TRACHEAL GROWTH AFTER SLIDE TRACHEOPLASTY

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Objective: Our goal was to investigate the effects of slide tracheoplasty on tracheal growth in newborn piglets. **Methods:** Slide tracheoplasty was performed on normal trachea ($n = 6$) and a model of tracheal stenosis resembling that seen in infants ($n = 6$). After division of the trachea at its midportion between the second cartilaginous ring above and the right upper lobe takeoff below (around 23 rings), the proximal and distal segments were incised vertically on opposite anterior and posterior surfaces and reconstructed together. **Results:** The reconstructed tracheas lengthened and their cross-sectional areas enlarged linearly at a rate of 0.94 cm per month and 1.55 mm²/kg, respectively, as the piglets grew over a 6-month period from 4.7 ± 0.6 to 64.4 ± 5.7 kg ($\pm$ standard deviation). Growth was not different between the two studied groups. There was no narrowing or late restenosis. The mean anastomotic cross-sectional area was overall 1.63 ± 0.28 times larger (range 1.2 to 2.7) than the cross-sectional area of the unreconstructed trachea. When the animals were put to death, all tracheal lumina were completely lined with normal respiratory epithelium and all layers were histologically intact; anastomotic trachealis muscles contracted less ($p < 0.001$) but relaxed similarly to those muscles lining normal tracheas. Tracheal blood supply was macroscopically and microscopically normal in both groups; however, newborn piglets had an almost twofold increased number of intramural capillary vessels as opposed to adult pigs ($p < 0.001$). **Conclusions:** Results suggest that slide tracheoplasty is not limited by the length of stenosis, provides a permanent enlargement of the cross-sectional airway diameter, does not compromise tracheal vascular supply, and does not impair tracheal growth as somatic growth continues. (J Thorac Cardiovasc Surg 1997;113:558-66)

Long congenital tracheal stenosis is life threatening in neonates and infants because it is almost invariably associated with complete tracheal rings and more than half of the cases involve more than

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50% of the entire tracheal length. The management of this malformation has included balloon dilation, segmental resection, and patch tracheoplasty with pericardium, anterior esophageal wall, costal cartilage, or other materials to expand the lumen after a longitudinal incision through the stenotic tracheal area.¹ However, segmental resection is inappropriate inasmuch as the juvenile trachea tolerates anastomotic tension less well than does the adult trachea.² Major disadvantages of patch tracheoplasty include the need for postoperative intubation for stenting and ventilatory support, repeated bronchoscopic treatments, development of granulation tissue, and restenosis.¹

An attractive alternative to patch tracheoplasty is slide tracheoplasty, recently introduced by Tsang and associates³ and Grillo.⁴ It divides the tracheal stenosis at its midpoint and includes a vertical incision on opposite surfaces of the proximal and distal narrowed segments. The two segments are

then slid together with a resulting quadruplication of the luminal cross-sectional diameter. Despite excellent early results,^{3,4} there still is reluctance to perform this technique in infancy and childhood because of the fear that the trachea will fail to grow commensurate with somatic growth.^{1,3-5} To address this question, we evaluated tracheal growth after slide tracheoplasty on normal and stenotic piglet tracheas.

Materials and methods

Animals and experimental design. Large White piglets, 1 to 2 weeks of age, were used for the study. The animals were divided (without randomization) into two groups of six animals each according to the time of performance of the slide tracheoplasty. In group 1, a slide tracheoplasty was made on the normal trachea. In group 2, a stenosis of the trachea was created, simulating the first stage of long congenital tracheal stenosis in infants and, at the onset of respiratory distress, a slide tracheoplasty was performed. All animals received care in compliance with the "Principles of Laboratory Animal Care" formulated by the National Society for Medical Research and the "Guide for the Care and Use of Laboratory Animals" formulated by the National Academy of Sciences (NIH publication No. 85-23, revised 1985).

Operative technique. Animals were premedicated with intramuscular ketamine hydrochloride (50 mg/kg) and anesthetized with intravenous sodium pentobarbital (10 mg/kg). After orotracheal intubation with a No. 3 or 4 polyvinyl chloride tube, anesthesia was maintained with inhaled halothane (Fluothane). All animals' lungs were ventilated (Labaz Inc. Ventilator, Chemin Cami-Saliè, Pau, France) with 100% oxygen at a tidal volume of 250 ml and a rate of 20 breaths/min. Ear venous catheters were placed for infusion of crystalloid solutions. Adequacy of ventilation was determined by means of a pulse oximeter.

Group 1. Through an anterior midline cervical incision, the right sternohyoid muscle was divided before its sternal insertion and the right cervical fat lobe was dissected. Both were then retracted cranially to obtain adequate exposure of the anterolateral surface of the trachea extending from the second cartilaginous ring above to the right upper lobe takeoff below. The intrathoracic trachea was mobilized by opening the pretracheal fascia, and traction sutures were placed to retract it superiorly. Next, the dissected airway was transected at its midportion, the proximal endotracheal tube was pulled back into the subglottic larynx, and one to two tracheal rings were excised (Fig. 1, A). Ventilation was obtained through a sterile cross-field tube inserted into the distal trachea beyond the orifice of the right upper tracheal bronchus. After circumferential mobilization of the dissected tracheal segments, two vertical incisions approximately 2.5 cm long each were then made on the posterior wall of the upper segment first and on the anterior wall of the lower segment next. The right-angled corners produced by the two divisions were then trimmed to create a gentle sloping corner extending from the meeting points of the

Fig. 1. Technique of slide tracheoplasty. **A**, After exposure of the cervical trachea (CT) from the second cartilaginous ring above to the right upper lobe takeoff below, the airway was transected at its midportion and one to two tracheal rings were excised. Two vertical incisions of approximately 2.5 cm each were then made on the posterior wall of the upper segment first and on the anterior wall of the lower segment next. **B**, The right-angled corners of the two divisions were then trimmed and **C**, then reconstructed together. The overall length of the cervical tracheas was 6.04 ± 0.5 cm (approximately four rings per centimeter of length).

vertical and horizontal incisions up to the meeting points of vertical incisions (Fig. 1, B). A stay suture was placed near the proximal flap, and the proximal and distal tracheal ends were then slid together after placement of a continuous 5-0 polydioxanone suture (PDS, Ethicon, Inc., Somerville, N.J.) on the posterior aspects of the two opposite flaps; after cervical flexion, the suture was tied and secured with two sutures, each placed on the entry end points of the continuous suture. Thereafter, several

Table I. Characteristics of piglets having a slide tracheoplasty on normal tracheas (group 1)

Piglet No.	Age (days)	Weight (kg)	Length of trachea*	CSA of trachea (mm ²)	Operation		Length: ST/trachea (%)
					Length of resection (cm)	Length of incisions (cm)	
1	10	6	7	30.1	1	5	85.5
2	7	4.5	6	29.3	1	5	100
3	7	4.5	6	29.3	1	4.5	91.7
4	7	5.5	7	29.3	2	5	100
5	8	5.5	5.5	53.1	1	4	100
6	7	5	6	29.3	1	4.5	91.7
Overall	7.6 ± 1.2	5.1 ± 0.6	6.2 ± 0.6	33.5 ± 9.6	1.1 ± 0.4	4.9 ± 0.4	94.8 ± 6.1

Overall data are expressed as mean ± standard deviation of number of observations. CSA, Cross-sectional area; ST, slide tracheoplasty.

*The length of the trachea extended from the second cartilaginous ring above to the right upper lobe takeoff below.

Table II. Characteristics of piglets having a slide tracheoplasty after creation of a stenosis of the tracheal lumen (group 2)

Piglet No.	Age (days)	Weight (kg)	Days to stenosis	Percent of stenosis	CSA (mm ²)		Length: Stenosis/T (cm)	Length: ST/T* (%)
					Normal	Midstenosis		
1	9	4.5	12	75	29.3	8.7	3.5/6 (58.3%)	91.7
2	5	3.3	7	83	19.3	2.9	3/5.5 (54.5%)	90.9
3	10	5	24	82	41.3	12.3	3.3/6 (55%)	91.7
4	10	5	6	80	31.4	8.2	4/6.5 (61.5%)	92.5
5	7	4	3	75	28.3	6.3	2.5/5 (50%)	90
6	7	4.5	7	82	27.3	6.1	4.5/6 (75%)	100
Overall	8 ± 2	4.3 ± 0.6	9.8 ± 7.1	79.5 ± 3.6	29.4 ± 7.1	7.4 ± 3.1	3.4 ± 0.7/5.8 ± 0.5	92.8 ± 3.6

Overall data are expressed as mean ± standard deviation of number of observations. The percentage of induced tracheal stenosis was evaluated at the time of the second operation by comparing the cross-sectional area (CSA) of the normal trachea to the CSA at midstenosis. ST, Slide tracheoplasty; T, trachea extended from the second cartilaginous ring above to the right upper tracheal lobe below.

*Includes segmental resection of the midstenotic cervical trachea.

individual 5-0 PDS sutures were placed around the entire oblique circumference and through the full thickness of the tracheal wall; knots were tied outside the tracheal lumen (Fig. 1, C). During reconstruction, the cross-field tube was safely and intermittently removed for brief periods of time to permit precise placement of the sutures. The cross-field tube was withdrawn, the original tube advanced to continue ventilation, and the two tracheal ends approximated. Frequently, the distal anterior tongue of the proximal tracheal wall telescoped into the distal trachea. Anastomotic sutures were then tied with the knots outside the lumen. The previously divided right sternohyoid muscle and right cervical fat gland were then interposed between the anastomosis and right carotid artery to avoid arterial erosion.

Group 2. A stenosis of the trachea was first created according to the method described by Cohen and associates.⁶ In brief, after an anterior midline cervical incision, the right sternohyoid muscle was sectioned before its sternal attachment and the underlying right cervical fat gland was resected. The thyroid gland was retracted and the anterolateral aspect of the cervical tracheal was ex-

posed extending from the second cartilaginous ring to the origin of the right upper lobe takeoff by opening the pretracheal fascia. Care was addressed to preserve the tracheal blood supply and the recurrent laryngeal nerves as well. Thereafter, the entire dissected trachea was separated from the underlying esophagus and the small esophageal arteries were coagulated selectively. The isolated trachea was then encircled with a 1 mm thick and approximately 4 cm long (corresponding to 16 tracheal rings) patch of polytetrafluoroethylene (PTFE, IMPRA, Inc., Tempe, Ariz.). So that immediate tracheal constriction could be avoided, the sheet circumference of the patch was left 5 mm greater than the tracheal circumference. The free margins of the PTFE were then sutured to each other with interrupted 4-0 polypropylene sutures (Prolene, Ethicon, Inc.). The wound was then closed with two-layers of 2-0 Vicryl continuous sutures (Ethicon, Inc.). At the onset of severe respiratory distress, the previous cervicotomy was reopened, the PTFE cuff incised and removed, and a slide tracheoplasty performed as described earlier after circumferential excision of the most stenotic segment.

Fig. 2. **A,** Tracheal specimens after removal of a 1 mm thick PTFE patch used to infold the membranous tracheas. **B,** As a consequence of the membranous infolding, all stenotic tracheas had complete tracheal rings with peritracheal fibrosis (arrow) but without acute inflammatory reaction of the tracheal mucosa. This picture is that usually seen in neonates who have noncomplicated long congenital tracheal stenosis with complete tracheal ring. (Toluidine blue and hematoxylin; original magnification $\times 10$.)

In both groups, the cervical incision was closed with two-layers of 2-0 Vicryl suture after single drainage of the cervical region.

Postoperative monitoring. All animals had lateral cervicothoracic x-rays films taken before the operation. Similar chest x-ray films were made immediately after the operation and monthly for the duration of the study. Animals received intravenous antibiotics (cephalothin, 500 mg daily) for the first 10 postoperative days. They were placed in cages and fed standard laboratory pig food and water ad libitum. Postoperative fiberoptic examinations were performed weekly.

Animals were put to death (using intravenous 26% pentobarbital sodium, 0.5 ml/kg) at 6 months of age, and

Fig. 3. The reconstructed trachea lengthens constantly (6.4 ± 1.75 to 11.5 ± 0.74 cm) and linearly ($p < 0.0001$) over a sixfold increase in age in all piglets ($n = 12$). The reconstructed trachea was arbitrarily called the *cervical trachea* and extended from the second cartilaginous ring above to the right upper lobe takeoff below.

the tracheas were removed and examined grossly, histologically, and pharmacologically.

Angiographic studies. So that differences in the vascular distribution could be assessed, the tracheas of newborn piglets (weight 4 to 5 kg) used for other experiments in our laboratory were harvested and their vascular network was evaluated both macroscopically and microscopically through selective angiography of the right subclavian artery according to our previously described technique.^{7,8} The angiograms were compared with those of study pigs having had a slide tracheoplasty and been put to death at the end of the protocol.

Histopathologic and pharmacologic studies. Postmortem specimens were fixed in 10% buffered formalin. After being embedded in paraffin, 5 μ m thick sections were stained with hematoxylin and eosin and assessed histologically in a blind fashion. The tracheal microcirculation was assessed at $\times 40$ objective magnification and defined as the number of blood capillaries lining into the chorion of the tracheal mucosa per 10 high-power fields.

Tracheal rings at the level of the anastomotic and normal areas were obtained when the animals were put to death; they were analyzed according to previously described methods.⁹ Muscular rings were carefully dissected from all other tracheal layers, gently cleaned, and maintained between an isometric force transducer and a fixed wire support in a 10 ml isolated organ chamber containing Krebs solution at 37° C. The Krebs-Henseleit solution in the organ bath consisted in NaCl 118, KCl 4.7, CaCl₂ 1.5, NaHCO₃ 25, MgSO₄ 1.1, KH₂PO₄ 1.2, and glucose 5.6 (in millimoles per liter). The solution was gassed with 95% oxygen and 5% carbon dioxide. The organ chambers were washed every 15 minutes by changing the bathing Krebs solution. Changes in isometric tension were recorded on a chart recorder. During a 90-minute equilibration period,

Fig. 4. There was no differential (p = not significant) growth of the reconstructed trachea between pigs belonging to groups 1 (closed circles) and 2 (open circle), and the relation between tracheal length and body weight was significant in both groups ($p < 0.0001$). The final lengths of the postmortem cervical tracheas were 8.5 ± 2.1 cm and 9.26 ± 1.6 for pigs belonging to groups 1 and 2, respectively ($p > 0.2$). The reconstructed trachea was arbitrarily called the *cervical trachea* and extended from the second ring above to the right upper lobe takeoff below.

resting force was kept at 1.5 gm. At equilibration, a cumulative concentration response curve to carbachol (10^{-8} to 10^{-4} mol/L final concentration) was produced. Isoproterenol was then added cumulatively (10^{-9} to $1.5 \cdot 10^{-5}$ mol/L) to promote muscle relaxation. Changes in force were measured from isometric recordings and expressed in milligrams. The maximal responses to carbachol or isoproterenol (E_{max} values) and the concentration of agonist yielding 50% of maximal response (EC_{50} values) were interpolated from the individual concentration-effect curves. Relaxation was expressed as the percentage of decrease in tension of carbachol-elicited constriction.⁸

Statistical analysis. The parameters used to assess tracheal growth included age (months), body weight (kilograms), length (centimeters), and coronal and sagittal diameter (centimeters) of the normal, midstenotic, and anastomotic trachea. They were evaluated at the time of the operation, each month after the operation (radiologically), and when the animals were put to death. Throughout the study, the length of the cervical or reconstructed trachea refers to the portion of trachea extending from the second cartilaginous ring (cervical portion) to the right upper lobe takeoff (intrathoracic portion). During the operation and on the postmortem specimens, the cross-sectional area (CSA) of the normal and anastomotic tracheas was calculated as follows⁶:

$$CSA (mm^2) = (a/2) \times (b/2) \times \pi$$

where a was the transverse and b the sagittal diameter. Postoperatively, the CSA of the normal trachea was

Fig. 5. The cross-sectional area (CSA) of the anastomotic (closed circles) and normal (open circles) tracheas increased linearly ($p < 0.0001$) at a rate of $1.60 \text{ mm}^2/\text{kg}$ and $1.51 \text{ mm}^2/\text{kg}$, respectively, as body weight increased.

determined from the linear regression equation that has been shown to relate the normal piglet's external tracheal circumference to its internal CSA⁶:

$$CSA \text{ of normal trachea } (mm^2) = -77.8 + 3.79 (2 \pi R)$$

The CSA of the midanastomotic trachea was indirectly calculated as follows:

$$CSA \text{ of the anastomotic trachea } (mm^2)$$

$$= CSA \text{ of normal trachea} \times$$

$$(1 - \text{percent of radiological anastomotic area})$$

Data are expressed as mean $\pm$ standard deviation of number of observations (n). Contraction-relaxation data were analyzed by one-way analysis of variance (repeated measures) with Fisher's protected least significance difference, Scheffé's F test, and Bonferroni's or Dunnett's t test for multiple comparison. Data were analyzed with a software package (STATISTICA, StatSoft France, Paris); p values < 0.05 were accepted as statistically significant.

Results

Operation. All animals survived the operation and had no respiratory distress during the 6 months after the operation. The characteristics of piglets belonging to group 1 are listed in Table I; as shown, they were operated on at the age of 7.6 ± 1.2 days and slide tracheoplasties included $94.8\% \pm 6.1\%$ of tracheal length, which corresponded to a mean of 23.3 ± 0.42 cartilaginous rings. Piglets belonging to group 2 (Table II) became symptomatic after 9.8 ± 7.1 days (Fig. 2), and at operation the tracheal lumina were re-

Fig. 6. Ratio of the cross-sectional area of the anastomotic (CSAA) to normal (CSAN) cervical trachea over the period study. It was not different between the two groups ($p > 0.2$). The CSAA and CSAN enlarged from 51.8 ± 31.8 to $170.6 \pm 25.1 \text{ mm}^2$ and from 15.8 ± 14.8 to $109.5 \pm 18.5 \text{ mm}^2$, respectively. Error bars represent the standard deviation.

duced by $79.5\% \pm 3.6\%$; a silicone T stent was left in place at the completion of the slide tracheoplasty and removed after 6.1 ± 1 days in four of six group 2 piglets. All piglets were extubated in the operating room.

Tracheal growth. The reconstructed tracheas lengthened linearly at a rate of 0.94 cm per month (Fig. 3) as the piglets grew over a 6-month period from 4.7 ± 0.6 to 64.4 ± 5.7 kg. This growth was not different between the two studied groups (Fig. 4). Similarly, the CSA enlarged steadily at a rate of $1.55 \text{ mm}^2/\text{kg}$ (Fig. 5). The mean CSA of the anastomotic trachea was overall 1.63 ± 0.28 times (range 1.2 to 2.7) larger than that of the normal trachea ($p < 0.001$). This ratio was higher ($p = 0.02$) in the first (2.01 ± 0.5) postoperative month but reached a plateau (1.57 ± 0.1) thereafter (Fig. 6). There was no tracheal narrowing or restenosis.

Tracheal vascularization, morphology, and function. Selective angiograms of the right subclavian artery showed that the macroscopic tracheal vascular network was similar in newborn and adult pigs and that the latter had, even after slide tracheoplasty, a typical and intact lateral vessel network supplying the trachea in a segmental fashion when the pigs were put to death. Conversely, the number of capillary vessels lining into the chorion of the tracheal mucosa anteriorly and membranous wall posteriorly was significantly higher in newborn pig-

Fig. 7. Micrograph of the anastomotic (A) and normal (B) trachea taken when the animal was put to death. A, The anastomotic tracheas assumed a more ovoid shape but displayed a normal respiratory epithelium even at the overlapping of the two spatulated tracheal tongues (arrowhead); note the absence of granulation tissue. B, By contrast, the nonanastomotic tracheas were circular. In this specimen, the anastomotic cross-sectional area was 1.88 larger than that of the normal trachea. (Toluidine blue and hematoxylin; original magnification $\times 10$.)

lets than in their adult counterparts (11.3 ± 0.2 vs 5.9 ± 0.9 , $p < 0.001$).

There was no evidence of differential growth in any part of the circumference of the normal and anastomotic tracheas. In some animals, the CSA was not completely circular but assumed a more ovoid shape, especially at the level of the telescoped anastomosis. Histologically, all tracheal lumina were completely lined with normal respiratory epithelium and normal surrounding cartilage (Fig. 7). Trachea-

Fig. 8. A, Contractile response of the anastomotic (closed circles) and neighboring normal (open circles) tracheal smooth muscles to increasing doses of carbachol. The maximal response (E_{max}) and concentration of agonist yielding 50% of maximal response (EC_{50}) of the anastomotic smooth muscles were 5340 ± 600 mg and $7.31 \text{ E-}7 \pm 0.5 \text{ mol/L}$, respectively; by contrast, the E_{max} and EC_{50} of the normal smooth muscles were 8540 ± 880 mg and $9.33 \text{ E-}7 \pm 1.28 \text{ mol/L}$, respectively ($p < 0.001$). B, Relaxation response of the anastomotic (closed circles) and neighboring normal (open circles) tracheal smooth muscles to isoproterenol (precontracted with carbachol). The E_{max} and EC_{50} were, respectively, $85\% \pm 3\%$ and $8.28 \text{ E-}7 \pm 0.85 \text{ mol/L}$ for the anastomotic and $83\% \pm 6\%$ and $8.28 \text{ E-}7.58 \pm 0.2 \text{ mol/L}$ for the normal smooth muscles ($p > 0.2$). Error bars represent the standard deviation.

lis muscles contracted significantly less in the anastomotic as compared with the normal tracheas ($p < 0.001$) but relaxed equally (Fig. 8).

Discussion

Slide tracheoplasty offers several clinical advantages for long congenital tracheal stenosis in small infants: (1) Reconstruction is performed with the patient's native tracheal tissues, so that the postoperative problems of graft materials is avoided; (2) even very elongated stenoses, such as an 80% lesion, require shortening of only half of the length of the stenosis, and this avoids reconstruction under tension; (3) the increase obtained by doubling the circumference appears to be adequate to provide marked and nearly complete symptomatic relief; (4) the operation does not result in ischemic and healing problems. Moreover, slide tracheoplasty can be accomplished in all ages with a cervical incision only and without cardiopulmonary bypass.^{3,4} However, because of its recent introduction in clinical practice and absence of long-term follow-up, the effect of growth on luminal size is yet not known.

To address this issue, we performed slide tracheoplasty on newborn piglets because their tracheal size is similar to that of a human neonate or infant, their tracheas resembles the human tracheas anatomically and functionally,^{10,11} and their growth during the first 6 months parallels the somatic growth of a newborn infant to the age of 18 years,¹² permitting the development of the reconstructed trachea to be fully evaluated. Moreover, the operation was performed on healthy tracheas and on a model of tracheal stenosis that has features closely resembling congenital tracheal stenosis seen in children, that is, posterior fusion of tracheal rings, inflammatory peritracheal reactions, and life-threatening airway obstruction.⁶

The results presented here provide evidence that slide tracheoplasty does not impair or distort tracheal growth, which mimicks the somatic growth of a growing animal model, and this despite involvement of up to 90% to 100% of the investigated trachea. These findings are particularly attractive because they provide experimental support of the hypothesis proposed by Tsang and colleagues³ and Grillo⁴ that even elongated strictures exceeding 60% to 80% of the total tracheal length can be repaired successfully simply because slide tracheoplasty shortens the involved trachea by only half of the original length. This coupled with the fact that we were able to successfully treat stenoses involving

100% of the investigated piglet trachea indicates that the length of the tracheal stricture is not per se a limiting factor for slide tracheoplasty. Encouraged by this experimental experience, we have performed slide tracheoplasty on three neonates whose long congenital tracheal stenosis from complete tracheal rings involved the entire trachea. Subsequent anastomotic growth and clinical outcome were excellent.

The efficacy of slide tracheoplasty is further proven by the absence of healing or narrowing problems, probably related to the slide technique by itself, differences in the tracheal microcirculation, the elasticity of animal and human newborn tissues, and the use of PDS sutures whose experimental¹³ and clinical¹⁴ advantages over alternative suture materials are widely known. Likewise, the *reconstructed* tracheas per se grew linearly and had a normal structure after the piglets' maximal body growth. More interesting, the growth observed in the areas of the overlapped spatulated halves of the anastomotic trachea was similar to that of the normal, neighboring trachea in terms of length and morphologic features. Taken together, these data confirm Burrington's findings¹⁵ that each tracheal ring has a growth constant that is uninterrupted when a linear incision is made. The approximate twofold increase in the mean CSA of the anastomotic segment that we observed is less than the quadrupled cross-sectional lumen observed in human beings after slide tracheoplasty.^{3,4} Species- or age-dependent variables may explain this difference. However, the piglets having a stenosis created before the slide tracheoplasty procedure demonstrated that slide tracheoplasty, like other types of tracheoplasty procedures, sufficiently enlarges the cross-sectional diameter to provide complete and permanent symptomatic relief.

The impact of slide tracheoplasty on the tracheal blood supply has not been previously studied. Whereas Tsang and colleagues³ had no ischemic problems after circumferential mobilization, Grillo⁴ was very concerned about ischemia and modified the original technique by incising the upper segment of the trachea posteriorly and the lower portion anteriorly and leaving the distal stenotic segment in situ without disturbing its blood supply. In our study, because of the length of the reconstructed trachea and to ensure a tension-free anastomosis, we minimized tracheal dissection but were obligated to perform a circumferential mobilization of more than 90% of the investigated trachea. In considering hypotheses to

explain why we had virtually no suture line dehiscence or ischemic problems in either group, one must examine the angiographic observations. In a broader context, there were no differences in the tracheal macrocirculation between the newborn and adult tracheas, as observable in human beings.^{7,16} Microscopically, however, there was an almost twofold increase in number of capillary vessels in the newborn tracheas as compared with their adult counterparts. That this more developed intramural microcirculation may nourish the reconstructed trachea, even after very elongated and circumferential dissection, may not be a utopian idea. These observations also serve to undermine traditional concerns about the role of the tracheal blood supply and tracheal surgery in children and provide an experimental rationale as to why Jonas¹⁴ and others¹ reported no healing problems even after resection of almost 50% of the trachea in children.

Histologically, all tracheas were intact when the animals were put to death, and their lumina were epithelialized with normal respiratory epithelium even over the reconstructed surfaces. The contractile activity of the anastomotic tracheal smooth muscle was reduced when compared with the tracheal muscle lining the normal trachea, reflecting perhaps the negative influence of postanastomotic scar tissue. However, it still was above its physiologic threshold⁹ and the relaxation capacity was not impaired. The approximate twofold increase in mean CSA of the reconstructed trachea halves indicates that tracheal growth is much greater at this level than on the normal trachea, probably because of tracheal cartilage growth induced by the longitudinal incisions; this is in accord with previous experimental observations with periosteal tracheoplasty.⁶ Burrington¹⁵ first demonstrated that cartilage growth occurs on the convex surface of the cartilage and the tip while continual remodeling progresses on the concave surface.

In conclusion, the chief findings of the present study are that slide tracheoplasty is not limited by the length of stenosis, provides a satisfactory and permanent enlargement of the cross-sectional airway diameter, and does not impair or distort the anatomic and functional growth of the trachea, which ultimately parallels somatic growth. Given these long-term results and its clinical attractiveness,^{3,4} slide tracheoplasty should be considered as an efficient tracheoplasty technique for neonates or infants with long congenital tracheal stenosis.

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