

STEROID AND ANTIHISTAMINIC THERAPY FOR POST INTUBATION SUBGLOTTIC EDEMA IN INFANTS AND CHILDREN

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A MAJOR complication of endotracheal anesthesia in children is laryngeal or subglottic edema. It is not so much the incidence but rather the catastrophic aspect of this entity which lead us to make suggestions for its prevention and treatment.

In its mildest form subglottic edema is evidenced by hoarseness or croupy cough persisting for several hours. Signs and symptoms of impaired gaseous exchange are not present.

In the more severe cases, in addition to hoarseness and croupy cough, inspiration is accompanied by retraction in the suprasternal area. As edema progresses and the degree of respiratory obstruction increases, intercostal retraction appears. The accessory respiratory muscles become active, and in thin patients the movement of the diaphragm, as it separates from the lower thoracic wall is easily visible. A crowing sound usually is present with inspiration. With increasing respiratory difficulty, the children become restless and show acute anxiety. Profuse sweating may start abruptly. If treatment is delayed, oxygenation is more and more difficult and the children become cyanotic. Complete obstruction may ensue, necessitating tracheostomy as a lifesaving measure.

ETIOLOGY

Two causative factors are generally recognized: (1) mechanical injury, and (2) the introduction of infection from either exogenous or endogenous sources.

Mechanical trauma is related, to a considerable extent, to the anesthetist's experience with pediatric patients. Inadequate depth of anesthesia or muscle relaxation and unneces-

sary roughness at the time of tracheal intubation may lead to laryngeal complications. The infantile larynx resembles a funnel; the narrowest point is situated at the lower border of the cricoid cartilage. It may be easy to insert a tube between the vocal cords, only to find that it is too large to pass the cricoid constriction. A tube left in this position may produce subglottic swelling.¹ If forced through the cricoid narrowing, the tube may cause ischemia of the laryngeal mucosa. This is followed by edema when the pressure is released after extubation. Because of a rubbing action, too small a tube also may be responsible for laryngeal trauma. The selection of the correct tube size, unfortunately, is not easy. Age, height, weight, and surface area are only fair guides in growing children. We find it of advantage to prepare two tubes, one smaller than the selected "correct" size. In our experience, the change to clear plastic tubes has decreased the incidence of respiratory difficulties following endotracheal anesthesia. Initially these tubes seem more difficult to insert than rubber tubes. If the selected tube is too large, it will buckle and cannot be forced through the larynx, providing an additional safety factor.

The prevention of exogenous infection is relatively simple. Tracheal tubes must be clean and free of pathogens. The tubes must be scrubbed with antiseptic detergent, thoroughly rinsed and sterilized by boiling or autoclaving. Storage in plastic wrappers facilitates preservation of cleanliness.

Spread of infection from endogenous sources is a more complicated problem. Following an acute upper respiratory infection, tracheal intubation should be avoided for four to six weeks if possible. It is difficult to recognize an incipient upper respiratory tract infection in a small child who cannot, or will not admit to a dry scratchy throat. It may be almost

Received from the Department of Anesthesia, Philadelphia General Hospital, and the Department of Anesthesia, University of Pennsylvania Schools of Medicine, Philadelphia, and accepted for publication July 12, 1961.

TABLE 1
DATA OF 10 PATIENTS TREATED FOR SEVERE SUBGLOTTIC EDEMA

Patient	Age (years)	Weight (pounds)	Sex	Operation	Duration of Anesthesia (minutes)	First Recording of Symptoms after Extubation (minutes)	Medication*	Onset of Response (minutes)	Clearing of Respiratory Symptoms Complete (minutes)
1	8/12	19	M	Skin graft to abdomen	120	45	Bena† 10 mg. Pred‡ 15 mg.	65	90
2 Traumatic intubation	1-5/12	27	M	Herniorrhaphy	90	50	Bena 15 mg. Pred 20 mg.	75	120
3	3-6/12	39	F	Ureteral transplant	210	90	Bena 20 mg. Pred 25 mg.	65	115 (Hoarseness 8 hours)
4	2	36	M	Bilat. ing. herniorrhaphy	205	40	Bena 15 mg. Deca§ 8 mg.	30	45
5 Recent upper respiratory infection	2	31	F	Ventriculogram Craniotomy	290	120	Bena 15 mg. Deca 5 mg.	20	80
6	4	42	F	Tendon transplant, Manipulation and cast right leg	145	175	Bena 20 mg. Deca 10 mg.	40	85
7	1-2/12	23	M	Skin graft to arm and thorax	165	55	Bena 10 mg. Deca 4 mg.	45	65
8	3-2/12	25	F	Ligation of patent ductus	100	60	Bena 15 mg. Deca 6 mg.	30	60
9	2-9/12	35	M	Osteotomy and bone graft left leg	170	75	Bena 20 mg. Deca 8 mg.	45	65
10	3-4/12	37	M	Explor. lap. and appendectomy	130	80	Bena 20 mg. Deca 8 mg.	30	150

* Medication given intravenously except for patient no. 1 which was given intramuscularly.

† Bena = diphenhydramine chloride.

‡ Pred = prednisolone.

§ Deca = dexamethasone-21-phosphate.

impossible to differentiate an acute coryza from a nasal discharge from crying. Yet, in the early stages of an acute upper respiratory infection the whole respiratory tract is susceptible to bacterial invasion. Undoubtedly, some post intubation edema has followed the

introduction of pathogens in the presence of a "cold" without recognizable, pathognomonic signs.

It must also be admitted that in spite of all precautions and use of a flawless technique, subglottic edema may still occur.

According to many, the onset of respiratory symptoms due to subglottic edema ranges from two to twelve hours. In our experience, however, the first warning signs appear within 40 to 175 minutes of extubation.

TREATMENT

At the earliest sign of a laryngeal complication, active therapy should be instituted and the child closely observed. Severe laryngeal obstruction requiring tracheostomy can be prevented, we believe. The exception is laryngeal edema following suboccipital craniotomy; here, we advise tracheostomy without delay.

Therapeutic measures include the following: (1) High oxygen concentration in the inspired air (60 per cent). (2) High humidity of the inspired atmosphere (close to 100 per cent). (3) Body temperature kept slightly below normal (cooled tent, or added ice packs). (4) Adequate parenteral fluid intake.

If the edema is mild, most patients will respond satisfactorily, albeit slowly, to these simple measures. If there is evidence of progression of symptoms with increasing restlessness, a more vigorous therapy is indicated, if tracheostomy is to be avoided.

In the wake of hypoxia restlessness increases further the demand for oxygen and leads to an exaggeration of the respiratory symptoms. Sedation is necessary, although concomitant depression of the respiratory center is undesirable. Intravenous diphenhydramine chloride (Benadryl) accomplishes this purpose well. The dosage should be carefully adjusted to the requirements of each child. A dose of 0.25 mg. to 0.50 mg. per pound of body weight usually eliminates restlessness and will induce a light sleep. Since there is always a possibility that an allergic response plays a role in the etiology of the laryngeal edema, some relief of the respiratory distress may occur. While useful in the control of symptoms, diphenhydramine alone seldom produces dramatic relief.

One of the therapeutic effects of the adrenal steroids is the control of inflammatory and allergic phenomena and swelling.² We have tried several steroid compounds. Prednisolone proved clinically effective (table I); however, the most rapid response was obtained

with dexamethasone-21-phosphate (Decadron).^{* 3} A single dose of 4 mg. for infants under one year of age and 8 mg. for children of the older age group is administered intravenously. A subsequent dose is rarely required. Definite improvement is noted from twenty to forty minutes after the administration of diphenhydramine and dexamethasone. Restlessness disappears and the children fall into a light sleep. Respiration becomes less labored, while pulmonary exchange improves as evidenced by disappearance of cyanosis.

Subglottic edema sufficiently severe that tracheotomy is advised is the exception rather than the rule. Table 1 lists data from 10 of 1,123 patients given endotracheal anesthesia during a period of 2½ years. We believe that tracheotomy would have been necessary had the combined therapy with diphenhydramine and dexamethasone been ineffective.

Two typical case histories follow:

CASE 1: A two year, 36 pound healthy negro boy without history of recent respiratory infection or other acute illness. The physical examination did not show any abnormality except a bilateral inguinal hernia. Preoperative medication consisted of pentobarbital 50 mg., morphine sulfate 1.4 mg. and scopolamine 0.15 mg., intramuscularly at 7:15 A.M. On arrival in the operating room half an hour later, the patient was awake, but quiet and drowsy. Anesthesia was induced at 8:20 A.M. with nitrous oxide, cyclopropane and oxygen by gravity flow; ether was added at 8:24 A.M. Seven milligrams of succinylcholine were given intravenously at 8:30 A.M. and a 22 F. Magill rubber tube was inserted into the trachea. Intubation was easy; there was no bucking, swallowing or coughing. Anesthesia was maintained with nitrous oxide 2.5 liters, oxygen 2.5 liters and ether. A nonbreathing Leigh valve was used. The surgical procedure started at 8:40 A.M., and a bilateral inguinal herniorrhaphy was completed at 10:35 A.M. Respiration was spontaneous throughout the procedure. At intervals the respiratory exchange was assisted. At conclusion of the operation, the patient was in light surgical anesthesia. As the endotracheal tube was removed, a slight cough occurred. Respiration continued to be regular and the exchange adequate. At 10:50 A.M. the patient was admitted to the recovery room. His reflexes were present and he responded readily to the spoken voice. He slept when undisturbed. The airway was patent. During the procedure the patient had received 150 ml. of 5 per cent glucose in water by vein.

* Supplied by Merck Sharp & Dohme.

At 11:15 A.M. the recovery room nurse noted that respiration had become noisy and the child had a brassy cough, when disturbed. At 11:20 A.M. there was definite inspiratory retraction, most marked in the supraclavicular area; the accessory muscles of respiration were being used. The rate of intravenous fluids was increased from 8 to 16 drops per minute. The patient was placed in a croupette with the humidity reaching 95 per cent within 10 minutes. By 11:40 A.M. the child became restless. Diphenhydramine hydrochloride 10 mg. was given intravenously; an additional 5 mg. were administered at 11:45 A.M. The child became quiet but respiration continued to be labored. The rectal temperature was 94.5 F. At 12:15 P.M. we believed that without relief of obstruction tracheotomy would be necessary. Dexamethasone-21-phosphate 8 mg. was given intravenously at 12:18. By 12:35 P.M. respiration was less labored, and retraction occurred only when the child cried. At 1:00 P.M. the child was asleep without evidence of respiratory distress. One hour later he was awake and crying. At 2:15 P.M. he was returned to the ward, to remain in the croupette until the following morning. By error the croupette was removed on arrival. By 6:00 P.M. the patient had left his bed and was walking around the ward. Convalescence was uneventful.

CASE 2: A two year old white girl for ventriculogram and craniotomy. Anesthesia consisted of thiopental and succinylcholine by vein, and endotracheal nitrous oxide and oxygen with the Ayre insufflation technique. Anesthesia lasted four hours and 50 minutes. The child was awake on arrival in the recovery room. Respiration was free and gas exchange adequate. Two hours later respiration became labored. Retraction in the suprasternal and supraclavicular area was marked. Direct laryngoscopy showed subglottic edema de-

creasing the airway by an estimated 70 per cent. The child was placed in a croupette and given diphenhydramine hydrochloride 15 mg. and dexamethasone-21-phosphate 8 mg. intravenously. She became quiet and a definite decrease of retraction was noted 20 minutes after medication. One hour later when respiration was no longer labored and retraction completely absent, direct laryngoscopy was repeated. Most of the subglottic edema had disappeared and the airway looked almost normal. The child was discharged from the recovery room five hours after admission. Her subsequent recovery was uneventful.

SUMMARY

Subglottic edema following extubation is a hazard of pediatric anesthesia. Etiologic factors have been outlined, prophylactic measures reviewed, and some new suggestions for treatment offered. In 10 cases, severe enough that tracheostomy was seriously considered, intravenous medication with diphenhydramine chloride and dexamethasone-21-phosphate proved effective. Within one to three hours the respiratory symptoms had cleared completely.

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