Off-Pump Replacement of the Pulmonary Valve in Large Right Ventricular Outflow Tracts: A Transcatheter Approach Using an Intravascular Infundibulum Reducer

ALIX MOLLET, ADELIN BASQUIN, BERTRAND STOS, AND YOUNES BOUDJEMLINE

Pediatric Cardiology, Assistance Publique des Hôpitaux de Paris, Necker For Sick Children, 75015 Paris, France

ABSTRACT: We report our experience of pulmonary valve replacement in animals with large right ventricular outflow tracts (RVOTs) using a percutaneous approach. We intended to implant a device to percutaneously reduce the diameter of the pulmonary artery (PA). Following its insertion, we intended to implant a valved stent inside the restriction. Animals were killed acutely (group 1, $n = 6$) and after a mean follow-up of 1 (group 2, $n = 3$) and 2 mo (group 3, $n = 9$). In group 1, all reducers were successfully deployed and allowed the reduction of the PA to a diameter of 12-mm. In one animal, the proximal part of the reducer did not reach its final configuration. Another reducer embolized when manipulating the stiff wire. The insertion of valves was therefore possible in 17/18 animals. One animal died from an arrhythmia during positioning of the valve. Angiographic evaluations showed no leak between devices and the pulmonary wall. During the follow-up, there was no device migration. At autopsy, reducers were fixed to the pulmonary wall and completely covered by a fibrous tissue. In conclusion, with the use of an intravascular reducer, implantation of a pulmonary valve is possible in sheep through a transcatheter approach when the RVOT exceeds 22-mm in diameter. (*Pediatr Res* 62: 428–433, 2007)

Early clinical human experience of transcatheter pulmonary valve replacement (PVR) has been reported recently (1–4). The device used for that application is a biologic valve harvested from the bovine jugular vein (Medtronic Inc) and mounted in a balloon expandable vascular stent (Numed, Inc). Due to valve availability, indications in humans are presently restricted to patients with a right ventricular outflow tract (RVOT) that does not exceed 22-mm in diameter. Unfortunately, most of the indications to pulmonary valve replacement are encountered in patients who underwent transannular patch repair of tetralogy of Fallot during infancy and who have pulmonary trunks that often exceed 30-mm in diameter. This makes PVR technically impossible with the current approach and valve design (5). To broaden the indications of PVR, we recently reported two approaches to reduce the diameter of the pulmonary artery to the available valve diameter. Firstly, we imagined and described a hybrid approach involving both surgeons and cardiac interventionists where the first step was to surgically perform a pulmonary artery (PA)

banding which could be followed by an off-pump PVR (6). Similar approach has been recently applied in humans by others (7–8). Secondly, we designed and recently reported the use of a preshaped self-expandable stent in a shape of a conduit with a central restriction of its diameter (9). Despite excellent preliminary results, several limitations to the study were noticed and prompted us to modify the device. Indeed, the anatomy of the RVOT varies greatly between patients making appropriate deployment of a 5.5-cm long device very unlikely. To encompass these difficulties and to reduce the invasiveness of the hybrid approach, this device was modified to make it implantable in the large majority of patients regardless of size and shape of PA. We then reported the use of a modified “pulmonary artery reducer” for PA banding (10). We report here our experience with the use of a similar device in another application. The aim of the study was to test this new device as to insert a pulmonary valve in sheep that have a pulmonary trunk of at least 25-mm in diameter.

METHODS

Device preparation for downsizing the diameter of the pulmonary trunk.

We designed and developed a self-expandable stent constructed from a single 0.22-mm nitinol wire in the shape of a conduit with a central restriction of its diameter (AMF, France). To shorten the device, both extremities are curved backwards to the central restriction, each part staying parallel and side-by-side as shown on Fig. 1. To guarantee the perfect sealing, we sutured a 0.1-mm PTFE membrane (AMF, France) with a 4.0 propylene thread on the inner side of the device (Fig. 1). The overall lengths of the deployed and crimped device are respectively 2 and 5.5-cm. To avoid the embolization of the reducer and the valved stent, spontaneous diameters of external and internal parts were carefully selected. We designed a reducer with extremities and central restricted part having a diameter of 35 and 15-mm respectively. We estimated that an over sizing of the device by 5 to 10-mm would enhance the mechanical fixation to the pulmonary wall. Similarly, the diameter of the central part was chosen 7-mm smaller than the valve to be inserted because we hypothesized that, by widening this restricted part, the balloon expandable stent would 1/ constrain the external part of the reducer adding forces to hold the reducer in place; and 2/ enhance the mechanical fixation between self and balloon expandable stents.

Preparation of the valved stent for pulmonary valve replacement. The valved stent was prepared as previously reported with the use of a 22-mm bovine jugular venous valve mounted in a balloon expandable CP stent (Numed Inc, Hopkinton, USA) (5). Valves were obtained from the slaughterhouse and constructed as a Melody valved stent (Medtronic Inc, Minneapolis, USA). Bicuspid and tricuspid valves were used for this experiment. All valved devices were stored in a glutaraldehyde solution until insertion.

Abbreviations: PA, pulmonary artery; PVR, pulmonary valve replacement; RVOT, right ventricular outflow tract

Received March 21, 2007; accepted May 10, 2007.
Correspondence: Younes Boudjemline, M.D., Service de Cardiologie Pédiatrique, Hôpital Necker-Enfants-Malades, 149, rue de Sèvres, 75015 Paris cedex, France; e-mail: younes.boudjemline@nck.ap-hop-paris.fr
This study was supported by the “Ville de Paris” grant 2006, Paris, France.

Figure 1. (A, B) The newly designed stent is shown uncovered (front and lateral views). The extremities have a diameter of 35-mm whereas central part is 15-mm. (C, D) A PTFE membrane is covering the extremities of the stent to assure the sealing of the device.

The delivery system. The delivery system consists of a front-loading long sheath, premounted with a 22-mm BiB (balloon in balloon) catheter (Numed Inc., Hopkinton, USA). Even though balloons are not necessary for nitinol stent deployment, the same delivery systems were used for balloon and self-expandable stents.

Repartition of the animals. Eighteen sheep weighing 55 to 80 kg were included. Animals were divided into three groups regarding the duration before sacrifice. We intended to implant a PA reducer as a first step, immediately followed by the insertion of a 22-mm biologic valve. Animals from groups 1 (6 animals), 2 (3 animals) and 3 (9 animals) were killed, respectively, just after valve implantation and after a follow-up of one or two months.

Percutaneous reduction of the diameter of the pulmonary trunk. Animals underwent catheterization for transcatheter implantation of a biologic valve in pulmonary position under general anesthesia. Anesthesia was induced with 10 mg/kg of thiopental and maintained with halothane in mechanically ventilated sheep. Cardiac and respiratory functions were monitored throughout the procedure. The right jugular vein was prepared for catheterization. Heparin (100 UI/kg) was administrated once during the procedure. Animals from groups 2 and 3 did not receive any long-term anticoagulation. All animals were treated according to the European regulations for animal experimentation (11). The study was approved by a local ethical committee for animal care (INRA, France).

Through the right jugular vein, a 5-French right Judkins coronary catheter (Cordis, Issy les Moulineaux, France) was advanced in the distal right PA. Through this catheter, a 0.035" extra-stiff guide wire (Amplatzter, Golden Valley, USA) was positioned distally. The reducer was loaded into the delivery system (Numed Inc., Hopkinton, USA), inserted over the previously positioned wire and advanced into the PA. The distal part was deployed by pulling on the external sheath while maintaining the dilator in position. To make the distal part fully configured (*i.e.*, applied to the pulmonary wall and surrounded by the distal part of the delivery system), the delivery system was pushed forward while holding the whole system together. The central part is subsequently uncovered, followed by the proximal part of the reducer. Its final configuration was obtained by pulling on the delivery system while maintaining the wire position. After the complete delivery of the reducer, the delivery system was retrieved leaving the device and the wire in position. The reducer was placed as close as possible to the native pulmonic valve. *In vitro* views of the deployment are provided in Fig. 2 and 3.

Percutaneous pulmonary valve replacement. The valved stent preloaded in a delivery system was advanced over the same wire and delivered as previously described inside the reducer (5). Briefly, while in position, the valved stent was deployed within the restricted part of the previously placed reducer by inflating the balloon of the delivery system. The balloon was then rapidly deflated and the delivery system retrieved leaving the device in position. Since radio-opacity of the reducer was used to position the valved stent, no injection of dye contrast was necessary during the positioning and delivery of the valve.

Figure 2. Photographs showing the delivery and configuration of the distal part of the device. (A) The reducer is shown loaded into the sheath. (B) The distal part is deployed by uncovering the device. (C) Once touching the wall of the plastic glass (representing the wall of the vessel), the sheath is advanced forward to configure the distal part. This causes the inversion of the distal part of the reducer in the same axis of the delivery sheath. (D) and (E) represent *in vivo* views of (B) and (C) (equivalent of a lateral view).

Figure 3. Photographs showing the delivery and configuration of the proximal part of the device. (A) The middle part is subsequently uncovered, followed by the proximal part of the reducer. (B) panel shows the proximal part in its deployed position. (C) Its final configuration is obtained by pulling on the delivery system while maintaining the wire position. (D) and (E) represent angiograms in lateral projection of (B) and (C) panels. Note the size of the PA trunk as compared with the size of the restricted area where the valved stent will be placed.

Cardiac catheterization and testing of the implanted devices. Right heart pressures (*i.e.*, RV and PA) were obtained before and after valvular implantation to determine the gradient across devices. No measurements were performed after insertion of the PA reducer to avoid any adverse mobilization of the device. Angiographic evaluation consisted of a PA injection distal to devices and a RV angiography to look for any paraprosthetic leak. Angiograms were performed using a marked pigtail catheter at two occasions: 1/ before the procedure to define the anatomy of the RVOT and to measure the size of the pulmonary trunk; and 2/ after the insertion of the valved stent to confirm the appropriate position and sealing of the reducer. Diameters were measured according to the markers of the pigtail catheter. Angiographic and hemodynamic studies were repeated before sacrifice of the animals. The study was not undertaken to appreciate the long-term function of the implanted valve but the long-term function of the reducer.

Table 1. Hemodynamic evaluation of animals from group 1

Table 1: Hemodynamic Evaluation of animals from group 1										
Group 1 Animal	PA diameter (-mm)				Systolic pressures (-mm Hg)				Outcome	
	At baseline	Inner diameter after reducer insertion	Outer diameter after reducer insertion	After valved stent insertion	At baseline		After valved stent insertion			
					RV	PA	RV	PA		
1	28.4	13.8	33.9	22	17	17	28	19	Success	
2	27.5	12.9	31.5	21	20	19	33	17	Success	
3	26.1	12	30	21	25	23	30	24	Incomplete configuration of the proximal disk	
4	30	15	34	NA	22	21	NA		Reducer embolization	
5	28.1	13.4	33.6	21.8	19	19	32	24	Success	
6	28	13.3	32.6	21.7	20	17	30	19	Success	
Mean	28	13.4	32.6	21.5	20.5	19.3	30.6	20.6		

RV, right ventricular; PA, pulmonary artery; NA, not applicable.

Graft retrieval. Grafts were explanted just after valve insertion in animals from group 1, one and two months after the initial procedure in animals from groups 2 and 3. Animals were killed and the thoracotomy was performed

thereafter. In a subgroup of animals (2 in each group), a left thoracotomy was performed before the sacrifice to look for the device with a beating and filled heart. Before harvesting, heparin (300 UI/kg) was given i.v. The heart and the lungs were retrieved in one block. The PA vascular tree was examined to determine the position of devices in relation to the pulmonic valve annulus. Devices were then harvested with a section of the PA, and rinsed to remove excess intraluminal blood. All devices were inspected macroscopically.

RESULTS

Insertions of devices in animals from group 1. Data are reproduced in Table 1. The mean size of the pulmonary trunk was 28-mm ranging from 26 to 30-mm. All reducers were successfully implanted. As expected, the mean diameter of the inner part of the reducer was 13.4-mm (range, 12–15-mm) whereas the external part measured 32.6-mm (range, 30–34-mm). In one animal, we delivered the proximal disk underneath the pulmonary valve. We, therefore, did not attempt to fully deploy the device to avoid slippage of the device into the right ventricle during its final configuration. As a result, the proximal part remained in its deployed position not parallel to the tubular part, which was more restricted than expected. It was, however, possible to deliver the valved stent inside this tubular portion, which resulted in a complete opening of the restriction (Fig. 4). In one animal, we lost the wire position during the advancement of the valved stent. The reducer embolized when we tried to reposition the stiff wire without retrieving the delivery sheath. Elsewhere, the insertion of valved stents was always possible without any problem or complication (Fig. 5). The placement did not require any injection of contrast dye since the reducer acted as a marker. After insertion, the inner part of the reducer was easily expanded at a mean of 21.5-mm (range, 21–22-mm).

Insertion of devices in animals from chronical groups (2 and 3). Characteristic and hemodynamic data of animals from groups 2 and 3 are shown respectively in Tables 2 and 3. The mean size of the main pulmonary artery was 27.4-mm, ranging from 25 to 30-mm. All reducers were delivered in the appropriate position. The diameter of the external part of the reducer was 30.8-mm (range, 27–34-mm). All valved stents were delivered successfully within the infundibulum reducer ($n = 12$). There was no early or late device migration. After insertion, the mean diameter of the inner part of the reducer increased from 11.2 (range, 10–12-mm) to 21.8-mm (range,

Figure 4. Angiograms showing incomplete deployment in the animal with the smallest PA diameter (A). (B) The proximal part (*) remains in its deployed position not parallel with the tubular part moderately lengthening the reducer. The valved stent is placed inside the reducer in good position.

Figure 5. Angiograms showing the insertion of the valved stent inside the infundibulum reducer. (A) Placement of the valved stent. (B) Inflation of the inner balloon catheter. (C) Inflation of the outer balloon catheter. (D) Aspect after full deployment of devices.

Table 2. Hemodynamic evaluation of animals from group 2

Table 2: Hemodynamic evaluation of animals from group 2											
PA diameter (-mm)					Systolic pressure (-mm Hg)						
Group 2 Animal	At baseline	Inner diameter after reducer insertion	Outer diameter after reducer insertion	After valved stent insertion	At baseline		After valved stent insertion		At sacrifice		Outcome
					RV	PA	RV	PA	RV	PA	
1	26.3	10	29.4	21.3	22	20	32	26	30	25	Sacrificed at 4 weeks
2	27.8	10.6	30.4	21.7	18	18	34	19	35	19	Sacrificed at 5 weeks
3	26.9	10	30	21.2	20	19	32	22	34	19	Sacrificed at 4 weeks
Mean	27	10.2	29.9	21.4	20	19	32	22.3	33	21	4.33 weeks

RV, right ventricular; PA, pulmonary artery; NA, not applicable.

Table 3. Hemodynamic evaluation of animals from group 3

Table 3. Echocardiographic examination of animals from group 3											
PA diameter (-mm)					Systolic pressure (-mm Hg)						
Group 3 Animal	At baseline	Inner diameter after reducer insertion	Outer diameter after reducer insertion	After valved stent insertion	At baseline		After valved stent insertion		At sacrifice		Outcome
					RV	PA	RV	PA	RV	PA	
1	28	11.2	31.2	21.8	24	24	34	19	36	12	Sacrificed at 2.2 months
2	28	12	32	22	22	21	32	18	30	20	Sacrificed at 2.2 months
3	25	10	27	21	19	17		NA			Died acutely from arrhythmia
4	27	11.6	31.3	22	18	17	26	18	30	19	Sacrificed at 2 months
5	26	10.7	28	21.8	20	20	25	19	26	18	Sacrificed at 2 months
6	28	12	32	22	21	21	28	22	26	20	Sacrificed at 2 months
7	30	13	34	22	22	19	32	18	30	19	Sacrificed at 2 months
8	29	13	34	22	22	19	34	24	30	20	Sacrificed at 2 months
9	27	13	31	22	22	24	26	22	24	18	Sacrificed at 2 months
Mean	27.6	12	30.1	21.8	20.7	19.6	29.6	20	29	18.2	

RV, right ventricular; PA, pulmonary artery; NA, not applicable.

21–22-mm). One animal had an arrhythmia during positioning of the valved stent. Balloon inflation/deflation was rapidly performed and followed by the retrieval of the delivery system. The arrhythmia was intractable and the animal died despite cardioversion. Autopsy showed a good position of both the reducer and the valved stent. No significant damage was found during inspection (no ventricular traumatism, thoracic bleeding, or hemopericarditis etc . . .).

Hemodynamic data ($n = 16$). Animals with device embolization ($n = 1$) and arrhythmia ($n = 1$) had no hemodynamic assessment following the procedure. Hemodynamic assessment in the remaining animals showed a mean systolic transprosthetic gradient of 9.9-mm Hg ($n = 16$) and 11.1-mm Hg ranging from 5–24-mm Hg ($n = 11$) respectively during acute and chronic evaluation (Tables 1–3). The ratio between the RV and aortic pressures was less than 35% in all animals. Acute and chronic angiographic evaluations revealed that the implants were in the desired position except in one animal from group 1 with the incomplete configuration of the device. These evaluations confirmed the sealing of the devices, showing no leak between devices and the pulmonary wall (Fig. 6). No stent fracture was observed.

Sacrifice. A planned sacrifice was performed in 16 out of 18 animals. One animal from group 1 had a device embolization, and one assigned to the group of two-month follow-up unexpectedly died during valvular insertion. Elsewhere, animals were killed as planned: after a mean time of one hour in animals from group 1 and 1.7 mo after insertion (0.8–2.2 mo) in animals from chronic groups. In group 1, coagulated blood

was trapped between the different parts of the reducer and the vascular wall. In chronic groups (groups 2 and 3, $n = 11/12$), all reducers were fixed to the pulmonary wall and completely covered by a fibrous tissue (Fig. 7). Small hematomas were found on the outer layer of the pulmonary wall but no perforation or blood extravasation was noticed. The valved stent was in good position within the restricted part of the nitinol stent. Valve leaflets were thin and perfectly mobile in acute group as well as in chronic implantations except in one animal where leaflets were thick and calcified.

DISCUSSION

Patients who underwent transannular patch repair of Tetralogy of Fallot during infancy usually have pulmonary trunks that exceed 30-mm in diameter, making the current transcatheter approach technically impossible (1–5,12–14). Trying to resolve this problem, we have investigated 2 different approaches to reduce the size of the PA trunk: 1/ externally with the help of the surgeon, and 2/ internally using an endovascular approach. We designed a self-expandable stent with two different diameters. To reduce the length of the device and allow for appropriate insertion in most patients, both stent extremities were designed to make a “U” turn or a 180° loop coming back to the middle of the restricted part of the device and in parallel with it. The diameter of the middle part was reduced to 15-mm to increase anchoring property. As a result, the implantation of a valved stent inside this central part has 2 advantageous effects. First, the nitinol stent tending to recover

Figure 6. Angiograms after dye injection showing the function of implanted devices (equivalent of a lateral view). (A, B, C) RV angiograms showing the perfect sealing of the device and the reduction of pulmonary artery diameter.

Figure 7. Macroscopic views (animal 1, group 3) showing the reducer and the valved stent explanted 2.2 mo after implantation. (A) The reducer is completely imbedded in the PA wall. (B) Aspect after dissection of the vascular wall showing covering the nitinol wire and fixation of the valved stent to the reducer. (C) Aspect of the valve: note the thin valve inside the stent.

its diameter adds some dynamic support to hold the valved stent in place. Second, the balloon expandable stent having more strength, the opening of the restricted part tends to constrain the external part, adding forces to hold the reducer against the vascular wall. We encountered only one embolization. This complication was more procedural-related than device-related. Indeed, the device was stable just after its insertion but during valve placement, we adversely lost the position of the stiff wire while advancing the valved stent. We tried to reposition the stiff wire without retrieving the delivery system. The device embolized during this maneuver. However, the occurrence of the embolization in the animal with the largest PA diameter of the series is not a hazard. It is highly probable that, beyond the procedural problem, the choice of the reducer was incorrect and that this animal would have required a larger device. It is certain that anchorage to the pulmonary wall is superior with the use of a larger device. However, a big discrepancy could be more deleterious than advantageous and hamper the deployment of the reducer. As shown in one animal of the series, the partial deployment of the proximal part had no impact on valve insertion and function. However, the use of a device that is between 5 to

10-mm larger than the PA diameter is probably the good choice but this should be precised with more experimental studies. The elasticity and diameter variation of the PA during cardiac cycle make the precise determination of the accurate diameter of devices more complex. In this preliminary work, only one size of device was available but this self-expandable stent can be manufactured to larger or smaller diameters to encompass all clinical situations. Even if we did not investigate the possibility to implant the valve as a one-step procedure with the implant being sutured inside the tubular part of the reducer, this is clearly a very reliable approach that needs to be explored. However, two limitations should be noticed here. First, because the device is chosen larger in diameter than the PA with a central part being reduced to 15-mm, it is difficult to predict the exact diameter of the central part where the valve would be sutured. This would need modification of the device with adjustment of the diameter of the central part. Second, the use of a balloon expandable stent is of interest adding some strength to hold the whole system in position and allowing precise and predictable expansion of the valve without any geometrical distortion.

Limitations to the study and unanswered questions. Since the aneurismal dilatation of the RVOT as seen in Fallot patients appears late after surgical repair, similar setting was impossible to achieve in animals and only animals with normal RVOT were studied. However, there should be no differences in the deployment of the device. With longer devices as reported with the first generation infundibulum reducer (7), it was obvious that, in a nonsymmetrical environment, the deployment could be hampered. However, with the present design, it is very unlikely. The shortness of the device, that is only 2-cm long, makes this design, at least theoretically, usable in all clinical settings. The precise placement of the device was difficult in the animal setting since we needed to impinge the function of the native pulmonic valve. In humans, this should be easier since it is indicated in patients with nonfunctioning pulmonary valve. The implantation of the reducer permitted the reduction of the PA diameter to the desired diameter without any clinically unacceptable elevation of right ventricular pressure. If in the same range, this elevation would be tolerated clinically in humans. However, it is likely that in clinical practice, one need to reduce the diameter of the pulmonary trunk by a ratio of two with a theoretical risk of creating a significant elevation of RV pressure. In those patients, it is obvious that the use of the largest biologic valve available is preferable since it would provide a greater valve area and less reduction of pulmonary artery diameter. Bovine jugular valves were used for these experiments because we used them in the past for our research on valvular implants but it is clear that the described pulmonary artery reducer can support any biologic valve. The only limitation is the possibility to reduce the thickening of the biologic valve enough to allow its insertion through a transcatheter course.

In conclusion, we report the design of a new self-expandable stent to percutaneously reduce the pulmonary artery to a diameter that allows for subsequent PVR through a transcatheter technique. If applicable in humans, the use of this reducer will lead to a considerable extension of the

present indications to transcatheter PVR, regardless of the anatomy of the PA.

Acknowledgments. The authors thank Philippe Marx who developed this new stent in cooperation with Younes Boudjemline.

REFERENCES

1. Bonhoeffer P, Boudjemline Y, Saliba Z, Hausse AO, Aggoun Y, Bonnet D, Sidi D, Kachaner J 2000 Transcatheter implantation of a bovine valve in pulmonary position: a lamb study. *Circulation* 102:813–816
2. Bonhoeffer P, Boudjemline Y, Saliba Z, Merckx J, Aggoun Y, Bonnet D, Acar P, Le Bidois J, Sidi D, Kachaner J 2000 Percutaneous replacement of pulmonary valve in a right ventricle to pulmonary-artery prosthetic conduit with valve dysfunction. *Lancet* 356:1403–1405
3. Bonhoeffer P, Boudjemline Y, Qureshi SA, Le Bidois J, Iserin L, Acar P, Merckx J, Kachaner J, Sidi D 2002 Percutaneous insertion of the pulmonary valve. *J Am Coll Cardiol* 39:1664–1669
4. Boudjemline Y, Agnoletti G, Piechaud JF, Iserin L, Vouhe P, Bonnet D, Sidi D, Bonhoeffer P 2003 Percutaneous pulmonary valve replacement: towards a modification of the prosthesis. *Arch Mal Coeur Vaiss* 96:461–466
5. Khambadkone S, Coats L, Taylor A, Boudjemline Y, Derrick G, Tsang V, Cooper J, Muthurangu V, Hegde SR, Razavi RS, Pellerin D, Deanfield J, Bonhoeffer P 2005 Percutaneous pulmonary valve implantation in humans: results in 59 consecutive patients. *Circulation* 112:1189–1197
6. Boudjemline Y, Schievano S, Bonnet C, Coats L, Agnoletti G, Khambadkone S, Bonnet D, Deanfield J, Sidi D, Bonhoeffer P 2005 Off-pump replacement of the pulmonary valve in large right ventricular outflow tracts: a hybrid approach. *J Thorac Cardiovasc Surg* 129:831–837
7. Berdat PA, Carrel T 2006 Off-pump pulmonary valve replacement with the new Shelhigh Injectable Stented Pulmonic Valve. *J Thorac Cardiovasc Surg* 131:1192–1193
8. Schreiber C, Vogt M, Bauernschmitt R, Prodan Z, Tassani P, Eicken A, Lange R 2006 Pulmonary valve implantation with a self-expanding stented valve: 1-year follow-up in the first patient. *Ann Thorac Surg* 82:2340
9. Boudjemline Y, Agnoletti G, Bonnet D, Sidi D, Bonhoeffer P 2004 Percutaneous pulmonary valve replacement in a large right ventricular outflow tract: an experimental study. *J Am Coll Cardiol* 43:1082–1087
10. Mollet A, Stos B, Bonnet D, Sidi D, Boudjemline Y 2006 Development of a device for transcatheter pulmonary artery banding: evaluation in animals. *Eur Heart J* 27:3065–3072
11. European convention for protection of laboratory animals 1999 Official Journal of European Community L222/29 to L222/37
12. De Ruijter FT, Weenink I, Hitchcock FJ, Meijboom EJ, Bennink GB 2002 Right ventricular dysfunction and pulmonary valve replacement after correction of tetralogy of Fallot. *Ann Thorac Surg* 73:1794–1800
13. Graham TP 2002 Management of pulmonary regurgitation after tetralogy of fallot repair. *Curr Cardiol Rep* 4:63–67
14. Discigil B, Dearani JA, Puga FJ, Schaff HV, Hagler DJ, Warnes CA, Danielson GK 2001 Late pulmonary valve replacement after repair of tetralogy of Fallot. *J Thorac Cardiovasc Surg* 121:344–351