

Caustic injury of the oesophagus. Sixteen years experience, and introduction of a new model oesophageal stent

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Abstract

Caustic ingestion can lead to oesophageal stenosis. We studied 251 patients, 205 of whom were children, in a sixteen-year period. Seventeen patients, of whom one was an adult, acquired deep burns in the oesophagus which had to be treated, to prevent the development of oesophageal strictures. These patients were treated with long-term stenting of the oesophagus with specially designed, silicone rubber stents, impregnated with silicone oil 20 cS, designed by one of us (R.N.P.B.) as the only treatment. Of all models, the twin-tube dilatator was the most satisfactory. No corticosteroids were administered. Only one patient developed a mild stenosis. It is therefore our opinion that, when life-saving operations are not indicated, twin-tube stenting of the oesophagus is helpful in treating caustic lesions of the oesophagus and will prevent stricture formation. Corticosteroids were not given in this series, and should be abandoned in the treatment of caustic lesions.

Key words: Caustics; oesophageal stenosis; Stents

Introduction

The results of the treatment of oesophageal lesions, due to caustic ingestion with lye are discussed. The use of different models of silicone rubber stents is evaluated and a new model silicone rubber stent is introduced (Figures 1 and 2). The silicone rubber of the stents is impregnated with silicone oil (20 centiStokes). This results in a 60 per cent increase in weight as the oil is absorbed by the stent (Berkovits *et al.*, 1978). During usage of the stent, the oil seeps out slowly and continuously in very small quantities, thus creating a system of weeping and boundary lubrication (Berkovits *et al.*, 1978). In this way direct contact between the stent and the mucous membrane is avoided, while mucus cannot adhere to the stent due to the film of silicone oil which covers it permanently. Tissue culture testing with human fibroblasts showed the material to be non-toxic (Berkovits *et al.*, 1987) and friction tests demonstrated extremely good friction properties, comparable with the friction of ice on ice (Bos *et al.*, 1973; Berkovits *et al.*, 1978; Berkovits *et al.*, 1987). Thus recovery and growth of mucous tissue is promoted, owing to the combination of low-friction, non-toxicity and the extremely pliable material of the stent.

Materials and methods

After inspection of the oral and hypo-pharyngeal cavity, endoscopy is performed with a rigid or a flexible endoscope (Hawkins *et al.*, 1980). The choice between a rigid or a flexible scope has to be made depending on the experience of the investigator.

To evaluate the condition of the stomach, of the pylorus and the proximal duodenum, flexible endoscopy has to be performed. The endoscopic evaluation is performed within 24 hours. Some investigators prefer to perform the evaluation later (up to 72 hours) because the extent of the lesions is then more pronounced. However, the danger of perforating the vulnerable oesophagus with the instrument then increases considerably.

The interpretation of the visible lesions is not always easy, and the extent of the penetration of the caustic agent is difficult to evaluate. Extensive lesions in the oral cavity are not always accompanied by lesions in the oesophagus or in the stomach. The opposite can also be true, when no lesions are seen in the oral cavity, but serious burns may be found further on in the digestive tract (Haller *et al.*, 1971; Kirsh and Ritter, 1976). Contrast X-ray investigation sometimes shows the absence of motility of the oesophagus, as lesions, extending into the muscular layer inhibit normal peristalsis of the oesophagus.

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Caustic lesions of the oesophagus are classified in three grades by Kirsh and Ritter (1976) and Kirsh *et al.* (1978):

Grade 1: Erythema and oedema. Grade 2: Ulceration, not circular. Grade 3: Circular ulceration, haemorrhages and necrosis.

Therapeutic developments

Traditionally a P.V.C. plastic stomach tube used to be introduced (Fr 12–16) for two to six weeks. After subsequent removal of the tube, endoscopy and contrast X-ray cinematography were performed. During the use of P.V.C. plastic stomach tubes, the plasticizer was observed to 'leak' out of the nasogastric tube, as a result of which the tube obviously hardens and becomes inflexible. This process is generally completed within two weeks. This plasticizer can exert a toxic effect on the mucous membrane (Bos *et al.*, 1973). The stiffened tube can then cause additional mechanical lesions in an already vulnerable oesophagus (We observed that P.V.C. nasogastric tubes used in intensive care treatment, can even cause haemorrhagic lesions in the normal oesophagus and stomach; such lesions disappear after replacing the P.V.C. stomach tube by a silicone-rubber one). In cases of severe caustic lesions, a gastrostomy is performed and a 'continuous' string introduced. In cases of exceptionally destructive lesions, oesophagectomy and/or gastrectomy may be tried as life-saving measures; such severe conditions were not seen in our patients series.

If a stenosis is developing or has developed, some authors advise intermittent dilatation of the oesophagus with different types of dilators. However, scar-tissue in the healing lesions is then ruptured and formation of new scar tissue will be unavoidable. Amazingly enough, this procedure occasionally seems to be successful.

In our opinion, a thin indwelling nasogastric tube will not prevent stricture formation and should not be used routinely, but only when the necessity to place a dilatator is as yet not evident. If this is the case, we prefer permanent dilatation with custom-made silicone oil-impregnated silicone rubber dilators for the treatment of cases of a developing or established stenosis.

Initially, a cylindrical siliconized silicone rubber stent (12 mm outer diameter in adults) was used for this purpose, for the treatment and the prevention of stricture formation (Bos *et al.*, 1973; Reyes and Hill, 1976; Berkovits *et al.*, 1978; Mills *et al.*, 1979; Tucker, 1979; Hawkins *et al.*, 1980; Claudel-Bonvoisin *et al.*, 1982; Dehesdin *et al.*, 1982; Berkovits *et al.*, 1987; Wijburg *et al.*, 1989; Anderson *et al.*, 1990).

In order to improve swallowing, we decided to introduce a ribbon dilatator. Our second patient (Table I) felt that this indeed facilitated food intake. To further improve swallowing we proceeded to construct a dilatator made of two thin tubes, only glued together at the proximal and distal end, both over a length of 2 cm. The total length of the twin dilatator for Case 2 was 36 cm; the diameter of each tube was 6 mm (Figures 1 and 2).

As expected, the two separate tubes sought their position post-cricoidally at the point of minimal pressure, to the left and the right of the midline. Since this also happened in the oesophagus, the space between the two tubes improved the efficiency of swallowing. This was actually confirmed in our second patient, the first patient to be treated with this twin-dilatator tube. After use of the ribbon-dilatator, finally the twin-dilatator was introduced, which she found to be still better for consuming solid food alongside the twin-dilatator. We hypothesise that the possibility of early eating and drinking will help to advance dilatation of the stenosis and it indeed improves the feeling of well-being of the patient.

TABLE I
CLINICAL DATA OF 17 PATIENTS FOLLOWING INGESTION OF CAUSTIC LYE SOLUTION

Patient no	Endoscopy	Treatment	Follow-up
1 4 years male	Grade 1 lesion throat Grade 3 lesion oesophagus	Gastrostomy, string Gastrostomy tube Intermittent dilatation Cylinder dilatator	16 years eating solid food
2 14 years female	Grade 3 lesion throat Grade 3 lesion oesophagus	Gastrostomy, string dilatation Cylinder dilatator Ribbon dilatator Twin tube dilatator	12 years eating solid food
3 30 years female	Grade 1 lesion throat Grade 3 lesion oesophagus	Gastrostomy, string Cylinder dilatator	10 years eating solid food
4 4 years male	Grade 1 lesion throat Grade 3 lesion oesophagus	Gastrostomy, string Cylinder dilatator Twin-tube dilatator	2 years eating solid food
5 5 years male	Grade 2 lesion throat Grade 3 lesion oesophagus	Gastrostomy, string Twin-tube dilatator	1 years eating solid food
6 4 years male	Grade 1 lesion throat Grade 3 lesion oesophagus	Gastrostomy, string Twin-tube dilatator	9 months eating solid food
7–17	Grade 1 lesion throat Grade 3 lesion oesophagus	Cylinder dilatator	6–8 years all eating solid food (16)

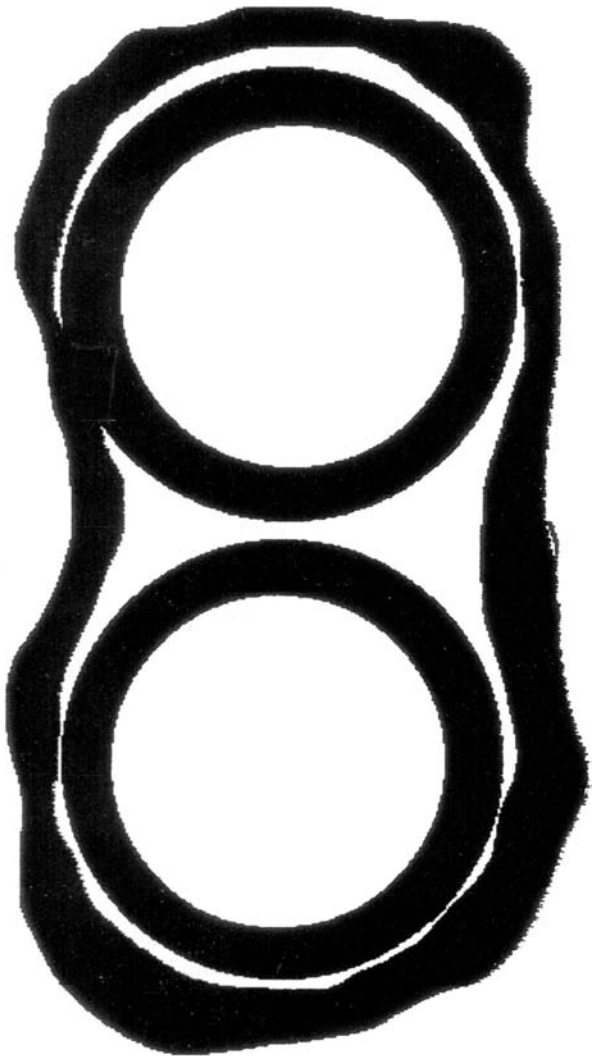


FIG. 1

Schematic cross-section of twin-tube oesophageal dilatator in the oesophagus.

Fixation and medication

The upper end of the dilatator is equipped with two threads, covered with silicone rubber, which are led out of each nostril and then knotted around the colummella nasi (Figures 1 and 2).

The end of the dilatator lies in the stomach and is equipped with a thread, also covered with silicone rubber. This thread is led out of the gastrostoma and fixed on to the abdominal skin.

The dilatator tubing is occluded proximally and distally; food intake takes place alongside the dilatator; in our patients, this never presented a problem. Initially, food was given through the gastrostomy, but oral fluid intake was commenced after a few days.

The tubes are custom-made, according to our specifications; length and diameter are individually decided upon and eventually changed in the course of the treatment during the three-weekly changings of the dilatator, repeated endoscopies can provide information on the progress of the treatment.

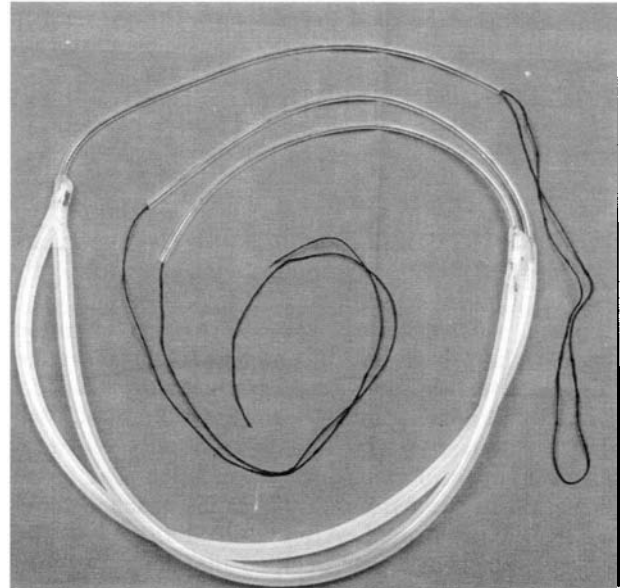


FIG. 2

Silicone-oil impregnated twin-tube dilatator. The two tubes glued together only at the upper and at the lower end. Two silicone-rubber covered threads coming out of the upper end for fixation around the colummella nasi, and one at the lower end, to be secured on the abdominal wall.

In order to prevent the growth of *Candida albicans* or other organisms into the dilatator material and into the damaged oesophageal mucosa, oral Miconazole gel (Daktarin, Janssen Pharmaceutical) was administered three hourly (2 cm gel) for the duration of the stenting. The schedule of the three-weekly changing of the dilatator was based on the observation that its lubricating properties are then depleted, and *Candida* tends to grow in the dilatator. If indicated (mediastinitis), antibiotics were given. The systemic use of corticosteroids remains a controversial issue; they were not administered to our patients (Haller *et al.*, 1971; Hukins *et al.*, 1980; Wijburg *et al.*, 1989; Anderson *et al.*, 1990). One must be aware of the possibility of reflux of gastric contents, which must then be treated with ranitidine or anti-reflux medication such as ranitidine.

Results

From 1975, we have treated 17 patients with stage 3 lesions (according to the classification of Kirsh). Out of a total of 251 patients who had ingested caustic substances, 205 patients were children.

Patients 1, 2 and 3 were treated in Rotterdam, The Netherlands. Patients 4 and 5 were treated in Cologne, Germany. Patient 6 was treated in Spain. Patients 7–17 were treated in Amsterdam, The Netherlands (Wijburg *et al.*, 1989). All patients were treated with cylinder dilatators except patients no. 4 and 5, to the design of the first author. Patients no. 2, 3, 4, 5 and 6 were additionally treated with ribbon type dilatators and with twin-tube dilatators. Diameter of cylinders: 10 to 12 mm in adults, 5 to 10 mm in children from age 5 to 10. Diameter of twin tubes: twice 4 to 6 mm (Figures 1 and 2). Duration of

dilatation: six weeks to twelve years (average three months).

Case report

Case 2

A female, 14 years of age, ingested liquid lye solution in 1983. Inspection of mouth and hypopharynx showed severe Grade 3 lesions. Endoscopy showed Grade 3 lesions of the oesophagus, the stomach was not involved. X-ray investigation showed that there was no peristalsis in the mid-third of the oesophagus. A gastrostomy was performed and a string introduced in combination with a P.V.C. nasogastric tube. Eventually, airway-obstruction developed, due to excessive scar tissue formation in the hypo-pharynx and a tracheostomy had to be performed. After consultation with the thoracic surgeon, it was decided that a resection of the oesophagus was impossible, due to the difficulty of making a satisfactory anastomosis in the scarred pharynx. The scarred hypopharynx which totally occluded the larynx, was incised left and right of the position of the invisible epiglottis after injecting the incision site with depot-corticosteroids. This procedure had to be repeated after three weeks.

A cylindrical dilatator (11 mm) was introduced in the oesophagus, which was replaced every three weeks. The larynx remained free of obstruction and the patient could be decannulated. Initially, food had to be given through the gastrostomy, soon drinking alongside the dilatator was possible. In a few months the patient could swallow solids alongside the dilatator. After two years we tried to remove the dilatator, but in a few hours the oesophageal stenosis was present again. A cylindrical stent was re-introduced. After five years this cylindrical stent was changed for a ribbon dilatator, to be replaced after six months by the twin-tube dilatator which led to a significant subjective improvement in food consumption. After twelve years, the stent has now been removed and patient is able to eat and drink normally.

Discussion

The tendency to develop a stenosis after caustic burns of the oesophagus is mainly related to the extent of the lesion. If the lesion is deeply penetrating, a stricture is more likely to occur. The individual reaction of the patient is also of some importance. It is our impression that the tendency to produce abundant scar tissue of the skin (keloid), plays a role.

The principle of conservative treatment of oesophageal strictures should no longer be associated with the application of repeated dilatations in the first place.

The final diameter of the stenosis will be determined by the diameter of the long-term dilatator. Experimental and clinical surgical evidence shows that scar tissue progressively contracts up to six months after the start of the healing process. Therefore, continuous dilatation has to be per-

formed for a long time, with a range in our patients from six weeks to twelve years (average three months).

Conclusion

The treatment of oesophageal strictures after caustic ingestion should be performed with long-term dilatation of the esophagus during a sufficiently long period. Of all models tried, the use of the silicone-rubber, silicone oil impregnated, twin-tube oesophageal dilatator is an improvement in this therapy. This dilatator allows greater comfort to the patient and facilitates swallowing of food and drink without causing pressure-induced post-cricoidal ulcers.

The alternative for the above-mentioned long-term dilatation therapy could be reconstructive surgery. These interventions are not always successful and carry some morbidity. When there are extensive lesions in the pharynx, a proximal anastomosis will pose problems, as stricture formation is inevitable at the anastomosis site, where the anastomosis has to be constructed in cicatricial tissue Meredith *et al.* (1988). However, if long-term dilatation fails, there always remains the possibility of surgical intervention.

It is advisable to consult the gastro-enterologist and the thoracic surgeon to decide which course of therapy will be of maximum benefit to the patient.

Finally, the risk of the development of an oesophageal carcinoma has to be taken into consideration. This risk amounts to one to four per cent up to thirty years after the caustic ingestion Hager *et al.* (1986). This should also be kept in mind, when considering the morbidity of primary surgery.

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References

- Anderson, K. D., Rouse, T. M., Randolph, J. G. (1990) A controlled trial of corticosteroids in children with corrosive injury of the esophagus. *New England Journal of Medicine* **323**: 637-640.
- Berkovits, R. N. P., Bos, C. E., Pauw, K. H., de Gee, A. W. J. (1978) Congenital cricoid stenosis, pathogenesis, diagnosis and method of treatment. *Journal of Laryngology and Otology* **92**: 1083-1100.
- Berkovits, R. N. P., van der Schans, E. J., Molenaar, J. C. (1987) Treatment of congenital cricoid stenosis. In *Progress in Pediatric Surgery*, vol 21, (Wurnig, P., ed.) Springer Verlag, Berlin, Heidelberg, pp 19-29.
- Bos, C. E., Berkovits, R. N. P., Struben, W. H. (1973) Wider application of prolonged endotracheal intubation. *Journal of Laryngology and Otology* **87**: 263-279.

- Claudel-Bonvoisin, S., Morgon, A., Bontemps, P., Moulinier, B., Pansu, M. (1982) Nouveau protocole thérapeutique pour prévenir les sténoses caustiques oesophagiennes: intérêt d'une sonde de calibrage en Silastic. *Acta Endoscopica* **12**: 191–197.
- Dehesdin, D., Andrieu-Guitrancourt, J., Ingouf, G. O., Peron, J. M. (1982) Brûlures caustiques de l'oesophage chez l'enfant. A propos de l'endoscopie initiale et du calibrage de principe. *Annales Otolaryngologiques* **99**: 497–504.
- Hager, J., Aigner, F., Menardi, G. (1986) Oesophagusverätzungen im Kindesalter. Analyse von Diagnostik und Therapie unter Berücksichtigung des eigenen Krankengutes (96 kinder). *Padiatrische Praxis* **34**: 455–467.
- Haller, J. A., Gibbs Andrews, H. G., Wite, J. J., Akram Tamer, M., Cleveland, W. W. (1971) Pathophysiology and management of acute corrosive burns of the esophagus; result of treatment in 285 children. *Journal of Pediatric Surgery* **6**: 578–584.
- Hawkins, D. B., Demeter, M. J., Barnett, T. E. (1980) Caustic ingestion. Controversies in management. A review of 214 cases. *Laryngoscope* **90**: 98–109.
- Kirsh, M. M., Ritter, F. (1976) Caustic ingestion and subsequent damage to the oropharyngeal and digestive passages. *Annals of Thoracic Surgery* **21**: 74–82.
- Kirsh, M. M., Petersen, A., Brown, J. W., Orringer, M. B., Ritter, F., Sloan, H. (1978) Treatment of caustic injuries of the esophagus: A ten-year experience. *Annals of Surgery* **188**: 675–678.
- Meredith, J. W., Kon, N. D., Thompson, J. N. (1988) Management of injuries from liquid lye ingestion. *Journal of Trauma* **28**(8): 1173–1180.
- Mills, L. J., Estrere, A. S., Platt, M. R. (1979) Avoidance of esophageal stricture following severe caustic burns by the use of an intraluminal stent. *Annals of Thoracic Surgery* **28**: 60–65.
- Reyes, H. M., Hill, J. L. (1976) Modification of the experimental stent technique for esophageal burns. *Journal of Surgical Resources* **20**: 65–76.
- Tucker, J. A., Yarrington, C. T. (1979) The treatment of caustic ingestion. *Otolaryngological Clinics of North America* **12**: 343–350.
- Wijburg, F. A., Heymans, H. S. A., Urbanus, N. A. M. (1989) Caustic esophageal lesions in childhood: prevention of stricture formation. *Journal of Pediatric Surgery* **24**: 171–173.

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