At first it was thought that here was a new disease, but this idea was rapidly dispelled when search of the literature concerning epiglottitis and supraglottitis showed that several papers had been published in the USA (Sinclair 1941, Alexander *et al.* 1942, Du Bois & Aldrich 1943, Davis 1947), where the condition was well recognized. There was also a full analysis of 8 cases by Miller (1946), based on admissions to the Los Angeles Children's Hospital. Rabe (1948) analysed 347 cases of infectious croup admitted to hospital between 1937 and 1946, of which 28 proved to be due to epiglottitis (*H. influenzæ* Type b).

Since then much more has been written, and attention drawn to the fact that Sinclair (1941) described 10 cases of acute laryngitis with septicæmia caused by *H. influenzæ*. It is of interest in this connexion that I have seen epiglottitis associated with meningitis. Saphir (1945) reported 5 cases of sudden death following œdema of the larynx and acute myocarditis, and McLorinan (1957, personal communication), in an account of 46 cases in Melbourne, has stressed that this organism is almost entirely responsible for the supraglottic form. In fact his statement that the diagnosis of supraglottic œdema is preferable to 'epiglottitis' may afford some explanation for the later confusion. On semantic grounds 'œdema' is not specific, whilst the suffix '-itis' is. Many other papers have since been published.

The clinical picture has been described by Camps (1953) and Jones & Camps (1957), and it should be mentioned that as far back as 1950 Jones and Camps met in the post-mortem room over 2 cases within a few days of one another. Since then Jones has seen and treated many cases in his practice and the condition is well described in medicolegal textbooks (Camps 1968, 1969).

Summary

(1) Acute epiglottitis is an infective disease which occurs in USA, Canada and Australia as well as in the British Isles in children.

(2) It is specifically caused by *Hæmophilus influenzæ* Type b.

(3) It has been extensively described in the literature and in adults by Potondi & Ribári (1969).

(4) Its existence in Britain has not been emphasized.

(5) Deaths have occurred and whether or not it has been diagnosed at autopsy has depended upon the recognition of its existence and proper microbiological examination.

(6) The exact mortality rate is not known, nor the true incidence of the disease except in localized areas.

(7) It underlines the fact that coroners' autopsies, when properly carried out, can contribute to medical knowledge as well as to criminal investigation.

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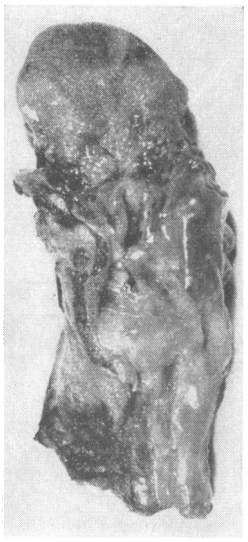
Acute Epiglottitis: A Personal Study over Twenty Years

Acute epiglottitis can be defined as an acute inflammatory œdema leading to acute laryngeal obstruction, occurring in the supraglottic area of the larynx and involving the epiglottis and hypopharynx. It is a well-recognized clinicopathological entity, especially in the USA, Canada and Australia.

Whilst working in a hospital near London 2 cases of this condition occurring in young children came under my care in one week of February 1950. The first case, a female child aged 18 months, died within a few minutes of admission to hospital. The second case, another female child aged 2 years, was admitted two days later; to relieve the acute respiratory obstruction the patient was intubated with an endotracheal tube and antibiotic treatment with tetracycline instituted. A few hours later the tube became blocked and the patient died.

Both cases came to coroner's post-mortem which was carried out by Dr F E Camps, the Home Office pathologist. I attended and was fortunate in being able to discuss both cases with him. He had become aware of the condition known as acute epiglottitis as a cause of sudden death in young children, and had seen 2 other cases at post-mortem in the previous few weeks. He demonstrated the acutely swollen tissues of the larynx and epiglottis, how the œdema stopped at the level of the vocal cords and that the trachea was unaffected. He suggested that to prevent death early tracheotomy was the treatment of choice. A further patient seen in December 1950 recovered after tracheotomy and antibiotic therapy. No more cases were seen until December 1955, when I was working in Bradford; 3 cases occurred within one fortnight and all recovered following treatment by tracheotomy and antibiotics. The most dramatic of these was



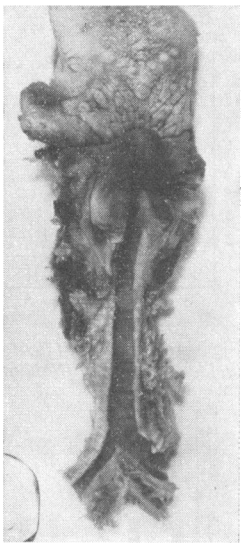


Fig 1 Fig 2

Fig 1 *Post-mortem specimen from case of acute epiglottitis, showing gross œdema of epiglottis, arytenoids and especially œdema in hypopharynx*

Fig 2 *Post-mortem specimen from a child aged 2½ years who died from acute epiglottitis. Specimen shows gross œdema of epiglottis and hypopharynx. Trachea and larynx below vocal cords clear of disease*

fortunate in being the second case admitted during this fortnight. The hospital theatre staff, pædiatricians and surgeons had been alerted by the case seen a few days previously; urgent tracheotomy resulted in recovery. Further cases were seen in 1956 and following the successful treatment of this small series a paper (Jones & Camps 1957) was published. Of the 31 cases then reported 26 were post-mortem cases seen by Dr Camps, 2 of these being the first cases seen by me. The remaining 5 were my cases and of these 4 had recovered and the one adult patient had died. Since 1957 I have continued my interest in acute epiglottitis, seeing an odd case from time to time with sporadic bursts of two or three cases in 1960, 1962 and the winter of 1967–68 and one in 1969. I have now seen 24 cases, 21 of these being in the Bradford area (4 adults and 17 children).

Recent reports in the British literature have stated that acute epiglottitis is a rarely recognized emergency (Andrew & Gardner 1963) and again that it is virtually a necropsy diagnosis in Britain (Johnstone & Lawy 1967). The purpose of this paper is to demonstrate that where there is a close association between the laryngologist and the pædiatric department these cases can be recognized at an early stage. They occur in Britain as elsewhere and following early recognition can be treated successfully by tracheotomy and antibiotics.

[Case reports of 24 patients were presented.]

Anatomy

The mucous membrane of the larynx is continuous above with that of the mouth and pharynx, below with that of the trachea. The epithelium is firmly adherent to the vocal cords, it is also attached but less firmly so over the laryngeal surface of the epiglottis and laryngeal surfaces of the arytenoid cartilages. Elsewhere the epithelium rests on a basement membrane beneath which is a layer of submucous connective tissue. This submucous layer is continuous above with the submucosa on the lingual aspect of the epiglottis and with that in the hypopharynx. The submucosa is narrow over the laryngeal aspect of the epiglottis but is very loose and lax over the lingual aspect of the epiglottis and in the hypopharynx. It is loose also in the subglottic region.

Pathology

As a result of the intimate adherence of the epithelium of the larynx to the vocal cords, any lesion above the glottis causing submucous œdema in this region cannot extend below the cords and similarly any submucous œdema occurring in the subglottic region cannot extend upwards beyond the vocal cords. Hence two different lesions can be distinguished: (1) Those occurring above the vocal cords. (2) Those occurring below the vocal cords.

(1) In supraglottic conditions causing œdema this is accommodated initially in the relatively large lax submucosa over the lingual aspect of the epiglottis and in the hypopharynx (Figs 1 & 2). Only later does the swelling extend to involve the supraglottic area of the larynx and cause respiratory obstruction, often of sudden onset. A feature of these cases is that the respiratory obstruction may get suddenly worse and cause death from asphyxia before help can be given. The exact mechanism of the obstruction causing asphyxia is not clear. It has been suggested that the swollen and loose supraglottic structures are drawn into the glottis and plug the lumen. There is a sudden sense of choking and the patient responds with frantic inspiratory efforts which impact the swollen tissues even more firmly.

However, it has been pointed out that the epiglottis and aryepiglottic folds are so swollen and rigid that this indrawing of œdematous tissue is unlikely. A more likely explanation of the sudden onset of asphyxia is that rigidity of the supraglottic structures plus swelling in the hypopharynx prevents the normal swallowing of saliva and that the aspiration of oropharyngeal secretions into the already dangerously narrowed airway results in acute respiratory obstruction and death.

The picture described above can also be seen in children who have sustained burns of the pharynx following inhalation of steam or ingestion of a

very hot beverage. In the condition known as acute epiglottitis the pathology explains the symptomatology. Initally there is a mild illness with soreness in the throat followed by difficulty in swallowing and then by respiratory obstruction of sudden onset. It is important to realize that this comes on late in the progress of the disease. The appearance belies the gravity of the condition and because of lack of distress in the early stages the physician is led to believe that he is dealing with a mild case of croup. Herein lies the danger for asphyxia can occur before help can be given.

(2) In the case of œdema below the vocal cords it may be confined to the subglottic area, the area of the conus elasticus or it can extend down the trachea and involve the bronchi. This has been called acute laryngotracheitis or laryngotracheobronchitis. In this paper this terminology has been confined to conditions occurring below the vocal cords. Where there is distinct narrowing of the airway at the subglottic level extending only a short distance, inflammatory swelling quickly causes respiratory obstruction and is most marked in the very young. Since the space in this region is limited, œdema immediately produces signs of obstruction and this is slowly progressive. This manner of development gives the physician a margin of safety and allows him to wait for possible improvement before resorting to operation for the relief of respiratory obstruction. Hoarseness may develop later.

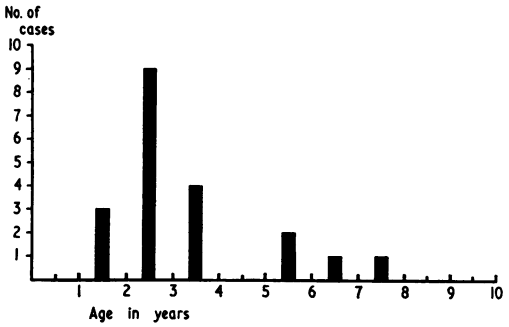


Fig 3 *Age in cases of acute epiglottitis: maximum incidence between 18 months and 3 years*

Fig 4 *Age in cases of* H. influenzæ *Type b meningitis: maximum incidence between 6 months and 2½ years*

Etiology

In acute epiglottitis the organism most commonly found is *H. influenzæ* Type b. With this organism it is believed that there is a generalized blood infection with local manifestations, most commonly meningitis, less commonly acute epiglottitis, middle ear infection and infection in the joints. These can occur in more than one site at the same time. In 2 cases reported by Camps (1953) patients had *H. influenzæ* Type b meningitis and died suddenly. Post-mortem revealed that acute epiglottitis resulting in acute respiratory obstruction was present in both cases, also reported in 2 of McLorinan's cases (1957, personal communication).

H. influenzæ is a small Gram-negative bacillus often markedly pleomorphic in appearance. It is a common inhabitant of the human upper respiratory tract, the majority of strains found in such a situation being noncapsulated. Capsulated strains are in a minority and can be divided serologically into six types designated by letters a–f. Such division depends on the fact that they differ in chemical structure of the polysaccharides of which the capsule is composed. Investigation in New York showed that 25% of children admitted to hospital had *H. influenzæ* in the upper respiratory tract, only 5% of these being capsulated. Of 41 capsulated strains, 31 were Type b and most of these came from children with severe upper respiratory tract infections, thus among the capsulated strains Type b seems to have the greatest pathological importance (Turk & May 1967). It is thought by some authorities that acute epiglottitis is invariably due to infection with *H. influenzæ* Type b, though Neffson (1949) thought that hæmolytic streptococci, pneumococci and staphylococci were also each responsible for a few cases. However, if these common organisms were responsible, one would expect acute epiglottitis to occur much more frequently. In the present series *H. influenzæ* Type b was isolated in 8 cases, other organisms found being staphylococcus in 4 cases and *N. catarrhalis* in 2; the swab most likely to give a positive result was that taken from the larynx. No positive blood cultures were obtained, possibly because treatment with intravenous antibiotics had been started before blood was taken for culture.

Incidence

Baxter (1967) reported 1,427 cases of respiratory obstruction admitted to Montreal Childrens' Hospital during the years 1951–65. Of these 1,324 were diagnosed as acute laryngotracheitis and 103 as acute epiglottitis (i.e. 8% approx.). Neffson

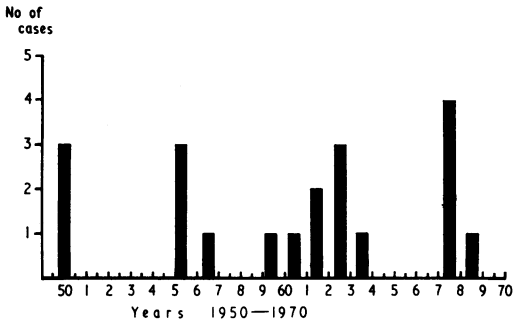


Fig 5 *Yearly incidence of* H. influenzæ *Type b epiglottitis*

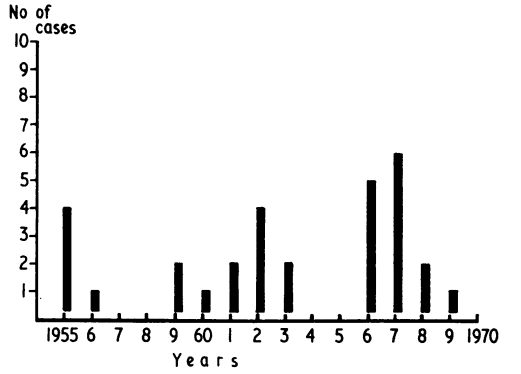


Fig 6 *Yearly incidence of* H. influenzæ *Type b meningitis*

(1949) reported acute epiglottitis in 5–10% of cases with acute laryngeal obstruction admitted under his care. In the present series there were 24 cases, 4 adults and 20 children. Of the children, 3 were seen in the London area and the remaining 17 in the Bradford area, the hospitals concerned serving a population of 850,000. The cases were seen during the years 1954–69. Over the same period 237 cases diagnosed as acute laryngotracheitis were admitted to the Bradford Childrens' Hospital, i.e. just over 7% of children admitted with laryngeal obstruction were suffering from acute epiglottitis. It is possible that the incidence of epiglottitis was a little higher, for only the most severe cases of respiratory obstruction were referred to the laryngologist.

Age incidence: The main incidence was between the ages of 1½ and 3½ years (Fig 3), an older age group than that of the main incidence of hæmophilus meningitis (Fig 4). It has been shown (Turk & May 1967) that there is an immunological explanation for the age incidence of infections due to *H. influenzæ* Type b. Blood samples taken from newborn and very young infants were nearly always bactericidal to a capsulated strain of *H. influenzæ* recently isolated from a case of *H. influenzæ* meningitis. With rare exceptions this bactericidal power was absent from the blood of children aged between 3 months and 3 years. It was more common in samples from older children and was present in all samples obtained from subjects over 10 years of age. It is assumed, therefore, that immunity to the relevant strain of *H. influenzæ* Type b was passively acquired by most babies from their mothers, was lost in the first 3 months of life and was usually restored by natural active immunization during childhood but in most cases not until the age of 3 years or even later. Why the main incidence in acute epiglottitis should be in an older age group than that for *H. influenzæ* Type b meningitis, I am unable to explain.

Yearly incidence: There was a great variety in the yearly incidence (Figs 5 and 6). In some years there were no cases, in other years they were more frequent. At times there was a tendency for a little group to occur as a minor epidemic, e.g. 3 cases in one fortnight of December 1955. Further cases in these minor epidemics came from the same area, 2 severe cases were from one practice within a short time of each other. The majority of the cases occurred in the winter months, the greatest number in this series being in December, followed by October and February, but the disease was not confined entirely to the winter months; a case was seen in May and 2 in August.

Sex incidence: Of the present series in children, 7 were females and 13 were males; if the adult cases are added, 8 females and 16 males. Other writers have noted a slight preponderance of males but attribute no particular significance to this.

Clinical Features

Initial symptoms are often mild and in only one of the cases described was there an illness of longer than twelve hours. In the typical case the patient was put to bed apparently well, only to wake up complaining of a sore throat; in one case earache was a prominent feature at the beginning of the illness. Later there is difficulty in swallowing. This is a marked feature in the few adult cases that were seen but is not a prominent feature in children. However, if the mother is closely questioned she may say that the child was given a drink and seemed unable to keep it down or swallow it. In one instance the inability to swallow and the bringing up of fluid was followed almost immediately by onset of respiratory obstruction, raising the possibility that inhalation of vomit had caused the obstruction.

Onset of the respiratory difficulty is sudden; at times it is of so dramatic a suddenness that the inhalation of a foreign body is suspected. Two of the cases described were sent initially to thoracic surgeons with this diagnosis before the true diagnosis was made and the laryngologist called in. In 2 other cases the patient had been put to bed off-colour or fretful, and 1½–2 hours later had been found very ill with extreme respiratory

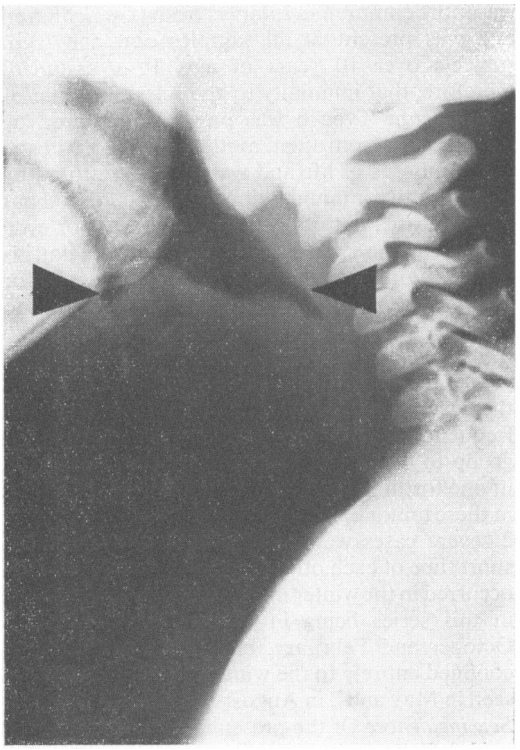


Fig 7 *Lateral X-ray of larynx in a case of acute epiglottitis, showing œdema of epiglottis and in hypopharynx. Narrowing of laryngeal air shadow*

difficulty. With such a history the possibility of acute epiglottitis should be considered. At any time the respiratory difficulty may suddenly worsen and result in asphyxia before help can be obtained. It cannot be emphasized too often or too strongly that stridor is a late feature in acute epiglottitis and warrants immediate admission to hospital for skilled treatment.

Length of history is important, especially in differential diagnosis between epiglottitis and laryngotracheobronchitis. In the former it is a matter of hours only, and in one child who came to coroner's post-mortem, the whole course from onset to death was six hours. An even more fulminating case of which the whole course was only four hours was reported by Camps (1953). Hoarseness is not a feature of acute epiglottitis but later the voice may become muffled and thick. In a classical case the patient is sitting up and leaning forward with head extended in order to get air past the swollen epiglottis, chest retraction is marked in the suprasternal and supraclavicular regions and later in the intercostal regions, and the degree of shock is out of all proportion to the duration of the illness. Absence of breath sounds indicates laryngeal obstruction. The presence of crepitant rales is good evidence that obstruction to the airway is not a major factor, and attention can then be paid to the presence of pneumonia. Movement of the alæ nasi has been noted and is marked in most of the cases with severe laryngeal obstruction. Marked restlessness, anxiety and an ashen grey pallor are features of the later stage of the disease, and early tracheotomy is needed for these patients.

The stridor is described as a low-pitched inspiratory stridor associated with a low-pitched expiratory rattle; later the inspiratory stridor becomes muffled and the expiratory component remains unchanged.

The diagnosis can be confirmed clinically by oral examination; pressure on the back of the tongue with a metal spatula will reveal the epiglottis, which in epiglottitis is greatly swollen and red. There is a difference of opinion as to the safety of this examination, as it might precipitate an increase in the respiratory difficulty and a terminal asphyxia.

The diagnosis can also be confirmed by indirect laryngoscopy and seeing the typical red swollen epiglottis. This is possible in older children but again may be a dangerous procedure unless done in hospital where all facilities for dealing with any increase in the respiratory obstruction are ready to hand. If there is still doubt, the diagnosis can be confirmed by direct laryngoscopy carried out in the operating theatre under anæsthesia by a senior anæsthetist. The diagnosis once confirmed, I would carry out an elective tracheotomy. X-ray examination can be of value but it is not necessary where the diagnosis is beyond doubt clinically, for any undue movement can precipitate asphyxia. A lateral view of the neck will show that the epiglottis and aryepiglottic folds are unusually swollen and appear to fill the hypopharynx, with the swelling abruptly stopping at the level of the vocal cords (Fig 7).

Leukocytosis is high in epiglottitis. In most cases WBC was above 16,000/mm^3, the highest being 31,000 in one of the severely ill patients.

Geographical Distribution

World distribution of reported cases is shown in Fig 8. Most papers on acute epiglottitis came from the New England states of America, from Montreal and Toronto in Canada and from Melbourne, Australia. It seems likely that the actual incidence exceeds the number of recognized cases. British literature also points in the same direction. Possibly there is a higher incidence of *H. influenzæ* in these areas but there is no evidence that the proportion of capsulated Type b is any commoner. In Britain the main reports have come from Lincolnshire, Newcastle, London and the Bradford/Leeds area. In the latter area *H. influenzæ* strains are likely to be recoverable from the nasopharynx and throat in 60–80% of a group of young children in normal health and

Fig 8 *World distribution of cases of acute epiglottitis, based on published papers. Most commonly recognized in Canada and in New England states of USA*

capsulated strains in 3–4%. In cases of upper respiratory illness there is an 82% carriage rate. This is above the ranges found by workers in other centres. It is possible that increased incidence of *H. influenzæ* is the cause of the apparent increased incidence of acute epiglottitis in the Bradford/Leeds area as compared with the rest of Britain. It is possible also that the number of cases is due to increased recognition in this area. *H. influenzæ* is found with strikingly low frequency in other parts of the world, especially Jamaica. Racial factors also might influence carriage rate.

Treatment

The first criterion of treatment is to establish and maintain a safe airway. Tracheotomy is the treatment *par excellence* in acute epiglottitis; it is below the site of obstruction and gives the patient an adequate airway away from the area of infection until the body defences plus antibiotics can overcome the disease. Being away from the site of infection through normal tissue, the tracheotomy causes very little trouble in post-operative care. It is true that the period of risk as regards danger of acute asphyxia is very short, not more than 2–3 days, and many papers have stressed that tracheotomy can be avoided, especially since hydrocortisone has been given to these patients. I feel, however, that tracheotomy is safer, particularly in a provincial hospital. Cases referred to me have already been seen by the pædiatrician, and only the more severe cases or those that are worsening are referred to the laryngologist. While many patients will survive with medical treatment only, there is a great risk of sudden complete airway obstruction and a policy of elective tracheotomy is preferable. In my opinion it is better to have a live child with a small scar in the neck than that even one of these cases admitted to hospital should die. This policy is stressed by Baxter (1967) and I fully agree. In the series described there were 24 cases: 4 adults with one death, and 20 children with 4 deaths. Two of these deaths were the first 2 cases seen; among children who had tracheotomy there was only one death and this case was complicated by the fact that the patient was suffering from phenylketonuria. Another died 45 minutes after admission to hospital, the diagnosis being confirmed by coroner's post-mortem. One further case of acute epiglottitis died before admission to hospital, diagnosis again being confirmed by coroner's post-mortem. In Leeds, 5 cases of acute epiglottitis have been seen by my colleagues, with one death; this was a child aged 2½ years admitted to hospital but not considered to need an immediate tracheotomy, who suddenly collapsed and died from asphyxia 2 hours later. A policy of earlier tracheotomy would certainly have prevented death in at least three of these fatal cases.

Decannulation of the tracheotomy tube took place 5–7 days later; in older children the epiglottis could be visualized by indirect laryngoscopy and settled down to normal in a few days.

Intubation: The second case seen was treated by intubation; despite dedicated care throughout, the tube got blocked and the patient died. The tube can cause laryngeal ulceration or later laryngeal stenosis. In my opinion, if I had done a tracheotomy, this patient would not have died. Nine of the patients who had tracheotomy were followed up, some of them years later; all were healthy teenagers and had suffered no disability from their elective tracheotomy.

Anæsthesia: I am indebted to Dr W E Arnold and to Dr G L Evans for carrying out anæsthesia necessary for tracheotomy. Both stressed that it was possible to lift up the epiglottis with the beak of the laryngoscope after induction with gas, oxygen and halothane. A small endotracheal tube was inserted which made the operation easy and elective. Operation under local anæsthetic is not advised. The restlessness of the child and the engorgement of the veins, plus the supraclavicular movements of the cervical pleura, made the operation considerably more difficult.

Medical management consisted in good nursing, adequate fluids, high humidity in the atmosphere and oxygen if needed. *H. influenzæ* is only moderately sensitive to antibacterial drugs and the infections it causes are in sites only moderately accessible to the drugs. In the cases where the organism was isolated, it was found to be sensitive to tetracycline and ampicillin. The development of resistance to antibiotics during treatment is rare in hæmophilus infections. Hydrocortisone 100 mg six-hourly was given in the early stages of the disease to cases occurring latterly, and the antibiotics were given intravenously.

Role of the Family Doctor

The most important task of the family doctor is to be aware of the possibility of acute epiglottitis.

Its early recognition and early admission of the patient depend largely on him. Faced with a case of respiratory distress he has to ask himself whether it is due to so-called croup or to an acute infection causing obstruction of the laryngo-tracheal airway. If the latter, he has to consider its etiology and, most importantly, whether it is safe to keep the patient at home. In spasmodic croup the child wakes up with sudden loud stridor and retraction of the chest wall. The condition looks alarming but quickly settles after sedatives. Next day there may be the same sequence. It is due to spasm of the vocal cords. In acute laryngotracheitis the patient shows signs of progressive respiratory obstruction. The initial symptoms and sudden onset of stridor in acute epiglottitis have been described above. The decision whether to keep the patient at home depends on the severity of the obstruction, its duration and whether the child's condition is worsening. If there is any doubt, it is better to err on the side of safety and send the child to an adequately equipped hospital. In 2 of the cases in my series, the family doctors took one look at the patients and brought them to hospital immediately. This played as great a part in preventing death as did the treatment carried out. The main cause of death in the cases which came to coroner's post-mortem was the fulminating character of the disease and the shortness of the history. Only by awareness of the nature of the disease and by its early recognition can death be prevented in these cases.

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Stridor in a Pædiatric Department

This is an analysis of 250 cases of stridor admitted to pædiatric medical wards in the City of Bradford from 1957 to 1969. These cases constituted 1·34% of 18,680 medical pædiatric admissions.

Table 1 shows the relative frequency of different types of stridor requiring admission, but does not necessarily represent total incidence in the area. The absence of stridor due to tetany, micrognathia and retropharyngeal abscess in such a relatively large series is surprising, but despite the relative frequency of rickets and neonatal tetany in Bradford during the period under review, and the occurrence of 2 cases of severe Pierre Robin syndrome, none of these presented with stridor. There were 9 cases of inhaled foreign body, but only one was associated with stridor. Although the male/female ratio in this series is higher than that described by Apley (1953), the two series are not strictly comparable.

Table 1
Classification of 250 cases of stridor

Etiology	*No. of cases*	*%*	*Male*	*Female*	*M:F ratio*
Congenital	39	15·6	25	14	1·8:1
Laryngotracheitis	124	50·0	93	31	3:1
Laryngotracheo-bronchitis	61	24·4	48	13	3·7:1
Acute epiglottitis	10	4·0	7	3	2·3:1
Diphtheria	1	0·4	1	0	
Spasmodic laryngitis	6	2·4	5	1	5:1
Pemphigus	1	0·4	1	0	
Vascular ring	2	0·8	2	0	
Cervical neuroblastoma	1	0·4	0	1	
Foreign body	1	0·4	1	0	
Hydrocephalus	2	0·8	1	1	
Associated with neurogenic swallowing dysfunction	2	0·8	2	0	
Total	250		186	64	2·9:1

The largest group in this series consists of cases of infective origin. Excluding cases of diphtheria and pemphigus, there were 201 such patients, of whom 124 were clinically classifiable as acute laryngotracheitis, 61 as laryngotracheobronchitis, 10 as epiglottitis and 6 as spasmodic laryngitis, by which I mean those cases where the patient wakened on 3 to 12 occasions with a stridor which ceased in six hours or less. Only one such case was pyrexial, and while there was a history of mild upper respiratory catarrh in all cases, the children were thought by their parents to be more nervous than average. It is reasonable on clinical grounds to differentiate between laryngotracheitis, laryngo-tracheobronchitis and spasmodic laryngitis, but one cannot say whether or not a child with severe laryngotracheitis is in fact suffering from epiglot-titis without obtaining a good view of the epiglottis; this is not easy in all cases, as to attempt this examination in an already ill child may sometimes markedly increase the degree of obstruction and necessitate immediate tracheo-tomy. One should therefore refrain from too thorough an examination unless prepared for the consequences. It follows that the total incidence of epiglottitis of all degrees of severity in this series cannot be known with certainty, but whenever this diagnosis has been made, the epiglottis has been visualized during anæsthesia for tracheotomy and, in one mild case, at laryngoscopy.

The annual incidence of inflammatory stridor varied considerably, but the majority of cases