Stent implantation of the arterial duct in newborns with duct-dependent circulation

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Aims Little is known about the medium term results after stenting of the arterial duct in neonates and infants with duct-dependent cyanotic congenital heart disease. We report the results of stent implantation of the arterial duct in 21 neonates and infants. The defects for which the arterial duct was stented included pulmonary atresia with intact ventricular septum, critical pulmonary stenosis, and more complex defects with associated reduced pulmonary blood flow.

Methods and Results Palmaz stents were used and successfully implanted in all the 21 patients. There were no major complications during the stent implantation procedure although two hospital deaths occurred 2 and 14 days after stent implantation. Cardiac catheterization was repeated electively 3 to 6 months after stent implantation. Stent stenosis due to intimal proliferation was noted in 11/13 patients who underwent recatheterization. Stenosis of the inner stent lumen ranged from 25% to 100%, mean 74%. Re-dilatation of the stent was required in five patients who were awaiting corrective surgery. In babies with pulmonary atresia or critical pulmonary stenosis, who also underwent additional balloon dilatation of the pulmonary valve, spontaneous closure of the stented arterial duct was well tolerated and when it occurred, the right ventricular size had increased and the circulation was no longer duct-dependent. In patients who required subsequent surgical corrective treatment, stenting of the duct allowed the definite corrective operation to be performed as the first surgical procedure. During the follow-up period, ranging between 2 months and 2 years, mean 8.7 months increased growth of the pulmonary arteries was seen in all the patients. No distortion of the branch pulmonary arteries was seen.

Conclusion In patients with cyanotic congenital heart disease stenting of the arterial duct is an effective alternative to surgical aorto-pulmonary shunts.

Introduction

Although primary corrective surgery is attempted in complex congenital heart disease, with a trend towards definitive surgery within the first year, the surgical creation of an aortopulmonary shunt is still used as a palliative procedure in neonates with duct-dependent pulmonary circulation and/or hypoplastic pulmonary arteries. However shunt-related complications such as shunt occlusion, shunt stenosis, distortion of the pulmonary arteries, pulmonary hypertension and differential growth of the right and left pulmonary arteries, as well as surgical adhesions, increase the complexity and risks of the final definitive surgery[1,2].

Stent implantation into the arterial duct has been proposed as a non-surgical alternative to aorto-pulmonary shunt surgery[5,6]. It offers the potential advantages of eliminating the need for palliative surgery, thus reducing the number of operations required, and optimizing the time of definitive surgical correction[3–6]. Stent implantation can be performed in newborns with hypoplasia of the pulmonary arteries and has the potential advantage that definitive correction can be performed as the initial surgical therapy without a previous thoracotomy.

Patients and methods

Between January 1994 and November 1996, stent-implantation of the arterial duct was performed in 21 neonates and infants with duct-dependent pulmonary circulation as an alternative to surgical palliation on an
intention-to-treat basis. Only those patients who were considered suitable for palliative treatment were included. Patients who required major surgical procedures in the neonatal period such as Damus Kaye Stansel operation or Norwood-type operation, palliative surgical right ventricular outflow tract reconstruction, conduit implantation or palliative interventional right ventricular outflow tract reconstruction were excluded. The procedure of stenting the arterial duct was approved by the hospital ethical committee. Parental informed consent was obtained in all the patients, and prior discussion and agreement on the management plan with the cardiac surgeon was obtained.

The age of the patients ranged between 1 day and 65 days, median 13-3 days. The body weight ranged from 1.76 kg to 3.97 kg, median 3.06 kg. The McGoon ratio (diameter of the right pulmonary artery at the pre-branching point plus that of the left divided by the diameter of the aorta at the diaphragm), based on angiography, ranged from 0.8 to 1.9, median 1.27.

The patients were divided into two groups: group 1 included eight patients, of whom five had pulmonary valve atresia and intact ventricular septum and three critical pulmonary valve stenosis. In these patients the mean age at the time of the procedure was 2.9 days (range 2-5 days) and the mean weight 3.02 kg (range 2.6-3.6 kg). The initial oxygen saturation ranged from 61% to 90%, mean 69% and the McGoon ratio was between 0.8 and 1.9, mean 1.4. The initial interventional therapy consisted of radiofrequency perforation of pulmonary valve atresia and balloon dilation of critical pulmonary stenosis, and these were performed 1 to 2 days before stent implantation, in order to assess the possibility of the patient not being duct-dependent.

The three patients with critical pulmonary stenosis had a hypoplastic right ventricle. The only difference between these three patients and those five with pulmonary valve atresia was a tiny hole in membrane-like structure instead of the pulmonary valve, so that radiofrequency perforation was not necessary. After the structure instead of the pulmonary valve, so that radio-pulmonary atresia was a tiny hole in membrane-like

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Table 1 Diagnoses and number of patients in group 2

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of patients</th>
</tr>
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<tbody>
<tr>
<td>P.atr.+VSD</td>
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</tr>
<tr>
<td>P.atr.+DORV</td>
<td>2</td>
</tr>
<tr>
<td>P.atr.+AVSD</td>
<td>1</td>
</tr>
<tr>
<td>P.atr.+AVSD+PAPVD</td>
<td>1</td>
</tr>
<tr>
<td>P.atr.+IVS</td>
<td>1</td>
</tr>
<tr>
<td>TGA (presented late)</td>
<td>1</td>
</tr>
<tr>
<td>TGA +LVOTO +VSD</td>
<td>1</td>
</tr>
<tr>
<td>TOF</td>
<td>1</td>
</tr>
<tr>
<td>PS+TA</td>
<td>1</td>
</tr>
<tr>
<td>PS+DORV+AVSD+LAI</td>
<td>1</td>
</tr>
</tbody>
</table>

A VSD = atroventricular septal defect; DORV = double outlet right ventricle; IVS = intact ventricular septum; LAI = left atrial isomerism; LVOTO = left ventricular outflow tract obstruction; PAPVD = partial anomalous pulmonary venous drainage; P.atr. = pulmonary valve atresia; PS = pulmonary stenosis; TA = tricuspid valve atresia; TGA = transposition of the great arteries; TOF = tetralogy of Fallot.

Selection of patients for stenting of the arterial duct

Echocardiography was performed to confirm the diagnosis of the underlying heart defect and for the initial evaluation of the patency and the anatomy of the arterial duct. At the time of cardiac catheterization, angiography was performed in all the patients to demonstrate the morphology of the arterial duct more accurately[7]. The anatomy of the arterial duct was delineated either by antegrade balloon occlusion angiography of the descending aorta distal to the origin of the arterial duct using a Berman angiographic catheter (Arrow 4F, M ount Holly, New Jersey, U.S.A.) or by selective angiograms directly into the duct using 4F catheters such as Cobra (Cordis, Roden, Netherlands), right Judkins (Cordis) or modified Amplatz catheters (Cordis). In patients with a tortuous duct a sitting up position with cranio-caudal tilt or a ‘laid-back view’ (caudally angled frontal X-ray tube) was used with individual angulations to delineate the arterial duct without superimposition of the duct and the great vessels. With the use of angiography and digital callipers, the anatomy and measurement of the maximal lengths and the minimal diameters of the arterial duct were achieved.

In group 1, 7/8 had arterial ducts which originated from the descending aorta and coursed to the pulmonary artery bifurcation in a curved, but not tortuous shape (Fig. 4). In one patient the duct originated from the under side of the aortic arch proximally and...
had an elongated course. In group 2, the morphology of the duct was more heterogeneous. In four patients the arterial duct had a slight and gentle curve, in three an elongated course and in six a complex tortuous course (Fig. 2). In 8/13 patients, the ducts originated from the distal arch and proximal descending aorta, in 3/13 the duct originated from the middle part of the aortic arch, in two the ducts originated from the left subclavian artery or from the innominate artery.

The maximum diameter of the arterial duct in group 1 ranged from 3.4 mm to 5.6 mm, mean 4.7 mm and the minimum diameter from 1.2 mm to 2.6 mm, mean 1.7 mm. The length of the arterial duct was between 12.7 mm and 19 mm, mean 16 mm. The maximum diameter of the arterial duct in group 2 ranged from 1.1 mm to 5.8 mm, mean 3.3 mm and the minimum diameter from 0.5 mm to 2.6 mm, mean 1.3 mm. The length of the arterial duct was between 6.5 mm and 21 mm, mean 10.3 mm.

Technique of stent implantation

The procedure of stent implantation in the arterial duct was performed under deep sedation with diazepam and ketamine in all the patients. In group 1, 4/8 of the patients were intubated, whilst in group 2, 10/13 were intubated. Immediately prior to stent implantation, after crossing the arterial duct with a guide-wire the infusion of prostaglandin E₁ was stopped in order to promote crossing the arterial duct with a guide-wire the infusion intubated. Immediately prior to stent implantation, after patients were intubated, whilst in group 2, 10/13 were ketamine in all the patients. In group 1, 4/8 of the patients were intubated under deep sedation with diazepam and

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Immediately before stent implantation, balloon dilatation of the arterial duct was performed with a coronary angioplasty balloon catheter varying between 2.0–3.5 mm diameters (Olimpix, Cordis). Balloon dilatation was performed in order to obtain a visual assessment of the anatomy of the straightened arterial duct and to ensure that the duct could be crossed by the delivery sheath and the stent. The diameter of the balloon used for pre-dilation was smaller than the diameter of the balloon used for the stent implantation. The stents used included Palmaz–Schatz coronary stents (Johnson & Johnson Interventional Systems, Miami, U.S.A.) and one Palmaz P104 renal stent (Johnson & Johnson Interventional Systems). The Palmaz–Schatz coronary stent is 15 mm long (before expansion) and consists of two 7 mm parts joined by a single 1 mm articulation. This stent can be divided into two equal 7 mm lengths by cutting the articulation. After dilatation to 4.0 mm diameter, the length of the stent shortens to 14.3 mm. This was carried out in 14 of the stents implanted in 12 patients. Palmaz–Schatz stents were implanted by using a 4 or 5 French sheath. The stents can be inflated to a maximum diameter of 4 to 6 mm. The Palmaz renal stent is 10 mm long (before expansion) and can be dilated up to 12 mm (9 mm diameter recommended). After dilatation to 6.0 mm diameter the length of the Palmaz renal stent shortens to 9.4 mm. A commercially available delivery system was used for implantation of the Palmaz–Schatz coronary stent (Johnson & Johnson Interventional Systems Co.). The Palmaz renal stent was implanted by using a 6 F long sheath (USCI, BARD GmbH, Dublin, Ireland).

Stent implantation required various approaches due to the variable origins and morphology of the arterial duct and variations in the cardiac defects. In group 1, stenting of the duct was performed from the femoral vein across the right ventricular outflow tract as an additional procedure 1 to 2 days after the initial intervention. After entering the pulmonary artery with an end hole catheter, a 0.014 inch guide wire (Hi Torque Floppy, ACS) was passed through the arterial duct into the descending aorta and over this guide-wire the delivery system with the coronary Palmaz–Schatz stent was advanced into the duct. After correct positioning of the stent across the duct, the long sheath was withdrawn and the balloon inflated in order to deploy the stent. In these patients, an attempt was made to implant the stent only at the pulmonary artery end of the duct.

In group 2, 8/13 patients with no continuity between the right ventricle and the main pulmonary artery, stent implantation was performed either percutaneously from the femoral artery or after cut down of the auxiliary artery depending on the position of the origin and the shape of the arterial duct. When the arterial duct originated from the middle of the aortic arch or the proximal part of the descending aorta, stent implantation was performed from the right or left axillary artery. When the duct originated from the descending aorta, stent implantation was performed from the femoral artery as described above (Fig. 1). The intention was to stent the whole length of the duct in these patients. For implantation of one Palmaz renal stent, a 6 French long sheath (USCI) was pre-shaped into a slight curve to avoid damage to the arterial duct during positioning of the sheath within the duct. The
renal stent was then mounted on a balloon catheter (Opta, Cordis; 6 mm diameter, 20 mm length), which
was positioned within the arterial duct, the long sheath
was withdrawn and the stent implanted by inflating the
balloon. This stent was implanted in a 61-day-old infant
with a body weight of 3.97 kg.

Repeat angiography of the duct was performed
in all patients after stent implantation to confirm the
stent position and to exclude persistent stenosis due to
incomplete stenting of the duct. In case of residual
stenosis, an additional stent was implanted to cover the
whole duct. Protrusion of the stents into the aorta or the
pulmonary artery was assessed by final aortograms.
After stent implantation the heparin infusion was con-
tinued (infusion of 400 U.k g
-1day
-1
for the first 48 h,
followed by 200 U.k g
-1day
-1
for 48 h). During the
follow-up, the patients were on antiplatelet agents. In
group 2, patients in whom the entire length of the duct
was stented, 2–3 mg . kg
-1 day
-1
acetylsalicylic acid
and 2–3 mg . kg
-1 day
-1
dipyridamole were adminis-
tered orally[8]. In group 1 patients, in whom only the
pulmonary artery end of the duct was covered by the
stent, only dipyridamole was given.

Results

In 21 patients, 32 stents were successfully implanted
during 25 interventional procedures. Of these stents, 31
Palmaz–Schatz stents with a length of either 15 mm or
7 mm were used. Fourteen of these were divided and the
stents were implanted using only one 7 mm part of the
stent as described above. One of the patients received a
single 10 mm long Palmaz renal stent.

In group 1, 11 stents were implanted in eight
patients. The procedure time ranged from 45 to 89 min,
median 61 min and the fluoroscopy time ranged from 20
to 46 min, median 29 min. Immediately after stent
implantation, the arterial oxygen saturation increased by a
median of 21% (range: 0%–34%) from 61%–90% (mean
69%) before to 83%–95% (mean 90%) after stent implan-
tation. In one patient, prostaglandin E
1
infusion was
stopped late and so there was no increase in oxygen
saturation after stent implantation. Between 35% and
87% of the length of the duct was covered by the stent in
seven of eight patients, because only the pulmonary
artery end of the duct was covered. Four of the patients
were intubated for the procedure and the assisted venti-
lation was continued for a period of 10 h to 2 days after
the procedure.

In group 2, eight stents were implanted from the
femoral artery and 13 after cutdown of the right (n=6)
or left (n=7) axillary artery. Ten of the 13 patients were
intubated during the procedure and were subsequently
continuing on assisted ventilation for a period of 6 h
to 30 days, median 6–6 days after the procedure. The
procedure time ranged from 42 to 93 min, median
71 min and the fluoroscopy time ranged from 12 min to
39 min, median 28 min. Immediately after stent implan-
tation, the arterial oxygen saturation increased between
5% and 34%, median 21% from 38%–80% (mean 62%)
before to 69%–88% (mean 76%) after stent implantation
without prostaglandin E
1
administration.

In this group, an attempt was made to cover the
whole length of the arterial duct. Between 89% and
100%, median 97–9% of the length of the duct was
covered by the stents. In two patients, stenosis at the
origin of the left pulmonary artery and in three cases
stenosis at the origin of the right pulmonary artery were

Figure 2 Patient with a long tortuous duct with complex duct-dependent heart

disease before stent implantation.
also stented simultaneously. In two patients a long and tortuous duct was shortened by straightening after stent implantation (Figs 2 and 3). One of these patients had had two previous aorto-pulmonary shunt operations at another hospital, but remained duct-dependent. Angiography showed both Blalock Taussig-shunts to be completely occluded and the arterial duct was therefore stented using a 10 mm Palmaz renal stent.

**Mortality**

In both groups, whilst there were no deaths related to the procedure, in group 2, two patients died 2 and 14 days after the procedure, one due to septicaemia and one due to low cardiac output and intracerebral haemorrhage. An autopsy in the latter patient showed an unguarded tricuspid orifice in addition to pulmonary atresia, a Gerbode defect and an absent main pulmonary artery. Unfortunately the stented duct was not retrieved for histopathological examination.

All of the surviving patients were discharged home 2 to 30 days (median 9.5 days) after the procedure. In 14 patients, ventilation time after the intervention ranged from 6 h to 4 days. Two patients needed prolonged ventilation for more than 4 days. In one patient, the ventilation time was 30 days because of tracheomalacia, and after extubation the patient was transferred...
to the referring hospital. In one patient with dysmorphic features, convulsions, tracheal stenosis and septicemia needed supportive ventilation for 25 days.

**Early reintervention**

In two patients, early reintervention was required due to recurrent cyanosis between 2 and 5 days after stent implantation. In the first patient, angiography revealed significant stenosis of the arterial duct, not covered by the stent, immediately at its junction with the pulmonary artery. In addition, severe stenosis of the left pulmonary artery was present. A further 15 mm long stent was placed inside the previous two 7 mm stents in order to cover the whole duct and the left pulmonary branch stenoses. The saturation increased from 54% to 78% after implantation. In the second patient, restenosis of the duct, at the aortic end with and within the articulation of the stent, occurred after implantation of a Palmaz–Schatz stent. Because the arterial oxygen saturation decreased to 60% after 5 days, two additional Palmaz–Schatz stents were implanted, resulting in an increase of the arterial oxygen saturation to 87%.

**Complications**

During the interventions the only notable complication was a temporary third-degree atrioventricular block in one patient. This resolved during the procedure. There were no instances of hypotension during the procedure and hypercyanosis occurred only during balloon inflation in the arterial duct. Arterial damage and subsequent arterial occlusion occurred in two patients after stent implantation. In one patient, the right femoral artery was occluded after insertion of a 5 French sheath. This was treated with additional intravenous heparin infusion for 48 h. In another patient, occlusion of the left subclavian artery was detected angiographically 3 months after the stent, which had been implanted from cutdown of the left axillary artery. The child was asymptomatic and no additional treatment was given for the occluded subclavian artery.

**Follow-up**

In group 1, the mean follow up time ranged between 0.75 month to 24 months (median: 8.7 months). In 6/8 patients, repeat cardiac catheterization was performed 3 to 17 months (median: 7-9 months) after the stent implantation. In all six of these patients, further elective dilation of right ventricular outflow tract was performed using 10-12 mm diameter balloon catheters (Opta, Cordis).

The stent was completely occluded due to intimal proliferation in 4/6 patients 4-5, 6, 13 and 17 months after implantation. In a further patient, the lumen of the stent was almost totally (90% stenosis) occluded by the intimal tissue after 3 months, whilst in one patient a 60% stenosis of the stent lumen was observed after 4 months (Figs 4 and 5). The oxygen saturation in each of these five patients remained above 96%. In two of the most recent patients, no repeat cardiac catheterization was performed because of the short follow-up time of 0.75 and 2.5 months.

In group 2, 7/11 surviving patients had repeat cardiac catheterization, of whom six had seven reinterventions 3 months to 18 months (median 6 months) after stent implantation. Four patients did not have repeat catheterization, because the stent had been implanted less than 3 months previously in three, and in one patient, the oxygen saturation was above 80% at the latest follow up.

The indication for re-intervention after stent implantation was decreasing oxygen saturation. The oxygen saturation in these patients decreased from 8% to 19%, mean 11% and ranged between 64% and 76%, mean 70%. In five patients, intimal proliferation was the cause of the decreasing saturation and resulted in the reduction of the inner lumen diameter of the stents to between 32% and 75% of the stent diameter 3 to 9 months, median 4-6 months, after stent implantation. Re-dilation of the stent was performed in three patients.
with a 4 mm diameter coronary balloon catheter (Olimpix), which was over expanded to 4·4 mm. One patient had two re-interventions because of stent stenosis. The first intervention was 9 months after stent implantation when the stent was redilated with a 6 mm Osypka balloon catheter. Nine months after this, a further balloon dilation was performed because of intimal proliferation causing stent stenosis. In 1/5 patients, because of additional stenoses of the left pulmonary artery at the site of the arterial duct 3 months after stent implantation, a 7 mm Palmaz stent, implanted in the middle of a Palmaz–Schatz stent at the initial procedure, had migrated distally and restenosis of the left pulmonary artery was noted. A further stent was implanted in this patient to overcome this stenosis. The remaining patient had no intimal proliferation by 3 months but the stent-diameter of 3·5 mm had become relatively small and was redilated to 4·4 mm with a Olimpix coronary balloon catheter.

After reintervention, in all these patients the oxygen saturation increased to between 79% and 88%, mean 83%.

Outcome

In group 1, all eight patients were discharged 2 to 7 days (median 3·7 days) after stent implantation. At the latest cardiac catheterization 3 to 24 months after stent implantation, no distortion of the pulmonary arteries was seen and the mean McGoon ratio in this group had increased to 1·9–2 (Fig. 6). At the latest follow-up, all the patients were well, and none have required any

Figure 5 The same patient after 4 months; the size of the right ventricle has increased, the stented arterial duct is narrowed by intimal proliferation.

Figure 6 Increased growth of the pulmonary arteries (McGoon ratio) after stenting of the arterial duct (mean values and standard deviation).

surgery. No right-to-left shunting at the atrial level was present even in those patients in whom the stented duct was closed due to intimal proliferation. At the same time the size of the hypoplastic right ventricle had increased.

In group 2, no distortion of the pulmonary arteries could be found and the McGoon index after the latest cardiac catheterization ranged from 1.2 to 2.1, mean 1.7. In seven of the 13 patients, definitive surgical treatment has been performed (univentricular repair in three; biventricular repair in four) between 2 weeks and 21 months after stenting of the arterial duct. Three patients are waiting for univentricular repair and one patient is waiting for biventricular.

There were no technical problems caused by the stents during the subsequent surgery. In one patient the whole stent was surgically explanted, in two patients a part of the Palmaz-Schatz stent, which extended into the branch pulmonary arteries, was removed and the stented duct surgically closed. In the other four patients, the arterial duct was ligated leaving the stent in place. The patients were extubated 1 to 12 days (median 4.5 days) after operation and discharged after 10 to 32 days (median 13.5 days).

### Discussion

Babies with a duct-dependent pulmonary circulation are usually treated with a systemic-to-pulmonary-artery shunt followed at a later date by either a single-stage or staged corrective surgery. This may ultimately result in biventricular circulation or if the anatomy is unsuitable for this, then univentricular circulation. The disadvantages of the initial shunt operation include a small but important incidence of complete occlusion necessitating a further palliative operation or distortion and/or stenosis of the shunted pulmonary artery, in the medium term, loss of perfusion to the upper lobe of the lung, differential growth of the pulmonary arteries and finally a thoracotomy scar[1,2]. Most, if not all of these disadvantages can be avoided by a non-surgical method of securing an alternative blood supply to the pulmonary arteries. Such a method may involve the implantation of metal stents in the arterial duct[4,6]. This will then avoid the initial palliative operation and allow the patient to undergo a corrective type of operation as the first surgical intervention. In very small newborns with a body weight of between 1500 g and 2000 g, with small pulmonary arteries and a McGoon ratio of less than 1.5, a shunt operation may pose some difficulties. In these babies, stent implantation may be an attractive alternative to aortopulmonary shunt surgery.

In the group of patients with pulmonary atresia or critical pulmonary stenosis combined with an intact ventricular septum and a hypoplastic right ventricle, a staged surgical management approach is often required. In these patients, antegrade flow into the pulmonary circulation from the right ventricle can be established by non-surgical techniques, such as conventional balloon dilation of pulmonary valve stenosis or radiofrequency valvotomy of the atretic valve[5]. This then allows a second stenting procedure of the arterial duct a few days later to augment the pulmonary blood flow. The additional advantage of this approach is that stent implantation in the arterial duct can be performed antegrade via the femoral vein, avoiding arterial catheterization. In our experience, even after establishing antegrade pulmonary blood flow, it has been impossible to withdraw prostaglandin E1 infusion. Oxygen saturation dropped down to 57% to 64%, mean 59% between stopping and re-starting the prostaglandin E1 infusion.

However, generally, the prostaglandin E1 infusion was stopped again after the arterial duct was crossed with a guide-wire. A balloon was inflated to pre-dilate the duct in order to facilitate the subsequent passage of the sheath or the stent mounted on the balloon. Thus, we recommend that the infusion be continued until the guide-wire crosses the arterial duct. Stopping the infusion at this stage promotes constriction of the duct thus securing the stent within it. It is recognised that these patients may only require an alternative source of blood supply to the lungs until the right ventricle has adapted or increased in size sufficiently to become capable of supporting the pulmonary circulation alone. We therefore set out to only cover a portion of the arterial duct rather than the whole of the duct. In six of these patients, the lumen of the stented arterial duct had become narrowed by intimal proliferation or was totally occluded after a period of 3 to 6 months. By this time the right ventricle had become capable of being the sole contributor to the pulmonary circulation and the occlusion of the stent had no clinical impact (Figs 4 and 5). It is unclear whether partial stenting of the duct or reduced ductal flow[9] resulting from an increase in right ventricular forward flow, may contribute to the rapid intimal proliferation in these patients. In this group, we avoided starting the patients on acetylsalicylic acid as part of any anticoagulation treatment, in order to avoid the possibility of causing closure of the unstented part of the duct.

In the follow-up assessment of the patients with pulmonary stenosis or atresia, the pulmonary arteries showed an increase in size to near normal (Fig. 6). At repeat catheterization, even when further balloon dilation of the pulmonary valve was performed because of residual gradients, no recanalization or re-dilatation of the stents was needed as the capacity of the right ventricle had increased. Thus, when spontaneous closure of the stented arterial duct occurred, there was no right-to-left shunt through the inter-arterial communication on colour Doppler echocardiography. Oxygen saturation was maintained above 96%. Thus interventional therapy might replace surgical therapy, thereby avoiding long-term prostaglandin infusion in patients with critical pulmonary stenosis or pulmonary atresia and intact ventricular septum, excluding those patients with right ventricular-dependent coronary blood flow.

Group 2 consisted of patients with more complex anatomy. In these, the stents were implanted using an
arterial route due to the origin and anatomy of the duct. When the arterial duct originated from the proximal part of the aortic arch, or proximal part of the descending aorta, stents were implanted via the left or right axillary artery. Because of the small size of the arterial vessels of these babies, the development of stenosis or complete obstruction of the vessels used for access is a well-known complication (two patients in this group). To establish secure aortopulmonary communication in these patients, we decided to stent the entire length of the arterial duct. This would help to maintain the patency of the duct for a longer period so that definitive corrective surgery could be delayed. However, in this group, it is important to be aware of the possibility of intimal proliferation causing shunt stenosis thus decreasing the oxygen saturation. Stenosis of the stents due to intimal proliferation were noted in 5/7 patients who underwent recatheterization. Recatheterization after 3 to 6 months is therefore recommended, although the timing may be determined by the patient’s oxygen saturation, clinical status and growth. The ability to influence and optimize the size of the stent by repeat over-dilation during the follow-up catheterization is one of the additional advantages of this approach, compared with conventional shunt surgery, although repeated X-ray exposure in this group is a matter of concern. The ability to influence and optimize the size of the stent by repeat over-dilation during the follow-up catheterization is one of the additional advantages of this approach, compared with conventional shunt surgery, although repeated X-ray exposure in this group is a matter of concern. The naturally occurring stenosis of the branches of the pulmonary artery could also be dilated and stented if present.

In this group of patients, the stented arterial duct fulfils its function as an aorto-pulmonary shunt and an increase in the size of the pulmonary arteries occurs resulting in a more favourable anatomy for the corrective operation, at which time intra-operatively the duct can easily be ligated. Curiously, there was no intimal proliferation in one child 3 months after stent implantation and no clinical indication for recatheterization in another patient 11 months after stent implantation. The reasons are not clearly understood. It is possible that in one patient with a slightly curved anatomy of the duct, which had been completely stented, the covering of the bridge in the middle of the Palmaz-Schatz stent prevented intimal proliferation.

There were two deaths in this subgroup of patients. Whilst there was one death 2 weeks after stenting, the cause could not be attributed to the intervention. Death occurred after massive cerebral bleeding and irreversible renal failure. Another child died 2 days after intervention because of septicaemia.

**Conclusion**

In patients with a hypoplastic right ventricle in association with pulmonary atresia or critical pulmonary stenosis, stent implantation temporarily augments the pulmonary blood flow in a similar fashion to the creation of a surgical shunt rendering its closure, surgical or interventional, unnecessary. Thus the interventional strategy in these patients could totally replace surgery. In patients with more complex duct-dependent congenital heart disease, interventional stent implantation of the arterial duct is an alternative to palliative shunt operation. Unpredictable intimal proliferation tends to limit the pulmonary blood flow so re-dilation of the stent and therefore repeated X-ray exposure may be necessary to maintain the patient in optimal condition. Increasing cyanosis would be an indication for re-catheterization with a view to re-dilation. However, at present, it is probably prudent to perform a repeat catheterization 3 to 6 months after stent implantation on a routine basis to forestall unwanted complete stent occlusion.

This initial experience suggests that securing pulmonary blood flow by stent implantation in the arterial duct may be considered a satisfactory alternative to surgical creation of an aorto-pulmonary shunt. Stent implantation in this group of patients carried no risk of serious complications and pulmonary artery distortion and stenosis as a result of the procedure was not observed. This contrasts with the experience with surgical shunts. In addition the advantages of palliation are obtained, namely gaining time for the child and the pulmonary arteries to grow but leaving the operative field for definitive surgery untouched. The timing of definitive surgery can be optimized just as with surgical palliation. At definitive surgery, ligation of the stented duct has presented no specific problems.

Future developments in the stent technology, for example more flexible stents, may broaden the applications to very tortuous ducts and increase the range of palliative options for augmentation of pulmonary blood flow in duct-dependent circulations. More experience of the approach is required before its place in palliation of congenital heart disease is fully established.

**References**


