SURGERY FOR CONGENITAL HEART DISEASE

PEDIATRIC TRACHEAL HOMOGRAFT RECONSTRUCTION: A NOVEL APPROACH TO COMPLEX TRACHEAL STENOSES IN CHILDREN

Jeffrey P. Jacobs, MD^{a*}
Martin J. Elliott, MD, FRCS^a
Marcus P. Haw, MD, FRCS^{a**}
C. Martin Bailey, FRCS^a
Claus Herberhold, MD^b
Sponsor:
Marc R. de Leval, MD^a

Purpose: Tracheal stenosis can be a life-threatening problem in children. Long-segment tracheal stenosis and recurrent tracheal stenosis are especially problematic. Tracheal homograft reconstruction represents a novel therapeutic modality for these patients. **Methods:** Cadaveric trachea is harvested, fixed in formalin, washed in thimerosal (Methiolate), and stored in acetone. The stenosed tracheal segment is opened to widely patent segments proximally and distally. The anterior cartilage is excised and the posterior trachealis muscle or tracheal wall remains. A temporary silicone rubber intraluminal stent is placed and absorbable sutures secure the homograft. Regular postoperative bronchoscopic treatment clears granulation tissue. The stent is removed endoscopically after epithelialization over the homograft. Twenty-four children with severe tracheal stenosis (age 5 months to 18 years, mean $\pm$ standard error of the mean = 8.18 ± 1.21 years) underwent tracheal homograft reconstruction. All but one had had previous unsuccessful reconstructive attempts. Ten lesions were congenital, nine were posttraumatic, and five were due to prolonged intubation. **Results:** Follow-up ranged from 5 months to 10 years (3.79 ± 0.70 years). Twenty patients survive ($20/24 = 83\%$), 16 without any airway problems. Four patients are still undergoing treatment. One patient requiring emergency extracorporeal membrane oxygenator support before the operation died 10 days after tracheal homograft reconstruction. Another patient with severe preoperative mediastinal sepsis died 3.5 months after tracheal homograft reconstruction. Two patients with functional airways died late of unrelated problems. **Conclusions:** Tracheal homograft reconstruction demonstrates encouraging short-term to medium-term results for children with severe recurrent tracheal stenosis. Postoperative bronchoscopic and histologic studies provide evidence of epithelialization and support the expectation of good long-term results. (J Thorac Cardiovasc Surg 1996;112:1549-60)

Tracheal stenosis can be a life-threatening problem in children. Certain types of tracheal stenosis are especially problematic^{1,2} including both long-segment congenital tracheal stenosis and recurrent

tracheal stenosis. Long-segment congenital tracheal stenosis, involving more than 50% of the trachea, is often associated with pulmonary vascular sling (25%), intracardiac lesions (20%), and right-sided aortic arch.^{3,4} Long-segment congenital tracheal stenosis often involves complete cartilaginous rings over the length of the stenosis.⁵ Although resection and primary end-to-end anastomosis has been the initial treatment of choice for most shorter stenoses, this treatment is considered difficult in long-segment

From Great Ormond Street Hospital for Children, London, United Kingdom,^a and the University of Bonn, Bonn, Germany.^b

Read at the Seventy-sixth Annual Meeting of The American Association for Thoracic Surgery, San Diego, Calif., April 28-May 1, 1996.

Received for publication May 6, 1996; revisions requested June 3, 1996; revisions received June 25, 1996; accepted for publication July 1, 1996.

Address for reprints: Martin J. Elliott, MD, FRCS, Cardiothoracic Unit, Great Ormond Street Hospital for Children, London, United Kingdom WCIN 3JH.

*Present institution: Miami Children's Hospital, Miami, Fla.

**Present institution: Harefield Hospital, London, United Kingdom.

Copyright © 1996 by Mosby-Year Book, Inc.

0022-5223/96 \$5.00 + 0 12/6/76252

Table I. Patient data

Patient No.	Initial diagnosis	Prior tracheal operation (yes or no)	Age at THR (yr)	Surgical approach	Current status		Length of follow-up (yr)
					Alive or dead	Airway problems	
1	Multiple trauma	Yes	16.61	Cervical	Alive	No	9.96
2	Multiple trauma	Yes	17.53	Cervical	Alive	No	7.47
3	Multiple trauma	Yes	18.31	Cervical	Alive	No	6.76
4	Multiple trauma	Yes	9.52	Cervical	Alive	No	9.11
5	Multiple trauma	Yes	6.36	Cervical	Alive	No	5.64
6	Multiple trauma	Yes	6.73	Cervical	Alive	No	8.16
7	Multiple trauma	Yes	11.53	Cervical	Dead	N/A	N/A
8	Multiple trauma	Yes	3.97	Cervical	Alive	No	5.03
9	LSCTS	Yes	4.13	Cervical	Alive	No	4.76
10	LSCTS	Yes	5.63	Cervical	Alive	No	1.37
11	Neurologic disorder	Yes	11.35	Cervical	Alive	No	4.41
12	Pneumonia	Yes	9.65	Cervical	Alive	No	0.39
13	Status epilepticus	Yes	13.35	Cervical	Alive	No	2.06
14	Toxic shock	Yes	15.50	Cervical	Alive	MNF	2.00
15	Pneumonia	Yes	17.97	Cervical	Alive	MNF	1.59
16	LSCTS	Yes	4.96	Cervical	Alive	No	1.38
17	Multiple trauma	Yes	5.58	Cervical	Alive	MNF	0.43
18	LSCTS	Yes	1.59	Cervical	Alive	No	0.44
19	LSCTS/PVS	Yes	5.10	Sternotomy	Dead	N/A	N/A
20	LSCTS/PVS	Yes	8.24	Sternotomy	Alive	No	1.88
21	LSCTS/PVS/VSD	Yes	1.46	Sternotomy	Alive	No	1.71
22	LSCTS/PVS	Yes	0.44	Sternotomy	Dead	N/A	N/A
23	LSCTS	No	0.42	Sternotomy	Alive	MNF	1.20
24	LSCTS/PVS	Yes	0.46	Sternotomy	Dead	N/A	N/A
No.			24				20
Mean			8.18				3.79
Median			6.55				2.03
Range			0.42-18.31				0.39-9.96
STDEV			5.91				3.12
SEM			1.21				0.70

LSCTS, Long-segment congenital tracheal stenosis; MNF, management not finished; N/A, not applicable; PVS, pulmonary vascular sling; SEM, standard error of the mean; STDEV, standard deviation; THR, tracheal homograft reconstruction; VSD, ventricular septal defect.

congenital tracheal stenosis.⁶ Numerous other treatment options have been proposed for long-segment congenital tracheal stenosis,⁵⁻¹⁶ but none have been uniformly successful.

Recurrent tracheal stenosis is a particularly challenging surgical problem in children. Recurrent scarring, impaired healing, infection, devascularization, and life-threatening anastomotic disruptions all are known complications of cases of recurrent tracheal stenosis.¹⁷ Recurrent tracheal stenosis is similar to long-segment congenital tracheal stenosis in that a variety of both nonsurgical and surgical options may be used, each with different success rates in different subgroups of patients.^{17, 18}

Tracheal homograft reconstruction (THR) with cadaveric human tracheal homograft represents a novel therapeutic option for children with either long-segment congenital tracheal stenosis, recurrent tracheal stenosis, or both. THR was initially

introduced by one of us (C.H.) in 1979 as a treatment for tracheal stenosis in adults.¹⁹⁻²¹ We described recently the application of THR to more distal tracheal and proximal bronchial lesions in children through the use of median sternotomy and cardiopulmonary bypass.²² We now report our total pediatric experience with 24 children undergoing THR as treatment for long-segment congenital tracheal stenosis, recurrent tracheal stenosis, or both.

Patients and methods

Patients. Twenty-four children (aged 5 months to 18 years, mean $\pm$ standard error of the mean [SEM] = 8.18 ± 1.21 years) underwent THR. All had severe life-threatening tracheal stenosis. Twenty-three of these children had undergone previous unsuccessful surgical reconstructive attempts. The remaining patient was referred to us at 2 months of age with tracheal stenosis and had a cardiorespiratory arrest after unsuccessful balloon

Fig. 1. The actual homograft is shown as it appears on arrival from the homograft bank. Also shown are two Dumon stents.

dilatation necessitating the institution of emergency extracorporeal membrane oxygenation (ECMO).

Ten lesions were congenital (long-segment congenital tracheal stenosis), nine were posttraumatic, and five were due to prolonged intubation. Eighteen patients (75%) required neck incisions only. Six children (25%) required sternotomy. Three patients (12.5%) without functional airways required stabilization with preoperative ECMO.

Further details about these 24 children are presented in Table I.

Homograft procurement and preparation. Cadaveric tracheas are removed within 24 hours of death either at multiorgan retrieval or at autopsy from donors aged 16 to 60 years. Contraindications are donors who are positive for hepatitis B or C or human immunodeficiency virus, donors who have died of viral diseases of unknown etiology, and donors who have had thoracic diseases or thoracic trauma involving the trachea.

Organ procurement is performed under clean but not sterile conditions. The trachea is removed circumferentially from the distal end of the larynx to include the first 30 mm of both bronchi. The trachea is then placed in isotonic saline solution for transport to the homograft bank. Tracheal homograft banks now exist in London, United Kingdom, and in Bonn, Germany.*

The trachea is stripped of overlying tissue and the trachealis muscle is removed. The remaining anterior cartilaginous portion of the trachea is immersed for 14 days in 500 ml of 4% formalin in compound sodium lactate solution.¹⁹⁻²² The trachea is then immersed for 56 days in 500 ml of 4 gm/L thimerosal (sodium ethylmercurithiosalicylate, a Methiolate-related compound) dissolved in Dulbecco phosphate-buffered saline solution. It

is then stored in acetone for a minimum of 10 days before its release for clinical use. All processing and final storage is in autoclavable and acetone-resistant Nalgene polypropylene bottles (Nalgene, a subsidiary of Sybron, Rochester, N.Y.). Five samples of the excised trachealis muscle undergo the same processing until the final stage in acetone, at which point they are used for microbiologic testing. They are tested for aerobic bacteria, anaerobic bacteria, fungi, mycobacteria, and hypothermic microorganisms.

Before insertion, the homograft (Fig. 1) is washed thoroughly in saline solution. Histologic studies have confirmed that all cells in the graft die and all major histocompatibility complex markers are lost.^{19, 20, 23}

Surgical technique. Two surgical approaches have been used. Eighteen patients with shorter (less than 50% of the trachea) and higher level tracheal stenosis had THR via a collar incision in the neck, with the use of a limited sternal split if access proved difficult. Median sternotomy was used eight times in six patients for THR for longer (greater than 50% of the trachea), more distal lesions (two children required a second THR: one child in whom the original homograft displayed persistent anterior collapse and one child in whom recurrent stenoses developed 14.5 months after THR). Cardiopulmonary bypass was used in seven of eight cases of THR via median sternotomy. Cardiopulmonary bypass facilitates the dissection for more distal lesions down to and beyond the carina, simplifies the requirements for intraoperative airway maintenance, and is particularly useful in small infants.^{13, 14, 22, 24-29} Three of the six patients treated with median sternotomy also required emergency preoperative ECMO when mechanical ventilation became impossible. Exposure of the trachea via a transverse neck incision is identical to standard approaches. By means of a mixture of sharp and blunt dissection, the trachea is exposed

*Details regarding these tracheal homograft banks may be obtained from the authors.

Fig. 2. The anterior part of the trachea is removed leaving only the posterior tracheal wall and trachealis intact. The homograft is cut to size and sutured into place with interrupted horizontal mattress absorbable monofilament sutures.

beneath the pretracheal fascia. Care is taken to avoid the head vessels and the pulmonary artery during dissection.

Patients undergoing an approach via median sternotomy are positioned supine, and the skin is prepared and draped to expose the neck and chest. Potential incision sites are marked with a surgical marker pen. A conventional median sternotomy is performed and the thymus excised. Before the pericardium is opened, the head vessels are dissected and vascular slings are passed around them to permit retraction. The pretracheal fascia is opened in the midline and the upper (normal) trachea is identified.

The pericardium is then opened longitudinally and stay sutures are applied. The superior vena cava, the aorta, the pulmonary arteries, and the innominate vein are mobilized and vascular slings or tapes are passed around them to facilitate retraction. Heparin is given and purse-string sutures are inserted into the upper left aspect of the aorta and the right atrial appendage. A flexible, wire-wound aortic cannula is preferred to permit safe and repeated repositioning. A right-angled Rygg cannula (Polystan A/S, Vaerlose, Denmark) is used for venous drainage. The bypass is run at a temperature between 32° and 37° C and with a hematocrit value greater than 35%.

The trachea is further exposed between the aorta and the superior vena cava. The incision in the pretracheal fascia is continued down to below the lower limit of the stenosis. A laryngeal release and bilateral hilar releases allow complete tracheal mobilization. Both the left and right main-stem bronchi can be mobilized if necessary. With these maneuvers, it is often possible to treat very-

Fig. 3. The stent is placed to support the homograft internally until reepithelialization has occurred, after which it is removed endoscopically usually 10 to 12 weeks after the operation.

long-segment stenoses, often extending from cricoid cartilage superiorly to below the carina inferiorly.

Once cardiopulmonary bypass has been safely established, the anterior trachea is incised longitudinally in the middle of the stenosis. At this stage, one must make a decision as to the preferred procedure, because it is still possible to perform either a slide tracheoplasty or another conservative operation if preferred. Once one has decided to perform THR, then the anterior incision in the trachea is continued both cephalad and caudad until normal trachea or bronchus is reached. The lateral walls of the narrowed segment are then removed, leaving only the posterior wall in continuity (Fig. 2).

It is often necessary to create a series of longitudinal, partial-thickness slits in the posterior remnant in patients with complete tracheal rings or rigid scar tissue down the posterior wall of the trachea. With cardiopulmonary bypass, electrocautery is preferred for these incisions to minimize hemorrhage. The rationale for these slit incisions is to create partially everted margins in the posterior wall in settings where that component is made of solid cartilage or complete tracheal rings. The everted margins reflect a splayed out posterior wall creating an advantageous morphology to receive the tracheal homograft.

One or two temporary silicone intraluminal stents (Dumon stents, Axiom, Lyon, France) (see Fig. 1) are then placed onto the posterior wall and sutured to the native trachea with 4 to 6 single absorbable monofilament sutures (Fig. 3). These sutures are placed at the upper and

Fig. 4. Bronchoscopic examination 13 months after THR revealing a completely epithelialized homograft with a normal caliber airway from cricoid to carina.

Fig. 5. Electron micrograph shows the appearance of the homograft in the first week after implantation. The absence of epithelium is apparent.

lower ends of the stents to minimize movement of the stents. The stent(s) will provide a good support to the tracheal homograft, which undergoes a period of softening in the early days after insertion. (Because oversized adult tracheal homografts are used even in small children, the Dumon stent should always fit. Our youngest patient was 5 months at the time of THR and received an oversized homograft, allowing easy placement of a Dumon stent with a 5 mm internal luminal diameter to stent an obviously larger airway.)

Before the next stage is begun, the endotracheal tube can be repositioned under direct vision to lie inside the upper orifice of the Dumon stent. The graft is then trimmed to reflect the defect in the anterior trachea before being sutured in place with interrupted horizontal mattress absorbable monofilament sutures. These horizontal mattress sutures join the homograft to the posterior tracheal remnant.

Intraoperative bronchoscopy is performed to confirm graft patency and to allow bronchial toilet. The suture line

Fig. 6. Eight weeks after THR, early epithelialization and budding can be seen on the surface of the underlying matrix.

Fig. 7. Two months after THR, the presence of ciliated respiratory epithelium is clearly demonstrated.

of the homograft anastomosis is sealed and made airtight with Tisseel fibrin glue (Immuno AG, Vienna, Austria). Cardiopulmonary bypass, if used, is withdrawn. Hemostasis and routine closure follow.

Postoperative care. All patients require prolonged stays in the intensive care unit and benefit from a multidisciplinary approach involving cardiac surgeons, intensivists, otolaryngologists, and especially a dedicated nursing staff. Personnel skilled in emergency bronchoscopy must

be readily available for treatment of acute airway problems. Frequent postoperative treatment with a rigid bronchoscope is necessary to clear granulation tissue for several weeks as the homograft undergoes epithelialization. Local temporary stenoses from exuberant granulation tissue may also be treated with balloon dilatation through the rigid bronchoscope. Postoperative antibiotic coverage includes 2 weeks of intravenous therapy and 4 additional weeks of oral trimethoprim. Inhaled steroids

Fig. 8. Patient outcome.* Twenty-three of the 24 patients had had unsuccessful prior tracheal reconstruction.

are used to help decrease granulation tissue. Adrenaline nebulizers also are used.

The intraluminal stent(s) support(s) the homograft for 10 to 12 weeks until the homograft hardens and reepithelialization has occurred. After bronchoscopic examination visually confirms that the granulation tissue no longer exists and that the inner surface of the homograft has undergone reepithelialization, the stent is removed. Endoscopic stent removal is usually not difficult because the absorbable sutures previously holding the stent will have dissolved. The stent can be grasped, rotated medially, folded onto itself, and withdrawn. After discharge from the hospital, short-term bronchoscopic follow-up is necessary. Immunosuppression is not used.

Results

Early mortality occurred in one patient (1/24 = 4.2%) who underwent THR at 5 months of age after 4 days of ECMO support. This child had undergone previous tracheoplasty at 2 months of age for long-segment congenital tracheal stenosis and returned with severe recurrent tracheal stenosis. ECMO was instituted as mechanical ventilation became impossible. Severe intraoperative pulmonary hemorrhage necessitated the continuation of ECMO in the post-operative period and the child died 10 days after THR of a catastrophic intraabdominal hemorrhage.

Late mortality occurred in three patients (3/24 = 12.5%); however, only one patient (1/24 = 4.2%) died late with airway (tracheal) problems. This infant with long-segment congenital tracheal stenosis had a sepsis-related anastomotic dehiscence after primary tracheoplasty and then had an unsuccessful patch revision. Subsequent mediastinitis necessitated ECMO support. This infant later underwent THR after 4 days of ECMO to allow for local control of sepsis. The airway stabilized sufficiently to allow separation from ECMO 3 days after THR, but the child died 3.5 months later of further sepsis and airway failure.

Two other patients died late with functional airways. One victim of multiple trauma died of cardiac failure despite a completely functional airway after THR. A second late death occurred in a child with recurrent long-segment congenital tracheal stenosis who died 18 months after THR of unrelated gastrointestinal problems despite a completely functional airway.

Twenty patients survive (20/24 = 83.3%). Sixteen patients are now free of symptoms and without airway problems. Four are still undergoing treatment. Follow-up has ranged from 5 months to 10

Fig. 9. Actuarial survival (Kaplan-Meier) after tracheal homograft replacement in children. *GI*, Gastrointestinal.

years (mean $\pm$ SEM = 3.79 ± 0.70 years). No patients required tracheostomy after tracheal homograft reconstruction.

The 16 children currently free of airway problems have all demonstrated stable and functional tracheal homografts, with bronchoscopic examination revealing reepithelialization of the lumen (Fig. 4). Histologic studies have confirmed the presence of ciliated respiratory epithelium (Figs. 5 to 7). No patients have had rejection or have required immunosuppression. No patients have had homograft calcification either clinically or radiographically.

Only one patient survives of the three patients who required preoperative ECMO.³⁰ At another institution, this 5-month-old infant had an unsuccessful attempt at balloon dilation for long-segment congenital tracheal stenosis and had a cardiopulmonary arrest after the procedure, requiring the initiation of ECMO. Four days later, THR was performed and ECMO was discontinued. The child was at home, without major airway problems, and growing normally.³⁰ However, tracheal stenosis recurred and was treated with a second THR 14.5 months after the original replacement. This child has since

been extubated. He is recovering well and awaiting stent removal.

The overall patient outcome, analyzed by tracheal disease and surgical approach, is depicted in Fig. 8. Fig. 9 demonstrates actuarial survival (Kaplan-Meier) after THR.

Discussion

Tracheal homograft reconstruction represents a novel therapeutic modality with encouraging short-term to medium-term results for children with severe long-segment congenital tracheal stenosis or recurrent tracheal stenosis in whom conventional management has failed. Numerous techniques have been described for the treatment of long-segment congenital tracheal stenosis and recurrent tracheal stenosis. These techniques include endobronchial stenting,¹⁸ aggressive balloon dilation,^{10, 11} pericardial patch tracheoplasty,^{12, 13} cartilage and rib graft tracheoplasty,^{14, 15} omental pedicle flap tracheobronchial reconstruction,¹⁶ and slide tracheoplasty.^{7, 9} In all series, an important subgroup of patients exists in whom conventional treatment fails. THR allows tracheal reconstruction when less of the patient's own tracheal tissue is available and allows

tracheal reconstruction when conventional treatment may be considered impossible or dangerous.

Other options for tracheal replacement in human beings have not been successful. Synthetic tracheal prostheses have led to an intense inflammatory reaction with airway obstruction, prosthesis migration, and erosion of major blood vessels often leading to fatal hemorrhage.⁸ Tracheal allografts not subjected to chemical preparation and transplanted in animal models are subject to immunologic recognition and rejection.^{31, 32} Allogenic vascularized transplants with the use of immunosuppression have been studied in dogs but have not been successfully applied to human beings.³³ Tracheal homografts, chemically prepared as described in this article, avoid the intense inflammatory reaction caused by synthetic tracheal prostheses and also avoid the need for immunosuppression.

Thimerosal-preserved cartilage had previously been investigated in the laboratory.³⁴ Because of the lack of a suitable alternative, Herberhold began placing chemically preserved tracheal homografts into adults, starting in 1979. The homograft possesses the characteristics of both avascularity and avitality.^{19, 23, 34} The chemical preparation has been shown to destroy the homograft's immunologic antigens.³⁵ Thus immunosuppression is thought not necessary. The chemically treated and preserved homograft acts as a biocompatible implant with no intrinsic cellular viability. It can be thought of as a skeleton, allowing fibroblasts to grow inward between cartilage rings and eventually permitting epithelialization from within the lumen. Immunologic investigation in human beings has demonstrated the absence of evidence of systemic immunoactivation or signs of clinical rejection after THR.²³

The success with THR in adults¹⁹⁻²¹ led to the eventual pediatric application of this technique. All but one of the pediatric cases of THR were in the setting of previous failed tracheal reconstructive attempts. Many of these patients had had several unsuccessful conventional tracheal reconstructive procedures, such as balloon dilatation, tracheal resection and reanastomosis, and pericardial patch tracheoplasty, at a variety of institutions. All were thought to be poor candidates for further standard conventional procedures because of the severity of their tracheal disease, the degree of tracheal thickening, and the large amount of scar tissue caused by previous interventions. Previous tracheal procedures often resulted in poor tracheal blood supply

and decreased tracheal mobility. Because of this lack of mobility and compromised tracheal blood supply, THR was used. The success of THR in the adult population along with the recurrence of stenosis after conventional techniques in these children made THR an appealing option.

The tracheal homograft has numerous advantages including adequate availability, nearly circumferential tissue for reconstruction, and a carina that may be used to permit extensive repair. In addition, the procedure can be repeated if required. Follow-up bronchoscopic and histologic studies have shown clear evidence of luminal epithelialization. Ciliated columnar respiratory epithelium has been shown to cover the lumen of the homograft.¹⁹⁻²⁴ Studies are now underway scientifically to evaluate long-term airway and pulmonary function.

An important possible disadvantage of THR is that the growth potential of the tracheal homograft is not known. Although the remaining autogenous trachea may grow, no evidence exists to suggest that the tracheal homograft itself will grow. However, because an oversized, adult-sized homograft has been used in children, problems related to lack of homograft growth have been avoided. Of course, THR can be repeated if necessary.

The role of THR in the management of tracheal disease is not yet established. THR certainly represents an important additional option for the treatment of severe long-segment congenital tracheal stenosis or recurrent tracheal stenosis. Our current approach for recurrent short-segment upper tracheal stenosis is THR. For recurrent long-segment congenital tracheal stenosis, if nonoperative treatment fails, we attempt to use slide tracheoplasty. If the length of the stenosis is too long or scar formation too severe for this option, we then would use some form of augmentation tracheoplasty, possibly with rib cartilage. We reserve THR for children in whom these other alternatives do not seem suitable. THR was used as primary surgical therapy in only one infant who had required emergency ECMO after unsuccessful balloon tracheoplasty. THR should currently be seen as an addition to the available list of therapeutic options for tracheal stenosis, not as a replacement for them.

Conclusion

THR is a novel therapeutic modality with encouraging short-term to medium-term results for children with severe long-segment congenital tracheal

stenosis or recurrent tracheal stenosis. Postoperative bronchoscopic and histologic studies provide evidence of epithelialization and support the expectation of good long-term results.

Illustrations were drawn by Becan Rickard-Elliott.

REFERENCES

- Cantrell JR, Guild HG. Congenital stenosis of the trachea. *Am J Surg* 1964;108:297-305.
- Hoffer ME, Tom LWC, Wetmore RF, Handler SD, Postic WP. Congenital tracheal stenosis: the otolaryngologist's perspective. *Arch Otolaryngol Head Neck Surg* 1994;120:449-53.
- Berdon WE, Backer DH, Wung JT, Chrispin A, Kozlowski K, deSilva M, et al. Complete cartilage ring tracheal stenosis associated with anomalous left pulmonary artery: the ring-sling complex. *Radiology* 1984;152:57-64.
- Binet JP, Langlois J. Aortic arch abnormalities in children and infants. *J Thorac Cardiovasc Surg* 1977;73:248-52.
- Andrews TM, Cotton RT, Bailey WW, Myer CM. Tracheoplasty for congenital complete tracheal rings. *Arch Otolaryngol Head Neck Surg* 1994;102:1363-9.
- Grillo HC, Zannini P. Management of obstructive tracheal disease in children. *J Pediatr Surg* 1984;19:414-6.
- Tsang V, Murday A, Gillbe C, Goldstraw P. Slide tracheoplasty for congenital funnel-shaped tracheal stenosis. *Ann Thorac Surg* 1989;48:632-5.
- Grillo HC. Tracheal replacement [editorial]. *Ann Thorac Surg* 1990;49:864-5.
- Grillo HC. Slide tracheoplasty for long-segment congenital tracheal stenosis. *Ann Thorac Surg* 1994;58:613-21.
- Hebra A, Powell DD, Smith CD, Othersen HB Jr. Balloon tracheoplasty in children: results of a 15-year experience. *J Pediatr Surg* 1991;26:957-61.
- Messineo A, Forte V, Joseph T, Silver MM, Filler RM. The balloon posterior tracheal split: a technique for managing tracheal stenosis in the premature infant. *J Pediatr Surg* 1992;8:1142-4.
- Idriss FS, DeLeon SY, Ilbawi MN, Gerson CR, Tucker GF, Holinger L. Tracheoplasty with pericardial patch for extensive tracheal stenosis in infants and children. *J Thorac Cardiovasc Surg* 1984;88:527-36.
- Cosentino CM, Backer CL, Idriss FS, Holinger LD, Gerson CR, Mavroudis C. Pericardial patch tracheoplasty for severe tracheal stenosis in children: intermediate results. *J Pediatr Surg* 1991;26:879-85.
- Van Meter CH, Lusk RM, Muntz H, Spray T. Tracheoplasty for congenital long-segment intrathoracic tracheal stenosis. *Am Surg* 1991;57:157-60.
- Willner A, Velez FJ. Rib-muscle flap for the repair of congenital tracheal stenosis. *Am Otol Rhinol Laryngol* 1994;103:601-8.
- Tsugawa C, Nishijima E, Muraji T, Matsumoto Y. The use of omental pedicle flap for tracheobronchial reconstruction in infants and children. *J Pediatr Surg* 1991;26:762-5.
- Weber TR, Connors RH, Tracy TF. Acquired tracheal stenosis in infants and children. *J Thorac Cardiovasc Surg* 1991;102:29-35.
- Bugmann P, Rouge JC, Berner M, Friedli B, LeCoultré C. Use of Gianturco Z stents in the treatment of vascular compression of the tracheobronchial tree in childhood. *Chest* 1994;104:1580-2.
- Herberhold C, Franz B, Bripohl W. Chemisch-konservierte menschliche Trachea als Prothesenmaterial zur Deckung trachealer Defekte. *Laryngol Rhinol* 1980;59:453-7.
- Herberhold C. Verwendung von konservierten homologen Luftrohrentransplantaten in der Chirurgie der Trachealstenose. In: Kastenbauer E, Wilmes E, Mess K, editors. *Das Transplantat in der plastischen Chirurgie*. Rottenburg: Karl Sasse OHG, 1986.
- Herberhold C. Transplantation von Larynx und Trachea beim Menschen. *Verhandlungsbericht der Deutschen Gesellschaft für Hals-Nasen-Ohren-Heilkunde. Europ Arch Oto-Rhinol-Laryngol* 1992;Suppl I:247-55.
- Elliott MJ, Haw MP, Jacobs JP, Bailey CM, Evans JNG, Herberhold C. Successful tracheal replacement in children using cadaveric human tracheal homograft. *Eur J Cardiothorac Surg* 1996;10:707-12.
- Bujia J, Pitzke P, Krombach F, Hammer C, Wilmes E, Herberhold C, et al. Immunological behavior of preserved human tracheal allografts: immunological monitoring of a human tracheal recipient. *Clin Transplant* 1991;5:376-80.
- Jacobs JP, Haw MP, Motbey JA, Bailey M, Herberhold C, Elliott MJ. Successful complete tracheal resection in a three month infant. *Ann Thorac Surg* 1996;61:1824-7.
- Woods FM, Neptune WB, Palatchi A. Resection of the carina and main-stem bronchi with the use of extracorporeal circulation. *N Engl J Med* 1961;264:492-4.
- Hall KD, Friedman M. Extracorporeal oxygenation for induction and maintenance of anesthesia in a patient with an intrathoracic tumor. *Anesthesiology* 1975;42:493-5.
- Hanagiri T, Shirakusa T, Okabayashi K, Ishikura Y, Shigematsu A, Sata T. Resection of tracheal carcinoma using partial cardiopulmonary bypass: report of a case. *Nippon Kyobu Geka Gakkai Zasshi* 1992;40:1285-90.
- Walker LK, Wetzel RC, Haller JA Jr. Extracorporeal membrane oxygenation for perioperative support during congenital tracheal stenosis repair. *Anesth Analg* 1992;75:825-9.
- Riley RH, Raper GD, Newman MAJ. Helium-oxygen and cardiopulmonary bypass standby in anesthesia for tracheal stenosis. *Anesth Intens Care* 1994;22:710-3.
- Goldman AP, Macrae DJ, Tasker RC, Edberg KE, Mellgren G, Herberhold C, et al. Extracorporeal membrane oxygenation (ECMO) as a bridge to definitive tracheal surgery in children. *J Pediatr* 1996;128:386-8.
- Biegel A, Muller-Ruchholtz W. Tracheal transplantation I. The immunogenic effect of rat tracheal transplants. *Arch Otorhinolaryngol* 1984;240:185-92.
- Biegel A, Muller-Ruchholtz W. Tracheal transplantation II. Influence of genetic difference and degree of sensitization on reaction to the tracheal transplant. *Arch Otorhinolaryngol* 1984;240:217-25.
- Khalil-Marzovk JF. Allograft replacement of the trachea: experimental synchronous revascularization of composite thyrotracheal transplant. *J Thorac Cardiovasc Surg* 1993;105:242-6.
- Hagerty RF, Calhoon TB, Lee WH, Cutting JT. Human cartilage grafts stored in methiolate. *Surg Gynecol Obstet* 1960;110:229-33.
- Bujia J, Wilmes E, Hammer C, Kastenbauer E. A comparison of class II antigenicity of human tracheal allografts stored in cialit and methiolate. *Laryngoscope* 1990;100:1337-40.

Discussion

Dr. Thomas L. Spray (*Philadelphia, Pa.*). I would like to congratulate the authors on bringing to our attention another therapeutic modality for patients with complex tracheal problems. Obviously there are several approaches to long-segment tracheal stenosis, including the use of more viable tissues, such as rib cartilage grafts for long-segment tracheal obstruction, as our group reported at this meeting last year.

I would like to ask several questions about this technique. I noted that about two thirds to three quarters of the patients had a cervical approach for the tracheal reconstruction. If you could use a cervical approach, was the length of trachea that was involved short enough to consider some other primary repair option? How many patients did you actually operate on and decide at the time of surgery that you could do a slide tracheoplasty or an additional resection and therefore abandon the use of the homograft?

The second question concerns the size of the homograft that you have used for this procedure. You mentioned that homografts were harvested from patients from 16 to 60 years of age, and yet you implanted them in patients as young as several months of age. Does the size discrepancy of the homograft cause any problem, especially in the lower portion of the trachea? How do you decide which size to use in any individual patient?

I noticed that several of your patients had long-segment tracheal stenosis associated with pulmonary artery sling. One of the problems with pulmonary artery sling and tracheal stenosis is that there is often a tracheal bronchus high in the upper portion of the trachea. How do you deal with that at the time of tracheal homograft implantation and stent implantation? Is there a problem with obstruction of the right upper lobe takeoff from the trachea?

Finally, I noted that all of these patients in your series had surgery for stenosis. Have you used this technique for patients who have localized or long-segment tracheomalacia? It seems that there could be wide application of this technique in tracheomalacia, especially in children.

Dr. Jacobs. Thank you, Dr. Spray, for your questions. First, in our series it is true that the majority of the patients were operated on by means of a cervical approach. Higher recurrent tracheal stenosis often involves a shorter segment. Therefore we can approach the lesion via a cervical approach and are inclined to proceed directly to THR. With the longer stenoses more distally, we more often consider other options, like rib, before proceeding to the THR.

In the past 3 months we have switched to a slide tracheoplasty from our initially intended THR in one patient. Slide tracheoplasty, pericardial tracheoplasty, and rib tracheoplasty are techniques that we do occasionally use. We try to choose whichever technique we believe is appropriate for the given anatomy.

Regarding the size of the homografts, it is true that the homografts that we use come from donors aged 16 to 60 years. We try to insert as large a homograft as is reasonable so that we will have reasonable luminal augmentation. Our homografts do not grow; consequently, we believe it is important to get the largest lumen that we think we can safely fit.

We try to mobilize the tracheal bronchus so that it is actually in the area of the carina and can be attached to the carina of the homograft. In actuality, the last patient who underwent a slide tracheoplasty had a tracheal bronchus. Part of the reason that we chose to use a slide tracheoplasty was because of that given anatomy.

Finally, we have not yet used this technique for tracheomalacia, although that is a very interesting thought. THR may play a role in tracheomalacia in the future.

Dr. Hermes C. Grillo (*Boston, Mass.*). We should be cautious about applying this technique as primary treatment for cases of long-segment stenosis because there are methods that have proven to be increasingly successful, namely, patch tracheoplasties with viable tissue, both pericardium and the more rigid cartilage. Since you are essentially using a piece of tanned tissue, formalin fixed, as a patch, I am not sure what its advantages are over autogenous cartilage, which also becomes epithelialized, or over slide tracheoplasty, which has the value of using the patient's own trachea and epithelium.

Chemically fixed pieces of tissue are replaced by scar ultimately, leaving some residual tracheal widening. Epithelialization has not generally been seen over extensive patch replacements with dead tissue.

A second question is, why do you need to do any release procedures when you do not resect any trachea? You are simply inserting a patch. There seems to be no reason to add the hazards of laryngeal release or of bilateral pleural entry for hilar release. I believe these are unnecessary.

Application of this technique to posttraumatic and postintubation stenoses puzzles me. In posttraumatic strictures, most of the time there has been no loss of trachea, just separation, and these ends can be brought together quite easily, even though they appear to be long-segment stenoses. In postintubation patients, we have found that more than 95% of strictures can be successfully treated by direct resection and reconstruction, even in children, unless the trachea has been damaged by previous unsuccessful therapy. Of course, in circumferential postintubation strictures the damage is full thickness and inserting a gusset in the front is not going to prevent recircutization circumferentially. This is a contribution that needs to be followed, and we will look forward to seeing how it works out.

Dr. Jacobs. Thank you, Dr. Grillo, for your questions and comments. We agree that there are many good options for the treatment of primary tracheal stenosis. Until now, with the exception of one case, we have reserved THR for use in cases of recurrent tracheal stenosis in which previous therapy has failed.

The homograft provides several advantages over a patch of rib or pericardium. First, its shape allows increased luminal augmentation, and its size allows reconstruction over a greater length. Finally, it has a built-in carina. If you need to extend the reconstruction down beyond the carina onto one or both main-stem bronchi, it is somewhat easier because the homograft has a carina of its own.

We use hilar and laryngeal releases to increase tracheal mobilization. Also, the hilar release is used only when we do a sternotomy approach. We use these release procedures because we are initially evaluating the trachea to see

if it is mobile enough after the previous operation to allow us to safely use another reconstructive option, such as a slide tracheoplasty. These release procedures are just part of our attempted technique to evaluate for other treatment options.

Finally, I agree completely that there are many good treatment options for primary posttraumatic and primary

postintubation stenoses, including slide tracheoplasty. In all of the cases of posttraumatic or postintubation stenoses in which we have used THR, it has been in a setting of a recurrence with bad scar tissue.

Thank you, Dr. Grillo and Dr. Spray, for your thoughtful questions and comments. I would also like to thank Martin Elliott for all his support and guidance.