CROUP AND ITS MANAGEMENT

BY

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A hoarse voice, obstructed stridulous breathing, and a characteristic harsh vibrant cough constitute "croup" which is due to laryngo-tracheo-bronchitis. The term is also used loosely to encompass the less common condition "supraglottic oedema" in which airway obstruction occurs not at the vocal cords but at the level of the epiglottis and aryepiglottic folds. Both conditions occur characteristically in infants and young children, and may cause complete obstruction of the airway.

The decline of diphtheria in immunized communities and the recent identification of formerly anonymous viral agents causing croup have changed the perspective of the condition, whose dangers are still emphasized by reports from pathologists to coroners' courts (Bowden and French, 1951 ; Jones and Camps, 1957).

The difficulty of predicting the course of the disease in an infant is well known to medical practitioners, but probably not to lay people, who underestimate the danger of croup. General practitioners are probably best placed to educate the community in this respect.

This review of the subject is based on experience at Fairfield Infectious Diseases Hospital, Melbourne, where diphtheria has been an uncommon cause of croup in recent years by contrast with the various respiratory viruses.

Laryngo-tracheo-bronchitis

Early symptoms of fever and hoarseness suggest that the larynx alone is involved, but this is by no means the only functional change dictating the management of laryngitis, which is almost invariably accompanied by inflammatory changes in the trachea and bronchi.

The severe effects of the disease are usually mechanical, producing airway obstruction. This is most commonly seen in infants and young children, whose respiratory channels are narrow, but also occasionally in adults.

Disproportionate laryngeal spasm, a notorious sequel to irritation in the vicinity of the larynx in children, aggravates the laryngeal narrowing caused by the inflammation.

The croupy cough, the evidence of fluctuating airway obstruction, and the rapidity with which complete occlusion may develop, are explained by the associated inflammatory changes in the trachea and bronchi. These lead to the accumulation in the trachea of voluminous quantities of tenacious mucus, whose evacuation is made difficult by oedema of the larynx. Transient aggravation occurs while mucus is passing through the narrowed aperture. Complete obstruction is ultimately precipitated by the impaction of tracheal mucus in the larynx.

A situation causing progressive increase in dyspnoea is established in advanced cases in which laryngeal narrowing hinders the expectoration of sputum that accumulates in the trachea, thus further reducing the airway so that the obstruction is no longer localized to the larynx. Pneumonia complicates the condition on occasions, also pneumothorax, which is rare.

Causal Agents

Infection is the usual cause of laryngo-tracheo-bronchitis, but on rare occasions anaphylactoid oedema of the larynx and spasm of bronchi in allergic individuals may simulate the condition. Infections causing the condition form three main groups: laryngeal diphtheria (bacterial toxin), viral infection, and bacterial infection.

The possibility of an inhaled foreign body should be considered if the onset is abrupt and the signs are atypical.

Laryngeal Diphtheria

In an unimmunized community laryngeal diphtheria is a common and most dangerous form of croup. Immunization in Australia has reduced this disease to a minor numerical status, but its potential remains, and since successful treatment requires the immediate administration of diphtheria antitoxin, this disease remains the prime consideration in a diagnostic classification (see below).

The term "diphtheritic laryngitis" is incomplete in that the membrane rapidly spreads to line the trachea and bronchi, even the small terminal bronchi.

Respiratory Virus Infections

The respiratory viruses attain major importance as a cause of laryngo-tracheo-bronchitis by virtue of their great prevalence and also their capacity to predispose to bacterial infection of the airways and lungs. A "virus group" has long been recognized by the observation of laryngo-tracheo-bronchitis due to the enanthema of measles, but until recent years the group was ill-defined. It is now apparent that the list of viruses associated with croup includes all the known respiratory viruses, the condition being one manifestation of respiratory virus infection, particularly in young children.

From November 1, 1959, to May 31, 1960, respiratory viruses were isolated from 50% of 159 children admitted to the Fairfield Hospital with croup. More than half of the remaining cases appeared to be viral in origin also, and those due to or aggravated by bacterial infection form about 10% of the group. The proportions, of course, vary with the prevalence of the various respiratory pathogens.

A concentration of severe and complicated cases of influenza at Fairfield Hospital during the pandemic of 1957 demonstrated the capacity of influenza virus to cause laryngo-tracheo-bronchitis (Forbes, 1958). About this time newly developed techniques for virus isolation revealed prevalent, formerly anonymous agents which have been named the croup-associated (C.A.) virus (Chanock, 1956 ; Beale *et al.*, 1958) and the haemadsorption (H.A.) virus types 1 and 2 (Channock *et al.*, 1958). These have been reclassified as follows:

Myxovirus parainfluenzae	type	1=H.A. virus type 2 and Sendai virus
„	„	„ 2=C.A. virus
„	„	„ 3=H.A. virus type 1 (Cook *et al.*, 1959)

During the 12 months preceding March, 1959, parainfluenza viruses type 1 and type 3 were grown from epidemic numbers of children admitted to Fairfield Hospital with laryngo-tracheo-bronchitis (Ferris, 1960 ; Forbes, 1960). More recently, parainfluenza virus type 2 (C.A. virus) has been isolated from similar cases (Fairfield Hospital, unpublished).

Influenza virus type B and adenovirus types 1, 2, 3, 5, and 7 have been associated with laryngo-tracheo-bronchitis; also certain of the enteroviruses appear to be causally related to the condition at times. Coxsackie virus type A9 has been isolated repeatedly from infants with laryngo-tracheo-bronchitis, and to a lesser extent E.C.H.O. virus types 14 and 10 (Fairfield Hospital, unpublished).

Thus the current list of virus agents causing laryngo-tracheo-bronchitis is as follows: influenza viruses (types A and B); parainfluenza viruses (types 1, 2, and 3); adenoviruses (types 1, 2, 3, 5, and 7) (incomplete); measles virus; E.C.H.O. virus types 14 and 10 and Coxsackie virus type A9 (tentative).

Bacterial Infections

Haemolytic streptococci group A and staphylococci are the bacteria most commonly associated with laryngo-tracheo-bronchitis. Infection with these organisms is characterized by the appearance of voluminous quantities of purulent tracheal mucus. The tracheal mucus produced by staphylococci infection is viscous and tenacious, so that its evacuation is extremely difficult.

The respiratory viruses described above probably predispose to most of these infections.

Pneumococcal infection is often superimposed upon respiratory virus infections, but pneumococci are a more frequent cause of pneumonia in such cases of severe airway obstruction, although impairment of the airway is at times an association of this dual infection. The danger of superimposed bacterial infection is greatest in infants and children who are carrying one of these organisms at the time respiratory virus infection occurs, in which case the history of illness may be short and severe.

Anaphylactoid Laryngeal Oedema

The condition of laryngeal oedema and bronchospasm is uncommon but may simulate infective croup. It is usually precipitated by the administration of an allergen such as horse serum to a sensitized person, but may also occur with the inhalation of a natural allergen or with infection.

A known allergic child or adult should be observed for at least one hour after the injection of a sensitizing antigen.

Supraglottic Oedema

Supraglottic oedema (syn. epiglottitis) is a generalized disease in which gross oedema of the epiglottis and aryepiglottic folds is associated with septicaemia (Sinclair, 1941; Alexander *et al.*, 1942; McLorinan and Ferris, 1956).* *Haemophilus influenzae* type B is the causal organism in the vast majority of cases. *Streptococcus haemolyticus* group A is associated with a small proportion.

The disease is exceedingly acute, the history of respiratory difficulty being invariably only a few hours. The associated septicaemia ensures early respiratory fatigue, and death may occur within eight hours of the development of laboured respiration.

Prior to the use of appropriate antibiotics, the development of *H. influenzae* meningitis was not an uncommon sequel in surviving patients. Jones and Camps (1957) have shown that inflammatory lesions in the vicinity of the epiglottis may be found in children dying of meningitis due to *H. influenzae* type B.

This was one of the conditions which formerly invoked the coroner's verdict, "smothered by bedclothes," in infants who were found dead in their cots in the morning, having gone to bed the previous evening with minimal or no symptoms (Bowden and French, 1951).

*During a five-year period ending in 1955, 33 cases of supraglottic oedema were admitted to Fairfield Hospital. Blood was cultured from 19 cases, nine of which yielded *H. influenza* type B. This organism was cultured from swabs of the throat or trachea of 24 cases. Tracheotomy was required in most cases. There were no deaths.

Symptomatology

Laryngo-tracheo-bronchitis and supraglottic oedema differ in certain symptoms and signs, so that supraglottic oedema may be identified clinically. The evidence of airway obstruction is common to both lesions.

Laryngo-tracheo-bronchitis

The development of "laryngitis" is usually preceded by a history of an infection of the upper respiratory tract often described by the relatives of the infant as a "cold."

The symptoms and signs of developing respiratory obstruction are summarized in the following list, in which the signs of increasing obstruction are given in order from above. These signs are cumulative and are associated with an increasing respiratory rate and increasing dyspnoea.

Stage 1	Fever Hoarseness of voice, often progressing to aphonia Croupy cough Inspiratory stridor when disturbed
Stage 2	Continuous respiratory stridor Lower-rib retraction Retraction of soft tissues of the neck Accessory muscles of respiration come into operation Respiration becomes laboured
Stage 3	Signs of anoxia and carbon dioxide retention Restlessness Anxiety Pallor Sweating Rapid respiration
Stage 4	Intermittent cyanosis Permanent cyanosis Cessation of breathing

In the development of obstruction the symptoms may fluctuate owing to transient plugging of the larynx with mucus from the trachea. When laryngeal obstruction has been prolonged, asphyxia due to fatigue is a great danger. The onset of laboured breathing is usually slow by contrast with the rapidity with which complete obstruction may develop in the later stages.

Provisional Diagnosis.—The patient is often seen early in this train of signs and symptoms. The suspicion of diphtheria has an important bearing on the management of a patient in that diphtheria antitoxin is administered on clinical grounds pending subsequent isolation of the organism by culture of throat or tracheal swabs.

If, on examination, faucial membrane is found in association with the laryngitis, diphtheria is an arbitrary diagnosis. On the other hand, if the patient appears toxic and the condition has been developing over a period of 36 to 48 hours, this diagnosis must be considered, even in the absence of faucial membrane, as the primary focus may occur in the larynx.

In many of the virus infections the throat may be palely reddened and the pharynx have a granular appearance due to patchy lymphoid hyperplasia. On the other hand, florid tonsillitis may be present in some cases of bacterial laryngo-tracheo-bronchitis. However, the

interrelation of these causes makes the use of antibiotics mandatory, so that, unlike diphtheria, early clinical distinction is not vital to management (see below). In the virus infections, the blood leucocyte count is often normal or low, with a predominance of lymphocytes.

Supraglottic Oedema

Supraglottic oedema differs from laryngo-tracheo-bronchitis in its onset (McLorinan, 1946). Since the larynx is not primarily involved in this condition, hoarseness and aphonia are not primary symptoms. The noisy obstructed breathing of supraglottic oedema does not develop as inspiratory stridor as it does in laryngo-tracheo-bronchitis, but as an expiratory snore due to vibration of the flabby swollen tissue of the epiglottis and aryepiglottic folds. The children are pale and toxic-looking owing to the associated septicaemia.

On examination, if the back of the tongue is depressed the epiglottis may often be seen as an oedematous red globular structure. If the epiglottis cannot be seen, the diagnosis may be made by "gagging" the child and passing a finger over the base of the tongue where the epiglottis is felt to be rounded or "marble-like."

In supraglottic oedema the progression of obstruction is rapid, inexorable, and aggravated by the associated septicaemia so that tracheotomy is almost inevitable.

Management

The basis of successful management lies in the early diagnosis of laryngeal diphtheria and supraglottic oedema, both of which should be considered in every case, in repeated observation and in the ability to recognize the degree of airway obstruction in its various stages

For the majority of patients with croup medical attention is sought in the earlier stages of developing obstruction. There are no criteria by which the future progress of the condition may be predicted except when diphtheria and supraglottic oedema, which almost inevitably cause severe obstruction, are present. This uncertainty makes constant observation of the patient with croup essential.

Patients with laryngitis and a croupy cough commonly fail to develop laboured breathing and do not progress beyond stage I of the symptomatology (see above). Patients at this stage may be observed at home, but parents should be made aware of the possibility of rapid development of respiratory obstruction so that they will review the child's condition from time to time, particularly at night.

The home treatment of the condition takes a suitably modified form of that described for the hospital management of more severe cases.

Patients in whom the symptoms of obstruction (for whatever cause) have progressed to stage 2, during which breathing becomes increasingly laboured, require observation in hospital.

Management in Hospital

At Fairfield Hospital these patients are nursed in a steam-tent (which has been found superior to various atomized wetting agents) to reduce evaporation of fluids from the respiratory tract, so minimizing the inspissation of the tracheal secretions, which are difficult to evacuate through a contracted larynx. The warm moist atmosphere lessens the patient's discomfort and ameliorates laryngeal spasm. These patients require constant nursing attention and reassurance to reduce their apprehension apart from the necessity for continuous observation. Ideally, they should be nursed and observed by staff with specialized experience in this field.

Dangerous Drugs.—The employment of oxygen, sedation, or atropine is perilous in this condition. The administration of oxygen, particularly under pressure, will disguise the symptoms even in the later stages of obstruction, so that the airway may suddenly be completely occluded by tracheal mucus without adequate prior warning. The use of oxygen is permissible only when a decision to perform a tracheotomy has been made or during the transport of a patient with severe respiratory obstruction. Sedation in all its forms carries the risk of respiratory depression and exaggerates the effects of fatigue in a patient in whom respiratory obstruction has been prolonged. Atropine increases the viscosity of tracheal mucus and so aggravates the difficulties of its evacuation.

Investigations.—Initially, the nose and throat are swabbed for bacterial culture and the throat also for viral investigation if facilities are available. When indicated by the presence of supraglottic oedema, blood is taken for culture if this can be done without causing distress. Later in management a blood leucocyte count is often helpful in distinguishing laryngo-tracheo-bronchitis of viral and bacterial origins, and *x*-ray examination of the chest is advisable to exclude associated pneumonia or pulmonary collapse.

Specific Treatment

Specific treatment with diphtheria antitoxin and antibiotics should be started immediately after samples have been taken for investigation on the basis of the provisional clinical diagnosis.

Diphtheria Antitoxin.—If diphtheria is suspected a dose of 30,000 units will be required to treat the laryngeal focus. This dose is supplemented according to the severity of a faucial lesion.

Antibiotics.—The majority of cases of laryngo-tracheo-bronchitis in this community at present are caused initially by a respiratory virus. These agents commonly predispose to secondary bacterial infection. The clinical distinction of the two situations is uncertain and bacterial aggravation of a primary virus infection may be so rapid in these more severe cases that it is advisable to assume that the combined infection is already established. Pneumococci and group A streptococci are the most frequent secondary invaders, but, when established, staphylococci may be particularly dangerous. Penicillin is the drug of choice for the first two pathogens and forms the basis of antibiotic treatment. In the less severe cases it may be used alone. Tetracycline is used in conjunction with penicillin to combat possible staphylococci and *H. influenzae* infections in cases of moderate severity. In this group of patients, who are admitted direct from their homes, the majority of staphylococci are sensitive to the tetracyclines, as are the haemophils. On the other hand, if the disease is severe or if the diagnosis of supraglottic oedema is made, so that the patient's life will be endangered by the delay in providing effective antibiotic treatment, intramuscular chloramphenicol, which has a broader and more certain cover, is used in conjunction with penicillin. Penicillin and erythromycin are used as adjuncts to diphtheria antitoxin to clear the foci of diphtheria organisms.

Indications for Tracheotomy

The plight of these patients may form a clinical emergency at the time of their admission to hospital, and if cyanosis is present (stage 4, see above) immediate tracheotomy is life-saving.

If, on admission, the symptoms have progressed to stage 3, with the accumulated signs of airway obstruction in conjunction with evidence of anoxia and carbon dioxide retention, tracheotomy is at times necessary, but two courses of management are open according to the duration of symptoms and the diagnosis. In those cases in which the development of the disease has been slow and restlessness appears to have been recently aggravated by laryngeal spasm, a trial of conservative treatment in a steam-tent may be successful. On the other hand, if progression of symptoms has been rapid and inexorable, or if rather severe obstruction has been prolonged so that fatigue is beginning to ensue, tracheotomy will be necessary.

The development and persistence of restlessness, anxiety, pallor, and sweating in association with an increasing respiratory rate in spite of appropriate early treatment, indicate the necessity for tracheotomy. The appearance of cyanosis, even if transient, calls urgently for tracheotomy. If supraglottic oedema is the cause, a tracheotomy is likely to be required.

Tracheotomy

A "high" tracheotomy at the site of the second and third tracheal rings is most suitable in these cases. The operation is performed under local anaesthesia, but if the patient is comatose anaesthesia is obviously unnecessary. Attempts to provide general anaesthesia are dangerous, and bronchoscopy is unnecessary, time-consuming, and traumatizing in severe cases. Attempts to pass a bronchoscope in supraglottic oedema can be lethal.

Management of Tracheotomy

The relief produced by tracheotomy in these cases is usually dramatic, but the effect is temporary if the patient is subsequently mismanaged. Unattended, the tracheotomy tube will rapidly become obstructed with mucus or membrane.

Surgical emphysema is a sequel to tube obstruction which forces the expired air to seek egress around the tube.

Considerations.—The tracheotomy provides not only an alternative airway but an avenue for the removal of secretions by suction from the trachea and bronchi. In using a rubber urinary catheter to aspirate the trachea it should be remembered that the catheter itself constitutes an obstruction to the airway while it is in the trachotomy tube. Improperly used, it may cause surgical emphysema. If the tube is obstructed the pulmonary air will be forced out of the trachea around the tube and into the cervical tissues by the inevitable coughing. The nasopharynx, which normally humidifies inspired air, is short-circuited by tracheotomy, and other means of performing this function must be found to prevent the drying of the respiratory mucosa and consequent inspissation of secretions.

Nursing Procedures.—The patient is nursed in a steam-tent as before to humidify inspired air. The inner tube is introduced into the tracheotomy tube to facilitate cleaning, which is carried out repeatedly. A saturated solution of sodium bicarbonate is introduced through the tube into the trachea to liquefy mucous secretions (the alkalinity favours this), to aid aspiration, and to humidify the respiratory mucosa. After this procedure the trachea is aspirated with a urinary catheter of suitable size.

Removal of the Tube

The tube may be removed when the volume of tracheal mucus has become negligible (the presence of the tube itself stimulates the production of tracheal mucus) and the laryngeal airway is competent. This is usually possible between three and five days. It is important that the tube be removed by the fifth day, as the risk of a "retained" tube is increased when it is allowed to remain for longer periods.

Removal of the tube must be regarded as a trial, and the child must be observed closely to detect persistent laryngeal obstruction which may require the reintroduction of another, perhaps smaller tube. It is a mistake to deliberately obstruct the tracheotomy tube for trial for long periods, as this is merely impairing the airway.

If difficulty is experienced (not common) in removing the tube it is preferable to use a series of tubes of diminishing size. In our experience, difficulty in removing the tube occurs when there is an associated pulmonary or laryngeal lesion.

The wound usually closes and heals quite rapidly after the tube is removed. Secondary suture does not assist healing or minimize scarring. A simple gauze dressing, perhaps with a topical antibiotic such as bacitracin, is sufficient.

Summary

This paper gives an account of the common causes of croup, some of which have been identified in recent years.

Laryngo-tracheo-bronchitis, which originates in three main categories—laryngeal diphtheria, and viral and bacterial infection—is distinguished from supraglottic oedema (syn. epiglottitis), which is usually associated with septicaemia and is caused by *H. influenzae* type B.

The symptomatology is described in conjunction with the management practised at Fairfield Infectious Diseases Hospital, Melbourne.

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