Fate of the Stented Arterial Duct

To the Editor:

In a recent issue of *Circulation*, Gibbs et al stated that duct stenting cannot be recommended in neonates and infants with duct-dependent congenital anomalies. We have concerns regarding the data and the conclusions of that study. In fact, we have different experiences and opinions about the effectiveness of ductal stenting, especially in neonates with hypoplastic left heart syndrome (HLHS). First, the data presented by the authors are limited to 8 patients with HLHS and 11 patients with duct-dependent pulmonary blood flow. Second, the technical details of stent implantation, as described by the authors in 1992 and 1993, have dramatically changed over time. The use of more flexible stents allows stent delivery without long sheath guidance. This avoids obstruction of blood flow through the arterial duct, which might lead to acute hemodynamic deterioration. Smaller catheters facilitate stent advancement. Perforation of the arterial duct, as is occasionally seen with Palmaz stents, occurs less because the flexibility of the new stents allows a smooth adaption to the vessel wall. Third, the goal of duct stenting must be precisely determined before the procedure. Usually, there is no reason for “long-term” duct stenting in HLHS (ie, for up to 30 months). Therefore, we agree with the comment by Ruiz and Bailey that we should be cautious about drawing firm conclusions before analyzing these results.

Concerning duct-dependent pulmonary blood flow, Schneider et al recently reported on 21 infants in whom ductal stenting was considered an effective alternative to surgical aortopulmonary shunts.

In our experience with a total of 40 stent implantations for maintaining duct patency, 29 stents were implanted in 25 patients with HLHS. Indications for stent placement were ductal stenosis despite prostaglandin E1 administration (n=14) or reaching outpatient status for those awaiting heart transplantation or a Norwood staged procedure. Because we observed stent dislocation and procedure-related arrhythmia followed by severe hypertension when we used Palmaz stents, we switched to the more flexible Jo-stents (Jomed), which we have used exclusively since 1997. There were no deaths from the catheterization procedure.

Ductal patency was achieved for up to 6 months, and 14 patients underwent successful transplantation. Six patients did not recover from postnatal cardiac shock and died awaiting heart transplantation for reasons not related to ductal stenting. Four patients had reconstructive surgery, 2 according to the classic Norwood procedure. In 2 patients, reconstruction of the aorta was combined with a bidirectional cavopulmonary shunt in one procedure at the age of 4 and 6 months, respectively. This new approach of “stage I and II of Norwood procedure combined in one” was based on bilateral pulmonary artery banding after ductal stenting. One patient is still palliated by the latter interventional/surgical approach.

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Response

We agree with Dr Michel-Behnke and colleagues that stenting the duct in hypoplastic left heart syndrome is relatively straightforward and maintains ductal patency; this was clearly stated in our article. None of the deaths in patients with hypoplastic left heart syndrome were related to stenting but, as in most countries, a shortage of infant donor organs was a major factor. Duct stenting surely cannot be recommended for these patients when long-term follow-up shows the results simply cannot compete with modern results of the Norwood operation. Michel-Behnke et al fail to tell us how the results of ductal stenting with a view to transplantation compare with the Norwood operation in Germany.

Duct-dependent pulmonary circulation is very different. It is not true that the technical aspects of stenting have changed “dramatically.” We never needed to use a long sheath inside the duct; we only used them to protect the stent’s passage through the heart in cases in which a venous approach was used. There has been little change in the size of balloon catheters (we used coronary angioplasty catheters), although new, more flexible stents offer a logical advantage. Neither has guidewire technology changed significantly: small (0.018-inch) wires with steerable, superflexible or hydrophilic wires were used in all our procedures. We were unable to recommend stenting in this group of patients because of aggressive neointimal hyperplasia and a clearly demonstrated risk of fatal ductal spasm, which can occur after even a single, gentle attempt at the passage of a guide wire across a tortuous duct. Again, the technique just does not compare with the results of aortopulmonary shunt surgery, except perhaps, in the rare subgroup of cases with bilateral pulmonary artery disconnection. Michel-Behnke and colleagues quote Schneider et al’s work in their support of continued use of ductal stenting, but Schneider et al also had a high incidence of intimal proliferation, with some ducts becoming completely occluded by neointima, even when the duct was fairly straight.

Future improvements in biomedical engineering might reduce neointimal hyperplasia, but the potential for fatal ductal spasm remains in patients with pulmonary atresia with a tortuous duct in whom some wire manipulation within the duct is inevitable. Ductal stenting is an attractive proposition in theory; in practice, aortopulmonary shunt surgery has lower risk and much greater freedom from early and medium-term reintervention.

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