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ORIGINAL ARTICLE

Modified Blalock Taussig shunt: a not-so-simple palliative procedure

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Abstract

OBJECTIVES: Thirty-two consecutive isolated modified Blalock Taussig (BT) shunts performed in infancy since 2004 were reviewed and analysed to identify the risk factors for shunt intervention and mortality.

METHODS: Sternotomy was the only approach used. Median age and weight were 10.5 (range 1–74) days and 2.9 (1.9–4.4) kg, respectively. Shunt palliation was performed for biventricular hearts (Tetralogy of Fallot/double outlet right ventricle/transposition of great arteries_ventricular septal defect_pulmonary stenosis/pulmonary atresia_ventricular septal defect/others) in 21, and univentricular hearts in 11, patients. Hypoplastic left heart syndrome patients were excluded. Two procedures required cardiopulmonary bypass. Median shunt size was 3.5 (3–4) mm and median shunt size/kg body weight was 1.2 (0.9–1.7) mm/kg. Reduction in shunt size was necessary in 5 of 32 (16%) patients.

RESULTS: Three of 32 (9%) patients died after 3 (1–15) days due to cardiorespiratory decompensation. Lower body weight ($P = 0.04$) and bigger shunt size/kg of body weight ($P = 0.004$) were significant risk factors for mortality. Acute shunt thrombosis was observed in 3 of 32 (9%), none leading to death. Need for cardiac decongestive therapy was associated with univentricular hearts ($P < 0.001$), bigger shunt size ($P = 0.054$) and longer hospital stay ($P = 0.005$). Twenty-eight patients have undergone a successful shunt takedown at a median age of 5.5 (0.5–11.9) months, without late mortality.

CONCLUSIONS: Palliation with a modified BT shunt continues to be indicated despite increased thrust on primary corrective surgery. Though seemingly simple, it is associated with significant morbidity and mortality. Effective over-shunting and acute shunt thrombosis are the lingering problems of shunt therapy.

Keywords: Modified Blalock Taussig shunt • Palliation • Mortality • Cyanotic heart disease

INTRODUCTION

While the classic Blalock Taussig shunt was a breakthrough in treating cyanotic heart diseases [1], it involves sacrificing antegrade flow to the subclavian artery with its attendant risks [2, 3]. In 1975, de Leval modified the technique, using a polytetrafluoroethylene interposition graft popularly known as the modified Blalock Taussig (BT) shunt (MBTS) [4]. While MBTS has become an established palliative procedure with progressive improvements in the outcome [5], growing experience has led to increasing thrust on primary corrective procedures. Palliative strategy has obvious disadvantages, such as the need for two operations, potentially two scars, possibility of distortion of the branch pulmonary artery, volume loading of the ventricles, lower diastolic pressures, etc. In spite of these, primary shunt palliation continues to be indicated in neonates with physiological pulmonary hypertension, which makes a bidirectional Glenn shunt untenable. Many centres also consider a primary neonatal correction of Tetralogy of Fallot to be riddled with risks and hence, still prefer to palliate neonates in blue spells.

While acknowledging its role even in the modern era, mortality of the MBTS procedure is relatively high, tending to be around 10% [6]. This report is based on 32 consecutive 'first time' MBTS palliations performed at our institution since 2004, with a view to analysing the risk factors for mortality, shunt thrombosis and need for decongestive therapy.

PATIENTS AND METHODS

Patients

Thirty-two MBTS procedures performed in neonates and infants at our institution since 2004 were analysed. MBTS performed in

patients with HLHS or as a part of other complex procedures, such as unifocalization, were excluded. Twenty-six patients were neonates and 6 were infants. Twenty-five patients were male. The demographic data are detailed in Table 1. Diagnosis was established using trans-thoracic echocardiography.

End points

Primary end points were mortality and shunt thrombosis. Secondary end points such as need for excessive inotropic support and cardiac decongestive therapy were also analysed. A brief analysis comparing Era 1 (2004–07) and 2 (2008–11) was performed to see if the results had changed over time and to identify the factors that may have accounted for that change.

Surgical technique

All MBTS procedures were performed through a sternotomy. Two procedures required cardiopulmonary bypass. After full sternotomy, the right lobe of the thymus was excised; the course of the brachiocephalic trunk up to its bifurcation was dissected. The right pulmonary artery up to the hilum was dissected. A bolus of 100 IU/kg crystalline heparin was administered. The brachiocephalic trunk was clamped with a Cooley clamp, and the distal right subclavian artery, temporarily with a ligaclip. A longitudinal arteriotomy was performed at the 'premarked' undersurface of the truncus to subclavian artery continuity. An obliquely fashioned end of the thin-wall Gore-Tex stretch vascular graft (W. L. Gore & Associates, Inc., AZ, USA) was sutured end-to-side to the arteriotomy. Clamps were released and good shunt flow through the anastomosis ascertained. The Cooley clamp was placed again to exclude the proximal anastomosis from the circulation. The shunt length was trimmed so as to avoid it being too long. The shunt lumen was flushed to remove microthrombi. The right pulmonary artery was excluded from circulation using two vascular clamps. The transversely fashioned distal end of the graft was anastomosed to the right pulmonary artery. The target arterial saturation was 75–85% (Q_P:$Q_s \approx 1$).

Patent duct: to ligate or not?

Our preference was to ligate the persistent ductus arteriosus (PDA) in all patients having forward flow through their main pulmonary artery. In shunt-dependent circulations, the patent duct was often circumvented and almost obliterated using a silastic sling and ligaclips. The aim of this manoeuvre was to allow a quick rescue by re-establishing ductal flow in case of a shunt thrombosis emergency.

Selection of shunt size

As a rule of thumb, a 3-mm graft was used for children around 3 kg or lower in body weight, whereas a 3.5-mm graft was used for children around 3.5 kg. The indication, whether palliating for a univentricular or a biventricular heart, influenced the size selection in borderline weight-class children. Fine regulation of flow and pressure was influenced by displacing the proximal inflow anastomosis either to the proximal subclavian artery or to the brachiocephalic trunk, as well as by slightly titrating the length of the shunt. The details of shunt size and positioning are summarized in Table 2.

Postoperative left open sternum

The primary goal was to close the chest at the shunt procedure. However, if there were any fears about the fate of the shunt (especially in totally shunt-dependent pulmonary circulation) or about the shunt getting squeezed behind the aorta, etc. the sternum was left open. It was believed that an open sternum lends itself to a quick response in case of an emergency when compared with a closed chest.

Anticoagulation

Anticoagulation regimen was decided on a case-by-case basis. Therapeutic heparinization was performed in high-risk shunt scenarios, such as shunt-dependent pulmonary perfusion, in

Table 1: Preoperative clinical characteristics

Variable	Median (range)
N	32
Age (days)	10.5 (1–74)
Weight at operation (kg)	2.9 (1.9–4.4)
Size (cm)	48 (39–52)
Body surface area (m^2)	0.2 (0.14–0.24)
SpO_2 (%)	85 (50–95)
Diagnosis	*n* (%)
TOF/DORV/TGA_VSD_PS, pulmonary atresia_VSD, Ebstein's anomaly	21 (66)
Single ventricle inclusive of pulmonary atresia_intact septum	11 (34)

TOF: Tetralogy of Fallot; DORV: double outlet right ventricle; TGA: transposition of great arteries; VSD: ventricular septal defect; PS: pulmonary stenosis.

Table 2: Shunt procedure details

Variable	*N* (%) except otherwise stated
No. of shunts (mm)	
3	8 (25)
3.5	19 (59)
4	5 (16)
Inflow	
Subclavian artery	12 (38)
Truncus brachiocephalicus	20 (63)
Outflow	
Right pulmonary artery	28 (88)
Left pulmonary artery	4 (13)
Heart lung machine	2 (6)
Absolute shunt size (mm)	3.5 (3–4)
Median shunt size/body weight ratio (mm/kg)	1.21 (0.9–1.7)

cases with shunt clipping (shunt size reduction) or technical problems encountered during shunt construction. Heparin infusion starting with 5–10 IU/kg/h, followed by therapeutic dose as early as 2 h postoperatively, was planned if surgical bleeding was not an issue. In effect, however, the therapeutic anticoagulation was often achieved later than 2 h. Shunts without complications and considered normal risk received aspirin in the long term.

Three patients did not have long-term anticoagulation/platelet inhibitor medication: 2 who died early and 1 who survived with early shunt thrombosis in a neonatal Ebstein's anomaly with antegrade pulmonary flow.

All patients were evaluated postoperatively with trans-thoracic echocardiography.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics 19 (SPSS, Inc., Chicago, IL, USA). Categorical variables are presented as numbers with percents and are compared using Fisher's exact test. Continuous variables are presented as median with range and are compared between groups using the Mann–Whitney test. *P*-values <0.05 were considered significant.

RESULTS

Mortality

Three of the 32 (9%) patients died at a median of 3(1–15) days. The details of patients who died are as presented in Table 3. Lower body weight ($P = 0.04$) and bigger shunt size/kg of body weight ($P = 0.004$) were significant risk factors for mortality (Table 4).

Shunt thrombosis

Acute shunt thrombosis was observed in 3 of the 32 (9%) patients, none of whom died. These 3 patients had Ebstein's anomaly with antegrade pulmonary flow (2.2 kg 3 mm right MBTS), double outlet right ventricle-hypoplastic left ventricle (LV)_pulmonary stenosis (2.9 kg 3 mm right MBTS) and unbalanced atrio-ventricular septal defect hypoplastic LV d-transposition of great arteries_pulmonary stenosis (3 kg 3.5 mm left MBTS). All shunt thrombosis occurred within the first 24 h and were diagnosed by echocardiography. Two patients had shunt revision with evacuation of the thrombus. One patient with stable saturation due to normal antegrade pulmonary flow was not subjected to shunt revision. Shunt thrombosis could not be statistically related to shunt size ($P = 0.1$) or shunt size/kg body weight ($P = 0.92$). Early anticoagulation regimen ($P = 0.33$), competitive blood flow ($P = 1.0$), diagnosis ($P = 0.27$), need for cardiopulmonary bypass ($P = 0.18$), increased inotropic support ($P = 0.71$) and shunt size reduction ($P = 1.0$) were not associated with acute shunt thrombosis.

Inotropic support

Need for adrenalin ≤0.05 and/or noradrenaline ≤0.05 and/or milrinone ≤0.75 μg/kg/min was defined as normal inotropic support. Accordingly, 6 of the 32 (19%) patients needed normal inotropic support and 23 of the 32 (72%) needed higher inotropic support. Three patients were without any ionotropic support.

Decongestive therapy

Need for cardiac decongestive therapy (over and above that of diuretics) was necessary in 10 of 29 survivors to discharge. Decongestive therapy was required in 2 of 19 (20%) biventricular hearts compared with 8 of 10 (80%) univentricular hearts ($P < 0.001$), obviously resulting in a significantly longer hospital stay ($P = 0.005$). Bigger shunts were associated with the need for decongestive therapy ($P = 0.054$).

Open thorax

The sternum was left open postoperatively in 11 (34%) patients.

Other complications

Postoperative complications included chylothorax, phrenic nerve palsy, necrotizing enterocolitis and abdominal bleeding with unclear focus in 1 patient each.

Corrective surgery

Of the 29 survivors, 28 (97%) have undergone corrective surgery with takedown of the BT shunt at the time of this study. Ten patients were subjected to a bidirectional Glenn anastomosis, while 18 underwent a biventricular repair. None of the shunt survivors died during or after the corrective surgery.

Fate of branch pulmonary artery

Seven of 28 patients (25%) had reconstruction of the branch pulmonary artery at the distal shunt insertion site. Residual stenosis after BT shunt takedown occurred in 6 patients. In 3 of the patients, the stenosis occurred despite the pulmonary artery being reconstructed, while in 3, the stenosis occurred without the artery being reconstructed.

Era

A brief comparative analysis between 12 shunts in Era I and 20 shunts in Era II is depicted in Table 5.

DISCUSSION

An ideal shunt helps promote uniform growth of the pulmonary arteries, without causing distortion. An excessive shunt results in significant diastolic run-off in the short term and elevated pulmonary vascular resistance or impaired ventricular and atrioventricular valve performance in the long term. Although various types of shunts have been described [1, 7–11], it is the MBTS that

Table 3: Mortality details

Diagnosis	Age (days)	Weight (kg)	Shunt size (mm)	Cause of death	Died (Postop day)
DORV_TGA_PS	19	2.5	3.5	Cardiorespiratory decompensation	15
Pulmonary atresia VSD, hepatopulmonary syndrome, catheter perforation and emergency shunt	4	2.2	3.5	Cardiorespiratory decompensation	1
Pulmonary atresia intact ventricular septum	6	2.4	4	Myocardial ischaemia	3 (ECMO)

DORV: double outlet right ventricle; TGA: transposition of great arteries; PS:pulmonary stenosis; ECMO: extra corporeal membrane oxygenation.

Table 4: Risk factors for mortality

Variable	Alive *n* (%)	Dead *n* (%)	*P*-value
N	29	3	
Weight (kg)	2.94 (1.9–4.4)	2.37 (2.2–2.49)	0.04
Diagnosis			
Biventricular hearts	19/29 (66)	2/3 (67)	1.00
Univentricular hearts	10/29 (34)	1/3 (33)	
Competitive pulmonary flow	21/29 (72)	1/3 (33)	0.22
Use of heart lung machine	2/29 (7)	0/3 (0)	1.00
Size of shunt (mm):			
3	8/29 (28)	0/3 (0)	0.47
3.5	17/29 (59)	2/3 (67)	
4	4/29 (14)	1/3 (33)	
Shunt size/kg body weight	1.19 (0.88–1.58)	1.59 (1.41–1.69)	0.004
Anticoagulation regimen:			
1 LDH	7/28[a] (25)	0/2[c] (0)	0.38
2 ETH	4/28[a] (14)	1/2[c] (50)	
3 LTH	17/28[a] (61)	1/2[c] (50)	
Long-term anticoagulation	7/28[a] (25)	0/1[b] (0)	1.00
Postoperative high ionotropes	20/29 (69)	3/3 (100)	0.52
Shunt size reduction	3/29 (10)	2/3 (67)	0.056
Shunt thrombosis	3/29 (10)	0/3 (0)	1.00
SaO_2 postoperative (day of operation)	83 (77–94)	85 (80–90)	0.97
Hospital stay	23 (5–95)	3 (1–15)	0.02

LDH: low dose (10 IU/kg/h) heparin; ETH: early therapeutic heparin; LTH: late therapeutic heparin.
[a]One patient data missing.
[b]One patient on immediate ECMO was not analysed for acute anticoagulation regimen.
[c]Two patients who died early were not analysed for long-term anticoagulation.

has become established as the procedure of choice [4, 12]. MBTS continues to be a subject of academic interest, because of persistent risks associated with this simple-looking procedure.

Sternotomy or thoracotomy

MBTS was classically performed through a thoracotomy. However, recent trends have shown increasing preference for a sternotomy approach [6]. A sternotomy saves the child from a second scar, avoids morbid damage to the thorax with prospects of late scoliosis, but more importantly, the target pulmonary artery being intrapericardial, it is more accessible for eventual reconstruction after takedown. Avoiding a thoracotomy in the prospective Fontan patients has an added advantage of reducing build-up of lung adhesions to the thoracic wall and the consequent development of systemic-to-pulmonary artery collaterals. Other disadvantages of a thoracotomy approach enumerated in the literature include Horner's syndrome, distortion of lobar branch pulmonary arteries and preferential flow to one lung with unbalanced growth [6]. Depending on the side of the thoracotomy, it may not be always possible to perform PDA ligation, but with a sternotomy, it is always possible. In the end, whether or not to close the duct remains a strategic decision [13].

The sternotomy approach does confront the surgeon with the challenges of a central run-off from the systemic artery leading to greater steal, low diastolic pressures, coronary malperfusion and pulmonary hyperperfusion. In addition, the often-used truncus brachiocephalicus to the right pulmonary artery shunt may be at danger of being squashed between the dominant aorta and the superior vena cava, for which the parietal pericardial reflection over the superior vena cava to the trachea should be divided to create space for the shunt.

Table 5: Comparison between eras

Variable	Era I (2004–07) *n* (%)	Era II (2008–11) *n* (%)	*P*-value
N	12 (38)	20 (62)	
Diagnosis			
Biventricular hearts	6/12 (50)	15/20 (75)	0.25
Univentricular hearts	6/12 (50)	5/20 (25)	
Weight (kg)	2.89 (2.2–4.4)	2.93 (1.9–3.8)	0.99
Size of shunt (mm)	3.5 (3.5–4)	3.5 (3–3.5)	0.002
Shunt size/weight	1.24 (0.91–1.69)	1.16 (0.88–1.58)	0.13
Presence of competitive blood flow	7/12 (58)	15/20 (75)	0.44
Use of HLM	0/12 (0)	3/20 (10)	0.52
Shunt distribution, mm (in %)			
3	0/12 (0)	8/20 (40)	0.001
3.5	7/12 (58)	12/20 (60)	
4	5/12 (42)	0/20 (0)	
Right vs left	11/20 (92) right 1/12 (8) left	17/20 (85) right 3/20 (15) left	1.00
Site of take-off (truncus brachiocephalicus vs subclavian artery)	10/12 (83) vs 2/12 (17)	10/20 (50) vs 10/20 (50)	0.08
Early anticoagulation strategy			
Low dose	3/10 (30)	4/20 (20)	0.71
Early therapeutic	1/10 (10)	4/20 (20)	
Late therapeutic (as defined in Table 4)	6/10 (60)	12/20 (60)	
Long-term anticoagulation			
Aspirin	8/10 (80)	14/19 (74)	1.00
Therapeutic	2/10 (20)	5/19 (26)	
Postoperative ionotropic support			
None	1/12 (8)	2/20 (10)	0.48
Normal	1/12 (8)	5/20 (25)	
High[a]	10/12 (83)	13/20 (65)	
Need for shunt reduction	3/12 (25)	2/20 (10)	0.34
Decongestive therapy (more than diuretics)	6/9 (67)	4/20 (20)	0.03
Duration of ventilation	2 (1–15)	1.5 (0–9)	0.53
ICU stay	4 (1–15)	5 (1–13)	0.39
Duration of hospital stay	18.5 (1–95)	22.5 (5–84)	0.69
Mortality	3/12 (25)	0/20 (0)	0.04
Shunt thrombosis	0/12 (0)	3/20 (15)	0.27
SaO_2 before takedown	82 (72–93)	81.5 (73–94)	0.66
Residual branch PA stenosis at shunt insertion site	4/9 (44)	1/15 (7)	0.05

[a]Normal ionotropic support is defined as adrenalin ≤0.05 and/or noradrenaline ≤0.05 and/or milrinone ≤0.75 μg/kg/min.

While the proposed alternative shunts such as Potts or Waterston/Cooley shunts were difficult to regulate, the MBTS flow is restricted by the size of the graft as also by the size of the inflow vessel.

While the Boston group [6] reported four times higher risk of shunt failures through a thoracotomy when compared with a sternotomy, Shauq *et al*. [13] have reported significantly longer ventilation time, inotropic support, intensive care unit (ICU) stay and hospital stay in the sternotomy group. These findings reflect the learning curve involved with shunts created through a sternotomy.

Competitive flow and PDA strategy

A completely left open duct may be difficult to regulate in the presence of a MBTS. With our technique of duct obliteration using a silastic sling, one retains the possibility of quickly restoring duct patency in the case of a shunt thrombosis. While a patent duct imparts significant safety in the case of a shunt failure, some reports have associated patent duct with shunt thrombosis [14]. Petrucci *et al*. (Society of Thoracic Surgeons [STS] database) have shown no association between closed duct and the risk of composite morbidity [15]. Closing or keeping the duct open during the MBTS procedure has advantages and disadvantages and, hence remains in the end, an individual decision.

Mortality

In spite of overall improvement in results [5], mortality reported ranges from 2.3 to 16% [15]. Our overall postoperative mortality was 9.4%. Low body weight ($P = 0.041$) and bigger shunt size/kg body weight ($P = 0.011$) were factors associated with postoperative mortality. There was a trend towards significance between the need for postoperative shunt size reduction and mortality ($P = 0.056$). These findings point towards over-shunting as a possible indicator of mortality in our series. The Boston group has reported a mortality of 9 of 102 (8.7%) patients, with indications that excessive pulmonary blood flow could have contributed to mortality in the sternotomy group. Multivariate risk factors for mortality in their analysis included small graft size, left MBTS and male sex [6]. The same group also suggested the use

of smaller (3.5 mm) shunts through a sternotomy approach instead of the 4-mm shunts for the thoracotomy approach. An STS database harvest study [15] has identified preoperative ventilation, pulmonary atresia_intact ventricular septum, univentricular hearts and weight <3 kg as risk factors for mortality. Pulmonary atresia with intact ventricular septum, when specifically analysed, did not come out as a significant risk factor for mortality in our cohort, probably because of small numbers. While Alkhulaifi *et al.* [16] identified weight <2 kg and preoperative ventilation, Rao *et al.* [17] identified restrictive atrial septal defect, univentricular physiology and postoperative intervention as risk factors for mortality.

Shunt thrombosis

Shunt thrombosis is a grave complication of the MBTS procedure. Our overall acute shunt thrombosis of (3 of 32) 9.4% corresponds with those of (9 of 76) 11.8% reported from Bristol and (14 of 102) 13.7% reported from Boston [6]. We could not show an association between smaller shunt size and occurrence of thrombosis, probably because of the small numbers. Tsai *et al.* [18] and Tamisier *et al.* [12] have suggested that young age and smaller size are significantly related to shunt thrombosis. Other reports have also linked weight <2 kg [16] and weight <3.6 kg [19] to shunt thrombosis. Gedicke *et al.* [14] have found weight <3 kg, high preoperative haemoglobin (>18 g/dl) and a postoperative patent duct as significant factors for shunt thrombosis.

Anticoagulation-coagulopathy

Although an association between an anticoagulation regimen and shunt thrombosis could not be established in our study, it does not belittle the role of postoperative anticoagulation, particularly in high-risk patients. Al Jubair *et al.* [20] have shown, less-shunt failure occurs if heparin is given before clamping. An early postoperative phase with a fresh anastomosis, coupled with phases of low systemic pressures, pulmonary hypertension, external compression and resulting stasis, can initiate thrombus formation. It is these uncertainties that can be positively influenced by early anticoagulation. Li *et al.* [21] have demonstrated a beneficial effect of acetylsalicylic acid in infants palliated with a shunt, with reduced incidence of shunt thrombosis and death. Another prospective study has shown the beneficial effect of haemodilution with a significantly higher shunt patency rate [22]. Rare coagulopathies, such as protein C deficiency [23], and primary antiphospholipid syndrome [24] have also been reported to cause shunt thrombosis.

Late shunt obstruction

We have not observed any late shunt thrombosis in this series of patients. This has been reported as a cause in up to 15% of out-of-hospital mortalities [14, 15]. Wells *et al.* [25] have observed >50% obstruction of the MBTS in 21% of their patients and have identified a shunt size of <4 mm to be a risk factor for high-grade stenosis (>50%).

Era

While the cohort did not change over time in terms of most demographic and procedural variables, shunt size was significantly lower in the latter half of the series when compared with the former half. Shunt thrombosis was higher in the later era, but did not reach statistical significance. These findings may indicate that small shunts are prone to shunt thrombosis. Mortality as well as need for cardiac decongestive therapy was significantly higher in the previous era. With time, while mortality was avoided, shunt thrombosis remained worrisome. While shunt thrombosis morbidity could be partially attributed to the technique, the importance of optimal intensive postoperative management cannot be overemphasized. Interestingly, in spite of smaller shunt size selection in the later era, transcutaneous saturation before shunt takedown was 82% in both the eras. This implies that the shunt flow was adequately regulated by the artery from which the shunt was sourced.

LIMITATIONS

This is a retrospective study with a small patient cohort, which may not be powered enough to identify all risk factors contributing to the various end points. The series being spread over a time frame of 8 years, even generalized improvements in operative technique and perioperative care may alone account for the improvements in outcome.

CONCLUSION

In spite of increasing confidence with primary neonatal intracardiac repairs, the MBTS continues to be indicated for malformations of the univentricular pathway. Although seemingly innocuous, the MBTS procedure is associated with significant morbidity and mortality. While small shunts may have a tendency to shunt thrombosis, large shunts may lead to pulmonary over-circulation and volume loading of the heart. Various studies have identified low body weight, small shunt size, over-shunting, univentricular hearts—specifically pulmonary atresia with intact ventricular septum, to be risk factors associated with postoperative morbidity and mortality. It appears that timely and efficient early anticoagulation as well as long-term antiplatelet therapy may help reduce the risk of early and late shunt dysfunction.

Conflict of interest: none declared.

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