ACUTE EPIGLOTTITIS IN CHILDREN
A Respiratory Emergency

BY
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SUMMARY
Reports of thirteen cases of acute epiglottitis are presented. Improvement in the mortality rate with adequate treatment is stressed. The diagnosis and management is briefly discussed. Particular regard must be given to the maintenance of the airway, the control of infection and use of steroids. In view of the rapid progression of hypoxia, and collapse, and the high mortality, acute epiglottitis presents a respiratory emergency requiring urgent intensive treatment.

“Croup” is the name given to a respiratory disease characterized by difficulty in breathing and a characteristic crowing sound. Earlier this century the main cause was probably diphtheria. Later, non-diphtheritic infections were recognized and labelled as “acute laryngotraceobronchitis”. Recently another entity has been increasingly recognized in which acute inflammation and oedema are localized to the supraglottic regions. This is called “acute supraglottitis” or “acute epiglottitis”.

History.
Although Lemierre, Meyer and Loplane (1936) were able to isolate Haemophilus influenzae as a cause of the infection leading to oedema and acute laryngeal obstruction, acute epiglottitis was first described by Sinclair in 1941. Subsequent reports by Du Bois and Aldrich (1943), Berenbeng and Kevi (1958), and Poole and Altman (1963) established it as a clinical entity. The incidence of the disease, in the British Isles would appear to be more common than formerly supposed (Andrew, Tandon and Turk, 1968).

Clinical features.
Characteristically, acute epiglottitis is a disease of previously healthy children of pre-school age. Rapidly progressing respiratory obstruction is its principal manifestation although Sinclair (1941) stressed the acute toxic nature of the illness with “prostration” or “shock” out of all proportion to the size of the local lesion. The mortality of untreated cases is very high but the outlook can be greatly improved by adequate management (see table I). Early diagnosis and treatment are therefore of paramount importance.

Typically the lesion is confined to the epiglottis, the aryepiglottic folds and the arytenoids. The tonsils, the pharynx and the true cords and trachea are not involved. If the adjacent respiratory tract is inflamed, e.g. tonsillitis, pharyngitis or laryngotraceobronchitis, then inflammation of the epiglottis is secondary and is much slower in onset.

Presentation.
There may be a short history of dysphagia or hoarseness. This is followed by an abrupt onset of dyspnoea and stridor, which steadily worsen. The child often sits upright with the mouth gaping to obtain relief. Unable to swallow he drools mucus. The inspiratory phase is prolonged and ends in stridor. In contrast the expiratory phase is short, silent and without obvious effort. The child may become unconscious. Within a few hours of onset the child is in extremis from respiratory obstruction, infective toxaemia and exhaustion. A pyrexia up to 41°C is common. Leucocytosis is usually present. Cervical lymph nodes are not palpable unless associated with tonsillitis or pharyngitis.


*Present address: Birmingham Accident Hospital, Birmingham.
### Table I
Number of cases of acute epiglottitis reported by various authors and percentage mortality

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Cases</th>
<th>1. Treatment of infection and artificial control of airway</th>
<th>Group II Medical Management</th>
<th>THERAPY</th>
<th>MORTALITY</th>
<th>MORTALITY (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinclair</td>
<td>1941</td>
<td>10</td>
<td>6</td>
<td>4</td>
<td>4</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Du Bois &amp; Aldrich</td>
<td>1943</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Rabe</td>
<td>1948</td>
<td>28</td>
<td>25</td>
<td>3</td>
<td>5</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Jones &amp; Camps</td>
<td>1957</td>
<td>29</td>
<td>3</td>
<td>26 (Post-mortem diagnosis)</td>
<td>26</td>
<td>0</td>
<td>26</td>
</tr>
<tr>
<td>Berenberg &amp; Kevi</td>
<td>1958</td>
<td>42</td>
<td>18</td>
<td>24</td>
<td>5</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Vetto</td>
<td>1960</td>
<td>37</td>
<td>12</td>
<td>25</td>
<td>3</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Present Series</td>
<td>1969</td>
<td>13</td>
<td>10</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>2</td>
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</table>

### Table II
Showing differential diagnosis of acute epiglottitis

<table>
<thead>
<tr>
<th>Disease</th>
<th>Onset</th>
<th>Cough</th>
<th>Hoarseness</th>
<th>Dyspnoea</th>
<th>Cyanosis</th>
<th>Inspiratory</th>
<th>Expiratory</th>
<th>Pyrexia</th>
<th>Toxemia</th>
<th>LARYNGOSCOPY</th>
<th>Chest</th>
<th>Airway film of neck</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Laryngotracheobronchitis</td>
<td>1–2 days</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+++</td>
<td>+</td>
<td>Subglottic oedema</td>
<td>+</td>
<td>Abnormal</td>
</tr>
<tr>
<td>Inhaled foreign body</td>
<td>immediate</td>
<td>±</td>
<td>+</td>
<td>+</td>
<td>±</td>
<td>±</td>
<td>±</td>
<td>±</td>
<td>±</td>
<td>May show atelectasis and other infective changes</td>
<td>Abnormal</td>
<td></td>
</tr>
<tr>
<td>Allergic Oedema</td>
<td>½ hr</td>
<td>++</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Supraglottic oedema</td>
<td>+</td>
<td>May show opaque foreign body</td>
</tr>
<tr>
<td>Ludwig's Angina</td>
<td>1–2 days</td>
<td>+</td>
<td>+++</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Submandibular brawny swelling</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>Acute Epiglottitis</td>
<td>2–4 hrs</td>
<td>+</td>
<td>+++</td>
<td>+</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>See text</td>
<td>Normal</td>
<td>Abnormal</td>
</tr>
</tbody>
</table>
Causative organism.

_Haemophilus influenzae_, type b, can be isolated from a throat swab in most cases (Andrew, Tandon and Turk, 1968). Occasionally other organisms appear to be implicated and these include streptococci, staphylococci, pneumococci and certain viruses; para-influenzae and respiratory syncitial virus (Poole and Altman, 1963; Charnock and Parrott, 1965).

Diagnosis.

Occasionally the enlarged and reddened epiglottis can be seen protruding above the tongue by direct inspection of the mouth (Sinclair, 1941), but a definite diagnosis is made only by direct laryngoscopy. The following features are seen:

(i) A marked redness and swelling of the epiglottis. In size and appearance it has been likened to a strawberry. There may be a lesser involvement of the arytenoids and aryepiglottic folds. No pus is visible in the affected area.

(ii) The swollen epiglottis protrudes posteriorly above the glottis forming a flap valve during inspiration.

(iii) The pharynx, the true vocal cords and the subglottic areas are not affected.

The urgency of the illness may make radiography of secondary importance in diagnosis. The radiographic features were described by Poole and Altman in 1963. A single recumbent lateral view of the neck with the head extended is taken during inspiration to visualize the airway (Dunbar, 1961). The radiographic features of the normal airway, the airway in laryngotracheobronchitis and in acute epiglottitis are shown in figure 1. Acute epiglottitis must be distinguished from acute laryngotracheobronchitis, inhaled foreign body, allergic oedema, and Ludwig's angina. (See table II.)

Management.

There are four aspects to the management of patients with acute epiglottitis. These are (1) the institution and maintenance of an adequate airway; (2) the control of the infection; (3) the reduction of the inflammatory oedema; (4) the general care.

PRESENT SERIES

Forty cases of croup were seen in children at Warwick Hospital in the period between January 1964 and December 1968. In seven of these the diagnosis was acute epiglottitis. The authors had also seen six further cases of acute epiglottitis at Sentralsykehuset, Trondheim, Norway (four cases), and at the Children's Hospital, Birmingham (two cases). The case reports in this series are summarized in table III.

Presentation.

The age of the children admitted with acute epiglottitis ranged from four months to eight years. All except one were under five years. The incidence was equal in both sexes. The duration of illness, before admission, ranged from two hours to three days. Nine of the thirteen patients were admitted within twelve hours, and twelve were admitted within twenty-four hours of the onset of symptoms. No patient had any significant previous medical history, except for two children whose parents described similar but less severe episodes a few months prior to the present illness, and wondered if there would be a recurrence. They could not be reassured.

Dyspnoea was the predominant symptom and was present in all the eleven patients who were seen alive. Inspiratory stridor and pyrexia were also present in all these cases. Cyanosis occurred only in six cases, and these were either unconscious or semi-conscious at admission. There were two cases who were semi-conscious but not cyanosed.

Diagnosis.

Acute epiglottitis was confirmed in all thirteen cases by direct laryngoscopy. A Macintosh laryngoscope with an adult blade was used. This provided a good retraction of the tongue and hence a wider view of the laryngo-pharynx. It was further confirmed at autopsy in the two patients who died. In eleven patients, the lesion was confined only to the epiglottis, whereas in two patients, one admitted at twenty-four hours and one at three days after onset, there was associated laryngotracheobronchitis and tonsillitis respectively. A throat swab was taken from all the eleven surviving children at laryngoscopy. _Haemophilus influenzae_, type b, was isolated from five patients.
Fig. 1

A Normal airway.

B Acute laryngotracheitis. Note typical swelling and narrowing in the subglottic region.

C Acute epiglottitis. Note ballooned hypopharynx and enlarged epiglottis almost reaching soft palate.
ACUTE EPIGLOTTITIS IN CHILDREN

and streptococci from one. No growth was obtained in three cases and no records were available for the remaining two patients.

Management.

In one child the respiratory obstruction was considered mild. The natural airway was maintained and she was nursed in a humidified oxygen tent. In the remaining twelve cases, artificial control of airway was accomplished as follows:

(a) Oral intubation alone (2 cases admitted with cardio-respiratory arrest).

(b) Immediate control by oral intubation followed by nasotracheal intubation (1 case).

(c) Endotracheal intubation followed by planned tracheostomy (4 cases).

(d) Nasotracheal intubation alone (5 cases).

Intubation was not always easy but never impossible, as reported elsewhere (Andrew, Tandon and Turk, 1968). In four patients who, initially, were treated by endotracheal intubation a tracheostomy was performed within twenty-four hours. These cases were early in the series. Prolonged endotracheal intubation was not at that time widely accepted. The advantages of prolonged intubation over tracheostomy have since become more apparent and the later cases in the series were successfully treated by this method.

Antibiotics.

Chloramphenicol was prescribed for five cases and ampicillin for four. In the child in whom acute epiglottitis was associated with laryngotracheobronchitis, penicillin and streptomycin were administered. The two who succumbed in the casualty department did not receive any antibiotic.

Sedation.

Phenothiazine derivatives were used for sedation, mainly trimipramine tartrate or promethazine. These were preferred to narcotics or barbiturates because they have antiemetic properties, produce minimal respiratory depression and have a prolonged action. Sedatives were initially administered intramuscularly, later by mouth. On one occasion, intramuscular paraldehyde was used to sedate a restless child.

Nutrition.

The disease was of comparatively short duration and parenteral nutrition was not required. In the first twelve hours no feeding was undertaken. This period was utilized to control the airway and the infection, and to provide adequate sedation. At twelve hours spoonfuls of cold orange juice were taken by mouth and continued as necessary. Hot drinks were not well liked. Ice cream was popular. The presence of an artificial airway did not affect the appetite or the process of swallowing. Oral feeding was withheld two hours prior to extubation and recommenced two hours later if the child's condition remained satisfactory.

Humidification.

Humidification was accomplished in one of four ways:

(a) Normal saline instillation—2–4 ml introduced into the tube every half-hour prior to suction.

(b) Air Shields "Croupaire", directed near the face of the patient.

(c) A steam kettle, when secretions were thick and viscid.

(d) An oxygen tent (Oxygenaire) with nebulizer, when oxygen enrichment was considered advisable.

Disposal.

Of the thirteen cases of acute epiglottitis, eleven survived. Cardio-respiratory arrest occurred in two children during transport from home. Resuscitation in the casualty department was unsuccessful. Of the eleven who survived, five were discharged home within three days and a further two by the eighth day. All of these were treated with nasotracheal intubation. The four children in whom planned tracheostomy was performed were discharged home between the tenth and thirteenth day. The cases who were intubated therefore recovered considerably more quickly than those with tracheostomy. This somewhat surprising finding may be coincidental; all these cases were late in the series, by which time the use of steroids had become routine.

Complications or sequelae.

In two cases it became necessary to re-introduce the nasotracheal tube some hours after extubation, on account of accumulating secretions which the
<table>
<thead>
<tr>
<th>No. of Cases</th>
<th>Age</th>
<th>Sex</th>
<th>Duration of illness (hrs)</th>
<th>Previous medical history</th>
<th>Clinical state at admission</th>
<th>Investigation of respiratory insufficiency</th>
<th>Diagnosis</th>
<th>Causative organism</th>
<th>Management of respiratory insufficiency</th>
<th>Management of infection and oedema</th>
<th>Complications or sequelae</th>
<th>Duration of stay in hospital</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8½ yr</td>
<td>Female</td>
<td>12</td>
<td>Measles</td>
<td>Semi-conscious</td>
<td>Dyspnoea</td>
<td>1. Laryngoscopy</td>
<td>Acute Epiglottitis</td>
<td>Haemophilus Influenzae</td>
<td>(a) Nasotracheal intubation (24 hrs) followed by (b) Tracheostomy (8 days)</td>
<td>Ampicillin (10 days)</td>
<td>Nil</td>
<td>10 days</td>
</tr>
<tr>
<td>2</td>
<td>2 yr</td>
<td>Male</td>
<td>14</td>
<td>Recurrent throat infections</td>
<td>Semi-conscious</td>
<td>Dyspnoea</td>
<td>1. Laryngoscopy</td>
<td>Acute Epiglottitis</td>
<td>Haemophilus Influenzae</td>
<td>(a) O₂ tent (2 hrs) followed by (b) Nasotracheal intubation (24 hrs) (c) Tracheostomy (7 days)</td>
<td>Ampicillin (10 days)</td>
<td>Developed rash on tenth day on trunk and face. Rash disappeared 3 days after stopping Ampicillin</td>
<td>13 days</td>
</tr>
<tr>
<td>3</td>
<td>5 yr</td>
<td>Female</td>
<td>3 days</td>
<td>Whooping cough and mumps</td>
<td>Conscious</td>
<td>Dyspnoea</td>
<td>1. Laryngoscopy</td>
<td>Acute Epiglottitis and acute tonsillitis</td>
<td>Streptococci</td>
<td>O₂ tent (Humidified) (2 days)</td>
<td>Ampicillin (7 days)</td>
<td>Nil</td>
<td>8 days</td>
</tr>
<tr>
<td>4</td>
<td>3 yr</td>
<td>Male</td>
<td>4</td>
<td>Acute epiglottitis (4 mths. before)</td>
<td>Unconscious</td>
<td>Dyspnoea</td>
<td>1. Laryngoscopy</td>
<td>Acute Epiglottitis</td>
<td>Haemophilus Influenzae</td>
<td>Nasotracheal intubation (3 days)</td>
<td>Ampicillin (7 days)</td>
<td>Nil</td>
<td>8 days</td>
</tr>
<tr>
<td>5</td>
<td>2 yr</td>
<td>Male</td>
<td>24</td>
<td>Premature birth (intubated for 1 day)</td>
<td>Conscious</td>
<td>Dyspnoea</td>
<td>1. Laryngoscopy</td>
<td>Acute Epiglottitis and Laryngotracheitis</td>
<td>No growth</td>
<td>(a) Nasotracheal intubation (12 hrs) followed by (b) O₂ tent (2 days) (c) Penicillin and Streptomycin (5 days) and steroids (3 days)</td>
<td>Nil</td>
<td>3 days</td>
<td>Survived</td>
</tr>
<tr>
<td>6</td>
<td>1 yr</td>
<td>Male</td>
<td>2</td>
<td>Sensitive to powders and perfumes</td>
<td>Unconscious</td>
<td>Dyspnoea</td>
<td>1. Laryngoscopy</td>
<td>Acute Epiglottitis</td>
<td>Haemophilus Influenzae</td>
<td>(a) Orotracheal intubation (armoured tube 4 hr) followed by (b) Nasotracheal intubation (24 hrs)</td>
<td>Chloramphenicol (3 days) Steroids (3 days)</td>
<td>Reintubated after 24 hrs of removal of endotracheal tube because of retention of secretions (6 hrs)</td>
<td>3 days</td>
</tr>
<tr>
<td>Case</td>
<td>Age</td>
<td>Sex</td>
<td>Duration</td>
<td>Previous Illness</td>
<td>Signs and Symptoms</td>
<td>Treatment</td>
<td>Course</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>3 yr</td>
<td>Female</td>
<td>2 days</td>
<td>No previous illness</td>
<td>Semi-conscious, Dyspnoea, Insp. stridor, Cyanosis</td>
<td>1. Laryngoscopy 2. Throat swab Acute Epiglottitis, Haemophilus Influenzae</td>
<td>(a) O2 tent (2 hrs) followed by (b) Nasotracheal intubation (12 hrs)</td>
<td>Chloramphenicol (3 days) Steroids (2 days) Nil</td>
<td>3 days</td>
<td>Survived</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>2 yr</td>
<td>Female</td>
<td>15 days</td>
<td>No previous illness</td>
<td>Unconscious, Cyanosed, Cardiac and Respiratory arrest</td>
<td>Laryngoscopy Acute Epiglottitis</td>
<td>(a) Oral intubation (b) Cardiac massage (c) Controlled respiration</td>
<td>Cardiotonic drugs Hydrocortisone Post-mortem</td>
<td>1 hr</td>
<td>DIED</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>4 mth</td>
<td>Male</td>
<td>2 days</td>
<td>Similar episode 3 months ago</td>
<td>Conscious, Dyspnoea, Stridor, No cyanosis, Pyrexia</td>
<td>1. Laryngoscopy under G.A. 2. Throat swab Acute Epiglottitis</td>
<td>No growth</td>
<td>Nasotracheal intubation Chloramphenicol (3 days) Steroids (5 days) * See below</td>
<td>5 days (1st admission) 4 days (2nd admission)</td>
<td>Survived</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>3 yr</td>
<td>Female</td>
<td>2 days</td>
<td>No previous illness</td>
<td>Conscious, mild dyspnoea, stridor, No cyanosis, Pyrexia</td>
<td>1. Laryngoscopy under G.A. 2. Throat swab Acute Epiglottitis</td>
<td>No growth</td>
<td>Nasotracheal intubation Chloramphenicol (3 days) Steroids (3 days) Extubated after 24 hrs No complications</td>
<td>4 days</td>
<td>Survived</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>18 mth</td>
<td>Female</td>
<td>24 days</td>
<td>No previous illness</td>
<td>Unconscious, Respiratory arrest, Cyanosed, Cardiac arrest</td>
<td>1. Laryngoscopy Acute Epiglottitis</td>
<td>—</td>
<td>Nasotracheal intubation Cardiotonic drugs Post-mortem</td>
<td>Nil</td>
<td>DIED</td>
<td></td>
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</table>

**RECORDS DEFICIENT FOR THE FOLLOWING TWO CASES**

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>Duration</th>
<th>Previous Illness</th>
<th>Signs and Symptoms</th>
<th>Treatment</th>
<th>Course</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>2 yr</td>
<td>Female</td>
<td>A few days</td>
<td>Semi-conscious, Cyanosed, Dyspnoea, Stridor</td>
<td>1. Laryngoscopy under G.A. 2. Throat swab Acute Epiglottitis</td>
<td>—</td>
<td>Oral intubation followed by tracheostomy</td>
</tr>
<tr>
<td>13</td>
<td>3 yr</td>
<td>Male</td>
<td>A few days</td>
<td>Conscious, Dyspnoea, Stridor, Pyrexia, Not cyanosed</td>
<td>1. Laryngoscopy under G.A. 2. Throat swab Acute Epiglottitis</td>
<td>—</td>
<td>Oral intubation followed by tracheostomy</td>
</tr>
</tbody>
</table>

* Extubation after 24 hours—reintubated 4 hours later because of stridor, extubated after 48 hours, sent home fifth day. Readmitted 2 days later with dyspnoea—laryngoscopy under G.A. Laryngotracheal tract sucked out—thick viscid secretions removed. Dyspnoea disappeared. Nursed in humidified O2 tent—Discharged four days later without any further sequelae.
child was unable to expel by coughing. Re-intubation was performed under general anaesthesia when the subglottic area was inspected, secretions aspirated, and a fresh tube reintroduced.

In one case a rash appeared on the face and trunk ten days after ampicillin treatment was commenced. This disappeared when the antibiotic was discontinued.

**DISCUSSION**

*Management of the airway.*

If the respiratory obstruction is mild, the child can be nursed with an oxygen enriched atmosphere in a tent. Effective humidification is essential. Sedatives are used with caution to calm the patient and reduce the unnecessary activity and hence oxygen requirements.

Deterioration in the clinical condition of the child, as shown by increasing stridor, increasing intercostal retraction and, in particular, exhaustion, warrant immediate control of the airway.

Emergency tracheostomy, although it may be life-saving, is associated with potentially lethal complications. Endotracheal intubation followed by planned tracheostomy avoids some of these hazards but the well accepted risks of tracheostomy remain (Smith, 1966).

If the nasotracheal tube inserted is small, e.g., less than 3 mm internal diameter, its patency is constantly threatened; if a larger tube is impossible to insert, then a planned tracheostomy is justified.

Prolonged nasotracheal intubation with a smooth non-toxic plastic tube is now well established as an alternative to tracheostomy, particularly in children (McDonald and Stocks, 1965; Allen and Steven, 1965; Rees and Owen Thomas, 1966; Smith, 1966). Prolonged intubation has certain advantages over tracheostomy. In comparison with tracheostomy, it can be rapidly performed in difficult situations, including acute epiglottitis. If rapid improvement occurs, the tube can be removed without further attention to the airway. Bleeding into the tracheobronchial tree is avoided (Harrison and Tonkin, 1968).

*Procedure.*

The endotracheal tube is chosen with care. Since acute epiglottitis is a localized supraglottic lesion, it only produces supraglottic obstruction. The inflamed epiglottis is displaceable: the true cords and subglottic area are normal. The size of the tube will depend upon the normal consideration; i.e. the size of the cricoid. It should be at least one size smaller than that which would be used for anaesthesia, to allow for mucosal oedema.

In most of the children the trachea can be intubated without anaesthesia because of their serious clinical state. Older children who are active and resist strenuously are anaesthetized prior to laryngoscopy. Premedication is not necessary because of the urgency required for control of the airway. An inhalation method of anaesthesia is preferred; halothane or cyclopropane is satisfactory. Because of the respiratory difficulty, induction is slower than normal. Muscle relaxants are contra-indicated as there may be difficulty in visualizing the glottis during apnoea.

The tube is securely fastened to reduce friction and to prevent dislodgement. Strict skilled nursing care of the airway is maintained.

The use of prolonged nasotracheal intubation in the management of acute epiglottitis presents two possible specific disadvantages. First, there may be some technical difficulty in the insertion of the tube due to the oedematous epiglottis. Secondly, the presence of the tube in direct contact with the infected tissues might be expected to aggravate the condition and prolong its course. Abbott (1968) discussing the complications of prolonged nasotracheal intubation in children, stated that permanent laryngeal changes were more common in patients with upper respiratory tract disease than with lower respiratory tract disease. His cases, however, were of laryngotracheobronchitis, which has a much more diffuse pathology. It is possible that with the localized pathological change of acute epiglottitis this generalization does not apply.

*Extubation.*

After 24 to 48 hours the infection and the oedema will have subsided sufficiently to consider the removal of the tube. The state of the epiglottis is first assessed by direct laryngoscopy. In addition, the following should obtain:

(a) The tube is loose and easily movable.

(b) The airway is clear.

(c) The temperature of the patient is normal.

(d) All equipment for re-insertion of the tube is at hand.
Management of the infection, oedema and inflammation.

The majority of cases are infected by *Haemophilus influenzae*, type b. The antibiotics of choice are ampicillin and chloramphenicol, and of these the latter is probably the more effective. Chemotherapy is commenced as soon as the clinical diagnosis is made. Bacteriological swabs are taken from the epiglottis and the surrounding structures. Blood can be taken for evidence of bacteraemia.

Steroids are administered to reduce the oedema and inflammation. Either hydrocortisone hemisuccinate 50 mg or dexamethasone 3–5 mg can be administered intramuscularly every eight hours. This dosage is reduced twenty-four hours after extubation if the airway remains satisfactory (Martensson, Nilsson and Torbjär, 1960; Novik, 1960).

Disposal and follow-up.

Normally the child is sent home in 3–7 days. Recurrence of dyspnoea at home necessitates readmission, laryngoscopy, aspiration, and observation.

If there are no complications after discharge, it is advisable to see the child in outpatients' clinic at periodic intervals for evidence of laryngeal complication due to the disease or instrumentation.

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REFERENCES


**L'EPIGLOTTITE AIGUE DE L'ENFANT: UNE URGENCE RESPIRATOIRE**

**SUMMARY**

Treize cas d'épiglottite aiguë sont rapportés. L'amélioration du taux de mortalité grâce au traitement adéquat est soulignée. Le diagnostic et les mesures à prendre sont brièvement discutés. Il est nécessaire d'accorder une attention particulière au maintien des voies respiratoires libres, au contrôle de l'infection et à l'emploi de stéroïdes. En vue de la rapide progression de l'hypoxie et du collapse, ainsi que de la mortalité élevée, l'épiglottite aiguë constitue une urgence respiratoire qui requiert une thérapie énergique urgente.

**AKUTE EPIGLOTTITIS BEI KINDERN**

**ZUSAMMENFASSUNG**