Laboratory Note

A Technique for Measuring Reactivity of the Glottis

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The sphincteric action of the larynx has been described in detail by Pressman. Because depression or absence of the protective reflexes of the larynx may lead to aspiration of foreign material into the lungs, a validated technique to evaluate this reflex closure of the glottic sphincter is necessary. In 1950, Hoglund and Michaelsson used anhydrous ammonia to initiate glottic closure. Gravenstein, Devlo, and Beecher used ammonia in 1954 to evaluate the effects of antitussive agents on experimental cough, as did Pontoppidan and Beecher in 1950 to measure the loss of protective reflexes in the airway with advance of age. They assumed that the response to ammonia was reflexive, but the site of afferent stimulation was not established. We have studied the action of this chemical irritant in developing a simple, clinically useful test of glottic competence. Our experiments show an upper airway site for afferent stimulation by ammonia.

Material and Method

A 9-liter Collins spirometer is connected by corrugated rubber tubing to the inspiratory port of an Ambu-Hesse valve with a disposable mouthpiece (fig. 1). At the junction of the valve and the corrugated tubing, we inserted the tip of a 10-gauge, 35-inch Intracath through the rubber, and to the other end of the catheter attached a three-way stopcock and a 50-ml glass syringe. The spirometer is filled with room air or oxygen; the syringe is filled with various amounts of room air and a commercially prepared 2 per cent ammonia gas-air mixture from a cylinder stored on the lower shelf of the cart.

The subject breathes via the mouthpiece with a noseclip in place. After recording a regular inspiratory pattern for several breaths on the spirometer, we inject a bolus of 50 ml of the ammonia-air mixture into the inspiratory tubing during exhalation so that the irritant gas will be incorporated into the next inspiratory volume. When the dose of ammonia given is sufficient to elicit the glottic closure reflex, a momentary halt in the inspiratory pattern is graphed; when insufficient, a normal inspiratory pattern appears, and when excessive, the subject coughs.

To determine the normal responses to this test, ten volunteers between 21 and 45 years of age were given 0, 2.5, 5, 10, and 20 ml of 2 per cent ammonia, with the balance to 50 ml being made with room air. After the normal threshold doses for glottic closure had been determined in five volunteers between 25 and 40 years of age, we anesthetized the upper airway, hypopharynx, and superior laryngeal nerves by topical application of 4 per cent lidocaine, using cotton pledges and a piriform sinus forceps. The amount of anesthetic used was not enough to produce systemic effects. As judged by voice changes and difficulty in handling secretions, adequate anesthetization of the upper airway and larynx was accomplished in a 20-minute period. We repeated the test using progressively doubled concentrations of ammonia. On a different day, the same five volunteers were given lidocaine, 150 mg, by intravenous infusion over a 20-minute period, and the ammonia test for glottic closure was repeated. This was done to determine whether the change in threshold

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TECHNIQUE FOR MEASURING REACTIVITY OF THE GLOTTIS

Fig. 1. A 9-liter Collins spirometer mounted on a cart carrying a tank of 2 per cent ammonia gas.

Fig. 2. Responses of ten normal subjects to threshold and excessive, i.e., resulting in cough, doses of ammonia gas diluted with air.
observed with topical anesthetization could be due to central depression of the reflex secondary to lidocaine absorbed into the circulation.

In addition, the glottis of each of four subjects were fluoroscoped in order to observe whether glottic closure occurred at the same time that a threshold response appeared on the spirometer tracing.

Results

The threshold doses of 2 per cent ammonia were 2.5 to 10 ml/breath in all ten normal subjects (fig. 2). Twenty ml produced cough in all subjects, and at no time did anyone respond to room air. The threshold doses necessary before and after topical anesthetization and intravenous administration of lidocaine are shown in figures 3 and 4, respectively. In four subjects, 20 to 40 ml of ammonia were necessary to elicit glottic closure following topical anesthetization, but all five responded normally after intravenous injection of lidocaine in spite of experiencing lightheadedness, tinnitus, or dysarthria. The fifth subject reacted to low doses on all occasions, but the dose needed after topical anesthetization was double the dose needed before.
At fluoroscopy the roentgenologist observed glottic closure at the same instant that a halt in inspiration was traced on the spirometer, and he remarked that the diaphragm momentarily stopped descending at this time. When the glottis did not close, the inspiratory tracing continued as a straight upward slope.

Discussion

From the results presented above, it appears that reflex closure of the glottic sphincter is initiated by ammonia gas, which stimulates afferent receptors located in the hypopharynx and larynx. The normal glottis gives a threshold response to a 50-ml bolus of 0.1 to 0.4 per cent ammonia gas added to the inspired tidal volume, but after topical anesthetization a response is not obtained until a higher concentration of ammonia is given. It could be hypothesized that the extremely soluble ammonia is reaching deep receptors in the glottic tissues that are not anesthetized by the lidocaine or that there are unanesthetized receptors further down the airway.

Because of the short period of exposure to the concentrations of ammonia used in this study, toxic effects did not result from inhalation of the gas.

This portable device simply and quickly determines whether the laryngeal sphincter is active and quantitates the amount of stimulus necessary to initiate the reflex. A control response is of the greatest value, however, since there is individual variability within the normal range of threshold dosage of ammonia. The technique has many uses in evaluating airway reflexes, which are sometimes depressed following tracheal extubation and after administration of anesthetic drugs.

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References


Obstetrics

BUPIVACAINE IN PERIDURAL ANESTHESIA  Bupivacaine, 0.25, 0.5, or 0.75 per cent, was used in 657 caudal or lumbar epidural anesthetics for delivery. Onset of pain relief occurred in 4 to 10 minutes. Duration was 3½ to 7 hours. When a continuous technique was used, reinforcing doses were needed in only 34 per cent of the patients receiving 0.25 per cent, and 13 per cent of patients receiving 0.5 per cent. Toxic reactions were infrequent and mild. Seventy-four per cent of the infants had one-minute Apgar scores of 8, 9 or 10. Addition of epinephrine to the bupivacaine usually prolonged the duration of the anesthetic. Bupivacaine appears to be a long-acting, safe local anesthetic very suitable for peridural anesthesia in obstetrics. (Moore, D. C., and others: Caudal and Epidural Blocks with Bupivacaine for Childbirth, Obstet. Gynec. 37: 667 (May) 1971.)