Tracheal lesions after tracheostomy

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There have been many previous reports of tracheal damage following tracheostomy (Björk, 1960; Beaver, 1961; Reading, 1958). It is, however, difficult to estimate the frequency with which these changes occur. The general impression from previous writings is that damage to the trachea is uncommon if certain precautions are observed, and that it seldom leads to serious complications. The findings here are based on the post-mortem examination of patients after tracheostomy; they show that macroscopic damage is in fact common. The site and degree of damage is noted and the findings are discussed in relation to certain factors that may affect the amount of trauma inflicted on the trachea.

CLINICAL MATERIAL

The observations are made on 23 patients who died during a series of 37 consecutive tracheostomies. Most of the patients had undergone open heart surgery and all except one died from causes unrelated to the tracheostomy. The indications for tracheostomy in the patients who died are shown in Table I.

TABLE I

INDICATIONS FOR TRACHEOSTOMY IN 23 FATAL CASES

	No.
Respiratory failure	16
Sputum retention	2
Prophylactic (respiratory failure)	2
Chest injury	1
Head injury	1
Instrumental rupture of cervical oesophagus	1

MACROSCOPIC CHANGES IN THE TRACHEA These are summarized in Table II. The trachea shows a progressive inflammatory process at the areas of maximum irritation. The mucosa at these sites becomes hyperaemic and then ulcerates to expose the cartilaginous rings. These are in turn eroded by the tube, and in extreme cases they become completely destroyed. This results in an

inflammatory process and pressure necrosis of the peritracheal tissues and may produce an oesophageal fistula or haemorrhage from an adjacent vessel. These effects are produced by mechanical and chemical irritation and local pressure.

The sites at which this reaction was found to occur were the stoma, the lower end of the tube, and the inflatable cuff when this was used. The lower end of the tube, which moves up and down with respiratory movements and with the movements of the tube itself, frequently ulcerated the carina (Figs 1 and 2) and occasionally the right main bronchus. In case 22 this finally resulted in three small broncho-oesophageal fistulae (Fig. 4). It is possible that some of this carinal and bronchial damage is produced by the suction catheter, although only soft rubber catheters were used in these patients.

It is seen that the ulceration in relation to the cuff begins on the anterior wall and is maximal there throughout its course. This is due to the unyielding cartilages that are found in the anterior two-thirds of the tracheal wall. The posterior wall is more yielding and more mobile with no cartilage, and the soft cushion of the oesophagus lies posteriorly. In only three cases was there macroscopic ulceration of the posterior third of the wall at the cuff (Fig. 3). In one of these the tube had been in position for 77 days, and a tracheo-oesophageal fistula resulted (Fig. 4).

Ulceration in relation to the stoma was unusual, probably because a large segment of tracheal wall was excised to allow the tube to be fitted without pressure around it at the stoma. When it did occur it was on the inferior rim of the stoma (Figs 2 and 3).

DURATION OF INTUBATION When death occurred within 24 hours of tracheostomy there were only minimal signs of mucosal irritation. If the tube was *in situ* for two days or more all the patients except two showed marked ulceration at one or more sites. There was a general tendency for the changes to be more severe as the duration of intubation was increased.

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TABLE II

Patient	Duration	Tube	Cuff	Stoma	Level of Cuff	Lower End of Tube	Remarks
1	1 hour	Rubber	Yes				
2	2 hours	Rubber	Yes				
3	9 hours	Rubber	Yes			Mucosal injection	
4	12 hours	Rubber	Yes				
5	12 hours	Rubber	Yes				
6	14 hours	Rubber	No			Mucosal injection	
7	15 hours	Rubber	Yes				
8	24 hours	Silver	No				
9	2 days	Silver	No				
10	2 days	Rubber	Yes	Mucosal ulceration inferiorly	Anterior wall to expose 4 cartilages	Mucosal ulceration of anterior wall and carina	
11	3 days	Rubber	Yes		Anterior wall to expose 1 cartilage		
12	4 days	Rubber	Yes		Erosion of 3 cartilages anteriorly in relation to the innominate artery	Mucosal ulceration of carina and right main bronchus	Hypotension; adrenaline drip 24 hrs.
13	4 days	Rubber	Yes		3 rings exposed in anterior wall	Mucosal ulceration of carina	Cuff released 2-hourly; hypotension and adrenaline drip 18 hrs.
14	4 days	Rubber	Yes		1 ring exposed in anterior wall	Mucosal ulceration of anterior wall	
15	5 days	Rubber	Yes	Cartilage exposed inferiorly	8 rings exposed in anterior wall, 4 showing actual erosion; mucosal ulceration of posterior wall	Mucosal ulceration of carina	Hypotension and adrenaline drip 24 hrs.
16	5 days	Rubber	Yes	Slight mucosal injection along the length of the tube			Adrenaline drip 24 hrs.
17	5 days	Rubber	Yes	Mucosal ulceration inferiorly	3 rings exposed in anterior wall	Mucosal ulceration right main bronchus	Cuff released 2-hourly
18	9 days	Rubber	Yes		5 rings exposed in anterior wall and eroded; mucosal ulceration posteriorly	Circumferential mucosal ulceration	Hypotension and adrenaline drip 24 hrs.
19	11 days	Rubber	Yes		6 rings anteriorly eroded in region of innominate artery		2-hourly deflation of the cuff
20	12 days	Rubber	Yes	Erosion of cartilage inferiorly	2 cartilages exposed anteriorly	Mucosal ulceration right main bronchus	
21	14 days	Silver 3 days; rubber 11 days	No		Mucosal ulceration along length of tube; no cartilages exposed	Mucosal ulceration of carina	
22	15 days	Rubber	Yes	Erosion of cartilage inferiorly	5 cartilages exposed in anterior wall, 2 with actual cartilage erosion		
23	77 days	Rubber	Yes	Almost complete destruction of all cartilages except the lowest two. Erosion of lower border of cricoid cartilage. Large tracheo-oesophageal fistula; 3 small fistulae from right main bronchus to the oesophagus. Erosion of innominate artery producing fatal haemorrhage			

Type of tracheostomy tube All red rubber tubes were of the Morratt Baker type with a fixed flange and a latex cuff (Fig. 5). The silver tubes were of Negus pattern in one patient and Parker type in another. The number of silver tubes does not allow a comparison to be made between these and the rubber tubes. The finding of so many severe reactions is, however, in keeping with the known irritant nature of red rubber.

Most of the patients were on positive pressure respiration for a variable period, and only four patients (6, 9, 11, and 13) were never given artificial respiration. It is difficult to attach the

respirator tubing to the tracheostomy tube without transmitting some drag to the tube, which increases its movement within the trachea. Suspension of the tubing from an angled or flexible arm clamped to the head of the bed was the method generally used in the patients reported here. The severe damage in patient 22 is largely due to an excessively low and deep tracheostomy which made it impossible to use a normal tube because of the curve of the trachea and the short length from stoma to carina. A cut down endotracheal tube was finally used, but no method was found to keep this firmly in position. The continuous

FIG. 1. Patient 21. Shows superficial mucosal ulceration along the whole length of the tube and at the carina.

FIG. 2. Patient 10. Shows mucosal ulceration at the inferior margin of the stoma. The cartilages of the anterior wall in relation to the cuff are exposed, and there is superficial mucosal ulceration of the carina.

FIG. 3. Patient 15. Shows mucosal ulceration to expose the cartilages at the inferior margin of the stoma and of the anterior wall at the cuff. There is also ulceration of the mucosa of the carina, and of the posterior wall at the level of the cuff.

FIG. 4. Patient 23. The white marker shows a fistula from the trachea to the oesophagus and the black marker shows the fistula connecting with the innominate artery. There are three small bronchopulmonary fistulae at the origin of the right main bronchus.

FIG. 5. A red rubber Morratt Baker tracheostomy tube with an inflated latex cuff.

movement of the tube within the trachea undoubtedly produced these remarkable destructive changes (Fig. 4). This was the only patient in whom the tracheostomy was directly responsible for death. In patient 21, who was not artificially respired and had a well fitting uncuffed tube, the changes produced were minimal in spite of 14 days' intubation.

Management of the cuff It has been suggested that regular deflation of the cuff for short periods will reduce the pressure necrosis of the mucosa. In most of the patients in this series the cuff was only deflated once a day as it was felt that the risk of inhalation of pharyngeal secretions made more frequent deflation undesirable. In the three patients in whom the tube was deflated at two-hourly intervals, there was no significant decrease in the reactions.

SYSTEMIC FACTORS Most of these patients were in a state of gross metabolic imbalance and circulatory failure, which may affect their resistance to pressure and irritation. The frequent occurrence of long periods of hypotension associated with compensatory vasoconstriction, sometimes enhanced by the use of vasoconstrictor drugs, is of particular importance. In these patients the circulation to the tracheal mucosa is grossly deficient. Some patients were also given steroids

in large doses, which would reduce their healing response to the irritative process.

LOCAL INFECTION All except two of the patients who died two days or more after tracheostomy had bacterial infections of the respiratory tract which was confirmed by culture. This was frequently a virulent infection with *Staphylococcus aureus*. The other two patients had monilial infections of the respiratory tract. The presence of such infections will increase the damage initiated by aseptic mechanical factors.

CLINICAL SIGNIFICANCE OF THE TRACHEAL LESIONS

It appears from this study that, although these changes are common in tracheostomies after two days or more, they seldom result in complications of clinical importance. Although 23 patients died in a series of 37 consecutive tracheostomies, the cause of death was related to the tracheostomy in only one patient, who died from erosion of the innominate artery on the seventy-seventh day (Figs 4 and 6). All the other patients died of some unrelated condition. Two other patients had haemorrhages from their tracheostomies. Patient 20 had massive bleeding on the fifth and seventh post-operative days. No other cause for this was found at necropsy apart from the ulceration of the

FIG. 6. Patient 23. Shows the innominate artery and its fistulous opening, marked with a black marker.

mucosa. Patient 18 had a small haemorrhage on the fifth day which may have been precipitated by anticoagulation therapy for dialysis on the artificial kidney. Patients 12 and 19 showed deep ulceration of the tracheal wall in relation to the innominate artery on the eleventh day, but this was not yet directly involving the artery. One of the patients who survived after tracheostomy had a large haemorrhage on the third day and a smaller haemorrhage on the sixth post-operative day, for which no cause could be found.

On these observations it therefore seems unlikely that the ulceration is of clinical significance unless intubation is continued for a long period. None of the surviving patients showed clinical evidence of late tracheal stenosis.

DISCUSSION

These findings suggest that with a red rubber tracheostomy tube damage to the mucosa is common. An inflatable latex cuff and artificial ventilation only increase this damage, which is maximal at the cuff but also occurs at the lower end of the tube and at the stoma. The tracheostomy tube produces its effect for a number of reasons. It is an irritant foreign body which produces an inflammatory reaction in the mucosa. Movement of the tube within the lumen of the trachea leads to mechanical trauma of the mucosa, and the pressure of the cuff produces mucosal ischaemia. The bypassing of the nasal passages results in deficient humidification and leads to drying of the mucosal secretions and poor lubrication of the mucosa. A fall in systemic blood pressure and the compensatory vasoconstriction which is sometimes augmented by vasoconstrictor drugs will increase the local ischaemia. Other systemic factors, such as renal failure or steroid therapy, may have an undesirable effect.

In spite of their frequency, these reactions seldom result in serious complications unless intubation is continued for long periods. It is not therefore a contraindication to tracheostomy when this is firmly indicated, but an inflatable cuff should only be used where artificial ventilation is required or when there is a risk of inhalation of pharyngeal secretions. Furthermore, the tube should be removed as soon as it is safe to do so. Now that suitable tubes of polyvinyl plastic are available it seems unreasonable to continue using the more irritant red rubber tubes. The tube described by Whittard and Thomas (1964) seems suitable. It is a Marrant Baker type of tube made of polyvinyl plastic with an inflatable cuff of

similar material. It has a fixed flange which reduces the movement of the tube within the trachea (Hewlett and Ranger, 1961). The general method of fixing the tube by two tapes from the flange tied around the neck is improved by the addition of two further tapes passed under the arms and around the chest (Matheson, Gardiner, Low, and Dudley, 1963). The connexion with the respirator presents the problem of allowing the patient sufficient freedom of movement without allowing the tubing to drag on the tracheostomy tube. Suspension of the tubing from an adjustable arm is the most satisfactory method of reducing this drag.

The risk of the end of the tube damaging the carina or entering the main bronchus and occluding the contralateral bronchus has been mentioned. Whittard and Thomas (1964) suggest the use of a blank flange inserted between the fixed flange and the stoma to reduce the effective length of the tube.

It is not possible to draw any valid conclusions from these observations on the importance of regular deflation of the cuff. The limited data available suggest that it has no effect in reducing the damage. Beaver (1961) says that regular letting down of the cuff may in fact increase tracheal damage by allowing increased movement of the tube within the trachea.

The prevention and control of respiratory infection and the maintenance of adequate humidification are difficult to achieve, but they may help to reduce the tracheal damage. The prophylactic use of broad spectrum antibiotics is often followed by infection with resistant staphylococci or Gram negative organisms and is of doubtful value. The regular culture of tracheal suckings and the control of antibiotic therapy by the estimation of sensitivities is essential. The routine use of an aseptic technique for tracheal toilet is, of course, essential but is not effective in preventing infection.

The careful control of the blood volume to maintain the systemic blood pressure without the use of vasoconstrictor drugs will improve the local circulation and reduce the effects of local pressure and irritation.

SUMMARY

In a series of 37 consecutive tracheostomies, 23 patients died. The trachea was examined at necropsy in each of these 23 patients.

The lesions were found at the stoma, the cuff, and the lower end of the tube. They increased with the duration of intubation.

Other factors that may influence the severity of these lesions are discussed.

Although tracheal damage is common after 48 hours' intubation it is rarely of clinical significance. Only one patient died as a result of his tracheal lesion.

It is suggested that the red rubber tube should be abandoned in favour of a polyvinyl plastic tube.

Further suggestions are made that may help to reduce the incidence and severity of the damage.

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