impact against the foreign body. Esophagoscopy is a true blessing, especially in cases of impacted foreign bodies. Much information, and that of a positive nature, can be expected from it. Foreign bodies may not only obstruct the esophagus, but also *perforate* it. Perforation may be caused by the instruments used in searching for the foreign body. In addition, perforation may be due to the breaking down of a carcinoma, or to ulceration. How is perforation recognised? No general answer can be given to this query. Subcutaneous emphysema may signal the occurrence of rupture—another symptom being hemorrhage—not only bleeding from the mouth, but also the rapid formation of a false aneurism in the neck. Anadale found an aneurism of the inferior thyroid caused by a perforating foreign body. Perforation may also show itself by producing an acute mediastinitis or pleuritis.

A few cases of *spontaneous rupture* of the esophagus have been placed on record. As a rule, it occurred in strong men habitually addicted to alcohol, the accident taking place soon after a meal, and proving rapidly fatal. The esophagus was probably previously diseased. The symptoms were: retching and vomiting; then pain localized at the point of rupture (the cardia); premonition of impending death, collapse, grave respiratory disturbances, and subcutaneous emphysema, starting in the clavicular region and rapidly extending over the entire body.

CHAPTER XI

STENOSIS OF THE AIR-PASSAGES AND OTHER DISTURBANCES OF RESPIRATION

Fabricius ab Aquapendente, the successor of Fallopias, teacher of anatomy and surgery at Padua, said in praise of the operation of bronchotomy, that the physician who performed this operation was like the god Æsculapius. The operation is undertaken to furnish an artificial means of entry for the atmospheric air in cases of stenosis of the larynx or trachea which endanger life. The success of the operation naturally depends upon creating a passage *below* the point at which the stenosis is situated. This applies to all cases of laryngeal stenosis and to stenosis of the upper part of the trachea. Occasionally a tracheotomy opening is made *above* the stenosis, but only when dealing with a compression of the trachea, which may be relieved by passing an elastic or inflexible tube downward, in order to again distend the air-passage.

Tracheal stenosis may be divided into three groups:

1. Obstruction of the lumen from within—*Obturation.*

2. *Compression* from without.

3. Pathological changes in the walls of the tube which produce a marked narrowing of the lumen—*Stricture*, in the narrower sense of the word.

Obturation usually is produced by foreign bodies; but croup may be classed with this variety, because of the bulky false membrane produced by the process, which may cause a temporary but complete stenosis.

Compression stenosis may be due to foreign bodies in the neighbourhood of the trachea, extravasations, tumours arising from the thyroid gland, thymus, esophagus, lymphatic glands, sternum, clavicle, spinal column, and to aneurisms.

Changes in the wall of the trachea are manifold. The exudative processes of the mucous membrane, croup, and diphtheria, which narrow the lumen by producing false membranes, may be placed in this class also, for they are accompanied by swelling of the walls. Swelling in the parenchyma itself must also be included, as it narrows the lumen by the products thrown out (similar to abscess), or by the œdema which accompanies it; suppurative perichondritis, necrosis, and tubercular, syphilitic, and typhoid ulcers of the larynx (inflammations in the vicinity of the larynx). Chronic infiltration or thickening; cicatrization, following ulcerative destruction; and lastly tumours, which project into the lumen, belong to this class.

Cicatricial stenosis best typifies those rare cases where a slight stenosis, gradually progressive, becomes dangerous to life. As a rule, however, the danger occurs *suddenly*, so as to take both patient and physician by surprise. In the case of foreign bodies, a sudden stenosis is, of course, to be expected. In compression stenosis, urgent symptoms are usually caused by a catarrhal swelling of the mucous membrane of the larynx and trachea at the site of constriction. In dealing with inflammatory processes we usually expect stenosis, but

the dangerous symptoms very often take the patient completely by surprise, corresponding to some unforeseen process in the inflammatory focus.

What are the characteristics of a dangerous STENOTIC ATTACK?

A typical attack of croup once seen is never forgotten. With the very first glance at a patient suffering from laryngeal stenosis we are impressed by the frightful efforts made by the little one. The child labours and strains; he fairly wrestles for air, and wildly endeavours to crawl up the wall, only to fall back among the pillows exhausted. Conscious of the obstruction, the little one tears at its neck and instinctively pries into its pharynx. Its face is a startling picture of anxiety and despair. Overwhelmed as it is, it utters neither cry nor scream, but lies absolutely speechless, giving vent only to deep, long-drawn, stridulous inspirations. Careful inspection discloses the fluttering of the alæ nasæ during deep inspiration, alternately widening and narrowing the nostrils. All the accessory respiratory muscles are driven to their utmost capacity; the neck muscles are on the stretch; the diaphragm constricts the lower thoracic zone, but is itself unable to descend. The epigastrium arches deeply inward with each inspiration. All, however, is in vain. Little air can pass the point of stenosis and reach the lung. The croup-membrane may hinder expiration also. If this is the case, the blood is forced out of the thorax at each expiration, and if this phase predominates the child grows cyanotic; otherwise, it is pale. After internal medicine has exhausted all its efforts, and the dyspnœa is still increasing, the surgeon is called in, and now has an opportunity to prove him-

self equal to Æsculapius. If tracheotomy affords no relief, the child grows quieter as the dyspnœa increases. Respiration becomes shallower, inspiration less audible, irritation of the periphery no longer meets with response, and carbon-dioxide poisoning causes death and puts an end to the agony.

The active exertions of the patient to obtain air; the full, noisy inspirations; the contraction of all the auxiliary muscles of respiration, and the deep pit in the epigastric region, are the most prominent symptoms of a stenotic attack of dyspnœa.

Surgery has its greatest triumphs in those cases in which the dyspnœa is caused by a Foreign Body.

We are called in to see a child suffering with marked signs of stenosis. The history shows that the attack was severe from the outset, and that the child was in good health until the very moment at which the air-hunger first appeared. In such a case, the sole explanation possible is that a foreign body has entered the air-passages. Our first endeavour consists in introducing the hand into the patient's mouth in order to make sure that the foreign body has not lodged in the aditus ad laryngem, or, if it is there, to remove it. If it is not there, and time is too pressing to permit of laryngoscopic examination, tracheotomy must be performed as rapidly as possible. A child was brought to Schuh's clinic in great danger of asphyxia. Schuh was informed that the child had been in the best of health, but suddenly had had the attack after playing with pebbles. Without wasting a second, he grasped his knife and performed a tracheotomy. A small pebble at once popped out from the wound. If there is time to make an examination, the larynx should at once be inspected.

If this is impossible, auscultation of the larynx, trachea, and lung may in some cases be of service, for the foreign body may produce a characteristic sound as it flies up and down the passage and strikes against the walls. Blood-tinged sputum is of some value, as it points to injury of the mucous membrane resulting from the passage of a rough body. Subcutaneous emphysema is of even greater importance, as it indicates perforation of the mucous membrane. Louis, in the middle of the last century, already called attention to this symptom.

A similar attack may be caused by a foreign body arrested in the esophagus and pressing forcibly upon the trachea. It may happen, during a meal, that too large a bolus is swallowed and remains stuck fast. Not infrequently false teeth are swallowed and stick in the throat. Therefore, in all cases of sudden dyspnœa accompanied by marked cyanosis, it is advisable to explore the esophagus.

It is hardly conceivable that sudden, severe attacks of dyspnœa can occur in a previously healthy person unless they are due to a foreign body. Attacks due to neuroses of the larynx (*Spasmus glottidis*) occurring in young children, appear suddenly, but the attack may pass off, and the child again be lively and well before a physician can be summoned. In other instances, the dyspnœa may persist for hours. In most cases we are able *to judge from the external circumstances* whether we have to deal with a foreign body or not; as, for instance, in the case of a child that is attacked by the stenosis while in its mother's arms, without having played with any object. If short attacks have preceded, the disease is unmistakably characterized. Greater difficulty is encountered if the attack takes place during

the course of an illness which has existed for several days. Here there is the possibility of laryngitis crouposa, catarrhal laryngitis, œdema of the glottis, or of a foreign body. Monti tells of a child that was supposed to be suffering with croup for three days. The child had a barking cough, croupy respiration, and was completely aphonic, and then developed a severe attack of suffocation. The examining finger, however, detected a small metal buckle which had become impacted in the glottis. But, as already emphasized, a foreign body can, as a rule, be taken for granted if a stenotic attack occurs without previous hoarseness and general malaise. Similar symptoms may, however, be noted in a child suffering from œdema of the glottis.

Laryngitis Crouposa and *laryngitis catarrhalis* are ushered in by fever, and stenosis is preceded by other warning symptoms. In some cases it is scarcely possible to distinguish one from the other; but, as the dyspnœa due to the catarrhal form never causes asphyxia, it is well worth while to know the diagnostic landmarks in order to be prepared for tracheotomy in the severer variety.

In *croup*, portions of the false membrane are either coughed up or can be seen upon the tonsils, and symptoms of a progressively advancing stenosis are usually present.

In *catarrhal laryngitis* the stenosis appears suddenly, but is of short duration. *Ascending croup* first gives the symptoms of an acute bronchitis, with the rapid development of cyanotic or cadaverous discoloration of the skin, and the evidences of carbon-dioxide poisoning. In the course of the following days laryngeal symptoms appear, just as in croup. Auscultation discloses incon-

stant râles, and small areas over which the respiratory murmur is inaudible, unaccompanied by any dulness.

Most of the children die, and but little relief can be obtained by performing tracheotomy.

An adult in the best of health may be attacked by the symptoms of stenosis. No swelling of the neck is visible; mouth, tongue, tonsils, etc., are normal. The patient is still able to talk. Sitting erect in bed, with neck stiffly extended, he briefly answers questions in a low, hoarse voice. Pain in the larynx, a change in the voice, or marked hoarseness and cough point to catarrhal laryngitis, the stenosis being due to collateral Œdema of the Glottis. The laryngoscope, which, as a rule, can be employed, shows the aryteno-epiglottidean folds distended into veritable sacs. If examination with the mirror is impossible, the finger introduced into the mouth can plainly distinguish these eminences when the tongue is strongly protruded. In such cases the patient's entire body must be examined, in order to find out whether œdema is present elsewhere, for people suffering with Bright's disease may have œdema of the glottis as the result of a catarrhal laryngitis. Such individuals show marked œdema of the lids after crying. Besides, it is well known that patients with kidney disease may have sudden attacks of dyspnœa, known as *Asthma urinosum*, and which is classified with bronchial asthma. Such cases are not infrequent. I saw a case in which, in addition to a moderate amount of compression stenosis due to goitre, severe attacks of dyspnœa occurred as the result of Bright's disease.

At the present day we use the term œdema *laryngis*, because the glottis is a cleft, and a cleft can not swell up. Boyle, who gave the first systematic description of the disease, believed the process to be a

non-inflammatory serous infiltration of the submucous tissues of the glottis. Later, it was found that œdema of the glottis most frequently occurred as the result of inflammation, and in consequence of this advance in our knowledge the terminology was altered. Therefore we speak of laryngitis submucosa purulenta, or laryngitis œdematosa, or laryngitis phlegmonosa—terms which designate the underlying condition, and which are consequently more accurate.

Exceptionally, acute infiltration of the submucous tissues, occurring without any previous disturbance, may suddenly threaten the patient's life. This is most often due to a foreign body arrested at the rima glottidis.

Ziemssen records such cases. A labourer ate his meal in great haste, suddenly developed marked dyspnœa, and died. At the necroscopy a small spicula of bone was found wedged in the entrance of the larynx, producing œdema of the glottis. In another case the trouble was due to the rib of a tobacco-leaf; in a third, to a splinter of wood.

Frequently the œdema develops in a more subacute fashion, the stenosis increasing in the course of several days or even weeks. The development of the condition can be watched, but the course is very treacherous. In consequence of some small irritation, the gravest symptoms may take us by surprise.

A minute abscess in the vicinity of the rima glottidis may imperil the patient's life. The symptoms which precede the attack are, darting pain, increased on swallowing, pain on pressing upon the larynx, and a moderate irritation of the throat producing an irritative cough. Suddenly dyspnœa develops. Often grave fears are not entertained, and in many cases the danger is transitory; but at the next attack the dyspnœa may be so severe that only the most rapid interference will save life.

The significance of PERICHONDRITIS LARYNGEA varies in different cases. The anatomical situation of the process must be kept in mind. Türck, who has done exceptional work both in the pathology of the spinal cord and in laryngoscopy, has carefully studied the variations in the aspect of the disease which depend upon whether the focus is situated on the arytenoid, thyroid, or cricoid cartilages. These points would be very difficult for a beginner to determine, especially as the diagnosis is largely based upon the laryngoscopic pictures. The following points will prove of value: Perichondritis laryngea is, except in very rare instances, due to typhoid, syphilitic, tubercular, or carcinomatous ulceration. The diseases which are the primary trouble—namely, ULCERS OF THE LARYNX—must therefore be considered.

Typhoid ulcers are the least frequent variety encountered, especially at the present day, for the primary disease runs a milder course when treated by hydrotherapy. The process may be likened to the bedsores which appear over the sacrum and trochanter.

Tuberculosis of the larynx is usually secondary to a tubercular process of the lung; only in rare instances is it primary. The ulcers show great variety in their course; but if we regard their clinical manifestations in reference to stenosis, we may sum up the subject by stating that the more advanced stages of ulceration are met with on the parts most exposed to the irritations occasioned by deglutition and phonation. The ulcers are therefore situated on the mucous membrane covering the vocal cords, that of the cartilages of Santorini, or of the aryteno-epiglottidean folds. The cartilages are laid bare, the joints invaded, the parts exposed become necrotic, so that most of the upper portion of the larynx may be destroyed.

Syphilis manifests itself in many ways. It is seen as syphilitic catarrh, condylomata lata, gummata, and ulcerations. Stenosis is caused by the cicatrization of the deep ulcers which result from the breaking down of gummata. They cover large areas and may be