A Silastic Prosthesis for Laryngeal Stenosis

B. KOTTON, P. KALISH

SUMMARY

A Silastic prosthesis anchored to a tracheostomy tube was used in the treatment of 5 children with subglottic stenosis. It did not cause any significant tissue reaction and obviated the problems of transfixion sutures. We treated 3 children successfully. Another died from complications owing to the tracheostomy. In the fifth patient the prosthesis was replaced by a Silastic T-tube. The use of the Silastic prosthesis is advocated for tough, dilatable laryngeal strictures, for which a T-tube is unsuitable.


Laryngeal stenosis can present with recurrent lower respiratory tract infections, hoarseness, stridor or decreased effort tolerance. Asphyxia and death may follow. One of the methods of treatment is to perform a tracheostomy, after which the stricture is dilated and an obturator is inserted for 6 to 9 months. Since Schmieglow described them in 1929, obturators have been constructed of different materials and various methods of fixation have been used to overcome tissue reaction and infection. Following the initial work of Kerr, we have developed a Silastic obturator anchored to a tracheostomy tube by means of a Vitalium wire loop. In this paper we wish to describe the construction and use of this prosthesis.

THE PROSTHESIS

The materials required to make the prosthesis are Silastic tubing of the appropriate diameter, Silastic adhesive type A, and Rocky Mountain (Vitallium) wire. Two prostheses are prepared. One is the correct diameter for the patient's age and one is smaller. The total length of the prosthesis is determined as follows: the distance, from the top of the tracheostomy stoma to the base of the epiglottis is measured on a 1:1 lateral X-ray film of the neck. To this is added the diameter of the tracheostomy tube plus 1 cm (Fig. 1a). Firstly, the wire skeleton is prepared by looping a piece of Vitalium wire at both ends. The lower loop is made first, its diameter being a little larger than that of the tracheostomy tube to be used (Fig. 1b). This loop is then slightly compressed from side to side so that it will just admit the tracheostomy tube (Fig. 1c). The small upper loop is made so that the length from the bottom of the lower loop to the top of the upper loop is the required length of the prosthesis (Fig. 1c). The wire skeleton is then inserted into a piece of Silastic tubing which is bevelled in a coronal plane at its lower end. The tubing is trimmed to allow the upper wire loop to fit flush with the top of the tube, while the straight wire passes through the centre of the bevelled area below (Fig. 1d). The tubing is filled with adhesive. The glottic and supraglottic portion of the prosthesis is clamped to form an anteroposterior keel (Fig. 1 e-f). The prosthesis is trimmed and rounded when
the adhesive has cured (Fig. 1 g - i). It is essential that the upper wire loop should not be exposed. The prosthesis is then ready for sterilisation (Fig. 2).

**INSERTION TECHNIQUE AND INDICATIONS**

The stricture is dilated with bronchoscopes. A silk thread is tied in a large loop to the lower loop of the prosthesis. This wire loop is partially compressed to allow it to pass through the stricture. By means of an up tilted laryngeal forceps, the free end of the thread is passed down a laryngoscope to the assistant at the tracheostomy stoma. He pulls it out through the stoma, using a Negus pusher as a pulley to protect the tracheal wall. The prosthesis is inserted through a laryngoscope by means of a straight artery forceps clamped to the keel, while the assistant exerts tension on the silk thread.

The assistant opens the wire loop with an artery forceps and passes a silver Negus tracheostomy tube through it. The silk thread is cut after the surgeon has ensured that the tracheostomy tube is through the loop.

The assistant requires routine postoperative tracheostomy care and the inner tube must be changed regularly. The outer tube may be changed if necessary. Towards the end of the treatment period the prosthesis becomes covered by a sticky, malodorous deposit. This is obviated by changing the prosthesis after 4 to 5 months. After 6 to 9 months the prosthesis is removed under general anaesthesia and the tracheostomy tube is spigotted. Postoperative physiotherapy may be necessary to re-establish the patient's normal cough reflex. Speech therapy is started at this stage. Six weeks later, before the removal of the tracheostomy tube, the patient is re-examined under anaesthesia to ensure that there has been no recurrence of the stricture. After another 6-12 months a final examination is carried out under anaesthesia. We have used this prosthesis in patients with tough dilatable strictures, for whom intralesional administration of steroids and dilatation were unsuitable. It can also be used as a laryngeal stent after laryngofissure.

**Fig. 1. Stages in making the prosthesis. Figures a, d, f - h are lateral views and b, c, e, i are frontal views.**

**Fig. 2. Front and side view of the prosthesis.**
CASE REPORTS

Patient 1

A 3-year-old boy had sustained a head injury and hemiplegia at 21 months of age. He was intubated endotracheally for 17 days. Soon after extubation he developed hoarseness, stridor and poor effort tolerance. On his admission to hospital an annular stricture 3 mm in diameter was found about 8 mm below the vocal cords. This necessitated a tracheostomy. The stricture was dilated to a diameter corresponding to that of a bronchoscope used for infants and a prosthesis with a diameter of 6 mm was inserted. Within a week the stricture was dilated to a size corresponding to that of a bronchoscope used for adolescents. A 9-mm prosthesis, narrowed in the glottic region, was inserted and left in place for 7½ months (Fig. 3). After removal of the prosthesis there was slight oedema at the site of the stricture and the mucosa of the vocal cords was thickened. A laryngoscopy 1 year later showed the stenotic segment to be a normally lined mucosal tube which comfortably admitted a bronchoscope with an outer diameter of 8 mm.

Patient 2

A 4-year-old girl had had a tracheostomy and right thoracotomy for a respiratory infection at another hospital 8 months before admission to our unit. She was extubated after 2 weeks. Six weeks later she developed stridor. Eight months later granulation tissue was removed from the trachea, but resulted in little improvement. On investigation we found an anterior, crescentic stricture of 4.5 mm immediately below the vocal cords. The mid-portion felt cartilaginous. A tracheostomy was performed and the stricture was dilated to a diameter corresponding to that of a bronchoscope used for adolescents. A 9-mm prosthesis with a supraglottic keel was inserted, and was removed after 8 months. The anterior wedge was still present but allowed the easy passage of a bronchoscope used for adolescents. The mucosa of the anterior two-thirds of the cords was oedematous. The rest of the supraglottic mucosa was normal. The lumen was unchanged at laryngoscopy 12 weeks after the removal of the prosthesis. The oedema of the vocal cords had subsided.

Patient 3

A boy was born prematurely (at 30 weeks) with a ductus arteriosus. He had several apnoeic attacks and congestive cardiac failure. He was intubated and connected to a respirator for an unknown period of time. When he was 1 month old, the ductus arteriosus was ligated. One week later he developed inspiratory stridor and had several cyanotic spells, which necessitated a tracheostomy. When he was 14 weeks old, a laryngoscopy showed an anterior, crescentic stricture of 2 mm immediately below the vocal cords, with a granuloma on the right arytenoid cartilage and tracheal granulations at the tip of the tracheostomy tube. The stricture was dilated with bronchoscopes and a prosthesis 6 mm in diameter was inserted. When the prosthesis was removed 7 months later, there was no sign of a stricture. The vocal cords were thickened but there was no posterior defect. A bronchoscope with an outer diameter of 6 mm passed comfortably through the larynx and trachea. When the patient was extubated 7 weeks later, there was still no sign of a stricture. The middle third of the vocal cords was oedematous. The child's airway has remained satisfactory for the past 7 months.

Patient 4

A girl, born prematurely at 33 weeks, suffered from apnoeic attacks and bradycardia. At the age of 17 days she was intubated and connected to a respirator for 1 week. On extubation she developed stridor and was re-intubated a week later for another 2 weeks. Since extubation failed again, a tracheostomy had to be done. Three months later she was found to have an almost complete, subglottic, crescentic stenosis with a hard wedge-shaped area just below the anterior commissure. The stricture was dilated with bronchoscopes. A prosthesis, 6 mm in diameter, with a supraglottic keel, was inserted. During insertion the upper wire loop was exposed, and this exposure caused ulceration of the epiglottis. The
prosthesis was changed after 1 month. The child was retarded, failed to thrive, had plagiocephaly and recurrent episodes of pneumonia. She was fed via a nasogastric tube. Unfortunately, after 14 weeks of treatment, she died from a blocked tracheostomy tube. At autopsy the trachea and oesophagus were found to be normal and there was no evidence of a stricture. The ulceration of the epiglottis had healed.

**Patient 5**

An 8-year-old boy who had had a surgically corrected craniostenosis sustained a head injury. He was intubated with a Jackson-Rees tube for 16 days, after which a tracheostomy was performed. After 5 weeks he was extubated and 3 weeks later he became hoarse and breathless and required a second tracheostomy. Examination revealed a 4-mm stricture at the tracheo-oesophageal junction. This was dilated to correspond in diameter to a bronchoscope used for adolescents. A prosthesis with a diameter of 10 mm was inserted. The prosthesis was too long, but did not cause any symptoms. When it was removed 4 months later, there were granulations over the false cords and on the posterior pharyngeal wall where the top of the prosthesis had rubbed it. The site of the stricture was ulcerated. There had been no dysphagia. Six weeks later the child was readmitted with a severe recurrence of the stricture. The site of his lesion was considered suitable for the insertion of a Silastic T-tube. Initially, a prosthesis had to be reinserted, because of the tightness of the stricture, A tracheostomy tube smaller than that previously used was inserted. After 5 days the prosthesis slipped down alongside the tracheostomy tube, and therefore had to be removed. A Silastic T-tube 10 mm in diameter was then successfully inserted and has been in place for 8 months. Another 2 infants who have subglottic stenosis as a result of intubation are at present undergoing this method of treatment.

**DISCUSSION**

The advantages of our prosthesis are that it is made out of Silastic, that it obviates the need for transfixion sutures, that it is introduced endoscopically, and that the inner tracheostomy tube can be changed and cleaned frequently without disturbing the prosthesis.

Since Silastic is chemically inert, it is not physically modified by soft tissue and does not react biologically with tissues. It is capable of resisting mechanical strains, can be shaped into any form and can be sterilised.

Transfixion sutures tend to break, introduce infection and produce scars while an endoscopic procedure is relatively short and does not endanger laryngeal development. We have not encountered the problem of leakage of food and saliva into the larynx and trachea.

The hazards encountered in the use of our prosthesis are those of a tracheostomy and these were responsible for the death of patient 4.

The problems which we encountered in our series of patients were dysphagia; nursing mistakes (leading to expulsion or slipping of the prosthesis); infection; head extension; communication and resocialisation difficulties and restenosis.

Dysphagia occurred in all except patient 5, the oldest. It was caused by the presence of the prosthesis and by operative trauma and lasted 2-3 days, during which time the patient was given a soft diet. Compression of the oesophagus by the prosthesis was shown on cine studies in patients 3 and 4. Patient 3 had to be fed via a nasogastric tube for 1 month. The dysphagia in patient 4 was further aggravated by neurological abnormalities which followed anoxia and the temporary ulceration of the epiglottis owing to an exposed upper wire loop. This necessitated feeding via a nasogastric tube throughout the treatment period.

The nursing staff should be warned never to change the outer tube. The surgeon knows how to find the wire loop and how to insert the tube through it. If the loop should be missed in this procedure, expulsion of the prosthesis will inevitably follow, which could be dangerous. We no longer change the outer tube.

In patient 5, after restenosis, the smallest available tracheostomy tube was used. This allowed the prosthesis to slip down alongside the tracheostomy tube.

This slipping was possibly aggravated by the excessively large lower loop of the prosthesis. The largest tracheostomy tube available should be used to avoid this problem.

For a time, patients 3 and 4 tended to lie with their heads extended. In patient 3 this tendency persisted for 5 months. According to Pracy, this is the favourite position of young infants with tracheostomies. In both our patients there was a past history of gross anoxia which could have accounted for this posture. No treatment was required. The children learnt to communicate by means of buccal speech and gestures. After extubation they passed through the following transition stages: (i) buccal speech, (ii) speech with breathy phonation, speaking mainly on inspiration, and (iii) true phonation with a breathy voice and normal breathing pattern.

Varying degrees of hoarseness and a vocal pitch lower than normal remained. Speech therapy was helpful. Patients 1 and 2 had to overcome their fears of the outside world as they stayed in hospital for prolonged periods. Resocialisation was started once the prosthesis had been removed. From the nursing and social point of view, home nursing is preferable if adequate tracheostomy care is available. This was clearly shown in patient 3, who would not eat and failed to thrive. When he went home he immediately started eating well and putting on weight. Restenosis is inevitable in some cases, regardless of the form of treatment used. This restenosis is more likely to occur with obturators if the recommended 6- to 9-month period of dilatation is not adhered to, as in the case of patient 5.

Laryngeal stenoses are produced by congenital, traumatic and inflammatory or neoplastic causes. Many laryngeal injuries in children are iatrogenic and there have been numerous reports of laryngeal stenosis after prolonged intubation. Four of our patients had definite histories of prolonged intubation and 1 patient (patient 2) must have been intubated for the duration of the thoracotomy at least, if not for longer. The pathophysiological factors
involved in the production of these strictures in children are the narrow larynx, loose mucosa, subglottic respiratory epithelium and surrounding cartilaginous ring. These are aggravated by the size of the tube, the presence of infection, the pneumatic piston effect of the respirators, the length of intubation period, re-intubation and systemic factors. Endotracheal intubation is often necessary, despite the complications which may result. Prophylactic measures consist of a short intubation period, a small tube size, reduced head movements, low cuff pressures with the cuff inserted distal to the cricoid cartilage, frequent aspiration of secretions and support of the patient's general condition. A tracheostomy, if necessary, should be performed within 2 - 7 days. In the debilitated child, it should be done after 48 hours. In our opinion, it should be performed on all children with upper respiratory tract infections who require endotracheal intubation for a period longer than 48 hours. Careful follow-up for possible late complications is necessary.

Three of our patients (2, 3 and 4) may have had an additional mild congenital stenosis, since there was a wedge-shaped cartilaginous abnormality below the anterior commissure. Patients 3 and 4 were born prematurely and patient 3 also had a ductus arteriosus.

According to Holinger and Brown, the main obstruction in congenital subglottic stenosis is usually 2 - 3 mm below the vocal cords and must be differentiated from a cartilaginous abnormality of the cricoid. These congenital stenoses may be brought to light by a superimposed infection or by intubation, as in the above cases. Granulomatous laryngitis may cause a stenosis in the absence of a congenital abnormality or without intubation.

Subglottic stenosis may be managed by tracheostomy alone, by dilatation, which may be combined with intraleisional or systemic administration of steroids, by dilatation with insertion of a solid obturator or a T-tube or by resections and plastic repairs. In mild cases a tracheostomy alone can result in growth overcoming the problem. Fearon and Cotton showed that 24% of their patients died from tracheostomy problems within 6 months (as did patient 4) and another 20% could still not be extubated. Recently there have been reports advocating laryngeal repairs in infants despite the warning of Bryce et al. and Conley that there could be possible adverse effects on laryngeal growth.

We advocate the intraleisional administration of steroids and dilatation in soft dilatable strictures and reserve the insertion of a prosthesis for very tough dilatable strictures or for patients who have failed to respond to the former method and are unsuited to the insertion of a T-tube.

The T-tube is preferred when the stricture is far enough below the cords to allow the upper limb of the tube to pass adequately through the stricture and still remain below the cords. The hollow tube then has the advantage of allowing normal respiration and speech.

In 3 of the 5 patients reported here, our prosthesis was used successfully. The fourth patient was progressing satisfactorily (as shown at autopsy) when she died from a tracheostomy problem. After the failure of our prosthesis in the fifth patient a Silastic T-tube was used, since the site of the stricture was favourable for this method of treatment. This T-tube is likely to fail if the recurrence of the stricture was caused by factors other than premature removal of the Silastic prosthesis. At present we are treating another 2 patients with the prosthesis. Resections and plastic procedures are reserved for patients in whom the prosthesis is unsuitable, or for patients in whom the stricture cannot be corrected by the prosthesis.

REFERENCES