardiac catheterization and intraoperatively during construction of a systemic–pulmonary arterial shunt. Postoperatively, PGE₁ can be used to augment pulmonary blood flow if flow through the shunt is temporarily inadequate because of elevated vascular resistance. If the shunt fails, PGE₁ may provide adequate pulmonary blood flow during reoperation if the ductus is still responsive. In the present series PGE₁ was used during reoperation in two premature infants. Had the drug been available, PGE₁ might have prevented four deaths due to shunt thrombosis.

The presence of a partially open ductus makes the diagnosis of shunt thrombosis difficult in the newborn. Because pulmonary vascular resistance is elevated, shunt murmurs, regardless of the type of shunt, cannot be definitely distinguished from ductal murmurs in the first few hours and days after operation.₁ ² The combination of hypoxia and acidosis raises the question of shunt adequacy; however, if ductal closure is delayed, these clear clinical signs may not develop for days or even weeks. All of our patients with inadequate or thrombosed shunts developed hypoxia (Pao₂ < 25 torr) and acidosis, but two other patients with adequate shunts also developed these signs and four others had at least one postoperative Pao₂ as low as 25 torr. These experiences underscore our inability to determine shunt adequacy in newborns who may have

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**Figure 2.** (A) A stenosed right Blalock-Taussig anastomosis made at 4 days of age, 53 months after construction. A second systemic-pulmonary arterial shunt was required. (B) An adequate left neonatal Blalock-Taussig anastomosis 72 months after operation. This shunt was ligated at the time of a successful corrective operation.

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**Figure 3.** Actuarial curve for shunt patency in 22 newborns who survived 30 days with a patent Blalock-Taussig shunt. One infant who died within her first year and did not have an autopsy is excluded. Seventy-eight percent of the shunts were still patent and adequate at 4 years.
an open or partially open ductus. When shunt thrombosis occurs, it nearly always develops within 24 hours of operation. Cardiac catheterization is the only means to prove shunt adequacy. In practice, if an infant is doing well and if a shunt murmur is eventually heard, there is no need for postoperative cardiac catheterization. However, if there is any question of shunt adequacy, we recommend an infusion of PGE₁ and immediate cardiac catheterization.

The best shunt for newborns with inadequate pulmonary arterial blood flow is not settled. The Glenn superior vena caval–right pulmonary arterial anastomosis is contraindicated because of elevated neonatal pulmonary vascular resistance and poor results. The Potts side-to-side descending thoracic aorta–left pulmonary arterial anastomosis has most of the disadvantages of the Waterston shunt, and it is also difficult to take down and may compromise the ductus during construction. The choice of shunt is between the Waterston ascending aorta–lateral pulmonary arterial anastomosis, PTFE prosthetic shunts and the Blalock-Taussig anastomosis.

The Waterston shunt has been successfully used in cyanotic newborns and is associated with low (0–10%) early mortality in some series, but a high mortality in others. This anastomosis is quicker and easier to construct than a Blalock-Taussig shunt, but the size of the opening is critical, with narrow limits of tolerance. Reoperation for thrombosis or heart failure is not uncommon. Late mortality is high in some series and the risk of subsequent pulmonary vascular disease is substantial. Furthermore, the Waterston anastomosis often results in selective flow to the ipsilateral lung and nonuniform growth of the pulmonary vasculature. A patch angioplasty of the pulmonary artery is generally required when the shunt is taken down. Growth of the anastomosis is unpredictable. Despite these disadvantages, before the advent of PGE₁, the Waterston anastomosis offered the lowest operative mortality of any systemic–pulmonary arterial shunt in the newborn.

Recently, expanded microporous PTFE prostheses have been successfully used to construct systemic-pulmonary arterial anastomoses in newborns. The reported early mortality in newborns ranges from 17–25%. These prostheses offer considerable flexibility in that both the systemic and pulmonary anastomoses can be made at convenient sites in the circulatory system. These shunts are easily taken down and offer protection against development of pulmonary vascular disease. The reported early shunt thrombosis rate is low, but the long-term patency rate is unknown. Shunt resistance is fixed, so pulmonary blood flow may be too high in young infants and too low in older infants who normally triple their birth weight in 12 months. The optimal length and diameter of the prosthesis for newborns are undetermined. Early results are promising, but more information regarding early and late patency, optimal size and length of the prosthesis, distortion of growing great vessels by the fixed length of the prosthesis and resistance to infection are needed.

The Blalock-Taussig anastomosis is generally favored when systemic–pulmonary arterial anastomoses are required in older infants and children. In our experience, early mortality of Blalock-Taussig anastomoses in infants 1 week to 2 years of age is 6% (three deaths in 52 patients). However, until microsurgical techniques were developed, results in newborns were extremely poor. The authors have performed a series of 14 infants younger than 2 weeks of age, with three early deaths (21%) and four late deaths (29%). In the present series early mortality (23%) and late mortality (19%) are similar. Analysis of our early deaths indicates that four (13%) were due to shunt thrombosis, which occurred in 19% of patients (including both premature infants). Shunt thrombosis was in part related to the diameter of the subclavian artery; the mean anatomic diameter of thrombosed shunts was significantly smaller than the diameter of patent shunts (table 2). Early shunt thrombosis remains a serious complication of neonatal Blalock-Taussig anastomoses.

A patent Blalock-Taussig anastomosis offers many long-term advantages for the cyanotic newborn. Congestive heart failure and late development of pulmonary vascular disease are uncommon after Blalock-Taussig shunts. Long-term patency is satisfactory, with 78% of neonatal Blalock-Taussig shunts in this series patent at 4 years. This patency rate is similar to that reported for older infants and children. Abrupt late closure of Blalock-Taussig shunts apparently does not occur. Blalock-Taussig shunts offer some prospect of growth, provide flow to both pulmonary arteries and are easily ligated at the time of definitive operation.

The results in this series, which covers an 8-year period and the work of four surgeons, are unsatisfactory, yet the reported experience of alternative shunts in newborns is equally poor or worse. The Waterston anastomosis has the lowest thrombosis rate, but the highest incidence of heart failure and late complications. Early results of PTFE shunts are encouraging, but early thrombosis is a problem and late results are not available. Early thrombosis of Blalock-Taussig shunts is a major problem; however, the advent of PGE₁ reduces the threat of early shunt thrombosis and permits time to perform an alternative shunt. The known long-term advantages of the Blalock-Taussig anastomosis favor this operation over alternatives when a satisfactorily large subclavian artery is available. Therefore, we recommend this shunt as the first choice in full-term, cyanotic newborns with subclavian arteries larger than 2.5 mm.

Acknowledgment
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