Long-term palliation of tetralogy of Fallot in dogs by use of a modified Blalock-Taussig shunt

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Objective—To describe a modified Blalock-Taussig shunt (mBT) procedure and assess its use in dogs with clinical signs associated with tetralogy of Fallot (TOF).

Design—Descriptive report.

Animals—6 dogs with severe TOF-associated clinical signs.

Procedures—Each dog had TOF (confirmed echocardiographically or angiographically) and underwent an mBT shunt procedure for surgical palliation of signs. The surgery was performed through a left fourth rib resection or a left fifth intercostal thoracotomy. The left subclavian artery was dissected free from surrounding mediastinal tissue. The main pulmonary artery trunk was exposed through an incision in the overlying pericardium. A shunt comprised of a 6-mm-diameter tube of expanded polytetrafluoroethylene (6 dogs) or a segment of carotid artery (1 dog) was sutured end to side between the left subclavian artery and pulmonary artery trunk.

Results—5 of the 6 dogs survived the immediate postoperative period. The dog that died shortly after surgery was the smallest of the dogs (weight, 2.9 kg [6.38 lb]) and had received the carotid artery autograft. Three dogs survived long term and 2 dogs died of unknown causes 6 years after undergoing the mBT shunt procedure. In all dogs that survived the mBT procedure, shunt patency was confirmed and quality of life appeared improved.

Conclusions and Clinical Relevance—These findings have suggested that the mBT shunt procedure safely provides long-term palliation of TOF-associated clinical signs in dogs. In addition, it may offer an effective low-risk and lower-cost alternative to open heart repair of TOF. (J Am Vet Med Assoc 2007;231:721–726)

Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>TOF</td>
<td>Tetralogy of Fallot</td>
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<td>RVOT</td>
<td>Right ventricular outflow tract</td>
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<td>BT</td>
<td>Blalock-Taussig</td>
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<td>mBT</td>
<td>Modified Blalock-Taussig</td>
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<td>PTFE</td>
<td>Polytetrafluoroethylene</td>
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Tetralogy of Fallot is one of several complex congenital cardiac abnormalities that result from developmental defects of the ventricular outflow region (conotruncus) in dogs. Breeding studies have revealed that conotruncal defects result from a single major gene defect in Keeshonds. Although the predisposition of the Keeshond breed to this disease is well documented, TOF also develops sporadically in many other dog breeds. The conotruncal defects that contribute to TOF include RVOT obstruction, ventricular septal defect, dextropositioning of the aorta, and secondary right ventricular hypertrophy. The pathophysiologic changes associated with this condition depend on the degree of RVOT obstruction and the proportion of blood that is shunted across the ventricular septal defect from the right ventricle. If the volume of shunting blood is sufficient, the dog will develop systemic hypoxia and cyanosis, progressive polycythemia, exercise intolerance, and weakness. Exercise or exertion can increase the volume of blood shunted across the septal defect, thereby increasing the severity of the hypoxemia. Undercirculation of the pulmonary vascular bed from birth can lead to underdevelopment or hypoplasia of the pulmonary arterial tree. It is also assumed that, as in humans, the RVOT obstruction can progress over time and therefore the clinical signs will gradually worsen. Dogs affected to this degree have a severely diminished quality of life and reduced life expectancy. Treatment of human neonates with TOF via early, complete surgical repair is now considered standard practice. This has only been considered safe and reliable within the last decade, as techniques and experience with cardiopulmonary bypass in neonates have developed. In humans, prior to the description of open heart surgical repair by Lillehei et al, the creation of an extracardiac systemic arterial–to–pulmonary artery anastomosis or shunt to increase pulmonary arterial blood flow was used as a long-term palliative measure. Some patients lived a long life after such procedures. After the description of open heart repair of the primary defects (closure of the ventricular septal defect and reconstruction of the RVOT), shunt procedures were used as a temporary measure prior to definitive corrective surgery, providing relief from symptoms, allowing
the patient to grow, and stimulating the pulmonary artery system to develop. Several shunting operations were described including the classic BT shunt, the mBT shunt, Potts anastomosis, and Waterston anastomosis. Procedures that involved the direct central anastomosis of the aorta to the pulmonary artery (eg, Potts and Waterston anastomoses) have been mostly abandoned because the high rate of blood flow through the anastomosis frequently resulted in congestive heart failure. Also, distortion of the pulmonary artery was common, and reversal of the shunt at the time of definitive repair was technically difficult. The classic BT shunt was also abandoned by some because of complications associated with kinking of the divided subclavian artery that resulted in shunt failure by mechanical or thrombotic occlusion. In addition, there was concern regarding failure of left thoracic limb development because of reduced blood flow.

Modification of the BT shunt by interposition of a synthetic tube of PTFE (and subsequently expanded PTFE) via an end-to-side anastomosis of shunt to subclavian artery and shunt to pulmonary artery was described by de Leval et al in 1981. This procedure allowed a larger-diameter shunt to be sutured in place, thereby reducing the risk of thrombotic complications while preserving blood flow to the thoracic limb. Because flow through the shunt was regulated by the subclavian artery orifice, congestive heart failure secondary to overperfusion was less likely to develop. The mBT shunt has been the subject of many subsequent publications, and a great deal has been learned about its use in humans. Long-term complications such as occlusion (particularly in shunts ≤ 4 mm in diameter), disparate growth of left and right pulmonary arteries, and distortion of the pulmonary artery at the site of shunt attachment (associated with growth of the infant) are recognized still as limitations of this technique in humans.

In dogs, complete repair of TOF has been reported by several authors but is not commonly performed. Palliative medical treatment (via administration of β-adrenergic receptor blockers, repeated phlebotomy, and oxygen supplementation) has occasionally provided satisfactory intermediate-term results. Successful balloon dilation of pulmonic stenosis provided good short-term palliation in 1 dog but surgical reconstruction of the RVOT caused congestive cardiac failure in another. Palliative shunting has been reported in 6 dogs clinically affected by TOF. Eyster et al described use of a BT shunt procedure in 1 dog; the palliation was effective for 1.5 years before shunt occlusion caused the death of the dog. Generally, the results of the BT shunt procedure have been inconsistent. Ringwald and Bonagura described 4 dogs that underwent a Potts anastomosis, of which 2 died shortly after the surgery and 2 survived long term. Of the 2 dogs that survived, 1 lived for 3.5 years and the other lived for 3 years after the procedure; both dogs had recurrence of clinical signs for a period of 6 to 12 months before death. Weber et al described a successful mBT shunt procedure in a dog in which a classic BT shunt had failed. That dog survived 7 years after the mBT shunt procedure and had improved quality of life and a stable Hct during that time.

The purpose of the present report was to describe the application of an mBT shunt procedure and assessment of its use in dogs with clinical signs associated with TOF. We hypothesized that the mBT shunt procedure would be technically feasible in dogs and that it would provide long-term palliation for those affected with TOF.

**Techniques**

**Patient selection**—Dogs with clinical signs of severe and progressive disease (severe exercise intolerance, cyanosis at rest, and increasing PCV over time), with echocardiographic or angiocardiographic confirmation of TOF, and without evidence of pulmonary artery hypoplasia or atresia were considered for this procedure (Figure 1). All dogs had been evaluated on multiple occasions by cardiologists who had determined that medical or conservative treatment was no longer beneficial. Owners gave full informed consent for the mBT shunt procedure to be carried out on their dogs.

**Anesthesia**—The anesthetic protocol used in each institution varied according to drug availability, disposition of the dogs, and personal preference of the anesthesiologists involved. All dogs received either an opioid alone (meperidine, 3 mg/kg [1.36 mg/lb]) or an opioid combined with an anticholinergic and a benzodiazepine (hydromorphone, 0.2 mg/kg [0.09 mg/lb]; glycopyrrolate, 0.01 mg/kg [0.005 mg/lb]; and midazolam, 2.5 mg/kg [1.14 mg/lb]) via IM injection. Anesthesia was induced by use of a combination of fentanyl (3 µg/kg [2.27 µg/lb]), etomidate (2.5 µg/kg), lidocaine (20 mg/kg [9.1 mg/lb]), and midazolam (2.5 mg/kg) administered via IV injection. Anesthesia was maintained via continuous rate infusion of fentanyl (0.7 µg/kg/min [0.32 µg/lb/min]) alone or in combination with isoflurane in oxygen delivered via a cuffed endotracheal tube.

![Figure 1—Digital subtraction cardiac angiogram of a dog with TOF (dog 6). The catheter (C) has been passed down the cranial vena cava and across the septal defect; the tip is in the left ventricle (LV). The contrast agent has simultaneously outlined the aortic arch (Ao) and the pulmonary artery (PA). LSA = Left subclavian artery, BCT = Brachycephalic trunk.](image-url)
During anesthesia, catheters were placed to permit central venous pressure and direct arterial pressure monitoring and quantification of urine production. Perioperative antibiotic was provided via IV administration of cefazolin\(^d\) (20 mg/kg, q 2 h) or cefuroxime\(^e\) (20 mg/kg, q 2 h) throughout the procedure.

**mBT shunt technique**—For each dog, a left lateral fourth intercostal or fifth rib resection thoracotomy was performed. Heparin (30 to 50 U/kg [13.6 to 22.7 U/lb]) was administered IV. The left subclavian artery was identified, and an approximately 4-cm length close to the aorta was dissected free from surrounding mediastinal tissues (Figure 2). The pericardium was opened to expose the lateral aspect of the left main pulmonary artery. A segment of 6-mm-diameter expanded PTFE vascular graft or previously harvested autologous carotid artery was cut to extend from the intended site of anastomosis on the subclavian artery to the left main pulmonary artery; the ends were cut at an angle so that the graft would lie flat once it was sutured in place. A Cooley or Codman vascular clamp was used to isolate a 2-cm length of the subclavian artery. A No. 11 Bard-Parker scalpel blade was used to make an initial incision in the left lateral aspect of the subclavian artery; this incision was extended to the desired length (measured against the cut end of the graft) by use of 60° Potts scissors. The graft was then sutured to the edges of the incision in the subclavian artery in an end-to-side manner with 5-0 or 6-0 polypropylene or PTFE suture in a simple continuous manner. The clamp was removed from the subclavian artery, and the graft was allowed to fill with blood. Then, either the clamp was replaced on the subclavian artery or a Hopkins bulldog clamp was placed on the graft itself to facilitate hemostasis. The pulmonary artery was opened between the jaws of a Cooley or Codman vascular clamp, and the graft was allowed to fill with blood. Then, either the clamp was replaced on the subclavian artery or a Hopkins bulldog clamp was placed on the graft itself to facilitate hemostasis. 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recorded PCV and serum total solids concentration, the
time at which those variables were evaluated in relation
to the procedure, and the last echocardiographic evi-
dence of shunt patency were recorded. Additional fol-
low-up information (regardless of whether the dog was
alive or dead) was determined via telephone conversa-
tions with the owners and their veterinarian.

Results

Six dogs underwent the mBT shunt procedure
(Table 1). Four dogs were evaluated and treated at the
Matthew Ryan Veterinary Hospital of the University of
Pennsylvania (dogs 1 through 4); 2 dogs were evalu-
ated and treated at the Queen Mother Hospital of The
Royal Veterinary College, London (dogs 5 and 6). The
smallest dog (dog 4) weighed 2.9 kg (6.38 lb); all other
dogs weighed > 10 kg (22 lb). In dog 4, a carotid artery
autograft was used to create the shunt, whereas the re-
maining 5 dogs received a synthetic (expanded PTFE)
graft (6 mm in diameter). All the dogs survived surgery.
Dog 4 developed anemia during the first 12 hours after
surgery and required a whole blood transfusion; it also
developed respiratory failure 24 hours after surgery
and required mechanical ventilation for a period of 36
hours. That dog was weaned from ventilator support
but shortly after weaning underwent a cardiac arrest
and died. Postmortem examination was not permitted.
Four of the 5 surviving dogs had a sustained decrease
in PCV, compared with the presurgical value, and all of
the surviving dogs had PCV values that remained stable
over time. In all the dogs for which data was available,
there was an increase in \(P_{aO_2}\) during the immediate
postoperative period, compared with the \(P_{aO_2}\) prior to
surgery. Two of the dogs that survived to discharge did
eventually die. According to the local veterinarians’ rec-
ords, dog 1 died at the age of 11.5 years (6 years af-
ter the mBT shunt procedure) as a result of congestive
heart failure, but no further details are available; dog 2
died suddenly at home at the age of 7 years (6 years af-
ter the mBT shunt procedure), but similarly no further
medical details were available. All other dogs were alive
at the last known follow-up (ie, 30 to 54 months after
surgery).

Discussion

To the authors’ knowledge, the 6 dogs with TOF-
associated clinical signs in the present report represent
the largest group to have undergone surgical palliation
described in the veterinary medical literature. Five of
the 6 dogs reported here survived the surgery and had
an uneventful postoperative recovery. Two of these dogs
subsequently died, but both survived for 6 years after
the mBT shunt procedure. Three other dogs were alive
at 36, 48, and 18 months after the procedure. The dog
that died shortly after surgery in the present study dif-
fered from the other dogs in 2 important aspects. First,
it was the smallest dog (weight, 2.9 kg; all other dogs
weighed > 10 kg), and second, it had a section of na-
tive carotid artery used to create the shunt rather than
a synthetic material. The decision to use carotid artery
rather than 3- or 4-mm-diameter expanded PTFE tub-
ing was made on the basis of experience in human neo-
ates, in which inadequate palliation following use of
4-mm synthetic shunts was evident in 23 of 34 (68%)
of patients because of partial or complete shunt occlusion 2 years after surgery. In the dog that died, size of the vessel and physical size of the dog made the surgical procedure more challenging. The procedure appeared to go well, and the dog's recovery was proceeding well initially. However, the dog died as a result of respiratory failure and subsequent cardiac arrest; because a necropsy was not permitted, the causes of those complications were not established, nor was patency of the shunt evaluated after surgery or death. The risk of surgery-related complications, such as suture occlusion or thrombus formation at the anastomotic site, might be higher in physically smaller dogs, and such complications could, in part, explain the postoperative events in that particular dog in the present study. Unfortunately, comments regarding the cause of death in that dog are speculative, and no firm conclusions or recommendations can be made on the basis of the experience with that patient. The poor outcome in that small-sized dog concurs with the subjective assessment of the procedure in small dogs (<10 kg) by Eyster. However, the fact that successful palliation of TOF via microvascular anastomosis of the internal thoracic artery to the pulmonary artery has been reported in a cat suggests that a technique that is different from that described in this report might be more appropriate for small dogs. The only data available for the 2 dogs that died 6 years after surgery was the owners' assessment of quality of life just before death. Cause of death would be important in evaluating the procedure reported here but unfortunately was not available. Even if both dogs had shunt-related complications (occlusion or excessive flow) that caused their death, their treatment could still be considered successful because they both lived 6 years after the procedure. In addition, both these dogs enjoyed a better quality of life after surgery, according to their owners. When compared with other palliative procedures reported in the veterinary literature, dogs that have undergone an mBT shunt procedure for palliation of TOF have appeared to have improved short- and long-term survival.

In the present study, objective data collected to assess the shunt success were limited to arterial PaO₂ before and immediately after surgery, echocardiographic evidence of shunt patency, and serial PCV evaluations. Comparison of immediate preoperative PaO₂ with immediate postoperative values revealed only a modest increase in PaO₂ following shunt procedure in 4 of the treated dogs. Preoperative values were derived from blood samples collected from conscious dogs, and factors such as the stress associated with arterial puncture could have influenced these results. In addition, postoperative samples were collected the day after surgery while the dogs were in an intensive care unit and could have been influenced by postoperative thoracic surgical factors such as pain, lung atelectasis, and analgesic drug treatment. All dogs in which this comparison could be made did, however, have improvement in PaO₂ as a result of the surgical procedure. In all of the dogs evaluated echocardiographically after surgery, turbulent blood flow in the pulmonary artery at the site of entry of the shunt was evident, and frequently the shunt itself could be identified. Such evaluations were done intermittently for each dog, but from the last known record of shunt patency for those dogs in which it was assessed, the interval of patency ranged from 8 to 15 months after surgery.

Indirect evidence of shunt patency can be derived from PCV evaluations. Hypoxia is a potent stimulus for hematopoiesis, and secondary polycythemia is a classic feature of untreated TOF. All the dogs in the study reported here had either high absolute PCV or high PCV relative to their age prior to surgery. All the dogs had a similar or lower PCV value 1 month after surgery, compared with the value before surgery. The PCV values were monitored long term on a more random basis, but all appear to have remained stable at periods from 8 to 36 months after surgery. The PCV in all dogs remained stable over the period in which it was measured. Clearly, PCV can be influenced by a myriad of factors such as dehydration and many disease states. In dogs that were otherwise in good health, a stable PCV could be considered good evidence of a functional shunt and may be more reliable than PaO₂ assessed in awake dogs, which is a measurement that could be easily influenced by stress during sample collection. Although the objective data collection was not standardized, there is reasonable evidence to suggest that the shunts in the dogs of this report remained patent and functioned well for an extended period of time after placement.

The mBT shunt, as reported here, has several advantages over shunting operations that have been previously reported in dogs. First, none of the dogs that survived surgery in the present study developed congestive heart failure as a short-term complication of the procedure. This fact suggests that use of the left subclavian orifice to regulate blood flow protected these dogs against excessive pulmonary overcirculation. In contrast, congestive heart failure was reported in 1 of 4 dogs that underwent a Potts aortocapillary anastomosis. Second, early occlusion of the shunt was not evident in any of the dogs that survived to discharge from the hospital, and evidence of shunt patency lasting years was available for several dogs. In contrast, thrombotic complications associated with the classic BT or Potts procedures have been reported. In the mBT shunt procedure described in this report, a shunt of greater diameter than the native left subclavian artery was used, and this probably helped to reduce the risk of thrombotic complications. In addition, the shunt material (expanded PTFE) and the antiplatelet treatment (aspirin) perhaps minimized the risk of thrombosis as a cause of shunt failure. Suture material and suture technique can also influence the formation of a thrombus at an anastomosis site. In the first 2 procedures, the shunt anastomoses were performed by an experienced cardiac surgeon, but subsequent surgeries were performed by trained veterinary surgeons either alone or with an experienced assistant. As with any surgical technique, results improve as experience with a procedure is gained. The authors conclude that this surgery is within the capability of any trained surgeon with vascular surgical experience. The third advantage of this procedure is that, should complications develop at the left subclavian anastomosis site, the vessel can safely be ligated. In contrast, if technical difficulties such...
as tearing of the aorta occur during the Potts or any of the central aorta–to-pulmonary artery anastomoses, massive hemorrhage can rapidly become life-threatening. Of course, if similar complications develop at the pulmonary artery anastomoses site, hemorrhage can be just as serious a problem. Because these procedures were performed on either adult- or nearly adult-sized dogs, distortion of the pulmonary artery at the site of shunt anastomosis secondary to growth of the animal would seem less likely, although this was not studied specifically; however, there was no echocardiographic evidence that this had developed in any of the dogs. As reported in humans,13,14 the mBT shunt procedure appears to be a superior procedure for palliation of the clinical signs of TOF in dogs.

Complete repair of TOF would be necessary to give an affected dog what would be considered a normal quality of life. Herrtage et al13 reported successful surgical correction of a dog with TOF but that dog died suddenly after 3 years of an apparently normal life. In another report,18 complete repair of TOF in an acyanotic dog was successful, and the dog was free of clinical signs for at least 3 years. Orton et al14 performed surgical correction of TOF in 2 dogs that remained free of clinical signs 2 and 3 years after surgery. It is likely that, as in humans, successful surgical repair will provide better quality of life and improved long-term results than shunt placement in TOF-affected dogs. At present, open heart repair is only available at a limited number of veterinary medical centers around the world, and generally the financial cost to the owner and the relative risk to the patient are both high. Whether open heart repair of TOF in dogs actually does provide a better quality of life and increase life expectancy, compared with other treatments, has not been rigorously investigated. Indeed, because of the small number of dogs that receive surgical treatment for this condition worldwide, it may never be possible to determine that one treatment is superior to another by direct comparison. Extrapolating from data available on treatment of TOF in humans, it is logical for veterinary surgeons to continue to pursue corrective surgery as a goal for affected dogs. Palliative surgery involving the mBT shunt procedure may, however, represent a relatively lower-risk and lower-cost procedure that will improve both quality of life and life expectancy for dogs affected with TOF.

References


