Use of a jugular vein graft in a modified Blalock-Taussig procedure in dogs

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Objective—To determine whether autologous jugular veins provide functional grafts with high 30-day patency rates in an experimental model of systemic-to-pulmonary shunting performed with a modified Blalock-Taussig procedure.

Animals—15 healthy Beagles.

Procedure—A segment of the left jugular vein was implanted between the left subclavian and pulmonary arteries. Echocardiograms were obtained prior to surgery, at day 4 to 7, and at day 30 after surgery. Selective angiograms were performed immediately after surgery and on day 30. Oximetric shunt calculations were made via terminal angiography prior to euthanasia. Gross and histologic evaluations of the grafts were conducted.

Results—Grafts were patent in 12 of 15 dogs 30 days after surgery as assessed via auscultation, color Doppler ultrasonography, angiography, and histologic examination. Echocardiographic analysis revealed compensatory eccentric left ventricular hypertrophy. Mean pulmonary-to-systemic flow ratio was 1.5:1. Histologic evidence of endothelialization of the anastomotic sites and vein graft arterialization was detectable at 30 days.

Conclusions and Clinical Relevance—Autologous jugular vein grafts were effectively used to create a systemic-to-pulmonary shunt by use of a modified Blalock-Taussig procedure. High patency, ready accessibility, low cost, and theoretical adaptative remodeling during patient growth make autologous jugular vein grafts a valuable alternative to synthetic materials. (*Am J Vet Res* 2006;67:174–179)

Tetralogy of Fallot is an inherited congenital disease characterized by a large VSD, dextroposition of the aorta, pulmonic stenosis, and secondary right ventricular hypertrophy. This congenital abnormality represents approximately 5.8% and 3.9% of the congenital heart defects in children and dogs, respectively.[1,2] Although a large variety of breeds may be affected, TOF occurs most commonly in small-breed dogs.[2] The severity and nature of clinical signs and rates of survival are primarily determined by the degree of obstruction of right ventricular outflow and the magnitude and direction of the shunt caused by the VSD.

Over the course of the past few decades, improvement in treatment techniques in symptomatic children has yielded a progressive increase in survival rates during the early neonatal period. Although an early, complete repair of TOF is preferred by some,[3] in many cases, a 2-stage approach has been advocated as the key to successfully managing these patients.[4] In the 2-stage approach, an early, temporary systemic-to-pulmonary shunt is created to lessen hypoxemia. In a second phase, corrective surgery can be performed with primary repair of the VSD, release of the pulmonic stenosis, and ligation of the previously created shunt.[4,5] To establish a systemic-to-pulmonary shunt, a subclavian-to-pulmonary artery anastomosis is considered the most suitable strategy.[5-9] This was originally achieved by use of a direct anastomosis between the subclavian and pulmonary arteries—the cBTP.[10] Although the cBTP was a landmark procedure for its time, its effectiveness was jeopardized by kinking and subsequent obstruction of the subclavian artery at its attachment to the aorta.[11] To circumvent this shortcoming, an mBTP was used.[7,11] In the mBTP, graft materials, such as woven polyethylene terephthalate or expanded polytetrafluoroethylene, were interposed between the subclavian and pulmonary arteries. With this procedure, decreased morbidity and mortality rates have been reported[12,13] in children affected with TOF. Although effective in reducing the risk of delayed obstruction caused by kinking, synthetic graft materials have shortcomings including infection, thrombosis, lack of simultaneous growth with the patient, and added cost of the graft material.[9,14]

Open-heart repair, although preferable in humans, is not readily available in veterinary medicine.[15-17] Consequently, systemic-to-pulmonary shunts are usually the definitive treatment in dogs with TOF.[18,19] Only sporadic case reports have documented the use of cBTP or mBTP in dogs. However, despite initial success, in most cases, shunts eventually fail because of obstruction attributed to kinking and thrombosis.[18,19]

In attempts to circumvent the limitations associatged with synthetic grafts, autologous veins have been successfully used as vascular prostheses for the treatment of children with TOF.[20-24] Therefore, the objective of this study was to evaluate use of a jugular vein graft as an alternative material for an mBTP in dogs. The

VSD	Ventricular septal defect
TOF	Tetralogy of Fallot
cBTP	Classic Blalock-Taussig procedure
mBTP	Modified BTP

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jugular vein was chosen because of its anatomic characteristics, ready accessibility via a minimally invasive approach, and limited effects on the patient.

Materials and Methods

Pre- and postoperative clinical evaluation—The study was approved by the All University Committee for Animal Use and Care at Michigan State University. Fifteen clinically normal adult male Beagles with mean ± SD body weight of 14.9 ± 1.0 kg were obtained from a commercial vendor.[a] During a 2-week acclimation period, dogs were evaluated by performing right and left parasternal 2-dimensional and M-mode echocardiograms. Left ventricular volumes were calculated from 2-dimensional long-axis views by use of the Simpson protocol.[25] Left ventricular mass was calculated from the 2-dimensional echocardiogram by use of the area-length method.[25] Systolic function was assessed by use of fractional shortening, ejection fraction, and end-systolic volume.[26] The same echocardiographic data were obtained 4 to 7 and 30 days after surgery, including color-flow Doppler ultrasonography to assess patency of the shunt.

Anesthesia and preoperative procedures—Each dog was premedicated with acepromazine (0.02 mg/kg, SC) and morphine (0.2 mg/kg, SC). Anesthesia was induced by IV administration of ketamine (5.5 mg/kg) and diazepam (0.27 mg/kg) and maintained with an inhaled mixture of oxygen and isoflurane. Dogs were given an epidural injection of morphine (0.1 mg/kg) prior to surgery. At induction and every 2 hours during surgery, each dog received cefazolin (20 mg/kg, IV). Before starting the surgical procedure, a 6-F femoral sheath[b] was inserted percutaneously in the left femoral artery to measure arterial pressure during surgery and perform an arterial angiogram after surgery.

Surgical technique—A longitudinal 6-cm skin incision, parallel to the course of the left jugular vein, was made on the lateral side of the neck. Papaverine (0.02 mg/kg) was applied topically over the jugular vein to control spasms when present. Minor tributaries to the vein were identified and ligated, the adventitia was removed, and the incision was covered with a saline (0.9% NaCl) solution–soaked sponge while the surgical approach to the thorax was performed. By way of a routine left fourth intercostal thoracotomy, the subclavian artery was identified and isolated with umbilical tape. The adventitia was removed via blunt and sharp dissection. The subclavian artery circumference was measured by use of a 2-0 silk strand of known length.

Heparin (100 U/kg) was administered IV immediately before ligation and removal of 4 cm of jugular vein. The harvested vein was immediately placed in a vessel containing 100 mL of chilled saline solution, 3 mL of heparin (1,000 U/mL), and 0.2 mL of papaverine (30 mg/mL). A 5-F red rubber catheter was inserted into the vessel's distal end and secured with a bulldog clamp for graft irrigation with the vessel solution under low pressure. After its length had been recorded, the vein was transected obliquely and spatulated with a 0.3-cm longitudinal incision in the proximal end to increase its diameter at the anastomotic site.

A bulldog clamp was applied around the subclavian artery at its origin. The artery was ligated distally with 2-0 silk suture at a distance of at least 2 cm from the clamp and was transected 1 cm from its origin. The subclavian stump was flushed with the vessel solution and spatulated as described. An end-to-end anastomosis with 6-0 prolene[c] was used for the subclavian–jugular vein anastomosis (**Figure 1**). During the anastomosis, the vessel was repetitively flushed with the vessel solution through the 5-F catheter.

The pericardium was incised to expose the pulmonary artery for partial occlusion with a Satinsky clamp. The pulmonary artery was incised with a No. 11 scalpel blade and Potts scissor for 1 cm along its longitudinal dimension. The distal end of the jugular vein was transected with an oblique angle and spatulated for 0.2 cm on the ventral side. Two 6-0 prolene sutures were used for the end-to-side anastomosis, which was started from the cranial side and completed on the caudal side (Figure 1). Circulation through the graft was reestablished by removing the Satinsky clamp followed by the bulldog clamp. Patency was assessed by palpation of a thrill. Simple interrupted sutures (6-0 prolene) were used to control minor leakage in 3 dogs.

In 12 of 15 dogs, a silicone Doppler cuff[d] was applied either around the subclavian artery, when possible, or at the level of the graft to measure flow velocity. The probe lead wires were connected to a sonomicrometer[d] to obtain a first reading of the flow velocity and subsequently tunneled under the skin. The circumference of the graft was recorded. Immediately after surgery, a 5-F end-hole angiographic catheter was advanced to the origin of the left subclavian artery. Under fluoroscopy, 20 mL of diatrizoate meglumine[e] (60%) was injected to assess graft patency.

The dogs were treated with heparin (75 U/kg, SC, q 12 h) for 5 days, followed by aspirin (5 mg/kg, PO, once daily) for another 5 days. Cephalexin (20 mg/kg, PO, q 8 h) was administered for 10 days.

Terminal procedure—Thirty days after surgery, dogs were anesthetized as described and propofol (0.4 mg/kg/min; constant rate infusion) was administered to allow room-air breathing for at least 15 minutes before obtaining samples for oximetry. An angiogram of the shunt was obtained to reassess patency. A 3-F catheter-tipped pressure transducer[f] was used to measure pressures in the left ventricle, pulmonary artery, right ventricle, and right atrium. Oxygen saturations were calculated on the basis of measured PO_2 of blood obtained from the aorta, right ventricle, right atrium, and distal portion of the main pulmonary artery. The shunt ratio was calculated by use of the following equation:

$$SR = \frac{S_aO_2 - S_{MV}O_2}{S_aO_2 - S_{PA}O_2}$$

where SR is the shunt ratio, S_aO_2 is the arterial oxygen saturation, $S_{MV}O_2$ is the mixed venous oxygen saturation, and $S_{PA}O_2$ is the pulmonary artery oxygen saturation.

In the 12 dogs with flow probes, the wires were exposed in an attempt to record flow velocity. Because of malfunction of the flow meter, no flow velocity data were obtained.

Postmortem evaluation—Dogs were euthanized, and postmortem gross and histologic evaluations were conducted to determine graft patency and remodeling. Sections of tissue were collected from the graft, both anastomoses, and the vessel underneath the flow probe; portions of the normal right jugular vein, brachiocephalic trunk, and aorta were obtained for comparison. Tissues were fixed in neutral-buffered 10% formalin and routinely embedded in paraffin. For histologic evaluation, sections were stained with H&E as well as with trichrome and van Gieson stains. Immunohistochemical analysis was performed with antibodies (dilution, 1:100) against smooth muscle actin, vimentin, muscle-specific actin, and factor 8.[g] For immunohistochemical examination, sections of tissue were deparaffinized and rehydrated by routine methods.[27] Antigen retrieval was accomplished by incubation of slides in antigen-retrieval solution[g] in a steamer for 20 minutes. Endogenous peroxidase was blocked for 15 minutes with 3% hydrogen peroxide. Nonspecific immunoglobulin binding was blocked by incubation of slides for 10 minutes with a protein-blocking agent[g] prior to application of the primary antibodies. Vimentin immunostaining was used to

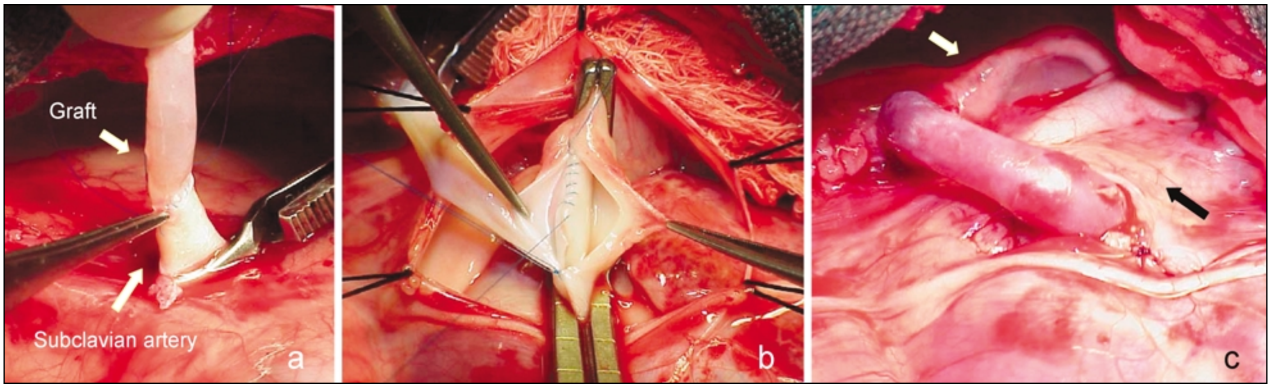

Figure 1—Photographs of anastomosis of the subclavian artery to the pulmonary artery with a jugular vein graft in a dog. Polypropylene suture (6-0) is used in a continuous pattern. (a)—Complete occlusion of the artery is achieved with a bulldog clamp. (b)—Notice that anastomosis is made to the medial side of the pulmonary artery. Partial occlusion of the main pulmonary artery is maintained with a Satinsky clamp. (c)—Jugular vein graft implanted between the subclavian (white arrow) and pulmonary artery (black arrow).

Table 1—Echocardiographic data (mean ± SD) obtained preoperatively (Pre), on day 4 to 7, and on day 30 after use of a jugular vein graft for anastomosis of the subclavian and pulmonary arteries in 14 dogs.

Variable	Pre	Day 4 to 7	Day 30
Left ventricular dimensions			
Diameter LVd (cm)	3.4 ± 0.3	3.8 ± 0.5*	3.8 ± 0.5*
Diameter LVs (cm)	2.4 ± 0.3	2.4 ± 0.3	2.4 ± 0.2
Volume LVd (mL)	35.2 ± 6.4	36.8 ± 7.5	42.5 ± 10.5*
Volume LVs (mL)	11.3 ± 3.1	10.2 ± 3.9	12 ± 4.4
Mass LV (g)	71 ± 14.4	82.1 ± 14.2*	85.6 ± 14.8*
Left ventricular ejection			
Ej frac (%)	59.7 ±5.1	68.2 ± 8.4*	67.6 ± 5.9*
Frac short (%)	29.9 ± 4	36.9 ± 7.6*	37.3 ± 5.1*
Left atrial dimensions			
Diameter LA (cm)	2.2 ± 0.2	2.3 ± 0.6	2.9 ± 0.7*
LA/Ao ratio	1.2 ± 0.2	1.2 ± 0.3	1.4 ± 0.3*
Heart rate (bpm)	104 ± 19.8	134 ± 19.6	125 ± 31.0

*Significantly ($P < 0.05$) different from preoperative value.

LVd = Left ventricle at end-diastole. LVs = Left ventricle at end-systole. Ej frac = Ejection fraction. Frac short = Shortening fraction. LA = Left atrium. Ao = Aorta. bpm = Beats per minute.

detect mesenchymal differentiation, desmin and smooth muscle actin were used for differentiating smooth versus skeletal muscle, and factor 8 was used for endothelial cell identification. A labeled streptavidin-biotin-peroxidase complex was used with 3,3'-diaminobenzidine substrate.[g] Sections were counterstained with Mayer hematoxylin. Positive immunohistochemical controls included normal arteries and veins to which the appropriate antisera were added. For negative controls, the primary antibodies were replaced with homologous nonimmune sera.

Statistical analysis—Results are reported as mean ± SD. Repeated-measures ANOVA was used to compare echocardiographic data. For dogs with shunts that were patent at 30 days after surgery, the Fisher least significant differences test was used to evaluate differences between individual days. For all analyses, $P < 0.05$ was considered significant.

Results

Fourteen dogs recovered from surgery without complications. In 1 dog, an occluded shunt was identified angiographically immediately after surgery. Subsequent exploratory thoracotomy revealed a mechanical obstruction at the level of the flow probe. Because of inability to reestablish flow, this dog was euthanized during anesthesia and data for the dog were not included. Shunt patency was confirmed in 12 of the remaining 14 dogs 30 days postoperatively and was always associated with a systolic or continuous murmur. Failure of 2 grafts was attributable to thrombosis; in 1 dog, thrombosis occurred at the junction with the

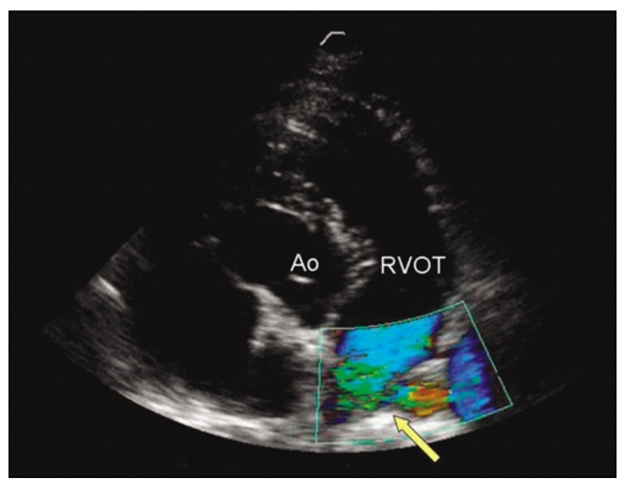

Figure 2—Right parasternal short-axis echocardiographic view of the right ventricular outflow tract (RVOT) and the aorta (Ao) in a dog 30 days after anastomosis of the subclavian artery to the pulmonary artery with a jugular vein graft. Notice turbulent blood flow that indicates variable blood velocities where the shunt flow enters the pulmonary artery (arrow).

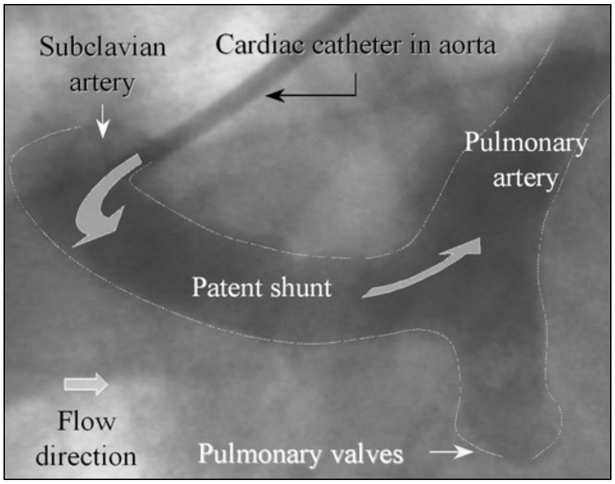

Figure 3—Arterial angiographic view of cardiovascular structures of the dog in Figure 2, 30 days after surgery. Notice that the shunt is patent and outlined by contrast material.

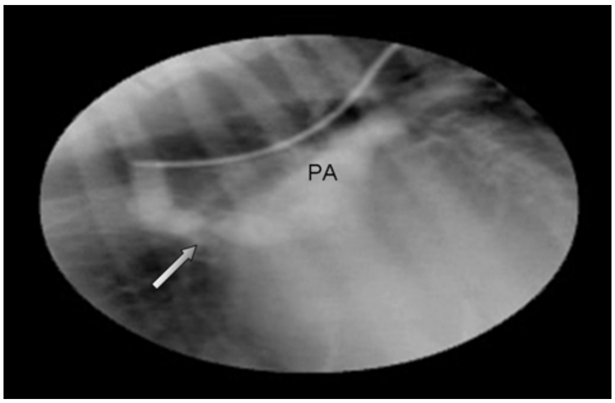

Figure 4—Angiographic view of cardiovascular structures in a dog 30 days after surgery. Notice a flow probe causing stenosis of a patent shunt (arrow). PA = Pulmonary artery.

pulmonary artery, and in the other dog, thrombosis occurred under the flow probe. The only other minor complication observed in this study was Horner syndrome, which occurred 2 days after surgery in 1 dog.

Vascular morphometric data were obtained during surgery. Mean graft length was 3.9 ± 0.5 cm, and mean graft circumference was 2.1 ± 0.5 cm. Mean subclavian artery circumference was 2.1 ± 0.3 cm.

Echocardiographic data—Echocardiographic data were summarized (**Table 1**). The shunt was consistently visible when patent (**Figure 2**). The shunt-induced increase in left ventricular stroke volume was accompanied by eccentric left ventricular hypertrophy (P = 0.006), whereas end-systolic volume was not significantly affected (P = 0.7). An increase in left atrial size was observed 30 days after surgery (P = 0.002). Ejection fraction (P = 0.002) and left ventricular end-diastolic volume (P = 0.005) were significantly increased. Cardiac remodeling was evident as early as 4 days after surgery.

Angiographic data—Immediately after surgery, shunts were patent in 11 of 12 dogs with flow probes and in all 3 dogs without flow probes. Thirty days after surgery, graft patency was evident in 9 of 12 dogs with flow probes and in all 3 dogs without flow probes (**Figures 3 and 4**).

Table 2—Vascular pressures (mean ± SD [mm Hg]) obtained 30 days after use of a jugular vein graft for anastomosis of the subclavian and pulmonary arteries in 14 dogs. Left ventricular diastolic pressures were recorded at end-diastole.

Pressure	Systole	Diastole
Arterial	99.0 ± 14.4	62.4 ± 10.8
Left ventricle	96.6 ± 16.3	8.3 ± 1.6
Pulmonary artery	27.2 ± 8.1	15.1 ± 7.9
Right ventricle	27.4 ± 10.2	8.7 ± 7.1
Right atrium	7.1 ± 2.1	5.4 ± 2.3

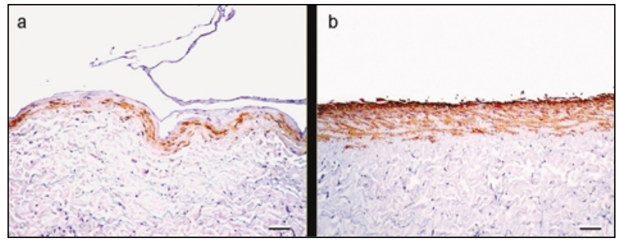

Figure 5—Photomicrographs of a normal jugular vein from a dog (a) and a jugular vein used for anastomosis between the subclavian and pulmonary arteries in a dog 30 days after surgery (b). Notice intense brown staining in the jugular vein used for anastomosis, which identifies smooth muscle actin in leiomyocytes that proliferated in the vein. Immunohistochemical stain; bar = 100 µm.

Oximetry and pressures—The shunt flow ratio was 1.5 ± 0.3. Pulmonary artery saturation was 84.9 ± 3.1%. No dog developed pulmonary hypertension during the 30 days after surgery. Pulmonary systolic and diastolic pressures were 27.2 ± 8.1 mm Hg and 15.1 ± 7.9 mm Hg, respectively (**Table 2**).

Histologic analysis—Graft remodeling was evident at gross examination. A progression toward arterialization was confirmed via histologic examination. Compared with the contralateral jugular veins, the grafts consistently had increased wall thickness secondary to increased numbers of elastic fibers, as confirmed by use of van Gieson staining, and spindle cell proliferation within the tunica media. The spindle cells yielded positive results for vimentin, muscle-specific actin, and smooth muscle actin—a staining pattern most consistent with leiomyocytes (**Figure 5**). The lumen of the graft was lined by a single layer of endothelial cells that stained with factor 8. In all dogs instrumented with flow probes, an additional finding was a focally decreased graft lumen (partial obstruction) caused by graft wall thickening (4 to 5 times normal) because of fibrosis (positive results of trichrome staining of affected areas) and localized inflammation at the level of the flow probes.

Discussion

Jugular veins used in an mBTP in dogs provided a high short-term patency rate (12/15), which was comparable to rates reported[7,9,11,14,28] in children (75% to 89%) who received synthetic grafts. Although we consider our patency rate acceptable, outcomes should be even better in clinical cases because placement of flow probes, which were responsible for 2 of the 3 shunt failures, will not be necessary. No important complications were associated with the use of jugular veins. Early remodeling of the graft and the heart occurred in all dogs.

Selection of the jugular vein was originally based on easy accessibility and limited effects on the patient. However, the study revealed that veins are well suited for an mBTP for additional reasons. Jugular veins are naturally of a diameter and length that facilitate creation of the shunt. The flow through the shunt meets reported[28] criteria that are needed in children to relieve clinical signs while avoiding pulmonary overload.

Vein grafts can be used in a variety of ways. In the originally described mBTP, an end-to-side anastomosis was performed between the graft and the artery. We chose to implant the vein graft as an end-to-end extension to the subclavian artery to take advantage of their similarities in diameter. An end-to-end anastomosis is probably technically easier in this location in dogs, diverts all the subclavian flow to the shunt, and maintains continuity in the direction of intraluminal blood flow, therefore reducing turbulence and the associated risk for intimal damage and thrombosis.

Danilowicz et al[20] reported that graft length is an important factor that affects shunt success. By adding length to the subclavian artery, use of the vein overcame the limitations and consequent failure associated with the cBTP. In our dogs, a mean graft length of 3.89 cm was sufficient to create a smooth curvature to avoid kinking. Although blood flow interruption was found in 2 shunts at the level of the flow probe, no kinking was identified in the subclavian artery or graft in any of the remaining dogs.

Graft diameter has also been reported[12,14] as a crucial element that influences successful outcome of an mBTP in children. Although large shunts increase the risk of congestive heart failure, smaller shunts provide inadequate palliation and are associated with high risk of thrombosis.[7,8] Although not detected in our study, distension of the vein graft might occur as a result of increased transmural pressures. Graft distension could promote turbulent flow and increase the risk of thrombosis. Vein diameter in this study (mean, 2.3 mm) matched the size of the subclavian artery (mean, 2.1 mm), thus facilitating the anastomosis and decreasing the risk of excessive shunt flow. The mean shunt ratio in this study was 1.5:1. This value is similar to ratio of well-tolerated shunts in children with cyanotic heart disease (mean, 1.3:1 [SD, 0.1]).[29] In immature patients, synthetic grafts must be oversized[d] at the time of implantation to allow for growth.[8,11] Veins could offer the advantage of synchronous growth with the patient, avoiding the risk of congestive heart failure associated with oversized grafts.[23] If growth is considered essential for maintenance of adequate flow over time, interrupted sutures might be preferred to the continuous pattern originally recommended in the cBTP.

Histologic examination revealed graft remodeling in the form of arterialization of the graft as early as 30 days after surgery. There was subendothelial proliferation of spindle cells, leading to increased wall thickness of the graft. These cells had a staining pattern most consistent with leiomyocytes. These changes may indicate early formation of a tunica muscularis and highlight the graft's capacity for adaptive remodeling and perhaps the potential for growth. Growth was not evaluated in this study but has been reported[23] for veins used in similar conditions in human patients.

Formation of a continuous endothelial lining between the graft and subclavian artery was also observed and could lead to several positive outcomes. Risks of secondary infections and thrombosis should be reduced, compared with synthetic grafts.[30-33] We identified thrombosis in only 2 dogs; this occurred at 4 to 7 days and 30 days after surgery. Decreased intensity of the murmur and echocardiography helped in diagnosis of the occlusion, followed by confirmation by angiography. In 1 shunt, thrombosis was identified at the level of the pulmonary artery. Abrupt interruption of flow in another shunt was detected at the level of the flow probe.

The flow probes, implanted for acquisition of flow data, were directly implicated in 2 shunt obstructions. One probe induced thrombosis, and the other probe caused kinking of the graft immediately after surgery. Without this technical complication, the patency rate might have been as high as 14 of 15. To our knowledge, this type of flow probe obstruction has not been reported before. A potential explanation may be shunt deformation attributable to probe interaction with a compliant vessel in an area subjected to repetitive heart and lung motion. The only other complication observed, transient Horner syndrome, is a known complication of mBTP in children and is caused by damage to the ansa subclavia during subclavian artery dissection.[11]

Although well tolerated in clinically normal dogs, the efficacy of vein grafts for patients with TOF has not been determined. Pulmonary artery hypoplasia, occasionally present in such patients, could potentially exacerbate the low tolerance to increased blood flow through the pulmonary vasculature, especially when larger shunts are used. The dogs used in this study did not have signs of congestive heart failure, as indicated by normal results of postoperative thoracic auscultation, left ventricular end-diastolic pressure measurements (< 12 mm Hg), and lack of radiographic signs of pulmonary edema. In addition, mean pulmonary artery pressure of 21.1 ± 8.0 mm Hg (ie, within reference range) was recorded and there was no evidence of pulmonary hypertension. Shunt flow, provided by veins, should be well tolerated in patients with TOF but will require evaluation in the context of naturally occurring disease.

Results of the study reported here indicated that autologous vein grafts can be safely and effectively used as an alternative to synthetic materials. The jugular vein is particularly well suited for an mBTP. It is readily available, matched in size to the subclavian artery, and should grow synchronously with the rest of the vascular system while undergoing adaptive remodeling. The similarity in circumference facilitates the creation of the anastomosis and may provide a natural restriction against excessive shunting.

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a. Covance, Kalamazoo, Mich.
b. Cordis Corp, Miami, Fla.
c. Ethicon Inc, Somerville, NJ.

d. Triton Technology Inc, San Diego, Calif.
e. Hypaque, Amersham Health Inc, Princeton, NJ.
f. Millar Instruments Inc, Houston, Tex.
g. DakoCytomation California Inc, Carpinteria, Calif.

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