STRIDOR IN INFANCY

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THE intralaryngeal causes of stridor in infancy may be classified under the headings of congenital anatomical abnormalities; tumours and cysts; inflammatory conditions; neurological abnormalities; trauma, at birth or later; and the presence of foreign bodies.

TABLE I

INTRINSIC LARYNGEAL CAUSES OF STRIDOR IN INFANCY

Congenital Anatomical Abnormalities	Tumours and Cysts	Inflammatory Conditions	Neurological Abnormalities	Trauma	Foreign Body
Congenital laryngeal stridor	Papillomata of the larynx	Acute laryngitis	Tetany (Laryngismus stridulus)	Birth injury	Vegetable foreign body
Webs of the larynx	Cysts of the larynx	Laryngo-tracheo-bronchitis	Neonatal tetany	Post-natal injury	Non-vegetable foreign body
Congenital stenosis of the larynx		Diphtheria. Post-diphtheritic stenosis	Recurrent paralysis	Injury caused by intubation	
		The exanthemata (Measles, whooping-cough)			

There are many points of difference between the infantile and the adult larynx, for the infantile larynx is, of course, far from being simply a miniature edition of the adult one, just as a baby is anything but a small adult.

These differences are both anatomical and physiological, and they explain why stridor is the presenting symptom of laryngeal disease in children as hoarseness is in adults.

Anatomical Differences

(1) Obviously the principal anatomical difference is in *size*. This difference is not only relative, but actual, for the lumen of the infantile larynx and trachea is smaller in proportion to the body as a whole than is the case with the adult, particularly with the male adult. The larynx may be regarded as an isthmus in the respiratory tract, and it is relatively narrower in the child than in the adult. The greater "choke", which is most marked in the sub-glottic region, will obviously predispose to the occurrence of stridor. Gabriel Tucker (1932), who devised a special forceps for taking laryngeal measurements during life, states that while a sub-glottic diameter of 6 mm. is normal, 5 mm. is reduced, and 4 mm. represents a definite stenosis. It must be remembered that before a

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diagnosis of stenosis can be established, it is necessary to correlate the size of the infant and the lumen of his larynx.

(2) A second and most important difference is in the *consistency* of the tissues of the larynx. It is obvious that in young children all the laryngeal tissues including the cartilaginous framework, the musculature, and the mucous and submucous tissues are softer than in the adult. The cartilage is softer and more pliable and the mucosa looser and less fibrous. The relatively narrow lumen, and the softness and laxity of the infant's larynx are important causes of stridor when the laryngeal tissues are further narrowed by inflammation, as in simple laryngitis and tracheitis and in acute laryngo-bronchitis.

(3) A third difference is in *position*. In the infant the larynx is placed high, and it descends continually during development. In a fœtus of five or six weeks the larynx is situated opposite the basi-occiput, but by the fourth month the lower border of the cricoid lies opposite the upper border of the fourth cervical vertebra. At seven months it lies about the middle of the sixth vertebra, and it is still found in this position at full-term. After birth further descent occurs until in adult life the lower border of the cricoid cartilage lies opposite the lower border of the sixth cervical vertebra, while the top of the epiglottis, which marks the upper border of the larynx, lies opposite the lower border of the third cervical vertebra (Negus, 1949).

As a result of the higher position of the larynx in infants, the line of entry of the air-current is straighter than in the adult, and the epiglottis less over-hanging. In later life, when the larynx has descended to its final position, the axes of the pharynx, larynx, and trachea meet at a more acute angle. This straighter air-entry in infants, with its obvious bearing on the production of stridor, is not accepted by all those who have written on the subject. For instance, Gabriel Tucker says: "In the infant the entrance to the larynx is at an angle from behind forward and downward toward the glottic lumen. With the descent of the larynx as the child grows the epiglottis assumes a more nearly vertical position, making the axis of the lumen at the entrance to the larynx more nearly in line with the subglottic larynx and trachea." However, examination of post-mortem material shows a straighter air-entry both in the fœtus and in the infant. It is possible that the error has arisen from the practise of removing the tongue with the larynx, as is usually done. When such a specimen is placed in the preserving fluid the tongue, instead of remaining in its normal position at right angles to the larynx, stiffens into a vertical position, and thereby pushes the epiglottis over the laryngeal vestibule like a lid. This reproduces what in fact normally occurs during growth, when the tongue slips over backwards into the pharynx as the larynx descends.

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(4) Finally, there are considerable differences in *shape*. The upper end of the larynx and trachea is funnel-shaped in the infant, the cricoid plate being tilted backward, while the tracheal lumen becomes smaller as it descends. The laryngeal lumen is smaller, as shown by the shortness of the vocal folds, and the trachea is narrower even than the larynx. This funnel-shape disappears in the older child and in the adult female, but reappears in a modified form in the adult male, the backward tilt of the cricoid being replaced by a forward tilt of the thyroid cartilage. The tracheal lumen, however, no longer diminishes as in the infant. Other differences are that the vocal folds are shorter ; and the epiglottis, while it overhangs the vestibule less than is the case in the adult, is narrower, and in the type known as the " exaggerated infantile epiglottis " is sharply folded in two longitudinally. This is erroneously supposed to be the type of larynx normally found in congenital laryngeal stridor. An increase in the obliquity of the angle of air-entry and these differences in shape are generally accepted as being, when present in marked degree, the cause of the disease known as congenital laryngeal stridor.

Another anatomical feature is the relatively large size of the ventricular air-sac in infants. This may have quite an important bearing on the production of stridor ; indeed there is a recorded case of fatal laryngeal obstruction in infancy in which enormously dilated laryngeal saccules were present.

Physiological Differences

In addition to these well-marked anatomical differences between the infantile and the adult larynx is the fact that the infant's nervous system normally is relatively irritable and unstable, and easily thrown into spasm. Relatively slight peripheral stimulation of the respiratory or digestive systems, acting on the phonation centre in the floor of the IVth ventricle *viâ* the widespread afferent fibres of the vagus, will cause spasm of the larynx. Hyper-excitability of the neuro-muscular apparatus of the larynx is thus an important factor in the production of stridor.

The infantile larynx therefore differs anatomically from the adult in four ways, which may briefly be summarized as relative size, consistency, position, and shape. When these anatomical infantile characteristics are present to an abnormal degree, the insufficiency of the laryngeal air-way and the softness of its walls may result in the syndrome known as congenital laryngeal stridor, or laryngomalacia.

When the normal infantile larynx becomes inflamed, as in diphtheria, and acute laryngo-tracheo-bronchitis, the already relatively small laryngeal lumen is further greatly reduced by the gross œdema permitted by the loose areolar tissue of the larynx, particularly in the subglottic area. Here again the presenting symptom is stridor. A third important

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cause is spasm, occurring in tetany, and commonly known as laryngismus stridulus.

Congenital laryngeal stridor is said by high authority to be a rare disease; of 11,076 live births in the Rotunda Hospital during the years 1947, 1948 and 1949, only two cases were seen. It is also said to be rarely fatal. This is true in so far that it is rarely a *direct* cause of death, but in ten recent cases sent to the National Children's Hospital, one death occurred from lung complications. This death occurred in hospital, and it is possible that a higher percentage of fatalities occur in children who are nursed at home. It is also possible that some of the considerable number of infants whose deaths are reported from "suffocation" or "overlaying" really die from some such cause as this.

The symptoms of congenital laryngeal stridor date from birth or soon after, and the stridor may be brought on by handling in such ways as bathing, changing clothes and feeding. It is constantly present, but subject to intermissions. The child may be lying comparatively quietly when some stimulus is applied. He is awakened suddenly, becomes cold or frightened, or the respiratory centre becomes stimulated by an accumulation of CO_2 in the blood after a period of quiet respiration; the larynx collapses and prevents proper air-entry, and the struggle begins, to end after a time with the child exhausted and pale. The nature of the sound varies, and is hard to describe in words. It is, however, usually low-pitched, and is constantly present, even during sleep. Like all laryngeal stridor, and in contradistinction to tracheal stridor, it is inspiratory in timing. Occasionally in severe cases a slight expiratory element is present, and this may synchronize with bulging of the tissues of the root of the neck (Norris, 1949); this may be caused by kinking of the larynx by upward pressure resulting in a further angulation of the air-entry at the laryngeal vestibule. Retraction of the soft tissues with inspiration occurs at the supra-clavicular and, to a lesser extent, in the epigastric region. This high retraction differentiates laryngeal obstruction from blockage lower down, as in pneumonia, when the retraction is greater in the epigastrium. Curiously enough, cyanosis is of infrequent and usually temporary occurrence, as a rule being seen only at the worst of an attack. Cases have been described in which tracheotomy was necessary; such an extreme degree of respiratory obstruction is seldom seen, nor is one often tempted to amputate the epiglottis, which suggests itself as a simple method of affording surgical relief.

As in all cases of stridor X-rays are an important part of the examination, but in congenital laryngeal stridor the information given is negative, its value being in the exclusion of other causes. Indirect laryngoscopy should be carried out and will give more information than it is usually credited with, but for a thorough examination and a full view of the larynx direct laryngoscopy is necessary. A point of technique is that the

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tip of the laryngoscope should not be placed below the epiglottis but in the valleculla.

The classical description of the laryngoscopic appearances of this condition is given in StClair Thomson and Negus's (1948) text-book. This description and the accompanying picture has been reproduced in the literature over and again for a period of forty or fifty years. It is based upon the observations of Lambert Lack and of D. R. Paterson and A. Brown Kelly. Lambert Lack is quoted as saying : " The epiglottis was sharply folded on itself, the two lateral folds being in close apposition and in some cases in contact. The aryteno-epiglottic folds were approximated, and thus the upper aperture of the larynx was reduced to a long narrow slit. The thin folds bounding this aperture seemed quite flaccid and flapped to and from on respiration. The inspiratory column of air striking down on these folds drove them together, and on expiration they again separated. In some cases, the 'purring' ones, the coarse vibration of these cords could be seen."

It is further stated in the same text-book that " By means of direct inspection D. R. Paterson and A. Brown Kelly demonstrated that the larynx, as seen clinically, is of an exaggerated infantile type. The epiglottis is very long and tapering and its lateral margins are rolled backwards so as to meet, and thus form a complete cylinder above. The greatly reduced entry to the larynx is bounded by the aryepiglottic folds which are too closely opposed to admit any but the slightest amount of air. The croaking noise is caused by the free and unsupported part of the posterior laryngeal wall and neighbouring loose tissue on the summits of the arytenoids, which is sucked forwards and inwards during inspiration."

This is, of course, a perfectly accurate account of the findings in a certain number of cases, although it is only true of a certain proportion. When, however, Lack asserts that he has " frequently examined the larynx in children and has never observed the malformation described except in association with this affection " it must be pointed out that this type of larynx is frequently seen in children who have never suffered from stridor, as those who intubate children for their anæsthetist, or who look down the laryngoscope when the anæsthetist is doing so, will agree.

The laryngoscopic appearances in children, stridorous or healthy, perceptibly varies only with the epiglottis, which varies from the " exaggerated infantile " type, which is flatly folded in two along its long axis, as in Lack's description, to the " omega-shaped " type, which approximates more to that which is seen in the adult. Half-way between these is the tightly-curled intermediate type described by Paterson and Brown Kelly.* These are probably all variations of the normal, the

* Chevalier Jackson seems to use the term omega-shaped for what I have called the " intermediate type ".

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infant type being the most unusual. Very often the adult epiglottis retains recognizable traces of its infantile shape.

Of twenty-one larynges of still-born infants recently examined, eleven had the normal omega-shaped epiglottis, eight had the cylindrical type, with increased curl at the tip, and only four had the laterally-folded upright type of epiglottis. Of ten cases of congenital laryngeal stridor seen at the National Children's Hospital during the last two years, five were adjudged to have the normal omega-shaped epiglottis, three to have increased curling at the tip, and the remaining two to have the laterally-folded " exaggerated infantile " epiglottis. These figures, although insufficient for proof, support the contention that the shape of the epiglottis is not the cause of congenital laryngeal stridor. The condition is perhaps more common in cases with the exaggerated infantile epiglottis, but an infant possessing a larynx of any one of these types may suffer from it.

The mechanism of the production of stridor, however, seems to differ with the type of epiglottis, which probably also has an effect on the *bruit*. There is nothing to add to Lack's admirable description in the case of the upright laterally folded epiglottis. In the case of the flat or " omega-shaped " epiglottis the mechanism is different. Here the epiglottis, under the pressure of the inspiratory stream, appears to fold transversely across its base, thereby coming down upon the vestibule as in the normal mechanism of swallowing.

Simultaneously with this inspiratory dipping of the epiglottis, the aryepiglottic folds are drawn anteriorly, not medially, while the soft superficial tissues covering one or other of the arytenoids is pulled across the middle line in front of its fellow. One gains the impression that the larynx is not nearly rigid enough to withstand the enormous strain.

Pancoast (1930), in his now classical article on the X-ray examination of the thymus in infancy, appears to describe a similar mechanism seen radiologically. He states " in the necks of many infants we have noticed that the shadow of the tips of the arytenoid cartilages closely approximate the lower margin of the epiglottis * * * * as the inspiratory phase is about to begin. This is the position normally assumed during the swallowing act * * * *. This appearance in infancy during respiration was at first regarded as an evidence of unusual soft-tissue relaxation and resulting collapse, but as it was later noticed in many clinically normal children, we have learned to regard it as abnormal and an evidence of collapse only when there is an associated lack of air in the laryngeal vestibule."

The theory that congenital laryngeal stridor is caused by an anatomically abnormally shaped larynx, must, therefore, be rejected for two reasons: first, that many cases showing an " exaggerated infantile " larynx are seen in infants who do not and never have suffered from stridor,

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and secondly, that a considerable number of cases of congenital laryngeal stridor occur in children with the normal " omega-shaped " epiglottis.

It is obvious that the stridor in these cases results from a valvular closure caused by the concentrated pressure of the inspiratory stream, and it is tempting to assume that this collapse results from immaturity causing undue flabbiness of the epiglottis and inefficiency of the posticus muscles. Chevalier Jackson (1936) presumably considers this the whole explanation, as it was he who suggested the term " laryngomalacia " for the condition. But congenital laryngeal stridor is not of particularly common occurrence amongst premature infants, which suggests that immaturity is not the whole explanation.

Schwartz (1944) considers that the disease is linked with micrognathia, glossoptosia, and other manifestations of arrested development of the first branchial (mandibular) arch. But well-developed micrognathia is not often seen in association with congenital laryngeal stridor, although it is fairly common in cases of cleft palate. It would seem that the underlying cause is much more likely to be tracheal stenosis combined with immaturity and softness of the tissues. It would obviously be very difficult to prove this, as so few of these cases come to post-mortem, and because there are so many relative factors of size and body-weight to be taken into account before a diagnosis of tracheal stenosis can be established.

Laryngismus Stridulus

The cause of the stridor in laryngomalacia or congenital laryngeal stridor is therefore the approximation of the flabby laryngeal tissues by the suction of the inspiratory air stream as it passes through the isthmus of the respiratory tract. As far as the larynx is concerned its causation is passive in character. In laryngismus stridulus the reverse is the case, for in this condition the stridor is caused by the spasmodic contraction of the adductor muscles of the larynx. It is, of course, merely one of the signs of a much larger clinical entity : tetany, or spasmophilia, resulting from rickets. Laryngismus stridulus occurs most often in under-nourished rickety children who frequently suffer from cœliac disease.

Rickets is not a common disease nowadays, but between the years 1942 and 1948, there was, in Dublin, a serious increase in its incidence, brought about in a curious way. It was found that the use of the whole wheat-grain in making bread, apparently a reasonable step in times of shortage, was responsible. The husk of wheat-grain contains phytic acid, which combines with ingested calcium to form an insoluble compound which is excreted. As a result a definite increase in the incidence of rickets took place. It was estimated that 25 *per cent.* of children under 4 years of age, and 43 *per cent.* between the ages of 18 months and

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2 years, suffered from the disease. Amongst the cases described were a number who developed spasmophilia.

The attack of laryngismus stridulus occurs suddenly, often at night. The child, who was previously lying quietly in bed, is suddenly afflicted with great respiratory distress. He sits up, if old enough to do so, clutching at the sides of his cot for support, struggling and gasping for breath. All the accessory muscles of respiration are called into play, the chest heaves, the nostrils widen, and the pupils dilate. The face becomes blue, and air is sucked in with loud crowing respirations. After a time the child takes a long deep breath and recovers. Chvostek's sign, Trousseau's sign, carpo-pedal spasm, opisthotonos, incontinence and other unpleasant manifestations may also occur.

It obviously would be difficult to obtain a good view of the larynx under these circumstances, and even if it were possible it is not likely that information of any value, diagnostic or otherwise, would be obtained. The Jacksons state that the larynx is usually of a violet or lavender hue because of cyanosis, and is in a state of sphincteric closure ; which is merely what one would expect. However, the sudden onset, the spasmodic nature of the stridor, and the associated signs and symptoms should make it quite easy to differentiate this condition from congenital laryngeal stridor. When the diagnosis has been made treatment may safely be left to the pædiatrician.

Tetany Neonatorum

As the incidence of rickets in older children diminishes, that of tetany with stridor in the newborn seems, if anything to increase. It is of interest if only because ten or fifteen years ago many pædiatricians stated categorically that tetany did not occur in the newborn. However, in the Rotunda Hospital during the years 1947, 1948, and 1949, of 11,076 live births, 48 developed neonatal tetany. Of these 48 cases, only one developed laryngismus stridulus, which seems to be a contrast to the experience elsewhere.

The symptoms of tetany neonatorum are quite well-defined. Soon after birth a child begins to appear irritable and easily stimulated, as shown by twitching and starting at noises. Following this convulsions appear, with all the familiar symptoms of tetany, including laryngismus stridulus. Chvostek's sign may be positive, but Trousseau's is usually absent. Stridor occurs occasionally and may be the first indication of the disease. Tetany neonatorum is, of course, quite different in ætiology to the tetany which occurs later in infancy, for it seems to be established that the cause in these early cases is defective function of the parathyroids. As the symptoms yield rapidly to appropriate medical treatment with calcium gluconate, vitamin D, sedatives, and parathyroid extract, the

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condition is of little interest to laryngologists except in the matter of diagnosis.

Acute Laryngitis in Childhood

In laryngismus stridulus, therefore, the stridor is caused by spasm. In the stridor of acute laryngitis of childhood another factor comes into play, for it is caused partly by spasm and partly by inflammation.

Every laryngologist is familiar with these cases. The child may have a slight cold. He goes to bed apparently well, but wakes up in the middle of the night, terrified, with a harsh croupy cough, severe inspiratory stridor, and dyspnœa. The attack subsides after an hour or so, and the child remains tranquil until morning, only for the sequence of events to be repeated on one or more subsequent nights. The syndrome has been unfortunately termed laryngitis stridulosa, but it is, in fact, merely acute laryngitis, with perhaps, sometimes a predominance of the spasmodic over the inflammatory symptoms.

These children are usually older, between the ages of three and seven or eight, and with tactful persuasion it is usually possible to get a good view of the larynx with the laryngeal mirror. The larynx as a whole will be seen to be injected and hyperæmic, but the striking feature is the " beefy-red " subglottic swelling. The diagnosis from diphtheria is easy, but measles and pertussis should also be borne in mind. One should also be alive to the possibility that the child may be developing acute laryngo-tracheo-bronchitis.

Acute Laryngo-Tracheo-Bronchitis

Since the decline in the incidence of diphtheria there has been in Dublin a marked increase of acute laryngo-tracheo-bronchitis. Whether this increase is actual or is merely due to better diagnosis is doubtful, for it is certainly no new disease. Morell Mackenzie, in his famous text-book published in 1880, states that " until diphtheria appeared in England in 1858, the term ' croup ' was employed to describe an acute affection of the larynx, believed to be inflammatory and non-contagious, in which false membrane was present."

The first modern description was published by Chevalier Jackson who, because of his pioneering work on endoscopy, was able to publish an accurate account of the disease in 1908. It is, of course, a disease which mainly affects children under three years of age, characterized by progressive laryngeal obstruction, and differing from diphtheria in that the mucosa is more œdematous and the secretion thick and tenacious. There is a tendency to the formation of inflammatory crusts in the air-passages which often results in bronchial plugging and atelectasis. It is a serious disease, difficult to treat, and has a high mortality, which is accentuated by the fact that mentally defective or weakly children with chest pathology

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in the form of enlarged hilar or para-tracheal glands seem more prone to develop it than well cared for, healthy infants. A number of cases occur in the post-eruptive stage of measles, and these for obvious reasons, tend to be more serious than those of primary origin.

Judging by the literature, the disease seems to be more common and perhaps more severe in America than here. Perhaps the drier atmosphere and air-conditioned houses of the United States causes a higher incidence in that country. It is probable that the dry air would tend to dehydrate the mucous membrane, and thereby prevent ciliary action and facilitate the penetration of organisms through the mucous membrane.

The disease is not of frequent occurrence in Ireland. Six cases only were admitted to Cork Street Fever Hospital, Dublin, during the last eighteen months, one of which required tracheotomy. All these patients recovered with the assistance of chloromycetin, which appears to be much more effective than penicillin or the sulphonamides, although it is of course impossible to draw conclusions from such a small number of cases. Recent reports seem to indicate that terramycin may be still more effective. For some years this condition has been treated with chloromycetin on the assumption that it is of virus origin, although there is as yet no proof that this is so, apart from negative bacteriological findings. In London and in Glasgow reports are available which show the chief findings at post-mortem to be redness and swelling of the arytenoids, inflammation of the trachea with blood-stained mucopurulent exudate, gross sub-glottic swelling, and pus in the bronchioles.

The onset of this condition is insidious, and, unlike laryngeal diphtheria, which, with vegetable tracheal foreign body, is the principal item of differential diagnosis, it is not rapidly progressive. Dyspnœa, with recession of the lower intercostals is the cardinal feature. Croupiness or a hoarse cry is not an early symptom, nor is cyanosis. The stridor is characteristically inspiratory and is sometimes crowing. The primary feature in the production of stridor in these cases is the relatively enormous swelling which takes place in the region of the *conus elasticus*. This will also cause dyspnœa and cyanosis, for as Negus has said, a swelling of the mucous membrane of one millimetre is a very serious matter in a child whose sub-glottic lumen is normally six or seven millimetres. The subglottic mucous membrane is not only swollen but is also ulcerated and covered in parts with a sloughy grey membrane. A serous exudate is poured out, which rapidly becomes mucoid, then purulent and perhaps sanguineous. It is markedly thick and tenacious, and exceedingly difficult to expectorate even by the robust adult (Jackson). The upper part of the larynx is not usually affected, but supraglottic cellulitis occurs in some cases. More often the trachea and bronchi are predominantly involved, and it is in these cases that pneumonia and atelectasis from plug formation form such a prominent part of the clinical picture.

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In the majority of cases the disease develops slowly, and this helps to distinguish it from diphtheria, in which the cry is thicker and the typical diphtheritic membrane is usually seen readily enough. Occasionally, however, one meets a fulminating case in which the infant is rapidly prostrated by an overwhelming infection. The laryngologist as a rule meets these cases first in the hospital ward, already installed in an oxygen or humidification tent, and they are usually very ill indeed. "As the disease progresses these patients become dehydrated, prostrated, and finally present a picture of complete collapse. The cyanosis disappears leaving an ashen pale skin and anoxic coma, with all the remaining energy being spent on breathing. Retraction may be so great that the sternum and thoracic spine seem to meet with each gasping inspiration" (Emery, 1950). It is a heart-rending scene. The onlooker is forcibly reminded of Hans Zinnser's statement that "infectious disease is one of the greatest tragedies of living things".

The medical treatment of acute laryngo-tracheo-bronchitis with chemotherapy and humidification raises points of great interest, but the instrumental treatment by tracheostomy and bronchoscopy is more the laryngologist's concern. Neffson (1937), who has had a very wide and successful experience of tracheostomy gives his indications under two headings. Under *signs of extreme obstruction* he gives: cyanosis going on to pallor; deep retraction; absence of breath sounds; and an anxious facies. Under *signs of exhaustion* he gives: tachycardia, impalpable pulse and heart failure; rapid, shallow, breathing; hyperpyrexia (105°-107° F.); stupor; coma; and convulsions. From this list of indications one must conclude that Neffson performs tracheostomy as a last resort. Others consider that if the obstruction is severe the operation should be done before signs of exhaustion appear. After all, it must be remembered that the obstruction to breathing in these cases may be as great below the tracheostomy as above it and that the primary object of the operation in most cases is to facilitate bronchoscopic suction and lavage.

There are a few points of technique which should be mentioned. The operation is made much easier by the prior insertion of a bronchoscope, first to aspirate excessive secretion and crusts, and then to steady the tiny trachea and bring it forwards. By doing so it is possible to avoid stripping the pretracheal fascia and the consequent risk of air being sucked into the chest. Pneumothorax is a very real danger in these cases, particularly if the opening is made too low down.

The immediate after-care of tracheotomized patients calls for devoted and skilled attention. Excess secretions, crusting and plug-formation have still to be combated. In the early stages of the disease the membrane is firmly adherent to the underlying mucosa, and if pulled off it leaves a raw bleeding surface. In the later stages the membrane swells into an

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amorphous sloughy mass which may separate off and which must be prevented from causing collapse of the lung. Opinions vary as to the best way of doing this, although the importance of keeping up a high degree of humidity in the oxygen tent is generally recognized. Some laryngologists regularly pass the bronchoscope through the tracheostome, instilling various solutions through the bronchoscope to loosen the crusts. This may be done as often as three or four times a day for a fortnight or more. Others prefer the use of a soft rubber catheter as being less traumatizing, and for the same reason only employ suction when the child is too weak to cough up the solution spontaneously. These measures are called for when the breath sounds at the bases disappear and the percussion note becomes dull, and particularly when to these signs are added a rapid increase in the rates of pulse and respiration.

The symptoms of acute laryngo-tracheo-bronchitis may be summarized as those of acute infection *plus* endogenous foreign-body formation. It is, therefore, easy to see how the presence of an exogenous vegetable foreign body in the air-passages might cause an error in diagnosis particularly if inflammation should supervene. Vegetable foreign body in the trachea or bronchi is, indeed, often given as an item in the ætiology of the disease, although its proper place is in the differential diagnosis. A vegetable foreign body which breaks down and becomes infected may, indeed, cause inflammation or allergic swelling of the air-passages with plugging and atelectasis, but acute laryngo-tracheo-bronchitis is a specific infection caused by an organism, probably a virus, which has not yet been identified.

Foreign Body

Non-vegetable foreign bodies can also cause mistakes in diagnosis, as is illustrated by a case seen recently. This child was aged nine months, and was in the process of being weaned. For its lunch on this occasion it was given a meal of beef-soup and potatoes, in the midst of which she suddenly coughed, and choked. Twelve hours later she developed severe and loud inspiratory stridor, and was admitted to the National Children's Hospital, Dublin, as a case of acute laryngo-tracheo-bronchitis. X-ray showed a linear shadow at the level of the vocal folds, but no corresponding shadow was at first seen on the lateral view. This caused a little confusion ; however, direct laryngoscopy showed a D-shaped lamina of beef-bone, so thin as to be almost transparent, which was removed without difficulty.

Shortly afterwards another child, aged $4\frac{1}{2}$, was admitted, with the history of having suddenly choked when playing on the beach. This child had double or " two-way " stridor, was cyanosed, and retraction of the supraclavicular and epigastric regions was present. On auscultation, and indeed without the stethoscope, a " slap" was heard at the end

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of each expiration. On X-ray and bronchoscopy this slap proved to be caused by a smooth ovoid pebble which was forcibly thrown at the lower aspect of the vocal folds at each expiration, only to be sucked back to the carina on inspiration.

Neither of these cases presented any difficulty in diagnosis, but had the foreign body in the second child been of vegetable nature it might have been more troublesome to determine the cause of the stridor. Chevalier Jackson's " asthmatoid wheeze " may be a helpful sign in these cases.

Laryngeal Web

Congenital webs of the larynx are another important cause of stridor in infancy, in most cases the stridor being brought on by exertion. The webs vary in size from those which completely occlude the larynx and are incompatible with life to a small membranous band stretching across the anterior commissure. Sometimes there is merely a small opening posteriorly, of two or three millimetres diameter. The anterior part may be quite thick (10-15 mm.), while the posterior free edge is thin, sharply outlined and regularly concave backwards. The position of the web is variable, and it may appear below the vocal folds or in the trachea.

The principal symptoms are inability to cry properly, the voice being weak and aphonic; and stridor, which may be inspiratory or both inspiratory and expiratory and perhaps accompanied by a " crow ". Difficulty in feeding, dyspnœa and cyanosis, with retraction above the clavicle and in the intercostal spaces may also be present. It is generally agreed that unless these symptoms are severe enough to endanger life the web is best left alone. For those upon which it is necessary to operate there are a number of ingenious methods to choose from, including division and the suspension of a silver plate between the cords (Haslinger); tracheotomy followed by laryngo-fissure and dilatation by an indwelling rubber tube (Killian); and simple incision along one cord (Jackson). The soundest procedure surgically probably is Negus's method of laryngo-fissure followed by Thiersch grafting.

Papilloma of the Larynx

Papillomatosis of the larynx is another cause of infantile stridor, and it can be a most troublesome, indeed fatal, condition. It produces much the same group of symptoms as laryngeal webs but is not usually seen until the child is over two years of age. Here again the site of the lesion varies, and once more the stridor may be either inspiratory or " two-way ", according to whether the papillomata occur above, in, or below the larynx.

Papillomata of the larynx, like warts occurring elsewhere in the body, are probably caused by infection with a virus, and, like warts elsewhere,

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they tend to disappear spontaneously at or before puberty. Treatment should, therefore, be limited if possible to local removal and the topical application of one of the various escharotic solutions which are recommended. Tracheostomy may be required, but it is important to remember that papillomata may occur in the trachea as well as in the larynx.

Characteristics of Stridor as an Aid to Diagnosis

In both congenital web and papillomatosis the stridor may thus be either inspiratory or double. This variation in the nature of the stridor is interesting, for it may be a guide to the site of the obstruction. Generally speaking, an obstruction to respiration at or above the vocal folds will cause inspiratory stridor, while below that level it is usually double or expiratory in timing.

The inspiratory nature of the stridor caused by lesions at or above the focal folds no doubt results from the valvular nature of the block. This is seen in congenital laryngeal stridor, whooping-cough, and laryngismus stridulus. In acute laryngo-tracheo-bronchitis the stridor is also inspiratory as a rule, although the obstruction here is partly, indeed mainly, subglottic. Thymic obstruction will also at times cause an inspiratory stridor, which differs from that of congenital laryngeal stridor in the important fact that it is not present during sleep (Norris). Similarly, supraglottic causes of obstruction will cause inspiratory stridor; for instance lingual cysts and choanal atresia, in the latter of which a peculiar clicking noise on inspiration is described. The newly-born infant's urge to breath through the nose is tremendous: when it is impossible to do so he still makes violent efforts at nasal respiration. He turns blue and chokes: finally he manages to take a breath through his mouth, and in the transition from attempted nasal to oral breathing this "click" is made. A similar mechanism may be observed in "hairy dermoid" of the naso-pharynx which is also a disease of the newborn.

When the obstruction to respiration is in the trachea, the nature of the sound changes. The stridor is now either double or predominantly expiratory, as in tracheomalacia, vascular ring, tracheal and bronchial foreign-body. A pronounced expiratory stridor may sometimes be heard in cases of pneumonia with little or no laryngeal or tracheal inflammation. Chevalier Jackson's "asthmatoid wheeze" in cases of vegetable foreign-body in the air-passages is another example of expiratory stridor caused by a low obstruction.

It may therefore be said that inspiratory stridor usually has its origin at or above the vocal folds, while expiratory or double stridor as a rule is caused by a lesion below this level. This is, however, a broad generalization rather than a hard-and-fast rule.

Table II is a classification of the nature of the stridor produced by various intra-laryngeal conditions.

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TABLE II.

Disease	Direction	Sustained or spasmodic	Age of onset	Presence during sleep	Voice
Congenital laryngeal stridor	Inspiratory	Sustained	From birth or soon after	Diminished	Clear
Laryngismus stridulus	Inspiratory	Spasmodic	From six months to two years	An attack will awake the patient	Clear
Tetany neonatorum	Inspiratory	Spasmodic	Soon after birth	An attack awakes patient	Clear
Pertussis	Inspiratory	Spasmodic	Variable	An attack awakes patient	Clear
Acute laryngitis	Inspiratory	Spasmodic	Variable, three to eight years	Occurs most often at night	Hoarse and strong
Diphtheria	Inspiratory	Sustained	Variable	Yes	Hoarse
Acute laryngo-tracheo-bronchitis	Inspiratory or "two-way"	Sustained	Variable	Yes	Hoarse
Congenital Web	Inspiratory	Sustained	Soon after birth	Yes	Weak
Papillomatosis	Inspiratory or "two-way"	Sustained	Not usually before two years of age	In severe cases	Hoarse

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