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Laryngotracheal obstruction complicating tracheostomy or endotracheal intubation with assisted respiration

A critical review

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A classification of airway obstruction after tracheostomy with assisted ventilation is proposed and is illustrated in Fig. 1:

1. Suprastomal obstruction
   - A. Cricoid (subglottic) stenosis: due to perichondritis and scarring
   - B. Infracricoid stenosis: due to backward displacement of the anterior tracheal wall above the stoma
     - (i) operatively, or
     - (ii) postoperatively.
2. Stomal obstruction
   - A. Fibrous stomal stenosis: due to cartilage loss from
     - (i) excessive removal of cartilage at operation, or
     - (ii) destruction of cartilage after operation by pressure necrosis and infection
   - B. Tracheomalacic stomal stenosis: due to cartilage loss from
     - (i) excessive removal of cartilage at operation, or
     - (ii) destruction of cartilage after operation by pressure necrosis and infection
   - C. Granulomatous stomal stenosis: due to granulation tissue polyp attached (usually) to upper margin of stoma
   - D. Stomal stenosis from double-barrelled trachea: due to forward angulation of trachea
   - E. Stomal stenosis: due to retropulsion of a Björk flap.
3. Infrastomal obstruction
   - A. Cuff stenosis: due to pressure necrosis by inflated cuff
     - (i) Fibrous
     - (ii) Tracheomalacic
   - B. Tube-tip stenosis: due to pressure necrosis by end of tracheostomy tube; possibly also due to damage by tracheobronchial aspiration.
4. Mixed Obstructions, e.g., obstructions at more than one site.

The term 'cricoid stenosis' is used in preference to subglottic stenosis because it fits in better with the terminology used in this classification, while providing an accurate description of the site of the obstruction within the ring of the cricoid cartilage. It is the condition referred to by Jackson (1921) as chronic laryngeal stenosis. Infracricoid stenosis affects the area of the trachea above the level of the stoma made to insert the tracheostomy tube into the trachea. Stomal steno-

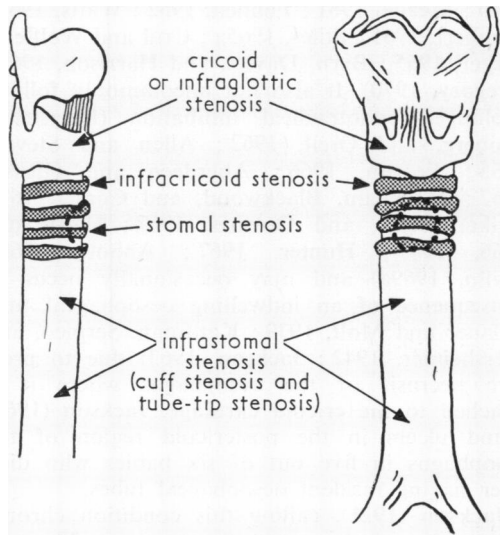

FIG. 1. *Diagram indicating the various sites of post-tracheostomy laryngotracheal stenosis.*

ses are located at the site of the stoma, while infrastomal stenoses lie below the stoma.

All the above varieties of stenosis can occur following the insertion of a non-cuffed tracheostomy tube, except for cuff stenosis, a very important variety. A cuffed tube, however, increases the incidence of stomal stenosis for reasons which will be discussed later.

There is considerable confusion with regard to terminology and the sites of stenoses in the literature. Thus, under the heading of subglottic stenosis after nasal endotracheal intubation, Taylor, Nightingale, and Simpson (1966a) discussed the case of a boy of 5 years in whom, they stated, the stenosis was in the trachea above the level of the stoma. In my terminology this was presumably an infracricoid stenosis.

SUPRASTOMAL OBSTRUCTION

CRICOID (SUBGLOTTIC) STENOSIS

PATHOGENESIS AND PATHOLOGY Cricoid stenosis, due to perichondritis of the cricoid cartilage, with prolonged inflammation and subsequent cicatricial contracture, ending in a stricture within the ring of the cricoid cartilage, has been known for many years to follow laryngostomy (Jackson, 1921 ; Abbey, 1960) or high tracheostomy, with resultant injury to the first tracheal ring and cricoid cartilage (Jackson, 1921 ; Jackson and Jackson, 1950, 1954 ; Jackson, 1963 ; Toremalm, 1960 ; Meade, 1961 ; Fennell, 1962 ; Watts, 1963 ; Lambert, 1965 ; Stiles, 1965a ; Crul and Wolffensperger, 1965 ; Byrn, Davies, and Harrison, 1967 ; Gregory, 1970). It also not uncommonly follows prolonged endotracheal intubation (Bergström, Moberg, and Orell, 1962 ; Allen and Steven, 1965 ; Fearon, 1966 ; Aberdeen and Glover, 1967 ; Markham, Blackwood, and Conn, 1967 ; Striker, Stool, and Downes, 1967 ; Hardcastle, 1966, 1967 ; Hunter, 1967 ; Abbott, 1968 ; Grillo, 1969a) and may occasionally occur in consequence of an indwelling oesophageal tube (Iglauer and Molt, 1939 ; Kaufman, Serpico, and Mersheimer, 1942 ; Jackson, 1963) due to pressure necrosis of the oesophagus where it is attached to the cricoid cartilage. Jackson (1963) found ulcers in the postcricoid region of the oesophagus in five out of six babies who died after having resident oesophageal tubes.

Jackson (1921), calling this condition chronic laryngeal stenosis, stated that in over 30 years of experience he had never been without cases of the condition, referred to him by tracheotomists who were unable to decannulate their patients, yet he had never had a case following tracheostomy on his own service. Of 200 cases of chronic laryngeal stenosis following tracheostomy or laryngostomy seen by him, 30 were caused by trauma or inflammatory disease (diphtheria, lues, typhoid fever or pneumonia) for which the operation was performed. These conditions gave rise to perichondritis and cartilage necrosis, leading to collapse of the cartilagenous framework of the larynx. Of the remaining 170 cases, no less than 158 (93%) were considered to have been caused by either high tracheostomy (126 cases) or laryngostomy (32 cases). Previously 85% of laryngotracheal stenoses were caused by division of the cricoid cartilage (Jackson and Jackson, 1954 ; Meade, 1961). In performing tracheostomy the first tracheal ring and cricoid cartilage must never be damaged. It must be remembered that these structures may be injured not only at the time of operation but also subsequently by the pressure of an ill-fitting tracheostomy tube inserted in a high stoma (Watts, 1963).

This very serious condition used to complicate tracheostomy fairly commonly but now rarely does so since the abandonment of the high operation as the result of the work of Jackson (1921). Today, however, cricoid stenosis occurs with some frequency as the result of the introduction of prolonged endotracheal intubation of infants and children. Thus Fearon (1966) reported an incidence of 8% in 72 children intubated for more than eight hours, while Markham *et al.* (1967) reported three cases among 156 infants and children, of whom 74 were long-term survivors. Striker *et al.* (1967) reported on 116 infants and children treated by prolonged nasotracheal intubation. Among 98 who suffered from respiratory insufficiency, without upper airway disease, three developed cricoid stenosis ; while among 18 who had upper airway obstruction, mostly laryngotracheobronchitis, no less than four did so. Abbott (1968), examining 26 of 30 children who survived prolonged nasotracheal intubation, found severe cricoid stenosis in two, and slight (asymptomatic) stenosis in four others ; this author gives a full bibliography. Hardcastle (1966, 1967) reported a case of almost complete cricoid stenosis accompanied by adherent vocal cords, and Allen and Steven (1965) reported one case among 61 children.

Cricoid stenosis is particularly common in infants and children. Their subglottic tissues are unusually intolerant of contact with foreign material (Jackson, 1921 ; Crul and Wolffens-

perger, 1965 ; Byrn *et al.*, 1967) and appear to be different in nature from those of the trachea (Jackson, 1921). In addition, the subglottic cricoid region is very narrow and unyielding. The larynx, including the subglottic region, is the narrowest portion of the laryngotracheal airway and has a cross-sectional area less than half that of the trachea (Jackson, 1921). In infants, subglottic measurements are even more significant than those of the rima glottidis. Tucker (1932) found the anteroposterior diameter of the infantile larynx at the level of the glottis to vary between 7 and 9 mm, whereas that of the subglottic space was only between 6 and 8 mm. He believed that a subglottic diameter of 5 mm was too small, and that one of 4 mm represented actual stenosis. Very little reduction in size, therefore, may cause serious obstruction. Furthermore, the subglottic space, being completely surrounded by the rigid ring of the cricoid cartilage, can afford no room for soft tissue expansion except at the expense of the airway. The subglottic tissues are quick to react to injury of any kind by swelling and scarring (Reading, 1958), and obstruction may rapidly become severe or complete.

The pathological changes produced in the larynx by resident endotracheal tubes, which may lead to cricoid stenosis, are well described by Bergström (1962) and Bergström *et al.* (1962) in a necropsy study of eight cases. They found that after about 12 hours of intubation diffuse redness and swelling, accompanied often by a mucopurulent membrane, developed, extending from the arytenoid region down to the vocal cords, that is as far distally as reliable inspection was possible. Ulceration was present after 16½ hours, and after 30 hours deep ulceration involved the underlying laryngeal cartilages. The ulcers were found especially over the arytenoid cartilages, the posterior parts of the vocal cords, and the dorsolateral part of the subglottic space (cricoid region). The epiglottis was also affected. The ulcers were principally oval or elongated in the long axis of the larynx. My colleague, Dr. R. M. E. Seal, has observed two necropsy specimens which exhibited almost identical features, characterized by paired symmetrical ulcers situated one above the other in the region just below the posterior ends of the vocal cords. The upper ulcers were located over the arytenoid cartilages, the lower ones over the lateral portion of the lamina of the cricoid cartilage just above its central point. The small area of normal mucosa between the ulcers on each side overlay the cricoarytenoid joint. One of the cases is illustrated in Fig. 2 ; microscopy of the ulcers showed loss of epithelium and necrotic changes but only minimal accompanying inflammatory changes (Fig. 3). The second case is illustrated in Fig. 14 and shows the scar of a single healed ulcer below the posterior end of each vocal cord. The appearances are consistent with the findings of Bergström (1962) and Bergström *et al.* (1962).

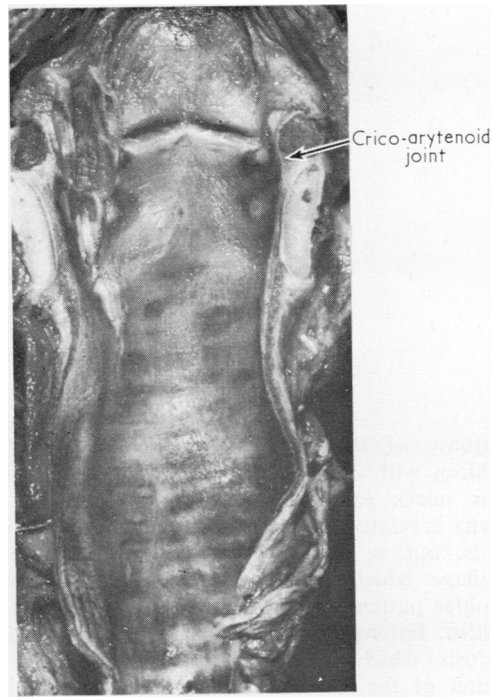

FIG. 2. *Larynx and trachea from a man aged* 46 *years. He died of cardiac arrest* 15 *hours after intubation with a cuffed endotracheal tube for treatment, by assisted ventilation, of irreversible heart failure due to mitral valve disease. The specimen shows bilaterally symmetrical paired ulcers below the posterior ends of the vocal cords. The upper ulcers lie over the arytenoid cartilages, the lower over the lateral parts of the lamina of the cricoid cartilage just above its middle. The upper ulcers measure* 3×4 *mm, the lower* 7×3 *mm. The normal mucosa between them overlies the cricoarytenoid joints (arrowed).*

The localization of the ulcers in the dorsolateral part of the larynx corresponds with the position of endotracheal tubes in the larynx, as demonstrated radiologically by Matzker (1953), but their surprisingly frequent incidence in the cricoid region, not commented upon previously, is attributed by Bergström *et al.* (1962) to the

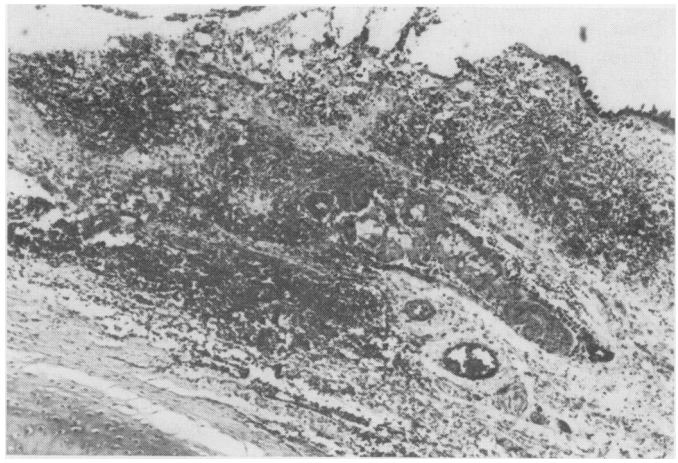

FIG. 3. *Microscopic section from one of the ulcers shown in Figure* 2. *There is loss of epithelium, except for some columnar cells (top right), necrosis of the tissues of the submucosa, including the mucous glands, and considerable haemorrhage, but only minimal inflammatory changes. The underlying cartilage is not involved. Necrosis and haemorrhage are more marked than in Fig.* 10. (*H. and E.* ×45.)

anatomy of the larynx and the tendency for children with endotracheal tubes *in situ* to keep their necks extended. The dorsal wall of the larynx is relatively pliable at the level of the vocal cords, but is rigid at the level of the cricoid cartilage which forms a rigid shield, especially in older patients in whom it is often calcified or ossified. Extension of the neck creates a cervical lordosis which causes the spine to force the lamina of the cricoid cartilage forwards against the endotracheal tube.

After the endotracheal tube has been removed the ulcers heal by cicatrical contracture and so may occasion a cricoid stenosis, symptoms of airway obstruction developing after some days or weeks (Hardcastle, 1966). In four cases reported by Striker *et al.* (1967), symptoms developed two to six weeks after extubation.

Damage to the larynx, leading to cricoid stenosis, may be predisposed to by upper airway disease because the endotracheal tube, acting as a foreign body, further irritates an already inflamed and oedematous laryngeal mucosa. Thus, as noted above, Striker *et al.* (1967) reported the development of cricoid stenosis in 4 of 18 children with upper respiratory obstruction treated by assisted ventilation via an endotracheal tube, as compared with only 3 of 98 children without upper airway disease so treated.

Nasotracheal tubes, however 'non-irritating', produce severe excoriation and ulceration, especially in the cricoid region, if left in place sufficiently long (Hardcastle, 1966). This is especially true if constant movement occurs between the tube and the mucosa, as during treatment by intermittent positive pressure ventilation (IPPV) (Hardcastle, 1966), or if the tube is too large (Markham *et al.*, 1967).

The site and character of the ulcers indicate that they are primarily pressure sores. The important factors in their pathogenesis appear to be pressure, movement, and infection, as in the case of lesions produced by inflated cuffs. Chemical irritation and other factors may play a part of lesser importance. The duration of intubation is probably highly significant. As indicated above, deep ulceration involving cartilage occurs within 30 hours.

INFRACRICOID STENOSIS

PATHOGENESIS Infracricoid suprastomal stenosis is caused by backward displacement of the anterior wall of the trachea above the level of the stoma (Reading, 1958; Hewlett and Ranger, 1961; Hardcastle, 1966; Ardran and Caust, 1963; Watts, 1963; Smythe and Bull, 1959;

FIG. 4. *Infracricoid stenosis, showing backward displacement of the anterior wall of the trachea above the level of the stoma.*

Johnston, Wright, and Hercus, 1967; Gregory, 1970; Dukes, 1970), as illustrated in Figure 4.

If the tracheostomy is not made sufficiently large when the tracheostomy tube is pushed through it, the upper edge of the stoma may be forced inwards, or buckled backwards, causing a permanent deformity (Reading, 1958; Hewlett and Ranger, 1961; Hardcastle, 1966; Gregory, 1970). In the postoperative period external pressure by the tracheostomy tube may force backwards the tracheal wall above the stoma, especially if the neck is hyperextended, or if the stoma is low (Reading, 1958; Ardran and Caust, 1963; Watts, 1963; Smythe and Bull, 1959; Johnston *et al.*, 1967; Dukes, 1970). Even a stab wound made with a blunt knife may buckle the upper margin of the stoma backwards in an infant (Reading, 1958). Backward displacement of the anterior wall of the trachea, sufficient to cause significant stenosis, by any of these means is especially likely to occur in infants and small children in whom the trachea is both narrow and soft, but the same appearance is occasionally seen in adults who have had a tracheostomy tube *in situ* for a considerable time (Ardran and Caust, 1963).

The mechanism of backward displacement of an infant's trachea above the stoma by angulation of the tracheostomy tube due to hyperextension of the neck is well illustrated in a case reported by Reading (1958). The trachea was incised transversely between the third and fourth rings. A small portion of the third ring was then excised to make a rounded opening of ample size to allow easy introduction of the tracheostomy tube so that this act did not force the upper margin of the stoma backwards. At necropsy the upper two tracheal rings were buckled backwards, creating a transverse ridge below the cricoid cartilage which was responsible for the failure to extubate the infant. This was caused by backward tilting of the upper end of the tube, associated with forward tilting of its lower end which had created a perforating erosion of the anterior wall of the trachea 14 mm below the stoma. This tilt of the tube arose because the infant had kept its neck persistently extended. The superior surface of the convexity of the tube had pressed upon the intact first and second tracheal rings, so buckling them backwards into the lumen of the upper trachea, while its tip had impinged upon the anterior wall of the trachea to produce a round perforating ulcer.

Reading (1958) pointed out that infants with air hunger often keep their necks extended. If this occurs during the first few days after a tracheostomy, because of partial obstruction of the tube, or for other reasons, an infracricoid stenosis is likely to result. Even if the posture is maintained for only a short time the cartilage may remain deformed and so create a permanent stenosis.

Ardran and Caust (1963) reported five infants with this type of stenosis, in four of whom it caused difficulty in excannulation. In all five infants lateral radiographs of the neck demonstrated the backward displacement of the anterior wall of the trachea above the stoma by the extratracheal portion of the tracheostomy tube, and they stressed the importance of taking lateral radiographs of the neck before and soon after inserting the tracheostomy tube. In all five patients inspiratory collapse of the suprastomal portion of the trachea occurred when the tracheostomy tube was withdrawn, so causing a serious problem, and in one the condition developed into a fibrous stenosis after several months.

STOMAL OBSTRUCTION

FIBROUS STOMAL STENOSIS DUE TO CARTILAGE LOSS

PATHOGENESIS It is clear from the classification that stenosis of the trachea at stomal level may be of several varieties. Stenosis due to too large

a defect in the trachea is caused by removal of too much cartilage by the surgeon when making a tracheal window or flap (Toremalm, 1960; Watts, 1963; Dukes, 1970), or because the opening enlarges after the tracheostomy tube has been inserted, due to pressure necrosis and infection of the cartilages. The condition is illustrated in Figures 5 and 17.

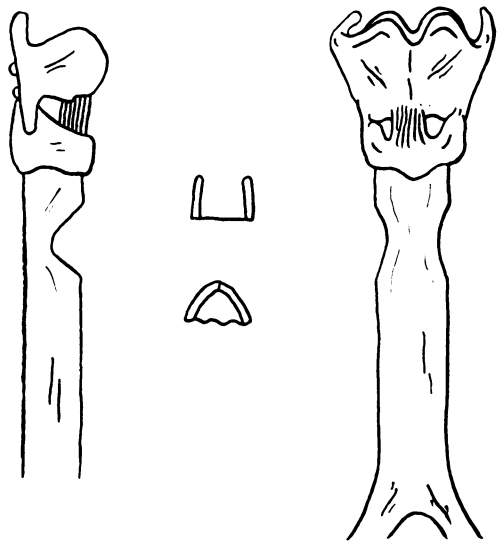

FIG. 5. *Fibrous stomal stenosis due to cartilage loss. Note prominent anterior shelf producing marked narrowing on the lateral view. The anterior view shows bilateral narrowing which is slight. The cross section shows that falling together of the anterolateral walls of the trachea, and contraction of scar tissue at the site of the stoma, cause the lumen to become small and triangular. The stenosis is non-circumferential.*

Watts (1963) showed by tracheography that in humans narrowing always occurred at the level of the stoma after removing a window from the trachea, and that in dogs the degree of narrowing depended upon the width of the defect, for example, the amount of cartilage removed. In this animal defects up to a centimetre in diameter caused minimal narrowing. The defects healed by a thin layer of fibrous tissue lined by normal respiratory epithelium, but no cartilage regeneration occurred, either macroscopically or microscopically (Watts, 1963; Pearson, Goldberg, and da Silva, 1968). As Dukes (1970) pointed out, any type of permanent defect left by merely extubating a tracheostomy will almost always lead to narrowing at the level of the stoma, which is probably most severe after a flap operation if the flap is not replaced. The opening must close by scar tissue which can contract to narrow the lumen of the trachea because of loss of its supporting cartilage rings at this site. This is illustrated in Figure 5.

Because of their soft and narrow trachea stomal stenosis of this variety is particularly liable to occur in infants and young children (Watts, 1963; Hunter, 1967; Dukes, 1970), and this is the reason why cartilage should never be removed when a tracheostomy is performed in these young subjects and one of the reasons why a flap should not be made.

After the insertion of a tracheostomy tube the pressure exerted by the tube on the margins of the tracheal opening will produce pressure necrosis of the cartilage rings, which will then sequestrate and disappear. This destruction will be enhanced by movement of the tube and by local infection, which inevitably occurs (Fennell, 1962; Jackson, 1963; Stiles, 1965a; Deverall, 1967; Pearson, 1969; Gregory, 1970). Infection arises because of the presence of a foreign body in an open wound, and because infected secretions pool in the region above the cuff from where they are not usually aspirated (Pearson, 1969).

The damage caused by tracheostomy tubes in the region of the stoma, especially at its lower margin, and principally due to pressure, movement, and infection, is illustrated in Figures 12 to 15. In Fig. 12 the stoma shows moderate pressure change, with baring of one cartilage at its lower margin, but little or no infective change. Figure 14 illustrates more pressure change at the lower margin of the stoma, with baring of two cartilages, but very little infective change. In addition, cuff lesions are present, and also glottic scarring and healing laryngeal ulcers from previous treatment by assisted ventilation. Figure 13 illustrates rather more stomal pressure changes, together with infective changes. Below the stoma four cartilages are bared, the uppermost of which is fractured in one place. The changes extend into the cuff area and are accompanied by superficial mucosal ulceration at tube-tip level. Figures 15 and 16 illustrate an extreme degree of destruction by pressure and movement combined with severe infection, creating a huge defect in the trachea involving the entire areas occupied by the stoma and cuff anteriorly, and extending upwards to the lower margin of the cricoid cartilage. The changes are described in detail later under 'Cuff Stenosis'. It is of importance that

the greatest damage was found when infection was prominent, and that infective lesions became less severe when anti-infective measures were made more rigid.

The changes described above follow the insertion of a tracheostomy tube into a linear incision, or an operative defect (window or flap) in the trachea which is too small for the tube. A tracheostomy tube must not fit too tightly otherwise damage to the tracheal wall will inevitably result from pressure necrosis, aggravated by movement of the tube and infection. On the other hand, too large an opening will necessitate excessive removal of cartilage.

Stomal stenosis of the above variety occurs after the use of both cuffed and non-cuffed tubes, but it more commonly follows the use of the former, probably because of circumferential occlusion of the submucosal tracheal circulation by the pressure of the inflated cuff. This devascularizes and devitalizes the segment of trachea between the cuff and the stoma (Pearson *et al.*, 1968). Movement transmitted from the ventilator may also be important.

The four cases of stomal stenosis of this nature described by Murphy, MacLean, and Dobell (1966) are of considerable interest. They all occurred within one year in patients aged between 18 and 41 years, who were artificially ventilated for 6, 12, 17, and 24 days respectively, and were thought to be due to the use of a metal tracheostomy tube fitted with a long cuff (36 mm in length) of the type fitted to oroendotracheal tubes. It was considered that the long cuff, when inflated, was partially extruded through the stoma, so exerting pressure on its edge, with the production of pressure necrosis and destruction of cartilage. This view was supported by the fact that when tracheostomy tubes fitted with similar cuffs were inserted into dogs, so that a part of the cuff occupied the stoma, inflation of the cuff for seven days caused tracheal stenosis in three out of four dogs in which a modified H-type tracheal incision had been made, and in one out of three dogs in which a circular window had been cut out of the trachea. After changing to a shorter cuff (18 mm long) in humans no further stomal stenosis occurred. This experience illustrates well the importance of cartilage loss in the aetiology of stomal stenosis.

PATHOLOGY (MORBID ANATOMY) As the result of loss of cartilage from the anterior and lateral walls of the trachea, followed by contraction of scar tissue at the site of the defect, a waist-line constriction develops which comprises the anterior and lateral portions of the tracheal lumen (Fig. 5). This creates a stricture which has a roughly triangular cross section (Pearson *et al.*, 1968). The membraneous part of the trachea is not affected, but when sufficient lateral narrowing occurs, the membraneous trachea may be thrown into longitudinal folds by shortening of its transverse width. Thus stomal fibrous strictures are typically non-circumferential and take the form of an anteriorly placed fibrous shelf (Pearson *et al.*, 1968 ; Grillo, 1969a). Severe narrowing occurs from before backwards, but little from side to side (Fig. 5). The pathological changes which occur in the region of the stoma due to pressure, movement, and infection are illustrated in Figures 12 to 15.

OSTEOMALACIC STOMAL STENOSIS DUE TO CARTILAGE LOSS

PATHOGENESIS In some patients, especially infants and young children, because of cartilage removal or destruction, the trachea becomes soft and flexible at the site where the stoma was made, and sometimes also below it where the inflated cuff had exerted pressure (Reading, 1958 ; Venables, 1959 ; Jackson, 1963 ; Deverall, 1967 ; Pearson *et al.*, 1968 ; Grillo, 1969a ; Cooper and Grillo, 1969). Sometimes this is accompanied by the development of a granuloma at the upper margin of the stoma (Smythe and Bull, 1959 ; Pearce and Walsh, 1961). Because the soft area of the trachea lies in the neck it becomes sucked in by the negative pressure generated during inspiration, with resultant inspiratory collapse of the mobile segment of trachea. This is an important cause of obstruction to respiration when infants are extubated (Reading, 1958 ; Venables, 1959 ; Fennell, 1962 ; Jackson, 1963) and may also cause difficulty in adults (Grillo, 1969a ; Cooper and Grillo, 1969). In two infants reported by Jackson (1963), the edges of the stoma were seen to sink inwards during inspiration, and obstruction was relieved when the lower edge of the stoma was retracted with a skin suture. In Jackson's experience this problem in babies was eliminated by changing from a tracheal window to a vertical incision in the trachea.

The pathogenesis of this condition is the same as that of fibrous stomal stenosis due to cartilage loss, but, instead of luminal reduction by contraction of scar tissue, the affected segment of trachea becomes soft and mobile, the condition known as tracheomalacia. This is more likely to

occur when a cuffed tracheostomy tube is used, as also is fibrous stomal stenosis due to cartilage loss.

STOMAL STENOSIS DUE TO GRANULOMATOUS POLYP (usually attached to upper margin of stoma)

PATHOGENESIS Stomal stenosis may arise because a granulomatous polyp develops at the stoma (Holinger, Novak, and Johnston, 1950; Pearce and Walsh, 1961; Atkins, 1964; Binns, 1964; Johnston *et al.*, 1967; Cooper and Grillo, 1969; Grillo, 1969a). Such polyps usually develop at the superior margin of the stoma (Pearce and Walsh, 1961; Johnston *et al.*, 1967), as illustrated in Fig. 6, but Calvet, Coll, Fournie, and Son Qui (1962) described one at tube-tip level caused by trauma from the end of the tracheostomy tube. These granulomata may follow the use of either cuffed or non-cuffed tubes and were reported many times before the introduction of cuffed tubes (Atkins, 1964). They are more common, and also more likely to cause obstruction, in children than in adults, and in children they may give rise to difficulty following extubation.

The aetiology of these granulomata is uncertain, but their probable cause is irritation of the tracheal epithelium by the tracheostomy tube and by food and secretions accumulating above the cuff, together with infection, which is almost inevitable at this site (Pearce and Walsh, 1961).

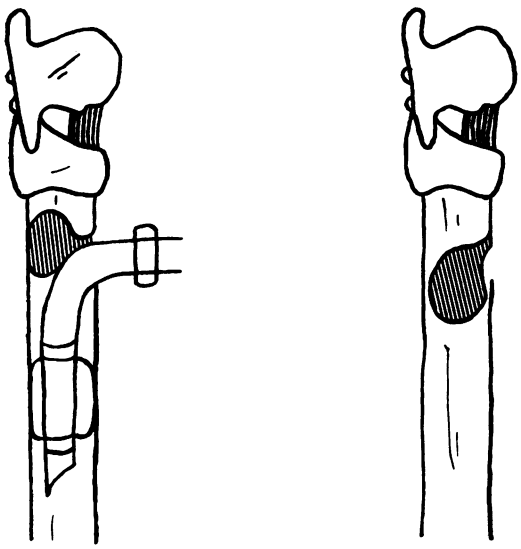

FIG. 6. *Granulomatous stomal stenosis due to granulation tissue polyp growing from the upper margin of the stoma. When the tube is* in situ *the polyp rests upon the upper surface of its curve. When the tube is withdrawn the polyp falls down like a ball valve to obstruct the trachea. This most often causes obstruction in children.*

PATHOLOGY The granulomata take the form of sessile or pedunculated polyps of granulation tissue attached to the mucosa, usually at the upper margin of the stoma (Fig. 6). They do not appear to be associated with weakening or inflammation of the deeper layers of the trachea in most cases (Johnston *et al.*, 1967), but they may occasionally be accompanied by a fibrous stricture (Hunter, 1967). Binns (1964) described a circumferential granuloma reducing the lumen of the trachea at the level of the stoma to the size of a pencil and accompanied by narrowing extending up to the level of the true cords. This author states that a granulomatous polyp inside the trachea is likely to be present if there is persistent discharge from the tracheostomy wound, or if granulomata appear externally at the wound.

The usual type of polyp attached to the upper margin of the stoma sits on top of the tracheostomy tube. When the latter is withdrawn the granuloma may fall down like a ball valve and obstruct the trachea (Fig. 6).

STOMAL STENOSIS FROM DOUBLE-BARRELLING OF TRACHEA DUE TO FORWARD ANGULATION

PATHOGENESIS AND PATHOLOGY Stenosis at stomal level in infants or children, due to double-barrelling of the trachea, has been reported by Clarke (1965) and Deverall (1967), and is illustrated in Figure 7. Clarke's patient was a child of unstated age. A large circular stoma was made in the trachea, the margins of which became adherent to the upper and lower margins of the transverse tracheostomy wound. Because of this adherence the trachea was buckled forwards until it resembled a double-barrelled colostomy. Deverall's patient was a girl aged 4 years, in whom a Björk flap had been sutured to the skin and subepithelial tissues. The tracheostomy tube was *in situ* for nine days. Contraction of scar tissue associated with the flap angled the trachea forwards, producing, as in Clarke's case, a deformity like that of a double-barrelled colostomy. Bronchoscopy revealed the trachea to be angled forwards at the stomal site and narrowed by granulation tissue. It was possible to pass a naso-tracheal tube across the area of double-barrelling, but the child died from secondary haemor-

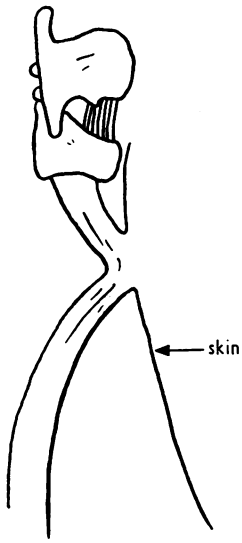

FIG. 7. *Stomal stenosis due to double-barrelling of the trachea. Note the forward angulation of the trachea to the adherent skin.*

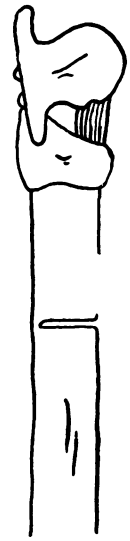

FIG. 8. *Stomal stenosis due to retropulsion of a Björk flap. This has been reported as a complication of tube replacement.*

rhage from the aortic suture line made for the repair of coarctation of the aorta.

The trachea of infants and young children is soft, mobile, and narrow. If a window made in it becomes adherent to the wound edges, or a Björk flap is fashioned and sutured to the skin or subepithelial tissues, contraction of scar tissue may angle the trachea forwards, so causing obstruction due to a double-barrelled deformity at the level of the stoma. This angulation gives rise to significant narrowing, which is enhanced by the development of granulation tissue and by puckering of the posterior wall of the trachea. In Deverall's patient stridor developed within a short time of extubation. I am not aware that stenosis of this variety has been reported in an adult.

STOMAL STENOSIS DUE TO RETROPULSION OF A BJÖRK FLAP

PATHOGENESIS In a leading article in the *British Medical Journal* (1969) retropulsion of a Björk flap during tube replacement was cited as a cause of dangerous obstruction. When the tube is changed, according to the writer of the article, the Björk flap may be detached and pushed into the lumen of the trachea. I have no personal experience of this accident of tube replacement and have not encountered a case report of one. Figure 8 depicts what presumably happens.

INFRASTOMAL OBSTRUCTION

CUFF STENOSIS

PATHOLOGY Many workers consider that stenosis at the site of the cuff is the commonest type of stenosis after tracheostomy and intermittent positive pressure ventilation (Ekedahl and Laage-Hellman, 1967; Johnston *et al.*, 1967; Shelly, Dawson, and May, 1969; Cooper and Grillo, 1969; Grillo, 1969a). This I believe to be true for adults, but stomal stenosis is probably at least as common in children and infants, and it is mainly in these young subjects that suprastomal stenoses occur.

These stenoses occur at the site of the inflatable cuff (Fig. 9) in consequence of pressure, movement, infection, and perhaps also chemical irritation and other factors such as systemic hypotension (Stiles, 1965a); Little, 1956; Pearson *et al.*, 1968) or steroid administration (Stiles, 1965a; Pearson *et al.*, 1968). Pressure by the inflated cuff is obviously a vital factor in their production (Watts, 1963; Bradley, Spencer, and Semple, 1964; Yanagisawa and Kirchner, 1964; Fraser and Bell, 1967; Gibson, 1967; Byrn *et al.*, 1967; Campbell, 1968; Jewsbury, 1969; Grillo, 1969a; Cooper and Grillo, 1969; James, Macmillan, Eaton, and Grillo, 1970). These matters will be discussed in detail later.

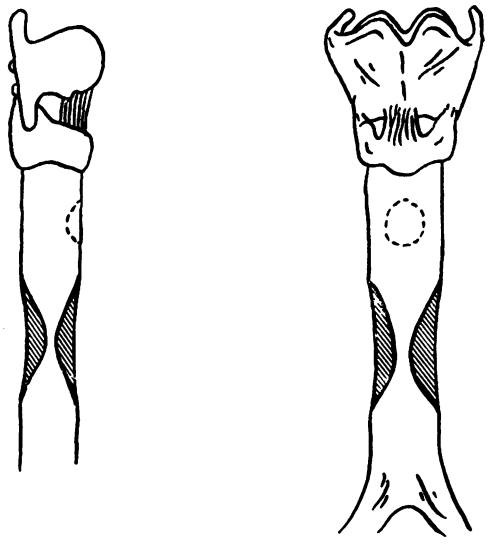

FIG. 9. *Fibrous stenosis at cuff level. Note that the stenosis is circumferential and associated with marked thickening. The stenosis is much more marked from within than appears from without. The site of the stoma is indicated by dotted lines.*

Damage to the trachea at the site of inflatable cuffs is common and severe (Stiles, 1965a; Ekedahl and Laage-Hellman, 1967; Gibson, 1967; Grillo, 1969a; Cooper and Grillo, 1969) and is well illustrated in Figures 10 to 16. Stiles (1965a) wrote that it was the general impression that damage to the trachea was uncommon if certain precautions were observed and that it seldom led to serious complications. His own work and that of others, including mine, has shown that this is, unfortunately, not true.

The typical fully developed stenosis at cuff level takes the form of a circumferential fibrous stricture, 1 to 3·5 cm below the stoma and 1 to 1·5 cm long (Grillo, 1969a; Cooper and Grillo, 1969; James *et al.*, 1970), leaving a lumen of between 2 and 5 mm in diameter (Fig. 9). The actual stenosis forms a firm circumferential web of dense fibrous tissue at the site of the cuff. From outside an hour-glass-shaped constriction can usually be identified, with loss of cartilage at its centre. Lesser degrees of damage are apparent for 1 cm or so proximally and distally. Sometimes no constriction can be seen from without, and even when one is present the lumen is narrower than one would suspect because the fibrous web causes more narrowing internally than is apparent externally (Fig. 9). The trachea is often firmly adherent to surrounding tissues at the site of the stenosis. The surface of the stenosis may be covered by granulation tissue or squamous metaplastic epithelium.

Microscopically the fully developed stenosis consists of dense scar tissue with only some attempts at epithelialization. In the most severe lesions there is total loss of mucosa, submucous glands, and cartilage, the whole thickness of the tracheal wall being replaced by fibrous tissue or granulation tissue of varying degrees of maturity. Lesser degrees of damage in adjacent areas include cartilage erosion, inflammatory infiltration, mucosal ulceration, and areas of epithelial squamous metaplasia. In many cases some damage is present between the fibrous web and the stoma, in the form of tracheitis, fibrosis or loss of cartilage rings. In some cases cartilage loss is so extensive that an area of tracheomalacia results (Grillo, 1969a; Cooper and Grillo, 1969).

The pathological changes which lead up to the fully developed fibrous stricture or area of tracheomalacia begin early. If the tracheostomy tube is present for two or more days superficial mucosal ulcers will be seen, while after 10 to 14 days changes are severe (Stiles, 1965a; Cooper and Grillo, 1969; James *et al.*, 1970). The changes which occur (Figs 10 to 16) are as follows.

The earliest change is a superficial fibrinous tracheitis which progresses to superficial ulceration. These ulcers increase in size and depth, and they eventually expose the cartilage rings. The bared cartilage rings then soften, fragment, and finally disappear, and if this happens over a large enough area the softened tracheal wall forms an area of tracheomalacia. From two to six cartilage rings may be bared in this way. If, in patients who subsequently develop a cuff stenosis, serial radiographs are taken while the tracheostomy tube is in situ, the trachea will often be seen to bulge at the site of the cuff (Gibson, 1967). This should warn the clinician to suspect the later development of stenosis. The trachea at the site of the inflated cuff may lose all cartilage and so become completely flexible and, therefore, both collapsible and distensible. Subsequent development of scar tissue converts the flexible area into a fibrous stenosis. The malacic area may involve not only the area of the balloon but also that between the cuff and the stoma.

The damage to the trachea at the site of the cuff begins on its anterior wall and is always maximal there (Stiles, 1965a; James *et al.*, 1970),

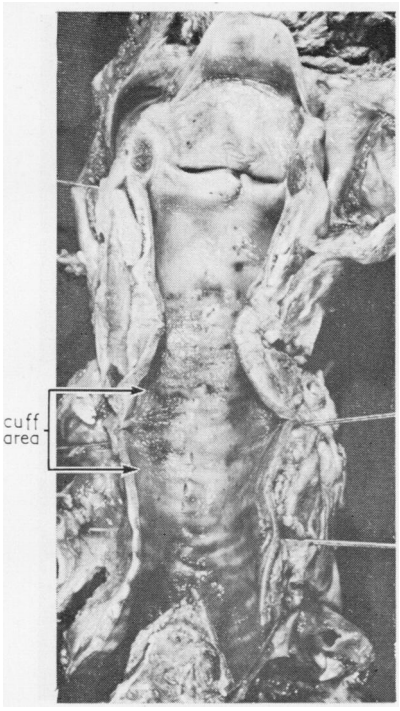

FIG. 10. *Larynx and trachea from a man aged* 62 *years who died on the operating table during aortic valve replacement for severe aortic stenosis with mitral regurgitation. Anaesthesia via a cuffed endotracheal tube had been administered for between four and five hours. Multiple superficial haemorrhagic ulcers are seen as dark patches in the cuff area (between the arrows) overlying cartilage rings* 7 *to* 11, *and situated between* 5 *and* 7·5 *cm from the lower margin of the cricoid cartilage. This indicates how quickly damage to the air passages can occur. The line of mucosal tears is the result of fractures of ossified cartilages during photography.*

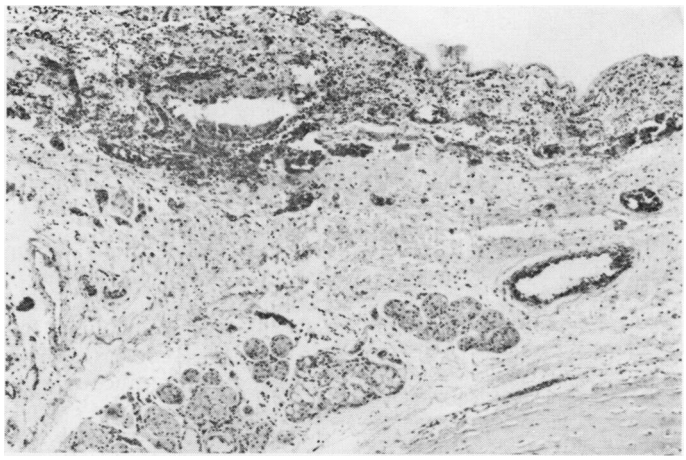

FIG. 11. *Microscopic section from one of the ulcers shown in Figure* 10. *There is loss of epithelium, the tracheal surface being formed by the basement membrane (top right). There is vascular dilatation with haemorrhage, and some leucocytic infiltration of the submucosa, together with small patches of early necrosis. The underlying cartilage ring (bottom right) is not affected. The changes are less severe than in Figure* 3 (*H. and E.* ×45).

as shown in Figures 10 to 16. This is probably due to the presence of the unyielding cartilage rings in the anterior two-thirds of the tracheal wall. The posterior wall is soft and more yielding and is cushioned by the oesophagus behind it.

The microscopic changes which accompany the macroscopic ones, leading up to the formation of stenosis, are as follows. Acute inflammation of the mucosa with haemorrhage and fibrin formation proceeds to ulceration over the cartilage rings. The ulcers enlarge and deepen to expose the cartilages. Inflammation then extends between and finally around the cartilage rings. The cartilages fragment and disappear, leaving ulcer craters in their place. The ulcers are floored by granulation tissue, but later they may be covered by squamous epithelium. In due course the tracheal wall is converted into fibrous tissue with loss of mucosa, mucous glands, and cartilage. In some cases ulceration extends through the whole thickness of the tracheal wall before stenosis has time to occur. If this happens anteriorly the innominate artery may be eroded, with fatal haemorrhage, while if it does so posteriorly a tracheo-oesophageal fistula may result. This condition is often fatal.

Figures 10 to 16 illustrate the pressure-infective changes produced by inflated cuffs and discussed

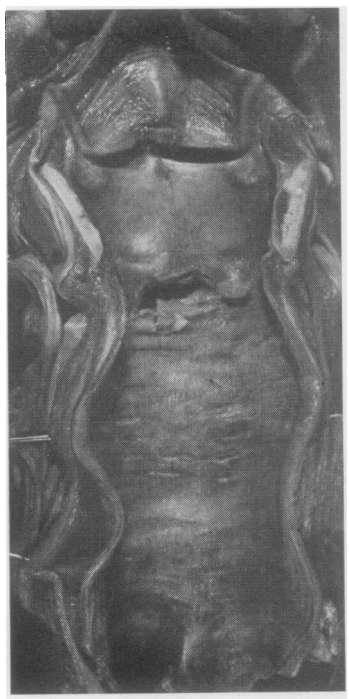

FIG. 12. *Larynx and trachea from a man aged* 45 *years who underwent replacement of the mitral and aortic valves. He died* 15 *days later due to paravalvar mitral regurgitation, haemolytic anaemia, pulmonary oedema, and uraemia. He was ventilated through a cuffed endotracheal tube for the first* 24 *hours. Seven days later he was reintubated and ventilated for* 48 *hours after which ventilation was continued through a cuffed tracheostomy tube. Artificial ventilation was therefore given for the first* 24 *hours and the last nine days. The stoma is fairly clean, with little evidence of infection, but one cartilage ring at its lower margin is bared. The cuff area shows some ulceration with baring of two cartilage rings. The scar of a healed ulcer is seen below the posterior end of each vocal cord.*

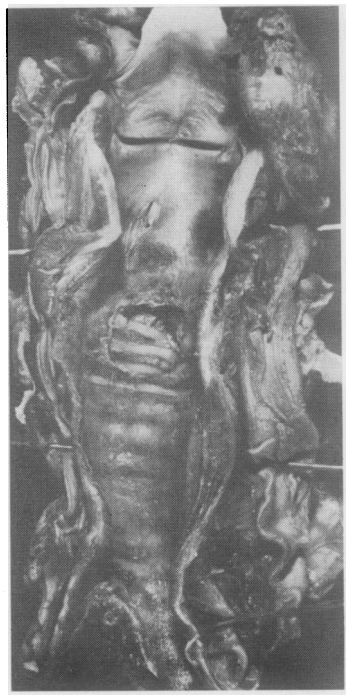

FIG. 13. *Larynx and trachea from a woman aged* 52 *years with severe restrictive and obstructive pulmonary disease, who was ventilated for* 14 *days between mitral valve replacement and death from confluent bronchopneumonia. Tracheostomy was performed* 48 *hours after operation. The lungs also showed small infarcts and haemorrhages. The stoma, situated below the third tracheal cartilage, shows moderate pressure-infective changes. Immediately below the stoma, extending into the area of the cuff, are four bared cartilages, the uppermost of which is fractured in one place. Further below, at tube-tip level, there is superficial mucosal ulceration on the anterior tracheal wall over the ninth and tenth cartilage rings.*

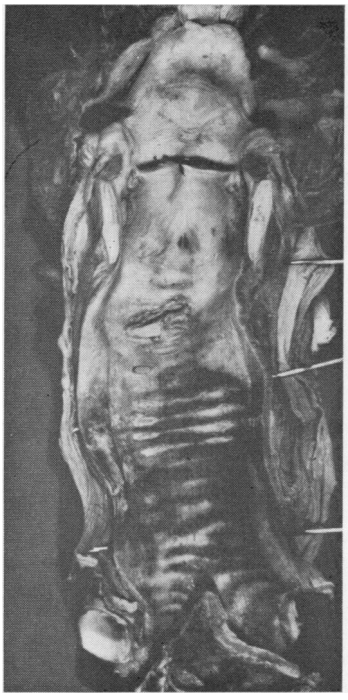

FIG. 14. *Larynx and trachea from a man aged* 49 *years who died of suppurative bronchitis* 20 *days after a second mitral valve replacement, having had cardiac arrests on the* 3*rd and* 13*th postoperative days. He was artificially ventilated for* 24 *hours after operation, for three days after the first cardiac arrest, and for seven days between the second cardiac arrest and death. Tracheostomy was performed four days before death. The stoma is clean, the first two cartilages below it being bared. The mucosa over the next three cartilages is intact, but the succeeding four are bared in the region occupied by the cuff. In addition to these recent lesions older lesions are seen in the larynx caused by endotracheal intubation at the time of the first mitral valve replacement. These consist of glottic scarring from the vocal cords downwards, and bilateral symmetrical healing ulcers below the posterior end of the vocal cords in identical positions to those shown in Figure* 2. *The upper ulcer measures* 4×3 *mm and the lower* 10×7 *mm.*

above. Figure 10 illustrates multiple superficial ulcers in the mucosa of the anterior tracheal wall, in the cuff area, occurring after only four to five hours of anaesthesia administered through a cuffed endotracheal tube. Figure 11 shows loss of epithelium, haemorrhage, some leucocytic infiltration, and small patches of early necrosis in the mucosa at the site of one of the ulcers. The underlying cartilage is intact. Figure 12 shows, in addition to the stomal damage previously described, some ulceration in the cuff area with baring of two cartilages after two periods of assisted ventilation of 24 hours and nine days. Figure 13 illustrates ulceration, with four bared cartilages, on the anterior wall of the trachea extending from the cuff area up to the lower margin of the stoma. This patient was ventilated for 14 days. Superficial ulceration is also present lower down at tube-tip level. Figure 14 illustrates, in addition to the stomal pressure lesion with baring of two cartilages, ulceration of the mucosa over the anterior half of the trachea at cuff level, with baring of four cartilages. The stomal and cuff lesions are separated by an area of intact mucosa overlying three cartilage rings. The longer standing laryngeal lesions, previously

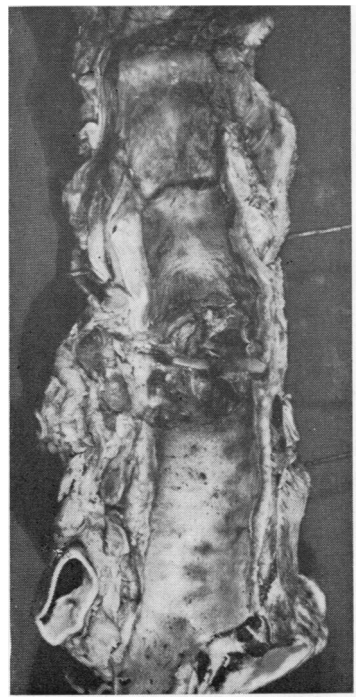

FIG. 15. *Larynx and trachea from a man aged* 65 *years with extensive complicated pneumoconiosis and left ventricular failure, who died* 24 *days after aortic valve replacement from infection of the mediastinum, lungs, and urinary tract. He was ventilated for the first four days through a cuffed endotracheal tube and thereafter a cuffed tracheostomy tube. The trachea shows a huge defect, measuring* 4×3 *cm in the fixed specimen, involving the whole area occupied by both the stoma and the cuff, and also reaching up to the lower margin of the cricoid cartilage. Six bared, distorted, and fragmented cartilages were visible in the necropsy specimen, and some cartilage is missing. Severe infective changes are present.*

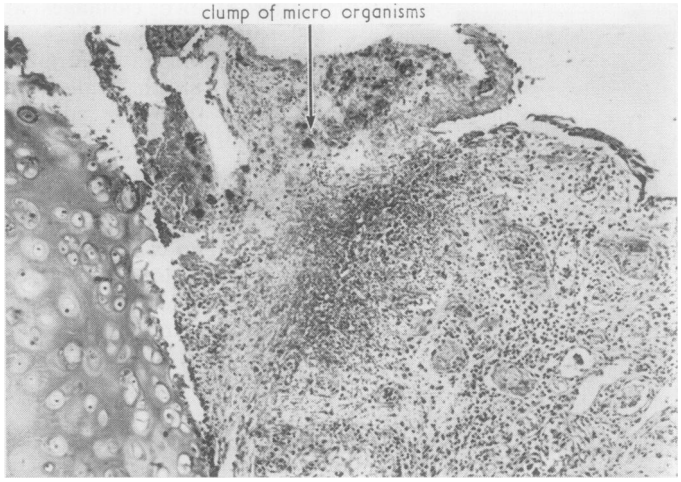

FIG. 16. *Microscopic section from lesion shown in Figure* 15. *There is loss of epithelium* (*top*), *and cartilage erosion both on the luminal surface and in the depths of the tracheal wall. At the latter site the cartilage is scalloped. Erosion in this area must be due to infection and not to pressure. There is adjacent acute inflammatory change, with visible, darkly staining clumps of micro-organisms* (*one of which is arrowed*). *Numerous venous thrombi are seen in the tracheal wall* (*mid right*), *forming foci for pulmonary microemboli and septic infarcts. The superficial tracheal lesions are a source of aspiration bronchopneumonia. Such septic infarcts and bronchopneumonia are both common in this type of case* (*H. and E.* $\times$90).

referred to, are also seen. This patient was ventilated for periods of 24 hours, 3 days and 7 days. Figure 15 illustrates gross destruction of the anterior tracheal wall by pressure, movement, and infection which created a huge defect. Figure 16 shows the microscopic findings. Of particular interest are the erosion and scalloping of cartilage in the depths of the tracheal wall, as well as on the surface, which must be of infective origin, numerous venous thrombi in the depths of the tracheal wall, and clumps of micro-organisms near the surface. These findings indicate the importance of infection in destroying the wall of the trachea, and also the importance of these infective lesions as a source not only for aspiration bronchopneumonia but also for infected infarcts of the lungs.

PATHOGENESIS As mentioned above, cuff stenoses occur in consequence of pressure, movement, infection, and perhaps also chemical irritation and other factors, such as systemic hypotension and steroid administration. These factors will now be examined.

Pressure Much thought has been given to the pathogenesis of the changes enumerated above, which may lead to perforation of the trachea, tracheomalacia or fibrous stenosis. One of the fundamental factors is pressure exerted by the cuff when it is inflated. This can be avoided in infants and young children in whom IPPV can be performed satisfactorily without the use of a cuff. In these young subjects cuffed tubes should not be employed (Jackson, 1963; Aberdeen, 1965; Gregory, 1970).

Important experimental work was conducted on dogs by Shelly *et al.* (1969). They assessed four variables, namely cuff volume, duration of inflation, pressure tolerance of the trachea, and hypotension (arterial pressure of 60 mmHg maintained for four hours). They named the minimal volume of air in the cuff required to prevent a leak, using an inflation pressure of 30 cmH_2O, as the minimal occluding volume (MOV), and the difference between the pressures inside and outside the trachea as the actual tracheal pressure (ATP). They found that when cuff inflation was maintained accurately at the

MOV for five days little significant damage was inflicted on the trachea, but that when twice the MOV was maintained for the same period three out of four dogs developed significant tracheal stenosis. They also found that an ATP of 50 mmHg maintained for five days produced no damage to the trachea, whereas one of 100 mmHg maintained for the same period caused stenosis in three out of four dogs. These workers considered that the significant factor was the relationship of the ATP to the mean arterial pressure. If the ATP exceeds the mean arterial pressure for long enough, ischaemia will probably occur and lead to necrosis of the tracheal epithelium and damage to supporting cartilage, ultimately resulting in stenosis.

Shelly *et al.* (1969) concluded that the primary cause of tracheal stenosis was ischaemia resulting from the ATP exerted by the cuff and the duration of its maintenance. From a practical point of view it appears from these experiments that a cuff inflated to the MOV continuously and for a prolonged period causes no appreciable damage to the trachea. The pressure exerted by a cuff inflated to MOV did not appear to be critical in these experiments, and in my view this is also true of humans. This is supported by the experimental work of Murphy *et al.* (1966). These workers fitted endotracheal cuffs, 36 mm long, over 1·25 cm hardened polyvinyl tubes, 3·75 cm long. These tubes were inserted into the trachea of three dogs, their pilot tubes being exteriorized through needle holes in the trachea of the dogs. After inflation of the cuffs continuously for 10 days the only change which occurred in the trachea was reddening of the mucosa which disappeared in one week. No stenosis developed during the four weeks of follow-up.

It may be of importance that the above experiments were carried out in dogs with normal lungs in which the occlusive pressure was relatively low, for example, 30 cmH_2O. In patients with lungs of low compliance for any reason, the pressures required to ventilate the patient may be high, and in such patients the risks of pressure necrosis caused by the cuff must be greater (Pearson *et al.*, 1968). Such a situation must be taken into account in clinical work.

Movement of tracheostomy tube in trachea Movement of the tracheostomy tube in the trachea, whether transmitted from the ventilator or caused by movements of the patient's neck or body, will cause shearing stresses where the inflated cuff is in contact with the tracheal wall. This is certainly an important cause of damage to the trachea (Gray, 1960 ; Jackson, 1963 ; Watts, 1963 ; Stiles, 1965a ; Gibson, 1967 ; Johnston *et al.*, 1967 ; Nicholls, 1968 ; Shelly *et al.*, 1969 ; Jewsbury, 1969 ; James *et al.*, 1970). It has been suggested that the length of the tracheal damage caused by the cuff may depend upon movement of the tracheostomy tube relative to the trachea, movement increasing the length of the damage (James *et al.*, 1970).

Infection The importance of infection has been discussed by many workers (Putney, 1955 ; Toremalm, 1960 ; Meade, 1961 ; Stiles, 1965a ; Gibson, 1967 ; Johnston *et al.*, 1967 ; Sara, 1967 ; Deverall, 1967 ; Campbell, 1968 ; Pearson *et al.*, 1968 ; Grillo, 1969a ; Cooper and Grillo, 1969). Infection of the wound and airways develops in nearly all cases of tracheostomy and IPPV (Stiles, 1965a ; Gibson, 1967 ; Pearson *et al.*, 1968). Gibson (1967) considered that trauma from the cuff and infection were the two main factors responsible for destruction of the full thickness of a segment of the tracheal wall leading either to malacia or stenosis. Johnston *et al.* (1967) thought that infection was one of the three major factors responsible for stenosis (the other two being pressure and movement), a view reiterated by Nicholls (1968). Putney (1955) and Meade (1961) believed it was important to leave the tracheostomy wound open lest abscess formation occur with spread of infection along facial planes to the mediastinum, leading to the production of tracheal stenosis. Stiles (1965a) stated that infection will increase the damage initiated by mechanical factors, while Grillo (1969a) and Cooper and Grillo (1969) considered that infection may be an accessory factor. Infection is usually established as early as two days after tracheostomy and is often mixed, the commonest organisms being *Staphylococcus aureus, Pseudomonas pyocyanea,* and coliforms. Numerous other bacteria and moulds may be demonstrated from time to time.

My observations of necropsy material strongly support the importance of infection in damage to the trachea following tracheostomy and IPPV. Examination of Figs 10 to 16 shows that damage at both stomal and cuff levels increases markedly when infection becomes prominent. The most extensive damage is shown in Fig. 15, which illustrates a huge defect in the trachea, measuring 4×3 cm in the fixed specimen. It involves the whole of the stomal and cuff areas anteriorly and extends up to the lower margin of the cricoid cartilage. The infective changes are severe and, as

indicated under 'Cuff Stenosis', the erosion and scalloping of the cartilages in the deep zones of the tracheal wall are considered to be due to infection and not to pressure. Furthermore the numerous venous thrombi in the depths of the tracheal wall (Fig. 16) must constitute important sources of infected pulmonary infarcts. Many clumps of micro-organisms are evident in the more superficial layers of the trachea. The evidence indicates that infection greatly increases the damage to the tracheal wall, and also that the infected tracheal lesions constitute an important source not only of aspiration bronchopneumonia but also of infected pulmonary infarcts, both of which are common in this type of case.

It must be accepted that infection will almost inevitably occur in the presence of an open wound and a large foreign body. It must also be accepted that damage to the mucosa and deeper tissues of the trachea, initiated by pressure and movement, will be increased by the infection which will be allowed ingress by this damage. Once tracheal cartilages are exposed by pressure necrosis of the overlying mucosa they will constitute avascular foreign bodies which will both encourage further infection and be destroyed by it.

In my view, the initiating factor in damage leading to stenosis at cuff level is mechanical, in the form of pressure and movement, but infection is an important secondary factor which aggravates the damage. Granted that this is so, every effort must be made to reduce infection to a minimum, but it must be realized that its complete prevention, in the presence of an open wound and a foreign body, and perhaps also an already infected bronchial tree, is unlikely to be achieved.

Chemical irritation Damage by the tube, at the level of the stoma, cuff or tip, may be increased by chemical irritation, so that tracheostomy tubes, and their cuffs, should be made of non-irritating material, despite the fact that similar damage may be caused by tubes made of any material (Grillo, 1969a) in consequence of pressure, movement, and infection. Both rubber and silver tubes produce chemical irritation (Salt, Parkhouse, and Simpson, 1960), while silver tubes have the added danger of rigidity and other side effects (Jacobs and Affeldt, 1956). Red rubber is an irritant material (Johnston *et al.*, 1967 ; Hunter, 1967 ; Gregory, 1970) and it should be avoided. Hunter (1967) stated that there had been a reaction away from red rubber tubes which may be irritant to skin and mucous membranes, while Byrn *et al.* (1967) recommended that rubber should be avoided for any form of intubation lasting more than a few hours. Latex and plastics appear to be the least irritating materials. Polyvinyl chloride (PVC) is probably the best material in this respect and has now replaced rubber in many branches of surgery because of lack of tissue reaction after long-term use (Gregory, 1970). Guess and Stetson (1968), however, showed that a stabilizer compound, organotin, can be extracted from PVC and is toxic to specific cell cultures and rabbit tissue. Changes in rabbit tissue after implanting PVC (Guess and Autian, 1964) are similar to those occurring in the glottic region of patients treated by long-term intubation. The United States Pharmacopeia, 1965 (Rendell-Baker, 1968) has laid down criteria for implantation tests which it has been recommended should be followed in Britain (*British Medical Journal,* 1969). In the meantime, PVC tracheostomy tubes, such as the adult Portex Bassett cuffed tube or the Great Ormond Street Hospital infant, non-cuffed tube, can be recommended.

Apart from the fact of chemical irritation, tracheostomy tubes should be of appropriate size and shape and as soft as possible, in order to minimize pressure effects at varying sites.

Hypotension Prolonged hypotension may well be a factor which will aggravate the damage caused by pressure, movement, and infection (Little, 1956 ; Watts, 1963 ; Stiles, 1965a ; Gibson, 1967 ; Pearson *et al.*, 1968 ; Shelly *et al.*, 1969). Prolonged hypotension produces vasoconstriction which compromises the minute circulation, so predisposing the trachea to damage by other factors. If Shelly *et al.* (1969) are correct in assuming that, if the actual tracheal pressure exceeds for long enough the mean arterial pressure, ischaemic damage to the trachea will occur and lead to stenosis, then prolonged hypotension must favour the production of stenosis. Every effort must, therefore, be made both to prevent and to treat hypotension. It is of interest in this context that 12 of 18 patients with tracheal stenosis studied by Pearson *et al.* (1968) had suffered from hypotension with systolic pressures of less than 90 mmHg for more than one hour. Several of the patients whose laryngotracheal lesions are illustrated in Figs 2, 3, and 10 to 16 suffered from periods of hypotension.

Steroid administration It has been suggested that the administration of steroids in high dosage and for long periods may impair the healing response to an irritative process and so favour the develop-

ment of tracheal stenosis (Stiles, 1965a ; Pearson *et al.*, 1968). The experimental work of Pearson *et al.* (1968) favours this suggestion, and of their 24 patients who developed tracheal stenosis after tracheostomy and assisted ventilation 13 received steroids in moderate to high dosage for more than a week. I have an open mind on the significance of steroid administration.

TUBE-TIP STENOSIS

PATHOGENESIS It is well recognized that stenosis may occur as the result of impingement of the lower end of the tracheostomy tube on the lower portion of the trachea (Watts, 1963 ; Ekedahl and Laage-Hellman, 1967 ; Johnston *et al.*, 1967 ; Byrn *et al.*, 1967 ; Hunter, 1967 ; Campbell, 1968 ; Grillo, 1969a ; Cooper and Grillo, 1969 ; Dukes, 1970). If the stoma is made too low, or the tracheostomy tube is too long, the lower end of the tube may ulcerate the carina, or enter the right main bronchus and either ulcerate it or cause obstructive collapse of the left lung or right upper lobe. Instead of producing tracheal stenosis, the end of the tracheostomy tube may ulcerate through the wall of the trachea. Because of the backward inclination of the trachea and the curvature of the tracheostomy tube, ulceration usually occurs through the anterior wall, especially when metal tubes are used (Watts, 1963), and it may perforate the innominate artery. Ulceration sometimes, however, affects the posterior wall, when it may create a tracheo-oesophageal fistula. Figure 13 illustrates, in addition to stomal and cuff lesions, superficial mucosal ulceration of the anterior tracheal wall over the ninth and tenth cartilages at the level of the tip of the tracheostomy tube.

The factors concerned in the production of stenosis by the end of the tube are similar to those for the cuff, especially pressure, movement, and infection, but perhaps also chemical irritation, hypotension, and steroid administration. Movement is undoubtedly important. The piston-like movement transmitted to the tube by the ventilator (Watts, 1963 ; Stiles, 1965a ; Gibson, 1967 ; Shelly *et al.*, 1969 ; Jewsbury, 1969) will cause the tube to move upwards and downwards in the trachea and may result in severe damage by the tip of the tube. Ulceration of the carina is common, and the right main bronchus may be affected (Stiles, 1965a).

Quite apart from the piston-like movement of the tube transmitted from the ventilator, angulation of the tube around a fulcrum at the stoma may occur and so cause the end of the tube to impinge upon the wall of the trachea. This was considered to be the cause of the tube-tip stenosis in two cases reported by Johnston *et al.* (1967). Both patients, one an adult the other a child, were very restless, ventilation was difficult, and the tracheostomy tubes were frequently displaced. This mechanism of damage to the trachea is well illustrated by the case of infracricoid stenosis reported by Reading (1958) and detailed previously, in which the lower end of the tube, rotated forwards by extension of the neck, created a circular perforating ulcer of the anterior wall of the trachea 14 mm below the stoma. Movements of the neck will cause movement of the tracheostomy tube in the trachea which may lead to damage by the end of the tube (Jackson, 1963).

Dukes (1970) believed that strictures of the trachea after tracheostomy most commonly arise from damage by the end of the tube as the result of continual movement during ventilation therapy. Hunter (1967) reported seven stenoses, two at cuff level and five at tube-tip level. The two patients with cuff stenoses had been ventilated through red rubber cuffed tubes, whereas four of the five patients with tube-tip strictures had been ventilated through cuffed plastic tubes. Hunter considered that the spherical cuffs fitted to the plastic tubes allowed the tubes to move around in the trachea. Table II does not support the high incidence of tube-tip stenoses.

It is possible that some stenoses located at or just below the tip of the tube arise from damage caused by rough, dirty or high-pressure aspiration of tracheobronchial secretions (Campbell, 1968). The technique practised should be gentle and aseptic.

In summary, it appears that most stenoses situated at the level of the end of the tracheostomy tube are caused by repeated movements of the tube within the trachea, either transmitted from the ventilator or occurring in consequence of tilting of the tube by movements of the neck or body of the patient, but some may be due to faulty methods of aspiration of the tracheobronchial secretions. No doubt damage caused by these mechanical factors will be aggravated by infection and chemical irritation and the other factors discussed with regard to cuff stenosis.

LARYNGOTRACHEAL OBSTRUCTION COMPLICATING TRACHEOSTOMY AND IPPV

INCIDENCE

From the literature I have been able to collect 215 cases of laryngotracheal stenosis following

tracheostomy and assisted ventilation. No claim is made that this collection is comprehensive. To these cases are added 17 collected through the courtesy of my colleagues in the London Society of Thoracic Surgeons. This makes a total of 232 stenoses. In 90 of these it has been possible to estimate the percentage incidence of stenosis following tracheostomy and assisted ventilation. The findings are shown in Table I.

TABLE I

INCIDENCE OF LARYNGOTRACHEAL STENOSIS FOLLOWING TRACHEOSTOMY AND ASSISTED VENTILATION

Author	No. of Tracheostomies	No. of Stenoses	Incidence %
Smythe and Bull (1959)	9	2	22·2
Toremalm (1960)	25 (4 weeks to 10 yrs)	1	4·0
Head (1961)	462	2	0·43
Meade (1961)	212	1	0·5
Mollaret *et al.* (1962)	447	36 (21 fibrous)	8·0
Oliver *et al.* (1962)	294 (all under 18)	3	1·0
Watts (1963)	212	1	0·5
Bradley *et al.* (1964)	29 (chr. bronch. and emphys.)	2	6·9
Crosby (1964)	50	6	12·0
Clarke (1965)	69	1	1·4
McClelland (1965)	389	0	0
Bargh and Slawson (1965)	217	1	0·5
Deverall (1967)	104	6	5·8
Ekedahl and Laage-Hellmann (1967)	259	4 (+3 C.X.R)[1]	1·5
Gibson (1967)	96	4 (+10 mild)[1]	4·2
Johnston *et al.* (1967)	325	12	3·7
Lord (1967)	280	2	0·7
Nicholls (1968)	64	3	4·7
Marshall (1969)	140	0	0
Dukes (1970)	110	3	2·7
Total	3,793	90	2·37

[1] See text.

It can be seen from Table I that the percentage incidence for various authors ranges from 0% to 22·2%, the average figure for the 3,793 tracheostomies being 2·37%. Although this figure may be biased towards the high side because I was, in general, collecting material concerning stenosis of the air passages, the incidence may, in fact, be much greater than these figures suggest, for they all relate to retrospective studies. Gibson (1967) stated that in addition to the four stenoses shown in the table there were 10 other mild ones, while Ekedahl and Laage-Hellmann (1967), in a careful radiological study of their patients, found three stenoses in addition to the four shown in the table. Only one prospective study has been reported to my knowledge, that of Pearson (1969). Among 58 patients who were treated by tracheostomy and assisted ventilation nine developed stenosis, an alarming incidence of 15·5%. The very high incidence reported by Smythe and Bull (1959) can be explained by the small number of cases and the fact that the patients were infants being treated for tetanus neonatorum, while both the stenoses were due to granulation tissue polyps.

Of the total of 232 cases, the site and type of stenosis were ascertained in 154, and were not ascertained in 78; the findings are shown in Table II.

TABLE II

SITE AND TYPE OF LARYNGOTRACHEAL OBSTRUCTION FOLLOWING TRACHEOSTOMY AND IPPV

Suprastomal obstruction		9
Cricoid	3	
Infracricoid	6	
Stomal obstruction		56
Fibrous, due to cartilage loss (one calcified—Kennedy (1968))	43	
Malacic, due to cartilage loss	2	
Granulomatous	9	
Double-barrelled trachea	2	
Retropulsion of Björk flap	0	
Infrastomal obstruction		87
Cuff	75	
Tube-tip	12	
Mixed		2
Cricoid and stomal	1	
Stomal and cuff	1	
Total		154
Site unknown		78
Total		232

The rarity of cricoid stenosis following tracheostomy nowadays is indicated in Table II and is in marked contrast to the situation in 1921 when Jackson condemned the then popular operation of high tracheostomy. As already indicated, most cases of cricoid stenosis seen at present follow prolonged endotracheal intubation.

It is probable that tracheomalacia is commoner than is indicated in Table II. It may occur at infracricoid stomal or cuff level, as the result of cartilage fracture or loss in consequence of either surgery or pressure and infection subsequent thereto. In some cases of fibrous cuff stenosis an area of tracheomalacia may be found between the fibrous stenosis and the stoma, and this area may be seen to collapse during inspiration on radiological examination (Grillo, 1969a).

The relative incidence of stomal and infrastomal stenosis varies with age. Stomal stenoses of all varieties are more frequent in children, whereas infrastomal stenoses are commoner in adults. Suprastomal stenosis and double-barrelled trachea especially affect infants and young children. The relative incidence of the various types of

laryngotracheal stenosis will, therefore, depend upon the type of patient being treated.

CLINICAL FEATURES

TIME OF ONSET OF SYMPTOMS The time of onset of symptoms is variable and is, to some extent, dependent upon the site and nature of the stenosis. Difficulty with ventilation or breathing may become apparent while the tracheostomy tube is *in situ* in suprastomal or tube-tip strictures, while in any variety of stenosis symptoms may occur at the time of extubation or at a variable period thereafter.

Cricoid stenosis may give rise to symptoms of respiratory obstruction at the time of extubation or at a variable period afterwards. In cases following endotracheal intubation symptoms usually start some days or weeks after the tube has been removed (Hardcastle, 1966; Striker *et al.*, 1967) because of the healing process and contracture which follows extubation. In four cases reported by Striker *et al.* (1967) the interval varied between two and six weeks. Infracricoid stenosis in infants usually gives rise to obstruction when the tracheostomy tube is removed, so causing difficulty in extubation, or within a few days. It is often mobile at first with resultant inspiratory collapse. Later, if not treated, it may become rigid.

Granulation tissue polyps growing from the upper margin of the stoma cause no obstruction while the tracheostomy tube is in place but are liable to obstruct the trachea when the tube is withdrawn. While the tube is *in situ* they rest upon its upper surface, but on its withdrawal they may fall down like a ball valve (Fig. 6) and so obstruct the trachea in the region of the stoma (Pearce and Walsh, 1961). These polyps may, however, cause no obstructive symptoms for several weeks, as in one of the two cases of Johnston *et al.* (1967), in whom symptoms developed four weeks after extubation, or in the case reported by Binns (1964) in whom a circumferential granuloma did not give rise to dyspnoea until four months after extubation.

The times of onset of symptoms of respiratory obstruction for the seven cases of fibrous stomal stenosis for which details were obtained were as follows: on extubation (1 case), and at 7, 12, 21, 23, 28, and 29 days, respectively, thereafter. Pearson (1969) reported 16 patients with fibrous stomal stenosis. The time of onset of symptoms was not given for each of these, but treatment started between 1 and 12 months after extubation, and in the first three months in all but three of them. It thus appears that many patients with fibrous stomal stenosis develop symptoms within one month of extubation, and that most of them do so within three months. Malacic stomal stenoses probably give rise to symptoms at the time of extubation or soon afterwards, and the same is true of stomal stenosis due to double-barrelled trachea. The 4-year-old girl with the latter condition reported by Deverall (1967) developed stridor a few days after the tracheostomy tube had been withdrawn. No appropriate details are available for the child reported by Clarke (1965).

Information regarding the time of onset of symptoms due to cuff stenosis was obtained for 38 cases. This varied from the moment of extubation (4 cases) to three years subsequently (1 case). The findings are shown in Table III.

TABLE III

TIME OF ONSET OF SYMPTOMS AFTER EXTUBATION IN 38 CASES OF CUFF STENOSIS

Weeks					Months		Years
0–1	1–2	2–3	3–4	4–12	3–6	6–12	1–3
8	5	6	5	7	3	2	2

It can be seen that 24 of the 38 patients (63%) developed symptoms within four weeks of extubation, and four of these became symptomatic at the time that the tracheostomy tube was withdrawn. For three of these four patients information is available regarding the duration of tracheostomy and IPPV. The periods were 10, 34, and 42 days. In each, therefore, tracheostomy and IPPV were maintained for a considerable period. Approximately one-third of the patients developed symptoms between one month and three years after extubation.

With regard to tube-tip stenoses information was available about the onset of symptoms in eight cases. In two (Johnston *et al.*, 1967, cases 11 and 12), respiratory obstruction occurred while the tracheostomy tube was still *in situ*. One was a 4-year-old girl with poliomyelitis who was ventilated through a silver tracheostomy tube. Ventilatory difficulty due to the stenosis became evident after 13 weeks. The other patient was a 44-year-old woman treated by tracheostomy and IPPV for acute haemorrhagic leuco-encephalitis whose stenosis became evident after 11 weeks when weaning proved impossible. Two patients with tube-tip stenosis developed symptoms of obstruction a few hours after extubation, one after seven

days, two after 14 days, and one after 26 days. In these six patients the period of intubation varied between 16 days and seven weeks. Thus all patients with tube-tip stenosis developed clinical tracheal obstruction either while being ventilated or within one month of removal of the tracheostomy tube. In every case the tracheostomy tube was resident for 16 days or more.

MODE OF PRESENTATION OF SYMPTOMS The mode of presentation of symptoms, as well as the time of onset, is variable. The onset may be very insidious, stenosis being almost complete before symptoms become severe (Miller, 1969), or symptoms may develop rapidly and create an acute emergency (Jewsbury, 1969). In most patients there is a latent interval after withdrawal of the tracheostomy tube before symptoms appear, but sometimes difficulty is experienced at the time of extubation, especially in the case of stomal and suprastomal stenoses, and occasionally a tube-tip stenosis will give rise to ventilatory problems during prolonged treatment by assisted ventilation. A latent interval when present is usually less than a month but may be as long as three years.

SYMPTOMS AND SIGNS Early symptoms include a dry repetitive cough (Binns, 1964 ; Deverall, 1967 ; Pearson *et al.*, 1968) and inability to raise the sputum above the stenosis (Pearson *et al.*, 1968 ; Grillo, 1969a ; James *et al.*, 1970). These symptoms occur at a stage when narrowing of the trachea is insufficient to cause dyspnoea or stridor. Inability to raise the sputum may result in acute obstructive episodes (Durcan, 1963 ; Grillo, 1969a; James *et al.*, 1970) or an attack of pneumonia (Deverall, 1967). Such an acute obstructive episode is well exemplified by a man of 20 years, with a tube-tip stenosis, reported by Durcan (1963). He developed transient dyspnoea and stridor 26 days after extubation and was admitted to hospital seven days later with increasing dyspnoea and stridor of 12 hours' duration.

These early features of tracheal obstruction require emphasis for they should lead to the suspicion of a developing stenosis, but their significance is often unrecognized.

In due course, often rapidly, the symptoms progress to dyspnoea and stridor, which may be accompanied by features of hypoxia, such as cyanosis, restlessness, and aggressiveness (Durcan, 1963 ; Binns, 1964). For these symptoms to develop a marked reduction in the lumen of the trachea is required. Pearson *et al.* (1968) stated that in healthy persons the tracheal diameter may be reduced by 50%, causing a reduction of luminal area of 75% without altering the exercise tolerance for ordinary activity. It is probable that the lumen of the trachea at the site of the stenosis must be less than that of the glottic region before appreciable dyspnoea and stridor appear (Dukes, 1970 ; and *vide infra*).

In cases severe enough to require surgery dyspnoea is almost always present, at first on exertion but later also at rest. The breathing becomes laboured and difficult, the accessory muscles of respiration may be brought into play, and inspiratory indrawing of the sternum and ribs may be seen (Jewsbury, 1969).

Stridor is usually, but not always, present in cases severe enough to require treatment (Deverall, 1967 ; Pearson *et al.*, 1968 ; Grillo, 1969a). In the less severe cases it occurs only on exertion or rapid deep breathing, but in the more severe ones it is present even at rest. In such cases the lumen of the trachea has a diameter of less than 3 to 4 mm (Pearson *et al.*, 1968). Stridor is especially common in children.

Obstruction of the larynx or cervical trachea produces inspiratory stridor, whereas obstruction of the mediastinal trachea or bronchi produces expiratory stridor. This may help to locate the site of the obstruction. The negative pressure exerted by inspiration will tend to collapse the extrathoracic portion of the airways, so increasing the severity of the stenosis, but will tend to dilate the intrathoracic portion of the trachea, so having the opposite effect. Expiratory stridor may be mistaken for asthma or bronchitis. According to Jewsbury (1969), the origin of the stridor can easily be located by the use of a stethoscope, even if there is coexistent pulmonary congestion or infection.

Pre-existing chronic obstructive airways disease, with cough and sputum, may not only increase the disability caused by tracheal stenosis but may also mask its development. The incidence after tracheostomy and IPPV of 2·37% (Table I) should alert the clinician to the possibility of the development of this serious complication in any patient who has been submitted to this treatment, especially if it has been prolonged.

INVESTIGATION

In any suspected case of stenosis of the larynx or trachea radiological and bronchoscopic examination are of great value in demonstrating the presence of a stenosis, and also its site and nature. Pearson *et al.* (1968), in a prospective study, also

examined the stoma, visually and digitally, and the proximal trachea with a nasal pharyngoscope.

RADIOLOGICAL EXAMINATION The value of radiological examination is stressed by almost all recent workers (Binns, 1964; Gibson, 1967; Johnston *et al.*, 1967; Deverall, 1967; Pearson *et al.*, 1968; Jewsbury, 1969; Grillo, 1969a), and an account of the techniques and findings is given by James *et al.* (1970).

Methods of radiological investigation include straight radiographs of the neck and chest, fluoroscopy with spot radiographs, fluoroscopy with spot contrast tracheograms, and tomography, with and without contrast medium in the trachea. There is a difference of opinion with regard to the superiority of contrast tracheography over air tracheography and tomography, but I have no doubt about the superior detail that contrast tracheography provides (Fig. 17). The technique, though simple, is important, for it is essential to show the upper trachea and cricoid region clearly and in multiple projections. This is an area which is not visualized by the usual methods used for bronchography. It is helpful to indicate the position of the stoma with a small radio-opaque marker placed over the centre of the tracheostomy wound, or the stoma itself if it is open.

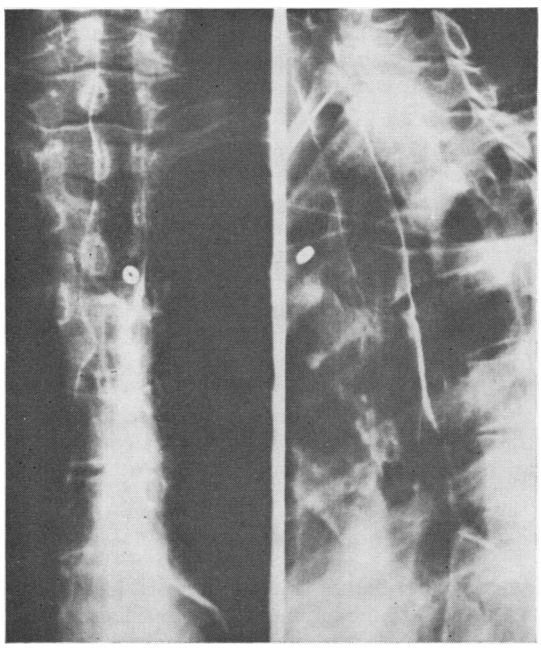

FIG. 17. *Tracheogram showing stomal narrowing, with anterior shelf formation in the oblique view.*

Radiographs of the neck and chest must be taken in anteroposterior, lateral, and oblique projections to demonstrate the larynx and the entire trachea. The importance of lateral radiographs of the neck in infants was stressed in the sections dealing with infracricoid stenosis. Multiple projections demonstrate more of the trachea in profile, while oblique projections remove soft tissue and bony shadows superimposed on those of the air passages. The radiographs should be taken to provide high contrast between air in the lumen of the trachea and the surrounding tissues. If the tracheostomy tube is still resident it must be removed, but all precautions must be at hand for replacement and suction should acute respiratory obstruction occur (James *et al.*, 1970). On occasions films taken with the tracheostomy tube *in situ* may provide useful evidence of cartilage destruction or overinflation of the cuff, either of which will result in a bulge at the site of the cuff (Gibson, 1967; James *et al.*, 1970) and will define the relationships between the parts of the tube and the stenosis.

Fluoroscopy of the neck and chest is useful for two reasons. First, it will help to distinguish fibrous stenosis from tracheomalacia. The former is fixed whereas the latter is mobile, so that the tracheal lumen alters during respiration, sniffing or coughing (Pearson *et al.*, 1968; Grillo, 1969a; James *et al.*, 1970). In some cases a fibrous stomal or cuff stenosis is accompanied by an adjacent area of tracheomalacia, as indicated in the section on pathology. The effects of inspiratory and expiratory effort on the lumen of the trachea depend upon the location of the malacic area. If it is in the neck it will collapse on inspiration, if in the mediastinum it will collapse on expiration. Abnormal mobility of the trachea can be recorded by the use of cine studies and videotape (James *et al.*, 1970). The second use of fluoroscopy is to allow spot films, with or without injection of contrast medium, to be taken in anteroposterior, lateral, and oblique projections in a manner calculated to give maximal information.

Stenosis may be indicated by a reduction of the lumen due to convergence of the thin tracheal walls, by a thin membrane projecting into the lumen almost at right-angles from the tracheal wall, or by an elongated soft tissue density associated with narrowing of the tracheal lumen. Fibrous stomal stenoses (Fig. 17) result from an anterior shelf, narrowing the lumen from in front, and are best seen in lateral projection in the lower part of the neck. The anterior indentation often lies a little lower than the radio-opaque

marker on the skin because the tracheostomy will have been performed with the neck extended. This position draws the trachea upwards. Cuff stenoses, and most tube-tip stenoses, form concentric narrowings behind the manubrium sterni. It is important to locate the stenosis relative to the upper margin of the manubrium sterni for this may have important implications for the surgical approach.

In some cases tomography in two planes may be very helpful, but I am convinced that contrast tracheography gives more detail, provided the technique used is satisfactory.

Radiological examination gives more accurate information than does bronchoscopy with regard to the site and length of the stricture, but its nature is assessed better by bronchoscopy (Pearson *et al.*, 1968).

BRONCHOSCOPY Bronchoscopy may be carried out by the usual technique through the mouth (peroral bronchoscopy) or the stoma (Pearson *et al.*, 1968) if this is still open (perstomal bronchoscopy). I find it easy to pass a bronchoscope through the stoma under local anaesthesia after removal of the tracheostomy tube. By this method examination can be made of the stoma and the trachea below it, but examination of the suprastomal region is difficult. Damage to the tracheal wall by the cuff or the end of the tube can be readily assessed at the time of extubation, and severe ulceration, with baring of the tracheal cartilages, will give warning of the possibility of a future stenosis.

When a stenosis is already present or suspected, radiological examination should be followed by peroral bronchoscopy. If the trachea is severely obstructed, and the patient is distressed, bronchoscopy should be performed under local anaesthesia, as general anaesthesia may be dangerous (Pearson *et al.*, 1968), and it is wise to proceed to tracheostomy or excision of the stricture should an acute obstructive episode be induced (Jewsbury, 1969; Grillo, 1969a). All should, therefore, be in readiness should such a course of action be demanded.

During bronchoscopy the cords and subglottic region and the entire trachea must be carefully examined for any of the varieties of stenosis previously described. Performing routine peroral bronchoscopy at or within a week of withdrawal of the tracheostomy tube, Dukes (1970) found some narrowing at the level of the stoma in 95% of window tracheostomies and in 62·5% of Björk flap tracheostomies which persisted eight weeks later. This narrowing was prevented by making a modified shorter and wider flap which was replaced at the time of extubation. In only 3 out of 110 cases, however, was stomal narrowing sufficient to cause a clinical stricture with signs and/or symptoms. The carina, and even the right main bronchus, may be damaged by a tracheostomy tube which is too long for the patient, or by faulty use of suction catheters.

Obstruction due to a granulation tissue polyp may be satisfactorily relieved by bronchoscopic removal, which may have to be repeated, and other forms of stenosis can be assessed. Thus it is possible to determine if the stenosis is rigid or pliable, if it is circumferential or non-circumferential, if its surface is inflamed, ulcerated or epithelialized, or if secretions are retained below it (Pearson *et al.*, 1968). Its site can also be estimated, but this is more accurately determined by tracheography. The effect of coughing on the lumen in the region of the stenosis can be readily assessed if the examination is performed under local anaesthesia. If the stenosis is at stomal, cuff or tube-tip level, and is rigid and tight, it should not be dilated. Bleeding and inflammatory swelling may be induced and lead to dangerous obstruction.

Johnston *et al.* (1967) stated that a granulomatous polyp is likely to be present whenever a tracheostomy tube has been *in situ* for any length of time. In such cases they advised bronchoscopy to be performed at the time of extubation.

APPROACH TO INVESTIGATION The two keystones to the investigation of cases of laryngotracheal stenosis following tracheostomy and assisted ventilation are radiology and bronchoscopy. The former should be conducted first and, with the aid of air or contrast tracheograms and tomograms, the presence, site, length, and rigidity or otherwise of the stenosis should be ascertained. Further information can then be gained by bronchoscopy, but if stenosis and respiratory distress are severe all should be in readiness to proceed either to tracheostomy or to excision of the stenosis should an obstructive emergency arise.

PREVENTION

CRICOID STENOSIS Cricoid stenosis is prevented by avoiding high tracheostomy, resident laryngostomy tubes, or prolonged nasotracheal intubation for assisted ventilation, especially in infants and young children. The rare occurrence of the con-

dition due to resident oesophageal tubes is a hazard which it is reasonable to accept.

In performing tracheostomy the first tracheal ring and cricoid cartilage must never be damaged. Should the original tracheostomy have been made too high in error, a tube should be inserted through another stoma lower in the trachea. If laryngostomy is performed as an emergency procedure under extenuating circumstances a tube passing through the cricothyroid membrane should never be left *in situ*. Once the obstruction has been overcome by laryngotomy, and the emergency has been combated, tracheostomy should be performed below the level of the first tracheal ring.

The prevention of cricoid stenosis occurring after the treatment of infants and young children by assisted ventilation through a nasotracheal tube requires special consideration. Intubation should be gentle, the number of changes of the tube should be reduced to a minimum, and the duration of intubation should be as short as possible. Bergström *et al.* (1962) advised that intubation should not exceed 24 hours, while Abbott (1968) stated that it should not continue for more than seven days. Other workers have used this method of treatment for prolonged periods. In view of the rapidity with which deep ulceration of the cricoid region can occur, and the seriousness of this variety of stenosis, I agree with those who take a conservative view. Frequent changes of the tube increase damage to the larynx and favour its infection from the nose and mouth. For these reasons the tube should be changed as seldom as possible. The size of tube used is important. Too large a tube is more likely to produce pressure ulceration of the cricoid region (Markham *et al.*, 1967). When the upper airway is healthy a tube one size smaller than that which completely occludes the airway is chosen. In cases of upper airway disease one should select the smallest tube that will permit adequate ventilation and suction, or avoid endotracheal intubation in favour of tracheostomy. Ventilation is adequate in this sense when there is no intercostal or sternal recession and no marked tachypnoea. As mentioned previously, upper airway disease predisposes a child to cricoid stenosis after endotracheal intubation (Striker *et al.*, 1967 ; Abbott, 1968). The tube should be non-irritant and not too hard, and movement between it and the air passages should be reduced to a minimum by all possible means (see Cuff Stenosis, p. 517). Extension of the child's neck should be avoided.

INFRACRICOID STENOSIS Infracricoid stenosis is prevented by making sure that the incision or opening made in the trachea is large enough to allow the tracheostomy tube to be inserted without force, and by being as gentle as possible during the introduction of a tube of suitable size and shape. In infants care should be taken to see that the neck is not allowed to extend after the tracheostomy has been performed. Dukes (1970) claimed that her method of making a tracheal flap, by ensuring an adequate opening in the trachea, reduces the possibility of obstructing the trachea by pressure of the tracheostomy tube on its anterior wall above the stoma. It is important to take lateral radiographs of the neck soon after performing tracheostomy in infants to ensure that the tube is not forcing the anterior wall of the suprastomal area of the trachea backwards (Ardran and Caust, 1963). If it is, another tube of suitable curvature and length between flange and trachea, and placed at a suitable angle, should be inserted, and the situation should again be checked by lateral radiographs of the neck. If this is unsuccessful the anterior wall of the trachea may be pulled forwards by a right-angled retractor inserted into the trachea above the tracheostomy tube. It has also been suggested that at the time of tracheostomy a steel wire suture should be inserted through the anterior wall of the trachea and that its ends should be brought out through the skin. If lateral radiographs of the neck show deformity the steel wire can be pulled until further films show that the deformity has been reduced (Ardran and Caust, 1963). If subsequent softening produces mobile stenosis with inspiratory collapse operative correction may become necessary, but in a small infant this may have to be deferred for some months or even years.

FIBROUS STOMAL STENOSIS DUE TO CARTILAGE DEFECT An important aspect of prevention of this variety of stenosis is the employment of the proper technique of tracheostomy, especially in infants. The degree of stenosis is directly proportional to the size of the cartilage defect made by the surgeon, or created subsequently, relative to the lumen of the trachea. In infants and small children no cartilage should be removed from the trachea (Watts, 1963 ; Jackson, 1963 ; Cavanagh, 1966 ; Hunter, 1967 ; Aberdeen, 1968 ; Dukes, 1970 ; Gregory, 1970). The surgeon should content himself with making a simple longitudinal incision, dividing three rings, usually the second, third, and fourth, but not the first. It is unwise to make either a window or a flap (Deverall, 1967 ; Johnston *et al.*, 1967), which also have the disadvantage in these small subjects that they may

cause double-barrelling of the trachea (*vide infra*).

In adults a window or flap should be made of such a size that it exactly fits the tracheostomy tube to be used. If too large, an unnecessary amount of cartilage will have been removed; if too small, pressure necrosis will subsequently enlarge it and will favour the occurrence of infection. Dukes (1970) advised replacing the flap and suturing it to the margins of the tracheal opening at the time of extubation. This is sensible advice. In addition to correct tailoring of the opening in the trachea all precautions must be taken to avoid movement of the tracheostomy tube, relative to the trachea, and infection. These matters are considered in detail under 'Cuff Stenosis'.

Stomal stenosis may follow the use of non-cuffed or cuffed tubes and IPPV; the use of inflated cuffs, however, increases the incidence of stomal stenosis due to enlargement of the operative defect in the trachea in consequence of destruction of cartilage by pressure and infection for reasons given previously. Thus cuffed tubes, in addition to causing stenosis at cuff level, also increase the risk of stenosis at stomal level. For these reasons cuffed tubes should not be used unless they are essential either to exclude pharyngeal lesions from the respiratory tract, as for instance in the presence of coma or laryngeal and pharyngeal paralysis, or to enable IPPV to be carried out. Cuffed tubes are not necessary for the latter purpose in infants (Jackson, 1963; Aberdeen, 1965; Gregory, 1970). If a cuffed tube is required the importance of not having too long a cuff which can prolapse through the stoma has already been pointed out. Should this happen, destruction of cartilage around the stoma by pressure necrosis greatly enhances the risk of stomal stenosis later (Murphy *et al.*, 1966).

OSTEOMALACIC STOMAL STENOSIS DUE TO CARTILAGE LOSS The causation and prevention of this condition are the same as they are for fibrous stomal stenosis and nothing needs to be added to what has already been said, except that infection is liable to cause widespread cartilage destruction.

STOMAL STENOSIS DUE TO DOUBLE-BARRELLED TRACHEA This condition is prevented by not making a tracheal window or flap when performing tracheostomy in infants and young children. It is the forward traction by scar tissue associated with a flap or a window adherent to the wound that causes the deformity. The fashioning of either a window or a flap in these young subjects, as already indicated, may also produce a stomal stenosis due to cartilage loss. In the case of a flap this variety of stenosis may be prevented, perhaps, by stitching the flap back into place when the tracheostomy tube has been removed.

STOMAL STENOSIS DUE TO RETROPULSION OF A BJÖRK FLAP The writer of the leading article in the *British Medical Journal* (1969) suggested that this accident can be avoided by suturing the lower margin of a tracheal stoma to the skin edge (Hewlett and Ranger, 1961) instead of fashioning a flap.

CUFF STENOSIS Cuff stenoses occur as the result of pressure, movement, infection, chemical irritation, and other factors.

Excessive or prolonged pressure by the cuff There is controversy as to whether inflated cuffs should or should not be deflated at regular intervals, and among those who practise intermittent deflation there is widespread difference with regard to the frequency and duration of deflations. For example, Gibson (1967) recommended deflation every half hour and at each tracheobronchial aspiration, Watts (1963) for one to two minutes each hour, Deverall (1967) and Johnston *et al.* (1967) for five minutes each hour, and Nicholls (1968) after 12 hours, and thereafter for one minute every four hours. A leading article in the *British Medical Journal* (1969) advised deflation for two minutes every hour. On the other hand, Beaver (1961) and Hewlett and Ranger (1961) advised against intermittent deflation, and Wilson (1966) and Marshall (1969) do not practise it.

I advise against intermittent deflation of the cuff for several reasons. It has been shown in humans by those who, like myself, do not deflate the cuff intermittently that this practice is safe provided that care is taken to inflate accurately to the MOV. Thus, in Marshall's series of 140 elective tracheostomies after open heart surgery, there was no case of stenosis or other serious injury to the trachea. The only death due to tracheostomy was from cardiac arrest following tube displacement. The experimental work of Murphy *et al.* (1966) and Shelly *et al.* (1969) is in line with this view. Intermittent deflation can be dangerous unless practised with skill and care. The cuff may not empty completely when the pilot tube is opened, either because the tube remains partly obstructed or because the elasticity of the cuff is imperfect, as it may if made of plastic material (Hunter, 1967). Such cuffs have to be aspirated flat. If they are not aspirated flat the

same amount of air as was injected during the first inflation will produce overinflation. This may happen progressively on each occasion. The only safe procedure is to assess the MOV accurately at each re-inflation. The original volume of air is never used as a guide. The second danger of intermittent deflation is that pharyngeal secretions which have accumulated above the balloon may be aspirated into the bronchi when the balloon collapses. Most tracheostomy tubes do not have a pilot tube arranged to aspirate the area above the inflated cuff, though one such tube has been described (Hunter, 1967). If intermittent deflation is practised, pharyngeal secretions must be aspirated before deflation and tracheobronchial secretions immediately after deflation. Pharyngeal aspiration is particularly important in comatose patients and in those with laryngeal incompetence or faciomaxillary injuries. The third danger of deflating the cuff is that increased movement of the tracheostomy tube is allowed in the trachea and further damage may thus arise (Beaver, 1961 ; James *et al.*, 1970). Finally, intermittent deflations add further to the work of a heavily committed nursing staff.

In order to reduce damage to the trachea by the pressure of inflated cuffs it has been recommended that cuffs should be elongated rather than spherical. This distributes the pressure more diffusely over a larger area (Lambert, 1965 ; Johnston *et al.*, 1967 ; James *et al.*, 1970), so avoiding small points of pressure which are more likely to inflict pressure necrosis on the tracheal wall. Too long a cuff, however, may prolapse through the stoma and so cause stomal stenosis (Murphy *et al.*, 1966), as indicated in the discussion on stenosis at stomal level. Cuffs which inflate only during inspiration (Martinez, 1964 ; Hunter, 1967 ; Johnston *et al.*, 1967) are probably inadvisable because of the increased movement of the tracheostomy tube which they allow. The cuff material should be as thin as possible.

The period of time for which IPPV was employed has been ascertained in 39 reported cases of stenosis at cuff level, including 10 of the cases in my series. The findings are indicated in Table IV.

Table IV indicates that the range of times is very great, namely 53 hours to 112 days, and that the period of IPPV may be quite short. In no less than 6 of the 39 cases (15·4%) it was less than one week, and in 13 cases it was between one and two weeks. On the other hand, in 10 patients IPPV was prolonged for more than four weeks. When a patient is being treated by IPPV, therapy

TABLE IV

39 CASES OF TRACHEAL STENOSIS AT CUFF LEVEL AFTER IPPV

Author	No.	Duration of IPPV (weeks)				
		Up to 1	1–2	2–3	3–4	More than 4
Atkins (1964)	2		1 (12)		1 (25)	
Johnston *et al.* (1967)	6	1 (6)	3 (8, 9, 10?)	1 (20)		1 (33?)
Deverall (1967)	4		2 (10, 10)	1 (21)	1 (28)	
Grillo (1969a)	13	2 (3, 4)	2 (9, 13)	1 (17)		8 (112, 38, 30, 57, 38, 35, 34, 60)
Jewsbury (1969)	2	2 (4, 4)				
James *et al.* (1970)	2		1 (10)	1 (14)		
Present study	10	1 (53 hr.)	4	3	1	1
Total	39	6	13	7	3	10

Figures in parentheses indicate the exact number of days or hours of IPPV in each case.

should be as short as possible, and care must be taken to avoid distending the cuff above the MOV at any time, and to reduce to a minimum both movement of the tube relative to the trachea and infection.

Movement of tracheostomy tube in trachea The piston-like movement transmitted from the ventilator can be reduced by suitable connexions to the tracheostomy tube, such as the Cobbs Suction Unit. Movements caused by alterations of position of the neck and body of the patient can be reduced by suitable suspension of the connecting tubing from an adjustable arm (Stiles, 1965a), magnetized connexions to the tracheostomy tube (Agerholm and Salt, 1965 ; Hunter, 1967), and suitable fixation of the tracheostomy tube to the patient (Watts, 1963 ; Matheson, Gardiner, Low, and Dudley, 1963 ; Stiles, 1965a ; Marshall, 1969). The tapes must be tied in knots at the back of the neck with the neck in the neutral position. Two further tapes passed under the arms and around the chest improve fixation (Matheson *et al.*, 1963 ; Stiles, 1965a) and may be a useful addition in restless patients. In infants, felt or plastic padding at the back of the neck will allow the tapes to be tied tightly without cutting into the skin (Gregory, 1970).

The use of elongated, rather than spherical, cuff balloons and the avoidance of intermittent deflations of the cuff, or of tubes fitted with cuffs which inflate only during inspiration (Martinez, 1964 ; Hunter, 1967 ; Johnston *et al.*, 1967), reduce movement of the tracheostomy tube in the trachea. The cuff should be placed near the end

of the tube in order to prevent movement of the tube at its tip as well as at the stoma, but not so near the end that it will prolapse over it and occlude it (James *et al.*, 1970).

Infection Despite the difficulty in preventing infection every effort must be made to do so. Tracheostomy should be performed under full aseptic conditions, the tracheostomy wound should not be sutured, certainly not tightly, around the tracheostomy tube (Putney, 1955 ; Meade, 1961 ; Watts, 1963), dry gauze dressings should be applied to the wound to prevent maceration of the skin (Watts, 1963), tracheobronchial aspirations should be conducted gently and aseptically, control of temperature and humidity of the inspired gases should be appropriate and aseptic, and clinical infection of the wound or bronchial tree should be treated with suitable antibacterial agents (Watts, 1963). Prophylactic treatment with broad-spectrum antibiotics is not advised as it is liable to lead to infection with resistant staphylococci, Gram-negative organisms or moulds. Some workers advise treating tracheostomy wounds with antibacterial agents such as chlorhexidine and polybactrin (Nicholls, 1968) or polybactrin and nystatin (Hunter, 1967), but the application of dry gauze and a strictly aseptic technique is probably just as efficacious. Barrier nursing (Gregory, 1970) may reduce the incidence of infection, but to be effective it has to be strict, and this is tedious.

Aspiration of tracheobronchial secretions is an important source of infection. The technique must be both non-traumatic and aseptic, for mechanical injury to the mucosa will allow the ingress of infection. Mechanical damage is minimized by the gentle use of soft rubber or plastic catheters, lubricated with water or non-greasy jelly (Lambert, 1965) and of a size which passes readily through the tracheostomy tube with plenty of room to spare (Watts, 1963). Suction should be applied only during withdrawal of the catheter (Gregory, 1970), and its force should be controlled by a Y-piece so as to avoid tearing the mucosa (Plum and Dunning, 1956 ; Watts, 1963 ; Gibson, 1967). All catheters must be sterile and introduced into the tracheostomy tube with the aid of sterile forceps by a no-touch technique.

Proper and aseptic control of the temperature and humidity of the inspired gases is of great importance in preventing infection by preserving ciliary action and preventing inspissation of secretions and mucosal damage, so causing crusting, sputum retention, and infection (Watts, 1963 ; Hunter, 1967 ; Chamney, 1969). The inspired gases should be delivered to the trachea at a temperature of 30 to 36° C and an absolute humidity of 30 to 40 mg of water per litre of dry gas (Chamney, 1969). If supersaturated mists are to be delivered by nebulization the droplets must be of the order of 0·3 to 2·0 μ in diameter and preferably not larger than 1 μ (Hunter, 1967), in order to penetrate the terminal bronchioles and alveoli. The water of water-bath humidifiers may become heavily contaminated (Phillips and Spencer, 1965 ; Gregory, 1970). This can be prevented by keeping the water at 60° C (140° F), which temperature will suppress the growth of all vegetative organisms (Hare, 1967 ; Hunter, 1967 ; Chamney, 1969). The temperature at which the gases are delivered to the trachea can easily be controlled by adjusting the length of the inspiratory tube (Sykes, 1970 ; Gregory, 1970). Filtration of the inspired gas by the bacterial filter described by Bishop, Roper, and Williams (1963) removes all particles with a diameter greater than 0·5 μ. In my view, the use of aerosols, detergents, trypsin, and other materials to reduce the tenaciousness of the sputum is less helpful than proper humidification, and they may damage the tracheobronchial mucosa.

Chemical irritation, hypotension, and steroid administration Chemical irritation was discussed at length under 'Pathogenesis', where it was pointed out that Latex and plastic, especially PVC, were the least irritant materials, whereas red rubber was much more irritating. Nothing further need be added here. Every effort should be made to avoid episodes of hypotension, and steroids should be administered with caution.

TUBE-TIP STENOSIS In order to prevent damage to the trachea by the end of the tube the tracheostomy should be correctly placed, and a tube of suitable length and curvature should be chosen. Metal tubes are especially likely to give rise to this kind of damage, and one must ensure that their angulation, as well as their length, is correct. Having made a stoma at a suitable site and chosen a tube of correct size, length, shape, and material, all precautions must be taken to avoid movement and infection, and scrupulous attention must be paid to the technique of tracheobronchial aspiration. These matters are discussed in detail under 'Prevention of Cuff Stenosis'. Finally, the tracheostomy tube should be removed as soon as possible.

TREATMENT

Treatment may be by conservative measures or by operative repair of the narrowed lumen. The latter

may be performed as the primary treatment, or after conservative treatment has failed. The forms of conservative and non-conservative measures which have been employed are as shown:

Conservative measures
1. Bronchoscopic or tracheoscopic excision of polyps
2. Dilatations, usually with a bronchoscope
   (*a*) Peroral
   (*b*) Perstomal
3. Tracheostomy
   (*a*) Distal (below stenosis), with or without dilatation or stenting, or stenting and grafting, of the stenosis:
      (i) Temporary
      (ii) Permanent
   (*b*) Proximal (above the stenosis)
4. Stenting (splinting)
   (*a*) Without grafting
   (*b*) With grafting
5. Antibiotics
6. Steroids
7. Combinations of the above

Operative correction
1. Resection and end-to-end anastomosis
   (*a*) Preceded by conservative treatment
   (*b*) Not preceded by conservative treatment
2. Resection and reconstruction
   (*a*) Preceded by conservative treatment
   (*b*) Not preceded by conservative treatment
3. Tracheoplasty without resection.

CONSERVATIVE TREATMENT The following measures have been used.

Bronchoscopic or tracheoscopic excision This is a very useful method of treating granulomatous polyps arising from the upper margin of the stoma (Pearce and Walsh, 1961 ; Atkins, 1964 ; Johnston *et al.*, 1967). Most workers advise doing this by the usual technique of peroral bronchoscopy, but Johnston *et al.* (1967), because of the danger of haemorrhage, advised excision through a tracheostomy incision and the tracheostome, with an endotracheal tube in place. In one case so treated the endotracheal tube displaced the polyp into the right main bronchus, from which it was extracted through the tracheostomy opening. In one of their cases bronchoscopic excision resulted in three haemorrhages, requiring treatment by blood transfusions and tracheostomy for one week. Twelve copper sulphate cauterizations were also required. The final result was good.

In general, good results are obtained by bronchoscopic excision, though it may have to be performed more than once. I am unaware of severe haemorrhage being reported apart from the case cited above.

Dilatations alone Successful treatment by intermittent dilatations alone has been reported in a proportion of cases by a number of workers (Johnston *et al.*, 1967 ; Pearson *et al.*, 1968 ; Nicholls, 1968 ; Campbell, 1968 ; May, 1969 ; Dolton, 1969), but this method is not likely to succeed in mature fibrous stenoses (Deverall, 1967 ; Pearson *et al.*, 1968 ; Grillo, 1969a ; Jewsbury, 1969 ; James *et al.*, 1970), especially at cuff or tube-tip level. Stomal stenoses appear to be somewhat more amenable (Grillo, 1969a). This may be because fibrous stenoses at cuff and tube-tip level are usually circumferential, whereas those at stomal level are not. Grillo (1969a) reported that no circumferential stenoses were amenable to conservative treatment (dilatations and/or stenting), while Pearson *et al.* (1968) reserved such treatment for mild stenoses and debilitated patients.

My experience confirms that dilatations are unlikely to succeed in fibrous stenoses at cuff level. Of five cases so treated only one was successful. One patient died, and failure in the other three led to the need for resection.

Conservative treatment, including dilatation, is of special value in infants and young children for two reasons. First, because of differential growth the stenosis often diminishes with increasing age (Borrie, 1960 ; Toremalm, 1960 ; Ardran and Caust, 1963), and, second, because resection with end-to-end anastomosis at this age is difficult, hazardous, and liable to be followed by recurrent stenosis (Borrie, 1960).

A second group of patients for whom dilatation may be advisable in the first instance are those in whom the stenosis is not yet mature, so that it is soft, or the mucosa at the site of stenosis is ulcerated or acutely inflamed. Resection and end-to-end anastomosis performed at this stage may lead to dehiscence of the suture line ; it may therefore be wiser to defer operation until the stenosis has become mature (Deverall, 1967 ; Pearson *et al.*, 1968). During the waiting period dilatations, or maintenance of the tracheostomy if it is open, may be useful.

Dilatations, with or without tracheostomy below the stricture, according to its severity, are probably the best way of treating cricoid stenosis, especially in infants and young children (Tore-

malm, 1960). It must be remembered, however, that the size of the dilator is limited by that of the cricoid ring. Excision of stenoses in this area is difficult because of the intramural course of the recurrent nerves (Demos, 1969), though it can be accomplished with success (Mollaret *et al.*, 1962; Grillo, 1969a). It may have to be combined with arytenoidopexy (Grillo, 1969a). This is a formidable operation in a young child. Currently most cricoid stenoses are seen in infants and children as the result of prolonged nasotracheal intubation. They should not occur after tracheostomy performed below the level of the first tracheal ring.

Multiple dilatations are usually required, and they may have to be repeated for months or even years (Johnston *et al.*, 1967), but occasionally one will suffice (Nicholls, 1968). Dilatations are usually carried out perorally with a bronchoscope, but they may also be performed through the tracheostomy (Dolton, 1969) in stomal or infrastomal stenoses. The treatment is not without danger for it may cause haemorrhage (Deverall, 1967; Jewsbury, 1969), or perforation of the trachea (Dolton, 1969), or of the right lower lobe bronchus (Johnston *et al.*, 1967). Furthermore, it cannot restore a normal sized tracheal lumen because the larynx limits the size of dilator that can be passed (Pearson *et al.*, 1968).

The following successes by dilatations alone have been reported: Pearson *et al.* (1968) six stomal stenoses; May (1969) two cuff stenoses; Johnston *et al.* (1967) two cuff stenoses; Nicholls (1968) two stenoses, probably at cuff level; Campbell (1968) one tube-tip stenosis; my series, one cuff stenosis. However, a high percentage of cases so treated have failed so that permanent tracheostomy or resection has become necessary (*vide infra*). The patient treated by Dolton (1969) died from tracheal rupture at the fourth dilatation through the stoma, while the patient in whom the right lower lobe bronchus was perforated (Johnston *et al.*, 1967) required right lower lobectomy.

In summary, treatment by dilatations alone should be considered for mild stenoses in debilitated patients, for infants and young children, for stenoses in the early formative stage when ulceration and acute tracheitis are present, and for suprastomal stenoses, which are usually seen in infants and young children. This method is likely to fail in severe, mature, fibrous stenoses, especially at cuff and tube-tip levels.

Tracheostomy In stomal or suprastomal stenoses tracheostomy may be performed at or below (distal tracheostomy) the stenosis as a permanent treatment for patients who are too ill for other forms of treatment (Mollaret *et al.*, 1962; Bradley *et al.*, 1964; Murphy *et al.*, 1966; Pearson *et al.*, 1968; Grillo, 1969a), or as a temporary measure while the stenosis is being treated by dilatations (Toremalm, 1960; Ardran and Caust, 1963), or by stents (Johnston *et al.*, 1967; Pearson *et al.*, 1968), or by stents and grafting (Borrie, 1960; James *et al.*, 1970), or in order to allow time for differential growth to ameliorate the stenosis in infants and young children (Toremalm, 1960; Ardran and Caust, 1963). Permanent tracheostomy may also be performed above (proximal tracheostomy) a cuff stenosis (Atkins, 1964; Johnston *et al.*, 1967; Pearson *et al.*, 1968) or a tube-tip stenosis (Mollaret *et al.*, 1962; Johnston *et al.*, 1967) in patients who are unsuitable for resection in view of their general condition. In such cases the tracheostomy tube must be passed through the stricture after it has been dilated.

In my series two patients were treated by distal tracheostomy. One of these (case 12) was a subglottic stenosis in a young adult, the other (case 22) a cuff stenosis in a patient who had had thymectomy performed for myasthenia gravis. The tracheostomy was re-opened and dilatations were also performed.

Ardran and Caust (1963) reported five cases of infracricoid stenosis in infancy. One did not require treatment. Three were treated by tracheostomy alone; in one of these the tube was removed after a year. Stridor and inspiratory indrawing persisted. A plastic-rubber half ring was inserted between the trachea and the pretracheal muscles with partial relief. The tracheostomy tube was still *in situ* in the other two infants at the time of the report. The remaining infant was treated successfully by numerous dilatations combined with distal tracheostomy for six months. None of the five children died. Johnston *et al.* (1967) treated an infant aged 1 year with an infracricoid stenosis by inserting a laryngeal metal stent above a tracheostomy tube. Stenting failed. The child was then treated with steroids, antibiotics, and gradual blocking of the tracheostomy tube. The latter was removed after six months. The boy remained well with slight stridor only on severe exertion. These cases illustrate the tendency for strictures in young children to improve with time as the result of differential growth.

In summary, tracheostomy may be used as permanent treatment for stenoses at any level in

patients too ill for operative correction, or as a temporary measure below a stomal or supra-stomal stenosis to allow conservative treatment for the latter by dilatations, stents or stenting and grafting.

Stenting (splinting) with or without grafting There are a few reports of the use of stenting, with or without grafting, in the treatment of post-tracheostomy strictures. Borrie (1960) reported the case of a boy of 14 months with a fibrous stomal stenosis. The tracheostomy was closed and another made at a lower level. A split-thickness skin graft was sutured to the inside of the trachea around a rubber tube. The stent was kept in place for six months and the tracheostomy tube for 17 months. Treatment was successful. The case of the boy with an infracricoid stenosis treated unsuccessfully with a metal stent, but no graft (Johnston *et al.*, 1967), has already been detailed. Pearson *et al.* (1968) successfully treated two stomal stenoses by introducing a laryngo-tracheal or tracheal stent and re-establishing a cervical tracheostomy. A third patient with stomal stenosis was treated by stenting with unsatisfactory results. James *et al.* (1970) treated a cuff stenosis by dilatations and an indwelling Silastic tube, followed by further dilatations and tracheostomy, in a patient so disabled by mitral stenosis that resection and anastomosis was considered to be contra-indicated. The result was reported as unsatisfactory but stable. Grillo (1969a) used tube splints in six patients, combined with dilatations in three of them, but all had to have subsequent resection.

It is clear that stents have been used on relatively few occasions and with variable results. The use of a stent or splint is merely a method of providing continuous dilatation, and many of the remarks made under that heading apply. Moreover the stent constitutes a foreign body. There may, however, be occasions when the use of a temporary stent or splint may be helpful at a time when resection is contra-indicated.

Antibiotics and steroids Appropriate antibiotics should be employed to combat established infection, and steroids may occasionally be helpful, especially in the early stages before the stricture has matured and become fibrous. Binns (1964) successfully treated a granulomatous stomal stenosis with three courses of terramycin, and Mollaret *et al.* (1962) claim to have cured 15 out of 36 post-tracheostomy stenoses with antibiotics and/or steroids, leaving 21 patients with fibrous strictures who either died of complications or were treated by resection or permanent tracheostomy. As mentioned later, Mathey *et al.* (1966) consider important the use of steroids after resection.

Combinations of conservative measures From what has already been said it is clear that several of the conservative measures discussed are often used in combination. Thus intermittent dilatations, tracheostomy, and stenting may be combined in various ways, while antibiotics and/or steroids may be used in addition. Conservative methods should be used only in suitably selected cases.

OPERATIVE CORRECTION There are three main forms of operative correction of laryngotracheal stenoses for consideration, namely, resection and end-to-end anastomosis, resection and reconstruction, and tracheoplasty without resection. The first of these should be chosen whenever possible (Miscall, McKittrick, Giordano, and Nolan, 1963; Belsey, 1950; Mathey *et al.*, 1966; Deverall, 1967; Pearson *et al.*, 1968; Grillo, 1969a; Jewsbury, 1969; James *et al.*, 1970).

Resection and end-to-end anastomosis Modern methods of mobilization (Grillo, Bendixen, and Gephart, 1963; Grillo, 1965, 1966, 1969a; Mathey *et al.*, 1966; Mulliken and Grillo, 1968) enable more than one-half of the trachea to be resected and yet allow end-to-end anastomosis to be achieved (Grillo, Dignan, Miura, and Scannell, 1964; Mulliken and Grillo, 1968; James *et al.* 1970), and so have almost eliminated the need for prostheses. This is certainly true of post-tracheostomy stenoses of a fibrous nature, for they are nearly always short, usually not exceeding 1·5 cm in length (Grillo, 1969a; Cooper and Grillo, 1967; James *et al.*, 1970), and it is very unusual to have to remove more than 4 cm of the trachea. Such lengths can easily be achieved (Barclay, McSwan, and Welsh, 1957; Miscall *et al.*, 1963). Difficulty may occasionally be experienced if stenoses are present at more than one site, or if failed reconstructive procedures have already been performed.

After resection of long lengths of the trachea tension on the suture line may be reduced by performing a Z-plasty (Narodick, Worman, and Pemberton, 1965; Worman, Starr, and Narodick, 1966), and this technique has been mentioned in connexion with post-tracheostomy stenosis by Deverall (1967) and Pearson *et al.* (1968). Two

hemispherical incisions are made through opposite halves of the circumference of the remaining tracheal wall, one above the other, and each between two cartilages. These incisions, which need not include the mucosa, result in window defects which are covered by a periosteal pedicle graft or a patch of synthetic material, such as knitted Dacron (Worman *et al.*, 1966). The grafts epithelialize early, soon become rigid, and are not liable to infection, and in these respects are superior to synthetic materials such as Teflon. The periosteal pedicle graft is prepared from the bed of a resected rib and is applied so that the periosteal surface is towards the tracheal lumen (Narodick *et al.*, 1965). This procedure should rarely be required for post-tracheostomy strictures.

The first resection with end-to-end anastomosis for post-tracheostomy stenosis was performed (successfully) by Forster, Molé, and Fromes (1958) and the second by Flavell (1959). Since then series and individual cases have been reported by many workers. I have collected 67 cases from the literature, to which I have added 11, making a total of 78 (Table V). The original case of Forster *et al.* (1958) is not included.

TABLE V

78 COLLECTED CASES OF POST-TRACHEOSTOMY TRACHEAL STENOSIS FOR WHICH RESECTION AND END-TO-END ANASTOMOSIS HAS BEEN PERFORMED

Author	No. of Cases	Died	Cause of Death
Flavell (1959) ..	1	0	
Binet and Aboulker (1961)	1	0	
Mollaret *et al.* (1962)	14	0	
Mathey *et al.* (1966)	3	2	Disruption of anastomosis; restenosis
Deverall (1967) ..	5	1	Endotracheal tube blockage; cardiac arrest
Byrn *et al.* (1967) ..	1	0	
Nicholls (1968) ..	1	1	*Ps. pyocyanea* septicaemia
Grillo (1969a) ..	18	1	Complicated case of pulm. stenosis and septal defect; erosion of anastomosis by cuff
Pearson (1969) ..	15	1	Subglottic oedema and cardiac arrest
Jewsbury (1969) ..	2	0	
Dolton (1969) ..	1	0	
Miller (1969) ..	3	0	
James *et al.* (1970)	2	0	
Present study ..	11	1	Ventricular fibrillation; coronary arterial disease
Total ..	78	7 (9%)	

In no less than 44 of the 78 cases shown in Table V, previous conservative treatment, mainly intermittent dilatations, sometimes with stenting, is known to have been tried and to have failed, indicating the high failure rate of these methods. Three of the 11 patients in my series had had previous dilatations. With present knowledge many of these patients would probably not have been submitted to such procedures.

Resection and end-to-end anastomosis is the treatment of choice for all fibrous stenoses at stomal, cuff or tube-tip level in all patients except those who are unfit to stand the operation or who are so young that the risks of restenosis, or the technical difficulties, are too great. It is probably wise to defer the operation until the stenosis has matured and acute ulceration and tracheitis have settled down (Deverall, 1967; Pearson *et al.*, 1968) in order to reduce the risk of dehiscence of the anastomosis.

Resection should always be circumferential (Mathey *et al.*, 1966; Deverall, 1967) even if the stenosis is non-circumferential, as it often is at stomal level. In such circumstances wedge resection is tempting, but it may produce puckering of the posterior membraneous wall of the trachea with resultant narrowing (Deverall, 1967). The only indication for non-circumferential excision of the trachea is a non-circumferential lesion involving most of the length of the trachea, in order to avoid the use of a tubular prosthesis (Mathey *et al.*, 1966). Such lesions do not occur after tracheostomy.

The results of resection and end-to-end anastomosis are good. As seen in Table V, only 7 of the 78 patients on whom the operation was performed died, a mortality of 9%. The causes of death in these seven patients are shown. Death from disruption of the anastomosis occurred in a 49-year-old man on the fourth day after operation. The fatal restenosis occurred in a 2½-year-old boy. Death from cardiac arrest due to blockage of the endocardial tube occurred on the fifth day after resection of her stenosis in a girl of 12 years who had had correction of Fallot's anomaly performed. The death from *Ps. pyocyanea* septicaemia occurred 27 days after resection of the stenosis and end-to-end anastomosis in a boy aged 2½ years. The patient of Grillo (1969a) was a man of 43 years who had been operated upon elsewhere for pulmonary stenosis and had developed a postoperative septal defect and a low tracheal stenosis which had been repaired with a Marlex patch. Subsequent resection and end-to-end anastomosis required reimplantation of the left main bronchus into the lower part of the right main bronchus (bronchus intermedius), and his postoperative condition necessitated respiratory support. The inflated cuff eroded the suture line with fatal results. The patient in my series

was a man of over 60 years who had had an aortico-carotid bypass graft inserted. Because of his arterial disease resection of the tracheal stenosis was performed under hypothermia. Irreversible ventricular fibrillation developed on the operating table, and necropsy showed severe coronary arterial disease.

Two of the deaths should have been preventable, those due to subglottic oedema and blockage of the endotracheal tube. The fatal restenosis occurred in a child of 2½ years. Resection at this age carried a danger of restenosis.

The results in the survivors were good and the complications were few. Thus results were recorded as good by Deverall (1967) in four out of five, by Grillo (1969a) in 16 out of 18, by Pearson (1969) in 12 out of 15, and in my series in 10 out of 11.

The non-fatal complications recorded are as follows. A boy of 8 years developed restenosis which was treated by dilatations. The result is not recorded. One patient developed wound dehiscence which required a staged skin-tube repair. One patient had a haemorrhage from the wound, and two became hoarse. Hoarseness was minimal in one and lasted for only two weeks in the other.

The two restenoses referred to above, one fatal, the other non-fatal, both occurred in children aged 2½ and 8 years respectively, indicating the tendency for this complication to occur in young people. It can occur also in adults, as in case 9 reported by Grillo (1969a) in whom resection performed elsewhere was followed by dehiscence of the anastomosis and a Marlex repair.

The important complications which occur in consequence of this operation are dehiscence of the anastomosis, restenosis, and generalized infection. Dehiscence and infection are likely to follow operation performed at an early stage when there is severe ulceration and acute tracheitis, whereas restenosis is likely to occur in infants and children.

Resection and reconstruction Resection and reconstruction, as already pointed out, should rarely be required for post-tracheostomy stenoses for they are short, and end-to-end anastomosis can be achieved after one-half or more of the trachea has been resected. Replacement of the trachea by solid prostheses, skeletonized prostheses of foreign material, viable homografts, non-viable autologous tissue or non-viable homologous tissue have all proved disastrous because of early leakage, granulation tissue formation, loss of ciliary action, late stenosis, infection, and rejection (Grillo, 1969a). In general, foreign material and a contaminated epithelial surface are incompatible. The best available prosthetic material appears to be heavy Marlex mesh (Beall *et al.*, 1963 ; Mathey *et al.*, 1966). To quote Beall *et al.* (1963), 'primary repair of tracheal defects is the method of choice and should be employed whenever possible. . . . I do not think we will ever have an entirely satisfactory tracheal graft. What I do believe is that when the occasion arises and we are forced to use a tracheal graft, heavy Marlex mesh is the best graft we have available today'.

There may be rare occasions when resection and reconstruction may be required, as when there are stenoses at more than one level, or when previous reparative operations have been performed and have failed. A few such operations have been reported for post-tracheostomy stenosis, using either skin-tube reconstruction (Gibson, 1967 ; Pearson *et al.*, 1968 ; Grillo, 1969a) or reconstruction with prosthetic material (Neville, 1969). Gibson (1967) treated four stenoses primarily by staged skin-tube reconstruction. The stenoses were more than 2 cm long and were considered to be too long for resection and end-to-end anastomosis, but with adequate mobilization strictures much longer than this, up to 6 cm in length, can be repaired by this method (Miscall *et al.*, 1963 ; Barclay *et al.*, 1957). At the time of writing three of Gibson's four staged reconstructions had been completed with excellent results. The patient on whom a staged skin-tube reconstruction was performed by Grillo (1969a, case 9) was a severe asthmatic who had had a complicated history before being treated by that writer. Resection and end-to-end anastomosis had been followed by wound dehiscence, replacement with a Marlex tube, restenosis at the tube suture line, and a local plastic operation. Staged reconstruction with a full-thickness autogenous skin tube supported by plastic rings (Grillo, 1965) was successfully accomplished. Two cases of staged skin-tube reconstruction were performed by Pearson *et al.* (1968). Their case 9 had a staged reconstruction for a stomal stenosis, a cuff stenosis having previously been treated by resection and end-to-end anastomosis on two occasions. Their case 19 developed wound dehiscence on the fifth day after resection and end-to-end anastomosis and was then treated by skin-tube reconstruction. Restenosis occurred after five weeks and was treated by dilatations, with unstated results. Neville (1969) advised using a

graft of Silastic with a Dacron sewing ring (Dow Corning) for long strictures, and perhaps bifurcation grafts for carinal lesions. He gave no results.

One of the cases in my series was treated by skin-tube reconstruction. The patient developed acute disseminated encephalomyelitis when between two and three months pregnant, for which she was treated by tracheostomy and assisted ventilation. At extubation on 1 February 1962 the patient became obstructed due to a cuff stenosis, and the tube was replaced with difficulty. She was delivered by Caesarian section on 28 May 1962 and skin-tube reconstruction was performed on 27 August 1962. She died on 11 September 1962. Necropsy showed severe pressure necrosis with cartilage erosion at the site of the cuff, and necrosis also at the site of the lower end of the tracheostomy tube.

Too few reports of resection and reconstruction for post-tracheostomy stenosis have been collected to assess the results. Of the seven quoted above in which results were given, one patient died, one developed restenosis, and five cases were successful. Several of the cases were complicated ones. This type of operation is probably needed only for double stenoses or after previous failed resections.

Tracheoplasty without resection There are a few reports of the use of tracheoplasty, without resection, in the treatment of post-tracheostomy stenoses. Thus Murphy *et al.* (1966) treated one of their four cases of stomal stenosis by tracheostomy below the stenosis followed one month later by a Z-plasty. After wide exposure the trachea was incised above and below the stenosis and flaps were constructed to swing across the stenosed area as a Z-plasty to widen the tracheal diameter. Tracheoscopy two weeks later showed some narrowing at the operation site but no significant stenosis. The writers stated that the operation seemed simpler than resection and end-to-end anastomosis. This type of reconstruction should not be confused with the Z-plasty (Narodick *et al.*, 1965; Worman *et al.*, 1966) previously described to relieve tension after resection and end-to-end anastomosis. Kennedy (1968) successfully treated an adherent, calcified stomal stenosis by incising it longitudinally and then inserting an oval-shaped free cartilage graft. Ekedahl and Laage-Hellman (1967) treated two of their four stenoses (sites not stated) by tracheoplasty. No technical details were given; the results were poor, and in both cases permanent tracheostomy was required. Oliver *et al.* (1962) treated one of their three cases of stenosis (site not stated) by tracheoplasty, but no details are given.

There is probably little indication for reconstruction of this type, though it might be considered in a non-circumferential stenosis at stomal level if problems arose making resection too hazardous.

TECHNICAL CONSIDERATIONS WITH REGARD TO OPERATIVE REPAIR

MANAGEMENT OF RESPIRATION Anaesthesia is begun with an endotracheal tube passed so that its end is above the stenosis. When the trachea has been mobilized and the stenosis located the trachea is divided below the stenosis and the distal stump is cannulated with a cuffed tube for continuance of anaesthesia (Mathey *et al.*, 1966; Deverall, 1967; Byrn *et al.*, 1967; Grillo, 1969a; Jewsbury, 1969). If the distal stump is short the cuff may have to be inflated in the right main bronchus. Although this will occlude the right upper lobe orifice adequate ventilation is still possible (Byrn *et al.*, 1967). After resection has been performed and the posterior two-thirds of the anastomosis has been completed the endotracheal tube is removed from the distal stump and the oroendotracheal tube is advanced beyond the incomplete anastomosis into the distal stump, or right main bronchus, for continuance of anaesthesia. The anterior layer of the anastomosis is then completed.

An alternative method of management is to establish cardiopulmonary bypass, after the necessary dissection, and during the period of tracheobronchial division and anastomosis. Neville (1969) used this method on 35 cases of benign and malignant disease of the trachea and carina. He stated that it was easy to use, and that there had been no complications directly relating to this technique. On the other hand, Grillo (1969a) thought that this was an unnecessary and potentially complicating manoeuvre. Mathey *et al.* (1966) failed to see the rationale of cardiopulmonary bypass because the difficult period ceases when the distal bronchial tree becomes available for cannulation. They conceded, however, that in the case of a seriously obstructed trachea institution of partial cardiopulmonary bypass before induction of anaesthesia could be helpful.

Cardiopulmonary bypass was not employed in any patient in my series, and, in my opinion, it must rarely be necessary. The two circumstances

in which it might be considered are unusually severe obstruction or the necessity for carinal resection. The latter can, however, readily be performed without cardiopulmonary bypass. In my view, the routine use of cardiopulmonary bypass would increase the morbidity and mortality in resections of post-tracheostomy stenoses.

PATHOLOGICAL CONSIDERATIONS With regard to resection three important aspects of the pathology of post-tracheostomy stenoses are their shortness (Grillo, 1969a ; Cooper and Grillo, 1969 ; James *et al.*, 1970), the frequency of severe associated peritracheitis (Miscall *et al.*, 1963 ; Grillo, 1969a), and the occasional difficulty in locating them at the time of operation. Although usually easy to detect as an hour-glass constriction with loss of cartilage at its centre (Grillo, 1969a ; Cooper and Grillo, 1969), or as an area of thickening of the tracheal wall (Deverall, 1967), sometimes no constriction can be seen from without in cuff stenoses (Grillo, 1969a), and the stenoses may be very difficult to locate.

The shortness of most stenoses means that resection with end-to-end anastomosis can nearly always be achieved. The severe peritracheal adhesions make mobilization difficult, and great care must be taken of the blood supply and of the recurrent nerves. The oesophagus is often adherent and it is, therefore, helpful to have an oesophageal tube in place (Grillo, 1969a). A useful way of locating the site of an infrastomal stenosis before starting the operation is to insert a fine needle through the skin and anterior wall of the trachea at the level of the upper border of the manubrium sterni and to observe its site, relative to the stenosis, with a bronchoscope.

BLOOD SUPPLY OF THE TRACHEA The blood supply of the lower part of the trachea is free so that the lower trachea may be extensively mobilized, and the carina transplanted, without endangering viability (Mulliken and Grillo, 1968). The blood supply of the upper trachea is less good. It is segmental, and there is no longitudinal vessel in man, as there is in the dog (Mulliken and Grillo, 1968). This part of the trachea is supplied most commonly from the inferior thyroid artery by upper, middle, and lower branches (Grillo *et al.*, 1964). The upper branch supplies chiefly the thyroid gland but sends a smaller branch laterally to the trachea, as well as supplying anastomosing vessels from the thyroid gland to the anterior part of the trachea. The middle and lower arteries enter the trachea laterally and subdivide anteriorly. Small vessels also travel posteriorly to anastomose with oesophageal vessels, which enter the posterior tracheal wall. Fine anastomotic arcades only join the three arteries along the lateral tracheal wall.

OPERATIVE APPROACH The great majority of reported operations have been performed through a cervical collar incision (Deverall, 1967 ; Pearson *et al.*, 1968 ; Grillo, 1969a ; Jewsbury, 1969), a sternal split, with or without a cervical collar (Binet and Aboulker, 1961 ; Mathey *et al.*, 1966 ; Byrn *et al.*, 1967 ; Pearson *et al.*, 1968 ; Grillo, 1969a), or a right thoracotomy (Flavell, 1959 ; Mathey *et al.*, 1966 ; Pearson *et al.*, 1968 ; Miller, 1969). Grillo (1969a) always used a cervical collar in association with a sternal split, and occasionally used an extension into the right pleura through the fourth intercostal space for mobilization of the right lung hilum and intrapericardial dissection of the pulmonary vessels. This extension is rarely required for post-tracheostomy strictures. Jewsbury (1969) used a collar incision, with a split of the manubrium in only one of his two cases of cuff stenosis. Borrie (1960) repaired a fibrous stomal stenosis in an infant with a stent and skin graft (*vide supra*) through a vertical cervical incision. The 11 resections with end-to-end anastomosis, and the tube-skin graft repair, in my series were all performed through sternal split incisions.

Stenoses at stomal level or above can be approached satisfactorily in most cases through a cervical incision. Thus of eight stomal stenoses repaired by resection and end-to-end anastomosis by Pearson *et al.* (1968), a cervical collar alone was used in seven and a sternal split incision was added in the eighth case. The authors claim that the cervical incision gave good or excellent exposure for all the stomal stenoses. In these high stenoses I advise starting the operation through a cervical collar and splitting the sternum later if necessary.

For stenoses at cuff or tube-tip level I advise a sternal split approach, either with a vertical extension into the neck, or with a small additional collar incision. Most cuff stenoses lie behind the upper part of the manubrium sterni and are difficult to approach from the neck, although this is evidently possible, for Grillo (1969a) resected eight cuff stenoses through a cervical collar incision. The anastomosis can almost always be made above the level of the innominate artery, and the left innominate vein does not usually require division.

The alternative incision which has been used for the repair of cuff stenosis is a right thoracotomy (Flavell, 1959; Mathey *et al.*, 1966; Pearson *et al.*, 1968; Miller, 1969). Flavell (1959) performed a right thoracotomy through the fourth intercostal space with the patient in the prone position, but other workers have used a standard posterolateral thoracotomy.

For strictures near the lower end of the trachea, as may be caused by the end of a tracheostomy tube, right posterolateral thoracotomy through the fourth or fifth intercostal space gives excellent exposure without damage to important structures. Mathey *et al.* (1966) claim that the lower end of the trachea and the proximal portions of the main bronchi can be exposed better through a sternal split incision than through a right thoracotomy, but the operation is an extensive one which requires division of the left innominate vein, median pericardiotomy, mobilization of the innominate and left common carotid arteries and of the aortic arch and upper part of the descending aorta, and bilateral pleurotomy for mobilization of the lung hila.

Although there is a difference of opinion regarding the operative approach to the various parts of the trachea I feel that a cervical collar is an adequate approach for the great majority of stomal and suprastomal stenoses, and sternal split for the great majority of cuff stenoses. Tube-tip stenoses may be approached through either a sternal split or a right posterolateral thoracotomy, according to their level. If very low and close to the carina, right thoracotomy is preferable.

MOBILIZATION OF THE TRACHEA There is a difference of opinion with regard to the extent and method of mobilization of the trachea. Some feel that mobilization of the trachea should be minimal, especially laterally, to avoid damage to the blood supply (Miura and Grillo, 1966; Mulliken and Grillo, 1968; Grillo, 1969a), and that dissection must be performed carefully adjacent to the trachea to avoid damage to the recurrent nerves, which are left undissected (Grillo, 1969a). On the other hand, Deverall (1967) advised mobilization of the trachea inside its fascial coat as far down into the mediastinum as possible, and Miscall *et al.* (1963) stated that good mobilization of the trachea allows plenty of trachea to be resected.

As guides to this matter, only enough trachea must be mobilized to ensure an anastomosis with minimal tension, but care should be taken to avoid injury to the recurrent nerves, the oesophagus, and, especially in the cervical trachea, the blood supply.

As mentioned previously, modern methods of mobilization (Grillo *et al.*, 1963, 1964; Grillo, 1965, 1966, 1969a; Mathey *et al.*, 1966; Mulliken and Grillo, 1968) permit more than one-half of the trachea to be resected and yet allow end-to-end anastomosis (Grillo *et al.*, 1964; Mulliken and Grillo, 1968; James *et al.*, 1970). Thus in the lower half of the trachea, exposed by right posterolateral thoracotomy, complete mobilization of the right hilum, with division of the right pulmonary ligament, will afford an extra 3 cm intrapericardial dissection of the right pulmonary vessels 0·9 cm, and division of the left main bronchus with its reimplantation into the lower part of the right main bronchus a further 2·7 cm, for a total of 6·6 cm (range 5·7 to 10 cm) (Grillo *et al.*, 1964; Mulliken and Grillo, 1968). Reimplantation of the left main bronchus is a complicated procedure and should be considered only if the carina has to be resected. These extensive procedures are rarely required for post-tracheostomy stenoses. For the upper half or two-thirds of the trachea cervicomediastinal dissection through a cervical collar or sternal split incision, or a combination of the two, with the neck flexed 15 to 35°, allows resection of 4·5 cm (range 3·5 to 6 cm). With added right pleurectomy and right hilar mobilization 5·9 cm can be resected (Mulliken and Grillo, 1968). Neck flexion adds 1·3 cm to the amount that can be removed. Mobilization of the cervical trachea requires division of the thyroid isthmus. The mediastinal trachea and proximal parts of the main bronchi beyond the site of pathology can be gently mobilized by blunt dissection with the finger.

The length of the trachea, as measured in adult cadavers, varies between 10 and 13 cm, with an average of 11·8 cm, and the number of cartilages varies between 11 and 18, with an average of 13·4 (Grillo *et al.*, 1964). It can be seen from these measurements that it is feasible to remove at least half the trachea and still rejoin the ends without prosthetic replacement.

MANAGEMENT OF AN OPEN TRACHEOSTOMY If the tracheostomy is open at the time of resection its management is of importance. If a cervical collar incision is made the tracheostomy opening in the skin should be included in the incision where possible. If the tracheostomy opening is too high for the low cervical collar incision it should be

included in a separate ellipse. If a sternal split incision is used it should be extended upwards to include the tracheostomy opening. If a combined mid-line and collar incision is employed the tracheostomy opening can readily be included in the incision. When a posterolateral thoracotomy incision is used the tracheostomy may be allowed to heal spontaneously without interference.

With regard to the tracheostomy opening in the trachea, this should be included in the resection when possible. This will be possible in stomal stenoses, and often in cuff stenoses. When this is not possible, and the approach has been through a mid-line or collar incision, or both, a new cutaneous opening should be sutured to its edges in order to exteriorize the chronically infected focus (Grillo, 1969a).

MANAGEMENT OF THE STENOSIS Once the trachea has been mobilized and the stenosis located, which may be difficult (*vide supra*), the trachea is divided transversely below the stenosis (Byrn *et al.*, 1967; Deverall, 1967; Jewsbury, 1969; Grillo, 1969a) and a cuffed endotracheal tube is inserted into the distal segment, after taking swabs for bacteriology (Deverall, 1967). The initial opening in the trachea below the main stenosis should be through an area of minimal narrowing to avoid the removal of more trachea than is necessary. The trachea is then opened transversely just above the stenosis, which is then examined and the amount of tracheal involvement is assessed. The smallest amount of trachea which will ensure a good lumen is then resected.

In order to reduce tension on the suture line traction sutures of silk may be inserted into the trachea above and below the area of resection (Miscall *et al.*, 1963), or the lower segment may be secured with tissue forceps and lifted upwards (Deverall, 1967). As already mentioned, flexion of the neck will reduce tension, and so will a mild Trendelenberg position. The anastomosis is made with interrupted sutures. The material is probably not important and several have been used including long-lasting catgut (Mathey *et al.*, 1966), chromic catgut (Pearson *et al.*, 1968), 4–0 Dacron (Grillo, 1969a), silk (Miscall *et al.*, 1963; Deverall, 1967), 3–0 Mersilene (Jewsbury, 1969), and No. 35 stainless steel (Pearson *et al.*, 1968). Grillo (1969a), using Dacron, placed the knots externally, and Deverall (1967), using silk, placed everting sutures, but I do not think that internal knots are a disadvantage. Any doubts on this can be resolved by using catgut.

Sutures are inserted into the posterior two-thirds of the anastomosis and tied while the distal stump is still intubated. This is made easier by the use of an armoured tube bent acutely (Byrn *et al.*, 1967). The endotracheal tube is then removed from the lower segment and the oroendotracheal tube is advanced by the anaesthetist across the suture line into the distal segment, and its cuff is then inflated for the continuance of anaesthesia. The remaining sutures are then inserted but are not tied. The oroendotracheal tube is next withdrawn into the proximal segment in order to relax the distal segment, and the sutures are then tied (Mathey *et al.*, 1966). In stenoses located in the neck the suture line may be reinforced with strap muscle (Grillo, 1969a).

RELEVANT POINTS IN POSTOPERATIVE MANAGEMENT A few such points are worthy of discussion.

Tracheostomy In general, postoperative tracheostomy, especially with a cuffed tube and assisted ventilation, should be avoided after resection and end-to-end anastomosis (Grillo, 1969a; Jewsbury, 1969) because of its many complications, and especially because an inflated cuff adjacent to the suture line may interfere with its healing and cause its disruption. For this reason one should be hesitant about advising resection for a patient assessed as requiring postoperative assisted ventilation. Permanent tracheostomy might be a preferable alternative. A high stenosis is an exception to this general rule. After laryngotracheal anastomosis a tracheostomy performed below the anastomosis gives added security in the immediate postoperative phase. This was employed in two such cases (cases 3 and 5) by Grillo (1969a).

Byrn *et al.* (1967) inserted a cuffed Latex tracheostomy tube through the refashioned tracheostome after resection and end-to-end anastomosis performed through a sternal split incision for a tube-tip stenosis. The distal end of the tracheostomy tube lay across the tracheal suture line. The tube was removed after four days. The patient recovered. Mathey *et al.* (1966) performed tracheostomy at the end of the operation in two of the three resections performed for post-tracheostomy stenoses, but no information was given as to whether or not the tubes were cuffed, or if ventilation was assisted. The first was a stenosis 1·5 cm above the carina in a boy aged 2½ years. Resection with end-to-end anastomosis was performed through a right

posterolateral thoracotomy, after which a high subcricoid cervical tracheostomy was established above the anastomosis in order to reduce tension on the suture line. The authors state that the individual patient will benefit greatly from this procedure. Their second patient was a 68-year-old woman on whom resection and end-to-end anastomosis was performed through a split sternal incision extending into the neck. Tracheostomy was then established 1 cm below the anastomosis. In neither of these two cases did tracheostomy impair healing of the anastomosis.

It is clear from the above that tracheostomy can be performed above or below the anastomosis without jeopardizing the suture line, but in general it should be avoided, and especial care should be taken to ensure that an inflated cuff is not placed adjacent to the suture line.

Endotracheal tubes Oral or nasal endotracheal tubes should be avoided after resection of tracheal stenoses because they may block, from secretions or kinking at soft palate level, irritate the trachea or damage the larynx (Deverall, 1967). The danger of a cuff near the anastomosis has already been stressed.

Moderate cervical flexion In order to relax the anastomosis, moderate cervical flexion was maintained in one case by Mathey *et al.* (1966), by the application of a plaster collar for 10 days.

Steroids Mathey *et al.* (1966) considered the use of steroids in the postoperative period important to prevent oedema of the suture line. They advised starting intravenous therapy on the day of operation, and then continuing administration by mouth for one month before reducing the dosage gradually, to prevent rebound congestion. I am unconvinced about the benefits of postoperative steroids.

SUMMARY OF APPROACH TO TREATMENT

CRICOID (SUBGLOTTIC) STENOSIS

Most of these stenoses occur in infants or young children after prolonged endotracheal intubation for assisted ventilation. This is a serious form of stenosis which is difficult to treat satisfactorily, and the child often suffers years of trouble before he outgrows it.

In severe cases, tracheostomy will have to be instituted below the stenosis, and the stenosis should then be treated by repeating dilatations, if this is possible. In the most severe cases, in which the orifice may be of only pin-hole size, dilatations may prove to be impossible to perform. In such cases permanent tracheostomy may be required, but, in some, differential growth may eventually allow dilatations to be performed and the tracheostomy tube to be removed. If stenosis remains severe, the possibility of resection of the stenosis, with laryngotracheal anastomosis, may be considered, but this should never be attempted in early childhood.

Mild cases may be treated by dilatations alone, without tracheostomy, aided perhaps by steroids or antibiotics. These are performed under general anaesthesia and will usually be required every few weeks in the early stages. Differential growth may ultimately eliminate the need for further dilatations. An alternative method of treatment for the milder cases is the insertion of an obturator, as in one of the three cases reported by Markham *et al.* (1967).

INFRACRICOID STENOSIS

If the measures detailed under 'Prevention' are either not taken or have failed, the infant should be treated by prolonged tracheostomy below the obstruction. During this time the stenosis may be treated by dilatations or stenting, aided, if thought advisable, by steroids and antibiotics. Dilatation of infracricoid stenoses is more satisfactory than that of cricoid stenoses because, in the latter, the ring of the cricoid cartilage limits the size of instrument that can be passed. These measures, together with differential growth, may enable the tracheostomy tube to be withdrawn in due course. If not, tracheostomy will have to be continued. At a later age resection of the stenosis could be considered.

STOMAL STENOSIS

Granulomatous polyps are best treated by bronchoscopic excision. This may have to be repeated. Mild fibrous stenoses may respond to repeated dilatations, but all severe ones should be treated by resection with end-to-end anastomosis as soon as they mature, and whenever the patient is fit enough. Resection and reconstruction with a skin tube may rarely be required in double stenoses or often previous reparative operations have failed. Prostheses should be used only as a last resort and should rarely, if ever, be required. If the patient is too ill for resection permanent tracheostomy will be required.

When fibrous stomal stenosis is accompanied by an area of tracheomalacia the latter should, if possible, be resected with the stricture. If this is not possible, stabilization of the soft area should be considered, for instance with plastic rings, after resection of the stenosis and end-to-end anastomosis have been performed. Pure tracheomalacic stomal stenosis, with resultant inspiratory collapse and obstruction, is an important cause of difficulty in extubating infants and young children. In such cases suturing of the upper margin of the tracheal opening to the skin may be helpful (Fennell, 1962).

Stomal stenosis due to double-barrelling of the trachea in a child is rare. I suggest that operative reduction of the deformity be undertaken by freeing the tracheal window or flap from the skin and subcutaneous tissues. If a flap had been made this should then be sutured back into place in the tracheal defect. It may be possible to pass an oroendotracheal or nasoendotracheal tube across the area of double-barrelling (Deverall, 1967). If so, the tube can be used for anaesthesia, and it could profitably be left *in situ* for several days to act as both a splint and an airway.

Stomal stenosis due to retropulsion of a Björk flap would require either removal or replacement of the flap.

CUFF STENOSIS

All severe cuff stenoses should be treated by resection and end-to-end anastomosis as soon as they are mature and whenever the patient is fit enough. Resection and reconstruction may occasionally be required if two stenoses are present, or if a previous reparative operation has failed. If the patient is not fit enough for resection permanent tracheostomy will be required. The tracheostomy tube must be long enough to pass through the stenosis and should be fenestrated so that the patient can speak.

TUBE-TIP STENOSIS

The principles of treatment of this variety of stenosis are the same as those for stenoses at cuff level.

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