ACUTE LARYNGOTRACHEOBRONCHITIS

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ACUTE laryngotracheobronchitis is a common disease which should be more widely understood. Invariably, it is associated with respiratory obstruction and frequently a tracheotomy under general anaesthesia is required. This paper deals with the disease from the viewpoint of the anaesthetist.

Acute laryngotracheobronchitis is an acute infective inflammation of the mucosa of the larynx, trachea, and bronchi. It is characterized by oedema of the larynx, toxæmia and a thick, viscid, non-membranous exudate in the tracheobronchial tree (1).

This disease affects children almost exclusively. The age incidence (2) ranges from three months to eight years, with 60 per cent of all cases occurring between one and three years of age.

The responsible organisms can be streptococci, either haemolytic or viridans, pneumococci, staphylococci, micrococci catarrhalis or haemophilus influenzae. Predisposing factors include a low humidity usually associated with central heating, sudden environmental temperature changes, and possibly allergy.

PATHOLOGY

When the pathological features of this disease are perceived, its seriousness becomes obvious. Under normal conditions, the mucosa of the larynx and tracheobronchial tree is separated from the cartilage by an abundance of loose areolar tissue containing rich vascular and lymphatic plexuses. In addition, the glottic aperture of children is relatively small.

With the onset of infection in the upper respiratory tract, an intense inflammation of the mucosa and submucosa occurs. The associated swelling is not the usual "watery" oedema but consists of an acute inflammatory cell infiltration. A reduction of the lumen ensues throughout the respiratory tree but the earliest and most marked obstruction is in the larynx. The swollen inflamed larynx causes hypoxia and laryngospasm, which increases the vascular engorgement and oedema and a "vicious circle" results. Retained sticky secretions due to a diminished cough reflex augment the obstruction.

Within a few hours, laryngeal oedema can progress to complete closure of the glottis. Almost all deaths in this disease are the result of anoxia, either from acute asphyxia or from the effects of prolonged hypoxia.

CLINICAL PICTURE

The clinical picture varies with the severity of the infection and the degree of respiratory obstruction. Two forms of the disease are seen, the mild and the

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severe, but they differ only in degree. In a severe infection with marked obstruction, the following disturbances may be manifested.

1. *The central nervous system.* Cerebral signs are the most sensitive indicators of lack of oxygen. The child shows inattention, restlessness, and delirium which is the result of depression of the cerebral cortex by hypoxia and toxæmia. Occasionally convulsions occur which are associated with the high fever.

2. *The cardiovascular system.* The pulse rate is rapid owing to apprehension and fever combined with the reflex response to lack of oxygen and retention of carbon dioxide. Terminally, the pulse may become extremely fast and weak or else become very slow owing to direct anoxic depression of the cardiac muscle. The blood pressure may be normal or slightly elevated.

3. *The respiratory system.* Dyspnoea is the most prominent feature and may be inspiratory, expiratory, or both. The accessory muscles are working maximally and there is marked indrawing of the supraclavicular, suprasternal, and intercostal areas, and retraction of the lower sternum. Many children assume a "knee chest" position in an attempt to gain relief.

The respirations are shallow owing to the difficulty in inspiration and expiration. The rate is rapid owing to the stimulation of oxygen lack and excess carbon dioxide. Although a normal pulmonary minute volume may be maintained, the actual alveolar ventilation is quite inadequate.

4. *Integumentary system.* The skin is usually pale and occasionally ashen. Cyanosis of the finger nails and lips is common.

The combined effect of prolonged hypoxia, toxæmia, restlessness, and laboured respiration is extreme fatigue. The child may become too exhausted to maintain the fight for air and quickly sleep into death. Therefore, in the severe form of acute laryngotracheobronchitis, tracheotomy must be performed immediately.

MEDICAL TREATMENT

Mild cases can be handled medically but since the disease can progress rapidly, constant competent observation is necessary during the acute phase. Mild cases are the most common and the following treatment is adequate.

1. A humid atmosphere is necessary. This can be achieved by placing the child in an oxygen tent fitted with a humidifier. The increased oxygen concentration helps to relieve the hypoxia and the high percentage of water vapour helps to prevent inspissation and retention of secretions.

2. Antibiotics must be administered promptly. Penicillin and streptomycin in the usual dosage are satisfactory.

3. Mild sedation is desirable. A short-acting barbiturate administered per rectum is valuable to allay apprehension and allow quieter and more efficient respirations.

Heavier sedation using large doses of barbiturate or opiates will mask the signs of progression of the disease. The central nervous system will be depressed, the respiratory effort weakened, the cough reflex depressed, and secretions will be retained. This may lead to bronchial obstruction associated with minimal signs of respiratory embarrassment, and quiet death may ensue. Atropine is contraindicated even if tracheotomy is contemplated as serous secretions are abolished.

SURGICAL MANAGEMENT

The evidence of progression of a mild case is shown by increasing restlessness, more laboured respirations, an increasing but weakening pulse which fails to respond to therapy, and diminishing air entry in the lung bases. Any one of these signs is an indication for a tracheotomy as soon as possible. Where reasonable doubt exists regarding the degree of respiratory obstruction, a tracheotomy should be performed.

In preparation for the operation, the child is placed on the operating room table and oxygen given during the draping. No anaesthetic should be administered at this stage, as even small amounts will produce either apnoea or laryngospasm. If an acute anoxic episode is added to a prolonged hypoxic period, death occurs promptly unless an airway is forced and artificial ventilation is carried out. It would be extremely difficult to pry open the mouth and force an endotracheal tube or even a Mosher tube through a tightly swollen larynx before the child was dead.

The method of choice is to open the child's mouth, insert a mouth gag, and pass a bronchoscope without any anaesthetic. A bronchoscope is most valuable because (*a*) it can be forced through the narrowed oedematous larynx, whereas an endotracheal tube would kink even with a stylet; (*b*) it establishes an airway which cannot be compressed, (*c*) it allows visualization of the trachea and bronchi and direct aspiration of secretions; (*d*) it allows the insufflation of oxygen. The lack of anaesthesia for insertion of the bronchoscope may seem to present difficulties but resistance is minimal, and on relief of the obstruction, the child immediately relaxes. A light ether oxygen anaesthesia is administered through the side arm of the bronchoscope. Only small quantities of the agent are necessary as the child is usually exhausted.

A tracheotomy (3) which is deliberate and unhurried can be carried out. A vertical midline incision in the lower neck is made, and using blunt dissection the trachea is exposed. The presence of a bronchoscope provides a rigid landmark and prevents injury to the oesophagus when the third and fourth tracheal rings are incised. The tracheotomy tube is gently inserted and the skin edges are loosely sutured.

Postoperatively the air entry in the lungs must be checked and then the child is returned to the humid atmosphere of his oxygen tent. Frequent aspiration of his respiratory tract through the tracheotomy tube should be carried out to prevent accumulation of secretions and bronchial obstruction. The tracheotomy tube is removed in four to seven days.

COMPLICATIONS

Serious complications are rare. If death occurs, however, it is usually the result of asphyxia. Any of the complications of an acute pyogenic infection can occur, such as endocarditis, meningitis, or septicaemia.

Pulmonary complications are commoner and may occur early or late. The early respiratory complications include pneumo-thorax, due to alveolar rupture from the high negative intrathoracic pressure consequent on excessive respiratory efforts. Occasionally, following relief of the obstruction, a temporary apnoea occurs. The

stimulation of respiration by oxygen lack and carbon dioxide excess is reduced and an interval is required for accommodation of the respiratory mechanisms to the new gaseous tensions of oxygen and carbon dioxide. Interstitial emphysema is common, usually the result of tight closure of the tracheotomy incision so that coughing forces air into the tissues.

Late pulmonary complications are usually the result of inadequate bronchial aspiration and include pneumonia, chronic bronchitis, lung abscess, and bronchiectasis.

SUMMARY

The disease, acute laryngotracheobronchitis, is discussed from the viewpoint of the anaesthetist. The age incidence and aetiology are mentioned and the pathological changes leading to respiratory obstruction with its consequences are outlined.

The main clinical features of a fulminating case are described.

The treatment of the mild case is outlined, it should provide a humid atmosphere, antibiotic therapy, and mild sedation.

A severe case is treated, in addition, by early tracheotomy. The preoperative and surgical management is presented. The postoperative complications are summarized.

RÉSUMÉ

L'auteur discute du point de vue anesthésique, la maladie appelée laryngotrachéobronchite aiguë. Il fait mention de l'étiologie et de l'incidence par rapport à l'âge de même qu'il décrit les changements pathologiques qui conduisent à l'obstruction respiratoire avec ses conséquences.

Les signes cliniques principaux d'un cas suraigu sont décrits.

Le traitement des cas bénins est donné. Il nécessite une atmosphère humide, les antibiotiques et l'emploi de légers sédatifs.

Un cas sévère nécessite en plus, au début, une trachéotomie. Les soins préopératoires et chirurgicaux sont présentés. Les complications postopératoires sont aussi données.

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