# ACUTE CAUSTIC SODA INJURIES OF THE OESOPHAGUS*

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The widespread domestic use of caustic soda, and the consequent exposure of large sections of the community to the corrosive, has created a distressing sociological problem. This was recognised by Chevalier Jackson (1921) who believed that adequate labelling of all containers of lye would materially lessen the dangers. In 1927 this was made compulsory in the United States, but according to Brown and Kiser (1942) the incidence of poisoning with caustics has continued to increase in that country.

The startling frequency of injuries in Johannesburg, a city of approximately 800,000 people, has formed the subject of a recent communication (Marchand 1954), in which the sociological aspects of the problem as seen in the Union were presented. The cases discussed were admitted to 4 hospitals in Johannesburg between July 1948 and July 1952 and came under the care of the Thoracic Surgery Unit directed by Mr. L. Fatti. During the following year a further 36 cases were treated, making a total of 152 cases in 5 years (Table I). Fifty-five of these patients were admitted with established oesophageal strictures and 97 were seen within a week of sustaining injury.

TABLE I. CASES TREATED BY THE THORACIC UNIT OVER A PERIOD OF 5 YEARS, BY RACE, SEX AND AGE (INCLUDING CASES ADMITTED WITH ESTABLISHED OESOPHAGEAL STRICTURES)

| | *Adults* | | *Children* | *Total* |
|---|---|---|---|---|
| | *Male* | *Female* | (*under* 14) | |
| European .. .. | 7 | 12 | 8 | 27 |
| Coloured .. .. | 9 | 31 | 13 | 53 |
| Indian .. .. | 3 | 11 * | 6 | 20 |
| African .. .. | 12 | 10 | 30 | 52 |
| Total .. .. | 31 | 64 | 57 | 152 |

* 1 Chinese.

The present communication deals with the clinical and therapeutic problems which we have experienced during the management of the 97 cases of acute caustic soda poisoning. All the cases were investigated according to principles formulated within the Unit and the eventual fate of every patient is known. A year has passed since the last case in the series was admitted to hospital.

## CLINICAL COURSE

For purposes of description the clinical course may be divided into 3 stages, viz. (*a*) the acute stage, (*b*) a latent period, and (*c*) the stage of organic stricture.

*The Acute Stage.* Dr. Emma Kohman-Ivy, a physiologist, accidentally swallowed 4 c.c. of a boiling caustic solution and has given a graphic account of her experience (Ivy 1922). Pain was immediate and severe, within 5 minutes she was unable to swallow, and after 20 minutes respiration was difficult. A sequence like this is usual, though patients are seldom able to describe them so concisely.

The duration of the acute symptoms varies with the severity of injury. The pain usually gradually diminishes over the course of 3 or 4 days, the vomiting ceases somewhat sooner, but the acute dyspnoea generally lasts only a few hours.

*The Latent Period.* By the 5th day all but the most severely injured patients are comparatively free from pain, are able to swallow fluids, and are buoyantly confident that the worst is over.

Patients with moderate injuries are usually able to take semi-solids by the end of the 1st week, whilst those with minor damage may have little obviously wrong after 2 or 3 days. With severe oesophageal injury temporary alleviation of dysphagia may never be experienced, whilst with moderate injury there is only occasionally a latent period of complete normality between the acute dysphagia and dysphagia due to organic stricture.

In this series only 4 of the 41 patients who developed circumferential strictures experienced an interval when they were able to swallow solids without any difficulty whatever.

*The Stage of Organic Stricture.* The time of onset and the degree of dysphagia depends upon the site and the rapidity of development of the stricture. In instances of severe oesophageal injury the primary dysphagia, due essentially to the pain of the corrosion, passes imperceptibly into the mechanical dysphagia due to the stricture. It is only with minor injuries that strictures may form so insidiously that the force of swallowing adapts itself to the gradually increasing resistance. In these instances months may pass before the victims become aware of swallowing difficulties.

## SIGNIFICANCE OF SIGNS AND SYMPTOMS

The question constantly in the mind of the medical attendant is whether or not the oesophagus has been corroded. In this series of 97 injuries, 51 patients (53%) escaped without developing significant strictures. Many of these, when first seen, appeared on superficial examination to be critically injured, whilst others, who later developed pronounced strictures, seemed little affected during the early stages. This is not surprising when one considers the different types of people involved. The hysterical suicide, who has little more than brushed her lips with the caustic, may be in great distress, whilst the stoic or the unimaginative may show little emotional upset.

The author has taken especial cognizance of the signs and symptoms during the early stages in the hope that they might assist in the diagnosis of oesophageal involvement.

*Shock.* When first admitted it is not uncommon for the victim to be in a state of shock but, once supportive

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emergency measures are instituted, recovery is usually rapid. Five patients remained in profound shock for more than 12 hours; 3 of them died and 2 developed very tight oesophageal strictures.

*Visible Burns.* It was once thought that the site and severity of visible burns would give a reliable indication of oesophageal damage. A severely corroded mouth is intensely reddened, with areas of desquamation and sites where the epithelium is opaque and coagulated. The lips, palate, uvula and fauces may rapidly become oedematous and the tongue may be so swollen that it protrudes from the mouth. Immediately after injury only a limited area may be involved, yet at a later examination the whole surface of the mouth may be found to be affected by the local extension of the corrosive action.

Amongst the 51 patients who did not develop oesophageal strictures, only 4 had no visible areas of corrosion beyond diffuse erythema of the lips and mouth. The others presented with injuries more or less comparable with those of the patients who later developed strictures (Figs. 1 and 2). All those who had taken solid caustic soda sustained severe oral burns, yet none developed serious oesophageal strictures.

Pharyngeal burns are of greater significance, but their absence in no way excludes the possibility of oesophageal corrosion.

Our experience is wholly in variance with Crowe's view (1944) that oral burns may be considered presumptive evidence of oesophageal injury.

*Pain.* The victims suffer appalling agonies during the first few hours but the intense pain rapidly dulls as the sensory nerve-endings in the mouth and pharynx are destroyed. When massive amounts have been taken this remission is temporary, for extension of the corrosive action beyond the confines of the alimentary tract may cause severe pain to recur. The suffering of patients who develop mediastinitis, peritonitis and pleurisy is only alleviated by profound narcosis. With the majority of cases, however, the significance of pain is difficult to assess because of the wide variation in the reactions of the different types of individuals.

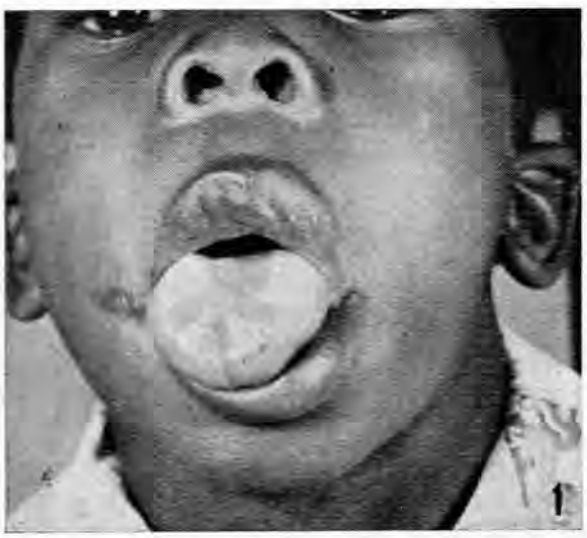

*Fig.* 1. Severe scarring of mouth and tongue following the ingestion of lye 1 year previously. He has developed microstoma and ankyloglossia. The oesophagus is unaffected (Fig. 2).

Burning pain of the mouth and throat, localized particularly to the angles of the jaw, often persists for days and hinders swallowing. Earache is frequent and is probably due to oedema of the Eustachian orifices. On 2 occasions middle-ear suppuration has occurred. Pleuritic pains are secondary to inflammatory changes within the lung resulting from inhalation of caustic at the time of injury or of vomitus or food during the days which follow.

Localization of oesophageal pain, particularly when arising from the upper third, is moderately accurate. Most patients complain of burning in the throat for several days and a diffuse burning substernal pain is common. Cervical and substernal pain does not necessarily imply that the oesophagus is severely corroded. Early oesophagoscopic examinations have repeatedly failed to establish such a correlation and on many occasions, though the patient had complained bitterly of discomfort, the oesophagus was found to be unaffected apart from erythema.

Abdominal pain is a frequent symptom. There are 2 common sites of pain: the region of the xiphisternum and an area to the right of the mid-line beneath the tip of the 9th costal cartilage. No precise correlation has been found between the mid-line epigastric pain and corrosion of the distal oesophagus. Right epigastric pain is probably secondary to pyloric irritation and 3 patients who complained of severe persistent pain in this region subsequently developed pyloric stenosis.

Pain is not a reliable index of corrosion of sufficient degree to cause strictures. Apart from the wide variations in the reactions of different people to a similar pain-producing stimulus, it has been our experience that pain may be slight when damage is considerable and may be severe in the absence of significant organic injury.

*Vomiting.* Whether the trauma be mild or severe, it is usual for patients to vomit shortly after swallowing lye. At first, the vomitus is intensely alkaline and altered blood is present in all but the mildest cases. Shreds of denuded mucosa are usually present, and on 2 occasions casts of considerable segments of the oesophagus were vomited towards the end of the 1st week (Fig. 3). Shreds of tissue, when examined microscopically, are found to consist entirely of epithelium but casts usually include other layers. Witthaus (1911) described an oesophageal cast composed of mucosa, submucosa and part of the muscular coat of the entire organ, including the lower pharynx and epiglottis.

Mucosal shreds denote severe penetrating injury but they do not necessarily derive from the oesophagus. Extensive areas of the epithelium of the tongue, palate and cheeks may exfoliate in the absence of oesophageal involvement. Casts of the oesophagus are easily recognized and indicate that a stricture is inevitable.

*Anaemia.* Eight patients became markedly anaemic within 10 days of injury. All had severe oesophageal corrosion.

Routine serial blood counts have been performed on 52 patients and a progressive decrease of the haemoglobin level has usually accompanied extensive injuries. Caustic

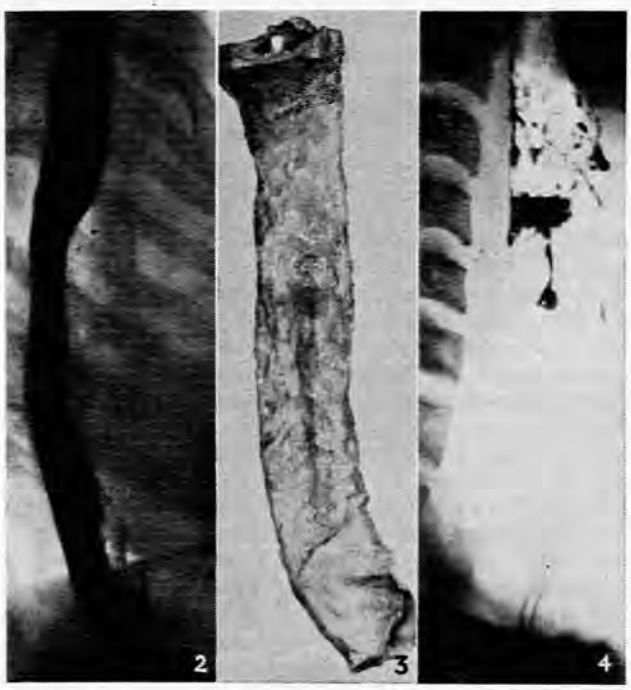

*Fig.* 2. Barium swallow of child shown in Fig. 1. There is no evidence of stricture 1 year after swallowing the lye.
*Fig.* 3. Oesophageal cast 15 cm. long vomited 4 days after the ingestion of lye.
*Fig.* 4. Lipiodol swallow 48 hours after injury. The contrast medium has entered the larynx and trachea.

soda has no direct haemolytic action and it would seem that serial haemoglobin estimations provide an indirect index of the extent of injury.

*Acute Dysphagia.* The dysphagia which follows immediately after corrosive injury is essentially a difficulty in initiating the swallowing reflex. Dr. Ivy experienced dysphagia 5 minutes after the accident and this persisted for 5 days. She localized the difficulty to the back of her throat and not to the oesophageal obstruction which she was to experience later. Carlson (1922) believes that the dysphagia is due to destruction or paralysis of the glosso-pharyngeal nerve-endings in the pharynx. These, he maintains, are the receptors of the stimuli which initiate the reflex stage of deglutition and if they are destroyed the co-ordination necessary for normal swallowing is lost.

Dysphagia may, however, be experienced in the absence of penetrating pharyngeal injury and here it would appear that the pain caused by attempts at swallowing is responsible. This view is suggested by the fact that an efficient surface anaesthetic will often enable these patients to swallow with greater freedom.

In cases of severe oesophageal corrosion, the direct action of the caustic on the oesophagus may contribute to the dysphagia.

Dysphagia due solely to oral pain and oedema is never complete and lasts from 3 to 5 days. When difficulty persists for longer periods the pharynx or the oesophagus (or both) are always involved. The reverse does not hold true, and on rare occasions a stricture has developed after a period of normal swallowing ability.

*Other Disturbances of Deglutition.* Attacks of coughing on swallowing may be so constant that a tracheo-oesophageal fistula is suspected. This we have never found and in 12 patients who experienced this distressing symptom it was possible to demonstrate, by lipiodol swallow, that the contrast medium entered the trachea *via* the larynx. There were 5 cases of death during the acute stages, in 2 of which it was possible to demonstrate such a spill-over radiologically. At necropsy the larynx, trachea and bronchi, in addition to the pharynx and oesophagus, were severely corroded. In the 10 other cases with such radiological findings, laryngoscopy and bronchoscopy confirmed that the rima glottidis and larynx, and occasionally the trachea, were involved. The paroxysms of coughing are therefore probably caused by incoordination of the swallowing reflex secondary to anaesthesia of the corroded pharynx and larynx.

Nasal regurgitation during swallowing occurred in 6 cases. These patients had extensive oral and pharyngeal burns with intense oedema of the soft palate and uvula. Two patients have persistent disability due to palatal scarring and both have the impaired phonation typical of a palatal defect.

*Laryngeal and Respiratory Symptoms.* The distressing sense of suffocation which may follow the ingestion of caustic soda is probably a result of laryngeal spasm due to irritation of the larynx. Such spasm prevents the entry of caustic into the air-passages but the inevitable relaxation with that sudden relieving in-rush of air may allow lethal quantities of caustic to enter the lungs. The 3 patients who died within 48 hours had extensive corrosive injuries of trachea and bronchi and advanced pulmonary oedema.

Respiratory complications due to other causes are frequent. When the laryngeal inlet is corroded and insensitive, inhalation of food and vomitus is a grave danger and broncho-pneumonia and atelectases may result.

Hoarseness, when present, signifies laryngeal involvement and one unfortunate girl has a permanently husky voice due to scarring of the vocal cords.

Hoarseness and respiratory complications indicate that the pharynx and probably the oesophagus have been involved, but give no clue as to the presence of the crucial factor of ulceration.

## DIAGNOSIS OF OESOPHAGEAL CORROSION

It is a fundamental requisite that involvement of the oesophagus should be proved before treatment is instituted. Only if this is established with exact certainty is it possible to assess the efficacy of the various methods of treatment. No reliance can be placed on the severity of oral injury and indeed no constant sign or symptom is pathognomonic of oesophageal corrosion of a degree sufficient to cause a stricture. There are, of course, cases where the true position is never in doubt, but in a large number one must rely on specialized investigations before a final decision can be made. Every survivor in this series was subjected to routine radioscopic and endoscopic examinations.

*Radioscopy*

Examinations by means of barium or lipiodol swallow are preferably performed within 48 hours of admission to hospital. When indicated, they were repeated at weekly intervals. The patients first swallow 5 c.c. of lipiodol whilst in the erect position. Should this be observed to enter the larynx, the examination is abandoned, with the certain knowledge that the lower pharynx, and probably the oesophagus, has been involved. On the other hand, if lipiodol passes into the oesophagus, 4 oz. (110 ml.) of a thin barium emulsion are given to the patient in the erect and supine positions. Considerable patience and sympathy may be required before this can be accomplished.

During the examination, the following points are noted: (*a*) the height of the barium column at the end of the swallow in the erect position; (*b*) the presence or absence of secondary and tertiary contraction waves, and (*c*) the rapidity of emptying of the oesophagus.

The observations have been of great interest but the method has not proved a reliable means of assessing oesophageal corrosion. In instances of severe injury several distinctive patterns of appearance have been produced:

(*a*) The contrast medium may enter the trachea (Fig. 4). This only proves that the laryngeal inlet has been injured but is of importance as a warning of the dangers of inhalation of food and secretions.

(*b*) Atonic oesophagus. In 6 instances the barium-filled oesophagus was widely dilated and no peristaltic activity was seen over a prolonged period of observation. In the erect position barium would accumulate in the inert oesophagus until a column sufficient to overcome the pleuro-peritoneal pressure gradient was reached. In the supine position the contrast medium remained indefinitely as a continuous column from mouth to cardia (Fig. 5). Serial observations showed that the atony persisted until stricturing began to distort the organ. In every case severe strictures resulted.

(*c*) The narrow rigid oesophagus. Seven patients, all of whom developed extensive tubular strictures, showed this appearance shortly after injury. In these cases the outline of the narrowed oesophagus was constantly irregular, and no peristaltic activity was evoked by attempts at swallowing (Fig. 6). These appearances are probably caused by oedema of the oesophageal walls and the fixed irregularities correspond with areas of mucosal ulceration.

(*d*) The irritable oesophagus. On 10 occasions irregular contractions of the lower oesophagus have been observed (Fig. 7). Three of these cases developed moderate strictures, 4 developed mild areas of localized fibrosis and 3 escaped without evidence of strictures. It is probable that tertiary contractions are an expression of oesophageal irritation due to superficial corrosive injury.

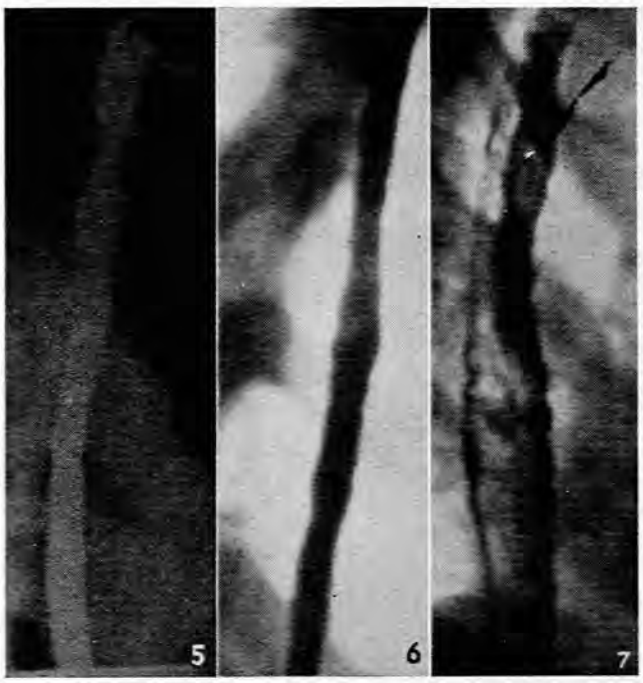


*Fig.* 5. Atonic oesophagus 60 hours after the ingestion of lye. The whole oesophagus subsequently stenosed.
*Fig.* 6. Narrowed and rigid oesophagus 30 hours after injury. An extreme stricture subsequently formed.
*Fig.* 7. Tertiary peristaltic contractions seen in a case with mild corrosive injuries of the oesophagus.

*Oesophagoscopy*

On the basis of our experiences it is felt that the only precise means of determining oesophageal involvement is to inspect the organ directly. This procedure has been critized because theoretically it is dangerous and because it is a specialized method of investigation which comparatively few physicians are qualified to use. In experienced hands immediate oesophagoscopy carries very little danger provided one is content to prove the existence and not the extent of damage. As for the second criticism, no one should presume to treat the acute caustic victim unless able to pass an oesophagoscope with gentleness and skill.

Unless oesophagoscopy is used as a routine method of investigation, the assessment of oesophageal corrosion remains presumptive. A great deal has been written about the treatment of acute caustic burns of the oesophagus and extravagant claims have been made about the value to the methods used, yet few of the authors have based their diagnoses upon direct inspection. Occasional workers (Blassingame *et al.*, 1947, Kernodle *et al.*, 1948, and Leegaard, 1945) have used oesophagoscopy during the acute stages but not as an indispensable need for correct diagnosis.

In this series oesophagoscopy has been performed on every patient admitted with a diagnosis of acute caustic poisoning, with the exception of 3 patients who were profoundly shocked and who died within 48 hours of admission to hospital. These examinations were made as soon as it was considered safe to administer a general anaesthetic. As a rule this was possible within 2-6 days of injury.

Ninety-four patients have been examined within a week of injury. Whenever oesophageal corrosion has been confirmed, the examination has been repeated at weekly intervals until healing has occurred or until

definitive treatment has been decided upon. At the first examination it is a rigid rule that inspection be confined to the detection of corrosion and no attempts made to advance beyond an area of mucosal ulceration. Even if the pharynx is severely involved it is possible to introduce the instrument into the oesophagus without danger. There has been no instance of untoward effect attributable to the examination.

The nature of the eventual stricture depends on 3 factors: (*a*) the depth of corrosion, (*b*) the circumferential extent of injury, and (*c*) the longitudinal extent of injury.

*The Depth of Corrosion.* Simple erythema of the oesophageal mucosa can be disregarded and the instrument may safely be advanced beyond an area with this appearance. If no ulceration is found, the oesophagoscope must be passed into the stomach before the gullet can be pronounced clear.

The appearances of oesophageal corrosion are the same as those of the mouth. Epithelium that is intact and viable is intensely reddened, but when destroyed it is swollen, opaque and grey in colour. By the 4th-7th day, when most of these examinations were done, the coagulated mucosa has desquamated, leaving dark red ulcers which bleed very readily. An oesophagus with coagulated epithelium, areas of ulceration, and granulations, is deeply involved and must inevitably heal by fibrosis.

*The Circumferential Extent.* Scarring is the sequel of mucosal ulceration but this may not be sufficient to cause a stricture. Ulceration confined to less than the total circumference is unlikely to produce significant narrowing, though localized shelves and luminal distortion may result. When the whole circumference is deeply ulcerated a stricture is inevitable.

*The Longitudinal Extent.* This is a factor of the utmost importance and one which it may be impossible to confirm by oesophagoscopy if the bounds of safety are not to be transgressed. On the lineal extent of ulceration depends the issue whether an innocuous ring stricture or a refractory tubular one will develop. If the walls of the corroded oesophagus are oedematous and rigid, the lumen may be held open by the beak of the oesophagoscope so that several centimetres of epithelium are visible. This only occurs with severe extensive corrosion and carries an ominous prognosis for the future patency of the organ. The absence of rigidity does not justify the assumption that a tubular stricture will not develop.

A difficulty which frequently arises is to determine whether severe burns are present beyond an area of partial circumferential involvement. It has proved a general rule that a burn of the cervical oesophagus, whether partial or complete, is associated with more severe involvement beyond. In the individual case time alone can provide the certainty, but the association of a cervical ring stricture and a tubular thoracic one is so frequent that it is reasonable to presume severe distal corrosion whenever limited cervical injury is observed. The lower the level of initial ulceration, the less likely is severe oesophageal injury to be present.

*Incidence of Oesophageal Involvement.* Ninety-four patients were subjected to early oesophagoscopic examination and in 26 instances circumferential ulcerations were seen. Two of these patients died and 24 have developed extensive oesophageal fibrosis.

In 37 cases no ulceration whatever was found and none of the patients in this group have developed a stricture.

In 31 instances areas of partial circumferential corrosion were seen. In 8 of these only the lower third of the oesophagus was involved and all have escaped without significant oesophageal narrowing. Six showed areas of partial corrosion of the upper thoracic segment, 2 of whom have developed multiple localized shelf strictures and 4 are asymptomatic. On 17 occasions partial cervical ulceration was encountered and further inspection of the oesophagus was abandoned. Ten of these patients developed localized cervical shelves or strictures associated with severe tubular strictures of the lower oesophagus. Two are asymptomatic and 5 developed multiple localized strictures which were dilated without serious difficulty (Table II).

TABLE II. CORRELATION BETWEEN FINDINGS DURING THE EARLY OESOPHAGOSCOPIC EXAMINATIONS AND THE EVENTUAL OUTCOME

| *Oesophagoscopic Appearance* | *Total No.* | *Deaths* | *Severe Strictures* | *Multiple Localized Strictures* | *Asymptomatic* |
|---|---|---|---|---|---|
| No Ulceration | 37 | — | — | — | 37 |
| Partial Circumferential Corrosion: | | | | | |
| Lower thoracic .. | 8 | — | — | — | 8 |
| Upper thoracic .. | 6 | — | — | 2 | 4 |
| Cervical .. .. | 17 | — | 10 | 5 | 2 |
| Severe Circumferential corrosion .. .. | 26 | 2 | 24 | — | — |
| Total .. | 94 | 2 | 34 | 7 | 51 |

## THE HEALING OF CORROSIVE LESIONS

The major damage is sustained at the moment of swallowing the corrosive. Caustic soda is rapidly bound to the destroyed tissues and within a few hours all corrosive activity has ceased.

The stages of healing correspond with those following thermal injuries. The inflammatory response around the devitalized tissue causes sloughs to separate and the resulting ulcers are the site of active growth of new capillaries and fibroblasts. When the defect has filled by granulation the cells of the surviving epithelium at the margins of the ulcers proliferate to cover the young vascular tissue. The growth of epithelium only proceeds satisfactorily in the absence of repetitive irritation and remains vulnerable until the epithelium has differentiated into stratified layers. When considerable segments of oesophagus have been denuded of mucosa regeneration may never be complete (Fig. 8).

Once the injured areas have been covered by epithelium the granulation tissue ceases to form and devascularization commences. Cicatrization begins within 10 days of injury but is a gradual process which varies with the extent of destruction.

When specialized structures are corroded there can be no regeneration except as scar. Muscle, mucous glands and nerve fibres never reappear within the destroyed areas.

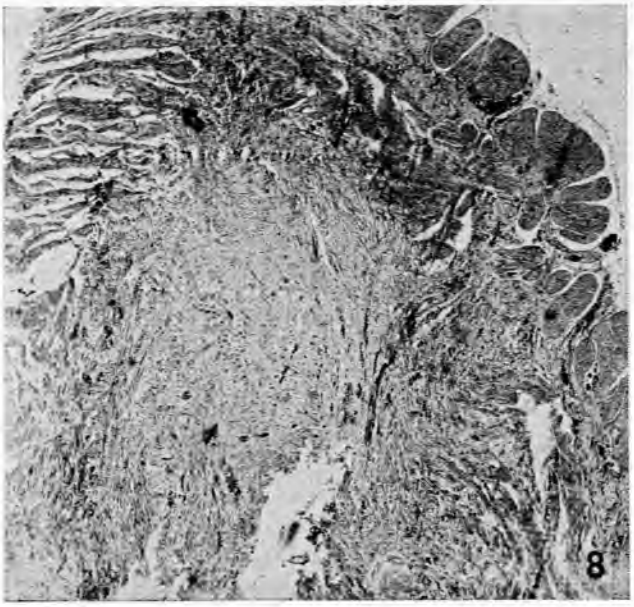

*Fig.* 8. Transverse section of a strictured oesophagus removed at operation 8 years after injury. The lumen, lined only by fibrous tissue, can be seen near the lower edge of the photograph. Magnification ×25.

Strictures are the end-result of repair and their severity is determined by (*a*) the extent and penetration of corrosion, and (*b*) the degree and duration of secondary infection and other factors which retard healing.

*The Extent and Penetration of Corrosion.* The greater the area of destruction, the longer it takes for the epithelium to regenerate and the greater the amount of granulation tissue that forms. When the gap is too great for the epithelium to bridge, extremely dense strictures result.

If the deep layers of the mucosa survive, healing will occur without fibrosis, but once the muscularis mucosa is destroyed scarring is inevitable. With severe injuries the whole depth of the oesophageal wall may be involved and even the extra-oesophageal tissue planes may be obliterated by fibrosis.

*Bacterial Infection.* Once the oesophagus has been severely traumatized, infection readily supervenes. Unless this is checked additional injury may be caused and healing delayed sufficiently to convert what may have been a mild stricture into one of great severity.

*Other Traumatizing Factors.* Apart from infection, the commonest cause of delayed healing is the early passage of a bougie. Regenerating epithelium is thin and delicate and when bougies are passed daily repeated injury is caused and fibrosis is correspondingly increased.

*The Rate of Healing.* Routine weekly examinations have provided the opportunity of observing the progress of corrosive lesions. It has been consistently noted that oral burns heal before oesophageal burns, and the lips sooner than the mouth or pharynx. In the oesophagus the lower injuries usually take longest to heal. The factors which may account for this orderly progression of healing from above downwards are:

(*a*) Exposure to air. Wallace (1949) has shown that thermal burns of the skin mend rapidly when exposed to freely circulating air. This probably accounts for the rapid improvement of corrosive injuries of the lips.

(*b*) The severity of the burn. When a bolus of food is swallowed, it traverses the pharynx and upper oesophagus with such great speed that contact with the mucosa is momentary. The force and rapidity of the primary contraction wave of swallowing is dissipated in the mid-oesophagus and further progression is effected much more slowly by secondary waves of peristalsis. The contact of caustic soda with the lower oesophagus may be still further prolonged if paralysis or spasm of the propulsive musculature is produced. These considerations adequately account for the frequent association of superficial cervical oesophageal corrosion with severe involvement of the lower oesophagus. With very concentrated solutions, however, even momentary contact will cause profund damage.

As a general rule, moderately severe burns of the cervical oesophagus have re-epithelialized by the end of the 2nd week and those of the lower oesophagus by the 3rd or 4th weeks. In such cases one can confidently expect to control the ensuing stricture by dilatation. When ulceration persists after the 6th week the patient will probably develop a long dense stricture, difficult to treat and dangerous to dilate.

## TREATMENT OF ACUTE CORROSIVE BURNS

Treatment should aim at (*a*) diluting and neutralizing the corrosive, (*b*) combating shock, (*c*) preventing pulmonary complications, (*d*) maintaining hydration and nutrition, (*e*) alleviating symptoms, (*f*) preventing infection, and (*g*) preventing stricture-formation.

*Dilution and Neutralization.* This has usually been carried out by relatives or friends long before a doctor is able to attend the victim. Milk is the favourite antidote but lemon-juice, olive oil and vinegar are also popular. If neutralizing agents are to be of use they must be given immediately. Concentrated acids and stomach wash-outs with large gastric tubes are dangerous forms of treatment.

*Treatment of Shock.* Emergency treatment is limited to the administration of morphia to allay pain and anxiety. In hospital it is the rule to give appropriate doses of morphine at 6-hourly intervals during the first 24 hours. General supportive treatment and if necessary intravenous infusions of blood, plasma or saline may be necessary. Very little else can be done during the first disturbing day.

*Prevention and Treatment of Pulmonary Complications.* The early deaths from ingestion of caustic soda are usually due to pulmonary complications. Nothing will prevent a fatal termination from pulmonary oedema when the bronchial tree has been flooded with concentrated corrosive, but breathing exercises and frequent postural changes will benefit the milder cases.

When the rima glottidis has been corroded there is grave danger of inhalation of food and vomitus. Formerly, when this occurred, oral feeding was withheld

for 4 or 5 days and hydration was maintained parenterally. We now believe that with conscious patients the danger is slight because the cough-reflex is capable of expelling intruding foreign material. Meals may be prolonged and punctuated by paroxysms of coughing, but this rapidly improves. The danger period is during sleep, particularly if induced by drugs, when the sensitivity of the laryngeal and tracheal mucosa is depressed. Whenever this danger exists the patient should sleep with the foot of the bed raised 18 inches and antibiotics should be administered prophylactically. Deep breathing exercises, effective coughing and postural drainage must be supervised at frequent intervals. Should atelectasis occur, bronchoscopic aspiration may be necessary.

*Symptomatic Treatment.* Pain is the main complaint during the early stages but if possible morphia should be avoided after the 1st day because of the dangers of pulmonary complications. Surface anaesthetics are useful when ulceration is slight, and dramatic improvement of pain and dysphagia may follow their use.

Frequent mouthwashes are in routine use and the teeth, gums and lips should be swabbed daily.

*Prevention of Infection.* From the long-term point of view, control of secondary infection is of cardinal importance. Chemotherapy has enormously improved the outlook and today 7 times as many people survive

TABLE III. MORTALITY COMPARED WITH THAT OF THE PRE-ANTIBIOTIC ERA

| *Series* | *No. of Cases of Acute Poisoning* | *Deaths in Acute Stages* | |
|---|---|---|---|
| | | *No.* | *%* |
| V. Hacker (1899) .. .. | 333 | 112 | 35 |
| Witthaus (1911) .. .. | 83 | 30 | 36 |
| Thoracic Surgery Unit, Johannesburg (1952) .. .. | 97 | 5 | 5 |

the effects of caustic injury as did so 50 years ago (Table 3). Control of infection is so important that there should be no hesitation in ordering several different antibiotics simultaneously.

*Maintenance of Hydration and Nutrition.* Maintenance of adequate nutrition may prove difficult during the first few weeks. Pain and oedema of the mouth and pharynx, paralysis, spasm and oedema of the oesophagus, and the tendency to inhale food and secretions, may make swallowing a difficult task. Frequently patients spit out copious quantities of saliva rather than suffer the discomfort of swallowing. Explanation and persuasion usually suffice to tide them over the first days but occasionally it is necessary to administer fluids rectally or by intravenous drip. After 48 hours the lessening of pain and oedema permits of progressively easier swallowing, and only in the most severe instances is there reason to consider gastrostomy. It is seldom necessary to continue with intravenous therapy after 2 or 3 days, but if so it is preferable to do a gastrostomy. We have twice had to have recourse to surgical measures within the first week; both patients were so frightfully injured that they eventually died.

## PREVENTION OF STRICTURES

### *The Salzer Regime*

In 1920 Salzer described a method of auto-dilatation with mercury-weighted bougies commencing within 2-6 days of injury. This work was publicized by Bokay (1924), who reported that 117 out of 132 patients treated by the method did not develop dysphagia. Gellis and Holt (1942) claimed that, of 41 children treated, only one developed a stricture. Kernodle, Taylor and Davison (1948) used the method on 21 patients of whom 5 developed strictures. Crowe (1944) reports that all 9 children in his series were free from strictures 6 months to 4 years after the accident. These results sounded impressive and we approached our first few attempts at Salzer's technique with enthusiasm.

The treatment requires the victim to swallow soft rubber bougies weighted with lead (Bokay) or mercury (Hurst), the object being to prevent the development of stricture rather than to effect dilatation. The first attempt is preferably made within 48 hours and the bougie must be completely swallowed before the 6th day has passed. A number-14 bougie is first swallowed and is retained in position for 5 minutes. The size is rapidly increased until, by the 6th day, a size-34 bougie is passed. This procedure is repeated twice daily for 2 weeks, after which the same bougie is swallowed once a day for a further fortnight. Thereafter, it is necessary to repeat the procedure twice weekly for 2 months and, finally, once a week for a further 6 months.

One can only marvel at the discipline and fortitude of the children of Austria and America. Martin and Arena (1939) are alone, amongst all who have written of the method, to mention the enormous amount of cajolery that their patients required to induce them to carry out the therapy to the end. They were successful with 3 children out of 50. Our success was even less. An attempt was made to persuade 6 children between the ages of 3 and 7 to swallow Bokay bougies. Doctors, nurses, social workers, physiotherapists and relatives were successively enrolled in an attempt to get that bougie down by the 5th day, and all in turn were defeated. After watching an exasperated volunteer endeavour to coax a bougie beyond a child's clenched teeth, one realized the wisdom of making the bougie of soft rubber. We have since used the technique exclusively on adults.

Five adults were treated by Salzer's method. They were selected because they were intelligent and willing to cooperate. All had swallowed concentrated caustic solutions and oesophageal involvement, of an extent which was considered sufficient to cause a stricture, had been confirmed by oesophagoscopy. The case reports are as follows:

*Case* 1. G.N., African female aged 17. Suicidal attempt on 15 March 1951.

On 19 March the patient was given a tablet of anethane to suck and was sedated with 1½ grains of luminal. Patience and encouragement was eventually rewarded by the successful ingestion of a size-15 Bokay bougie, which was retained for 5 minutes. This was repeated twice daily under sedulous supervision and was always accompanied by retching, spitting and pleading.

On 21 March a size-30 bougie was retained. This was passed twice each day but never easily and with ever-increasing resentment.

On 24 March oesophagoscopy revealed a persistent severe burn of the cervical oesophagus; the mucosa was denuded and the granulations bled as soon as they were touched.

On 28 March she was unable to swallow the size-30 bougie and no amount of cajolery was effective in getting her to pass it through the cervical oesophagus. She was then given a size-20 bougie and this she swallowed easily. Oesophagoscopy confirmed that a stricture had commenced to form, 17 cm. from the upper incisor teeth.

By 2 April the patient was unable to swallow this bougie and on 10 April the treatment was abandoned after she failed to swallow a size-10 bougie and had been almost reduced to a state of nervous prostration. She developed very extensive strictures and eventually a successful oesophago-gastrostomy was performed.

*Case* 2. L.D., Coloured female aged 23. Accident on 8 April 1951.

Oesophagoscopy on 11 April revealed circumferential mucosal ulceration, 20 cm. from the upper incisors. The Salzer regime was started on 13 April, when the patient successively swallowed size-10, size-15 and size-20 bougies with the greatest of ease. Thereafter she swallowed size-30 bougies twice daily until 21 April, when she experienced pain on passing the tube. When the tube was removed she vomited some blood-stained material. Oesophagoscopy on 22 April confirmed that a stricture had begun to form at 20 cm.

On 23 April she was given a size-20 bougie, which she was able to swallow satisfactorily. By 6 May 1951 she was again having difficulty and thereafter her treatment was supplemented by weekly antegrade dilatations under general anaesthesia. Barium swallow demonstrated tubular strictures in the upper thoracic and retrocardiac segments.

This patient did not return to out-patient clinic until 14 December 1952. She was then extremely thin and a very narrow stricture had developed. She had abandoned the treatment a month after discharge on 17 July 1951 as she was then swallowing well, but slight dysphagia began 2 months later. She then tried in vain to swallow her bougie, and over the course of a year the dysphagia got steadily worse.

*Case* 3. B.M., African male aged 25. Suicidal attempt 23 November 1950.

Oesophagoscopy on 27 November showed circumferential corrosion, 21 cm. from the upper incisor teeth. The treatment was started on 28 November and he was able to swallow a size-30 bougie immediately. This he continued to do each day until 2 January 1951, when he began to experience difficulty. Oesophagoscopy demonstrated a ring stricture at 20 cm. and a bougie encountered a second stricture at 27 cm. Thereafter he swallowed a size-20 bougie daily and this was supplemented by weekly dilatation, under anaesthesia, to size-26.

He was discharged on 18 February 1951, 3 months after the suicidal attempt, but neglected to swallow his bougie and was readmitted on 27 March 1951. Since then he has returned periodically for dilatations.

*Case* 4. A.H. African male aged 35. Accident on 4 September 1951.

On 5 September, oesophagoscopy revealed circumferential corrosive injury 25 cm. from the upper incisors. He only succeeded in swallowing the size-20 bougie on 8 September. The subsequent performances were nerve-racking for all concerned, as he never took kindly to the method. Frequently the bougie would be withdrawn blood-stained and after oesophagoscopy on 17 September, when persistent ulceration was seen, the treatment was abandoned. Barium swallow at that time showed that already a stricture had developed.

*Case* 5. A.G., Indian female aged 30. Suicidal attempt on 18 December 1950.

Oesophagoscopy on 21 December showed corrosion immediately below the crico-pharyngeal sphincter. The regime was commenced on 23 December and by 25 December she was swallowing a size-30 bougie. Each operation was courageously performed, even though considerable pain and bleeding were caused. On 7 January 1951 she experienced difficulty and barium swallow outlined a ring stricture at the thoracic inlet and a tubular stricture below the level of the aortic arch. The treatment was then abandoned.

It is difficult to gainsay the many excellent reports of the Salzer treatment, but in our experience the method has no effect whatever in altering the clinical course. We believe that the treatment cannot prevent the formation of a stricture whilst, by repeatedly traumatizing the ulcerated areas, it may actually do harm. It is hard to believe that daily bouginage can be so effective as to prevent the contraction of developing scar, yet this is what Blassingame, McArthur and Atkinson (1947), Gellis and Holt (1942) and Kernodle *et al.* (1948) claim for the majority of their cases. These workers have confined the treatment to children and in this connection Table IV is instructive.

TABLE IV. RESULTS OF TREATING CHILDREN WITH ACUTE CAUSTIC INJURIES

| | *Total Cases* | *No. to Develop Strictures* | % | *End Result Satisfactory (No. Dysphagia)* | % | *Type of Treatment* |
|---|---|---|---|---|---|---|
| Bokay-Salzer .. | 131 | 15 | 11·4 | 116 | 88·6 | Salzer |
| Gellis and Holt .. | 41 | 2 | 5 | 39 | 95 | Salzer |
| Kernodle *et al.* .. | 21 | 5 | 19 | 16 | 81 | Salzer |
| Thoracic Surgery Unit, Johannesburg .. .. | 36 | 8 | 22·5 | 33 | 91 | Dilatation when healed |

In each series all children with oral injuries were subjected to treatment. In the Thoracic Surgery Unit (Johannesburg) series all 36 children had oral injuries, but after early diagnostic oesophagoscopy only 8 were detained for treatment.

Every child in our series had an early oesophagoscopic examination and, even though they all had oral burns of varying severity, only 8 had sufficient oesophageal damage to produce strictures. Thus only 22% of children with oral injury sustained sufficient oesophageal corrosion to cause strictures. These unfortunate victims were treated by dilatations, at a time when we considered this was safe, and all but 3 were readily controlled. This policy of treating only those who are liable to develop strictures, has resulted in permanent freedom from dysphagia in 91% of all acute cases admitted. This success rate can be favourably compared with the published results of the Salzer treatment.

The logical objection to these comparisons is that our series includes 28 children who had little or no oesophageal injury, but the advocates of the Salzer regime have exercised little discretion in the selection of cases included in their reported series. Bokay and Salzer treated all cases with oral burns without direct visual proof of oesophageal corrosion. Gellis and Holt do not describe any attempts at precise diagnosis, and Blassingame *et al.* and Crowe state that oral burns should be taken as presumptive evidence of oesophageal involvement. It has been shown that mouth burns, even of very severe degree, are frequently found in the absence of injury of the oesophagus. It is obvious that to institute the Salzer method simply on this basis is to subject the majority of victims to unnecessary treatment.

*Other Methods*

It is difficult to abandon hope in the possibility of preventing the formation of a stricture. The logical

conclusion after reviewing the failures of the Salzer method, is that the scar has a great deal of time to contract in the intervals between bouginage. Leegaard (1945) has evolved a method of continuous internal splinting and has used it in 15 cases, though he does not give his criteria for diagnosing oesophageal damage. With children under the age of 1 year he passes a nasal bougie, but in all older individuals the tube is passed into the stomach through an external pharyngotomy opening and serves the double purpose of splintage and feeding. He does not discuss his results in any detail and we have not been inclined to treat our patients in this way.

*The Effect of ACTH in Preventing Strictures*

It is well known that surgical wounds heal with difficulty in animals under treatment with ACTH (Ragan, Grokoest *et al.*, 1949). This is thought to be due to decreased fibroblastic proliferation and inhibition of collagen maturation (Ragan, Howes *et al.*, 1949). It was therefore hoped that ACTH might have a beneficial effect upon caustic oesophageal strictures by preventing excessive fibrosis and delaying contraction of the scar.

Four adult patients with recent caustic injury were selected for the treatment. They were given daily intravenous infusions of 20 mg. of ACTH in a litre of glucose and water, for one week. Thereafter, the dose was decreased to 15 mg. daily for the second week and 5 mg. daily for a further 2 weeks. The solution was administered very slowly, an attempt being made to have the vacolitre last about 8 hours. The technique, though irksome for the patient and needing constant supervision, was considered to be the most reliable means of instituting what was then a somewhat novel therapy. Penicillin was given throughout the period of treatment. No untoward effects attributable to the hormone resulted. The following are reports of cases in which ACTH was administered:

*Case* 1. A.G., Indian female aged 30. Attempted suicide on 18 December 1950.

Treatment was begun the day after admission to hospital. On 24 December an oesophagoscopy was done and circumferential corrosion was seen to be present immediately below the crico-pharyngeal sphincter.

The patient was able to swallow fluids until 27 December, when she began taking porridge and other semi-solids. An attempt was made to get her to swallow a size-30 bougie and this was accomplished on 29 December. Oesophagoscopy was repeated on 3 January 1951, when persistent ulceration was seen 19 cm. from the incisor teeth. The walls of the organ appeared thickened and stiff, but the lumen had not contracted to the extent which one would have expected from the previous examinations.

On 10 January the patient, who had swallowed the bougie daily, reported that she had vomited blood after the procedure that morning. Bouginage was then abandoned. On 15 January oesophagoscopy was repeated and the findings were essentially the same as those of the examination 12 days earlier. The ulcerations had not lessened in extent and were still shaggy and unhealthy in appearance. The lumen had remained widely patent.

The treatment ended on 28 January and at that time the patient was swallowing very well. Oesophagoscopy next day revealed that ulceration was still present at 19 cm., though there appeared to be some slight regeneration of epithelium as compared with the previous examination.

Autobouginage was recommenced on 2 February 1951, but the patient was only able to swallow a size-18 dilator.

On 4 February oesophagoscopy showed that epithelium had covered the upper ulcerations and a ring stricture had now formed. This was dilated so as to permit the passage of a child's oesophagoscope, which was arrested at 25 cm. by a second stricture. The face of this second narrowing looked healthy.

Barium swallow on 6 February confirmed the presence of a ring stricture at the thoracic inlet, and a tubular stricture starting at the level of the aorta.

The patient was allowed home on 22 February 1951 with instructions to continue swallowing the bougie. She was last seen on 11 August 1951 and, though barium swallow confirmed the presence of the double strictures, these were still widely patent.

*Case* 2. L.S., Coloured female aged 33. Attempted suicide on 28 May 1951.

The treatment was commenced on the day of admission. On 30 May oesophagoscopy demonstrated that the crico-pharyngeal sphincter was in spasm. An infant's oesophagoscope was manipulated through, and 2 inches beyond the mucous membrane was seen to be corroded.

On 2 June an attempt to swallow a Bokay bougie failed, but the patient succeeded with a size-15 bougie on 7 June.

On 14 June oesophagoscopy showed that the spasm of the crico-pharyngeal sphincter had relaxed. The mucosa at 19 cm. had healed without a stricture, but at 20 cm. persistent unhealthy ulcerations were seen.

The patient was able to swallow with comfort and weekly oesophagoscopic findings remained essentially unchanged throughout the period of treatment, which ended on 2 July 1951.

On 13 July 1951 oesophagoscopy was repeated. An established stricture, with healed epithelial lining, was visualized at 21 cm. The passage of a size-16 bougie imparted a sensation of resistance to the operator.

The patient had swallowed a size-15 bougie throughout, and this she continued to do. Unfortunately on 20 July she had a sudden recurrence of delusions, hearing the same voices which had originally commanded her to attempt suicide. It was found necessary to commit her to a mental institution, where the medical officers were at pains to oversee her treatment. Her mental condition was successfully treated with electro-convulsive therapy, and we saw her again on 23 January 1952, when she was still swallowing the bougie daily.

Barium swallow demonstrated the presence of a ring stricture at the thoracic inlet and a retro-cardiac tubular stricture. The passage of a bougie imparted a sensation of resistance as firm and unyielding as a stricture of similar extent, untreated by ACTH.

*Case* 3. V.H., Coloured female aged 48. Attempted suicide on 25 August 1951.

This was a mild case, in which circumferential ulceration was sustained at 25 cm. (aortic arch). The treatment was started immediately on admission and weekly oesophagoscopies were performed. Again it was noticed that re-epitheliazation of the denuded mucosa was not so rapid as usual and that infection was persistent. One would have expected healing to have been completed in this case by the end of the 2nd week, but this was only observed 10 days after the ACTH was discontinued. In view of the limited damage, no attempt was made to get the patient to swallow a bougie and, whereas throughout the period of treatment with ACTH no narrowing of the affected segment had been seen, 10 days after its cessation a definite ring stricture had formed.

The stricture has been controlled by autobouginage and on 14 May 1952, when the patient was last seen, the narrowed segment was adequately controlled.

*Case* 4. J.M., African male aged 28. Accident on 31 August 1951.

The treatment was commenced after oesophagoscopy on 1 September, when the gullet was seen to be severely corroded at 24 cm. (aortic arch).

By 13 September a definite stricture had commenced at 24 cm., where the mucosa was still ulcerated. The following week the stricture was more evident. When a bougie was passed the walls were felt to be soft and yielding and the lumen was dilated to size 24 with the utmost ease. On 8 October, one week after the ACTH was stopped, the mucosa was completely healed. The stricture then readily admitted a size-18 bougie and presented no resistence to efforts at dilating it to size 24 (Fig. 9).

On 19 October 1951 the patient was discharged. He was readmitted on 28 December 1951, having lost 30 lb. in weight, and confessed that he had neglected to swallow his bougie. At endoscopy a stricture was seen at 22 cm., which would admit a size-8 bougie (Fig. 10). The walls were hard and unyielding and re-

dilatation was effected with considerable difficulty over the course of 6 weeks.

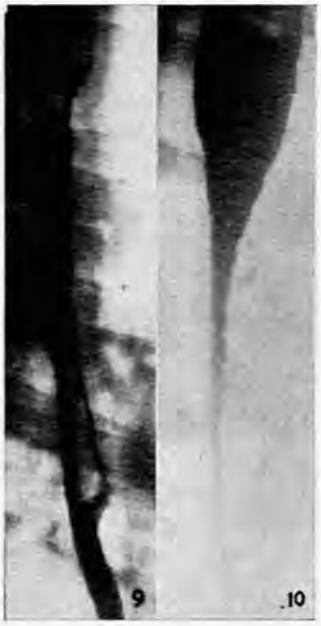


*Fig.* 9. Effect of ACTH. This radiograph was taken 8 weeks after injury and 1 week after cessation of ACTH. Severe corrosion was proved by early oesophogoscopy. The lumen remains widely patent.

*Fig.* 10. Effect of ACTH. Same case as Fig. 9, 10 weeks after cessation of ACTH therapy. A long dense tubular stricture has formed.

Our experience with ACTH is limited. A definite effect was accomplished in the initial stages, and strictures were delayed in their formation. In case 4 the stricture had commenced by the end of the 2nd week but was extraordinarily soft and dilatable. This effect upon the regenerating fibrous walls of the affected regions was maintained as long as the hormone was given.

It was noted in these 4 cases that infection persisted much longer than in untreated cases of comparable severity. Though this is probably due to the delay in healing, it will be recalled that Selye (1950) has reported that ACTH decreases the resistance to infection. Retardation of epithelial regeneration has been an undoubted result, again not an unexpected finding in view of Green and Bullough's demonstration that ACTH has the effect of depressing or suppressing mitosis in epithelial cells (1950).

ACTH may be a two-edged sword. It was noted in every case that ulceration persisted long after healing would ordinarily have been expected. Though penicillin therapy keeps gross infection in check, there is the danger that, so long as ulceration persists, destruction of specialized cells may continue, which can only be replaced by fibrosis. ACTH seems to suspend the processes of generation and maturation of fibrous tissue just so long as it is in circulation, and thereafter normal evolution continues unchecked and the scar inevitably contracts.

*When to start Dilatation*

Experience of acute caustic burns of the oesophagus has led to the conviction that, once the corrosive action penetrates into the oesophageal walls, scarring is inevitable. Nothing can prevent this and there is no practical method of avoiding subsequent contraction of the scar. Bouginage remains the sheet-anchor of treatment and the earlier this can be started the greater the chances of success. However, trauma may be caused by too-early instrumentation and this consideration, with the accompanying hazard of infection, must be weighed against the difficulties of dilating a tough, narrow stricture. It is now our practice to attempt dilatation as soon as weekly oesophagoscopic examinations show that the stricture face has epithelialized. An appropriate-sized, well-lubricated bougie is gently passed through the stricture and on withdrawal is examined for blood-staining. Should this be found, the presumption is that granulations are still present and attempts at dilatation must be postponed for a further week. The early regular passage of a fine bougie has the additional advantage of maintaining a straight lumen through the stricture, thus circumventing the grave danger of dilating a tortuous track.

In cases with mild limited injury, dilatation has been started as early as the 2nd week, whereas with severe damage 6 weeks or longer may pass before treatment can be attempted. With such a policy it is sometimes necessary to perform a gastrostomy before dilatation is attempted, and in such cases retrograde bouginage is preferred (Tucker 1931). No case in this series has had an oesophago-gastrostomy without a prior attempt to dilate, but in several instances of severe injury it would have been kinder and less hazardous had dilatation never been attempted.

## SUMMARY OF RESULTS

Ninety-seven patients with acute caustic soda poisoning have been treated by the Thoracic Surgery Unit in Johannesburg during the past 5 years.

Five patients died in the early stages, 3 within 48 hours of admission and 2 during the 2nd week. At necropsy all these patients showed evidence of pulmonary corrosion, and death was due to pulmonary oedema and bronchopneumonia. The 2 patients who survived into the 2nd week had developed mediastinitis and peritonitis following necrosis of the oesophagus and stomach.

Ninety-four patients had oesophagoscopic examinations within a week of injury (3 fatal cases were too ill for this) and, though all had injuries of the mouth, only 57 were found to have oesophageal corrosion. These 57 were examined at weekly intervals, and in 16 of them healing of the corroded area without significant scarring had occurred before the 2nd week had passed. Forty-one patients have therefore developed strictures and 5 have died (47% of total) whereas 51 escaped serious oesophageal involvement. With children, most of whom took solid caustic or weak solutions accidentally, the escape rate was even higher. Only 8 out of 36 (22%) developed strictures.

It had been hoped that a method would be found effective in preventing the development of strictures. The Salzer technique and ACTH therapy were tried but were ineffectual in preventing this dread sequel. One is forced to conclude that, once the oesophagus has been severely damaged, a stricture is inevitable. The standard treatment now is to control infection with antibiotics and to commence dilatation only when the epithelium has regenerated to cover the denuded areas of the oesophagus. With severe extensive strictures this may

never occur and in these cases it is hopeless to expect dilatation to succeed in maintaining an adequate permanent lumen.

Three more patients have died as a result of treatment, one owing to a leaking jejunostomy, one after oesophagogastrostomy, and one from gastric haemorrhage following dilatation of her stricture. The total mortality has therefore been 9%. With the passage of time there may be further deaths, for patients with oesophageal strictures are subject to many complications throughout their lives.

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