The E₂¹ E₂¹ genotype plasma cholinesterase that she possesses may well have been the cause of her postoperative problem.

References

Increased Respiratory Resistance after Ultrasonic Humidification of Anesthesia Gas

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Humidification of anesthetic gases has been advocated to prevent drying of secretions and alterations of cells lining airways. Cheney and Butler found increased respiratory resistance in patients when they received ultrasonic humidification with half-physiologic saline solution via a tracheal tube and in patients with airway disease breathing mist from a face tent. Patients without airway disease who breathed ultrasonic mists of water, half-physiologic saline solution, or saline solution from a face tent did not have increased resistance. We noticed that humidification of anesthetic gases with ultrasonic mist caused a prolonged expiratory phase in normal patients as well as those with chronic bronchitis. This study quantitates the effects of ultrasonic humidification of anesthetic gases on total respiratory resistance in a group of healthy patients and in a group of asymptomatic patients with increased resistance.

Methods
Subjects of the studies were 24 adult surgical patients who needed general endotracheal anesthesia. Patients who had histories of pulmonary disease were excluded. Anticholinergic drugs were not given for premedication. Anesthesia was induced with thiopental and maintained with nitrous oxide-oxygen and narcotics. Complete muscle paralysis was obtained with d-tubocurarine (0.4 mg/pound) and the trachea was intubated with a cuffed tube. Control studies were done after surgical stimulation had begun to include any possible bronchodilator effects from increased catecholamine release.

Compliance and resistance for the total respiratory system were derived from flow, volume, and transthoracic pressure measurements obtained during thoracic inflation and subsequent passive exhalation by a method described previously. Control measurements were made at 3-minute intervals while the patients were being ventilated with dry gas from the anesthesia machine. After five control measurements had been made, the inspired gas mixture was humidified using an ultrasonic nebulizer (Monaghan, 670). This unit was set to deliver 1.5 ml/min of mist into the inspiratory gas flow.
Fig. 1. Changes in respiratory resistance after ultrasonic mist: • = water; △ = 0.45 per cent saline solution; ○ = 0.9 per cent saline solution. The resistance values were measured at a flow rate of 0.5 l/sec.

By using clear plastic endotracheal tubes, the mist flow could be monitored visually from breath to breath. The patients were ventilated with the humidified gas mixture for 20 minutes, and then five additional measurements of respiratory mechanics were made at 3-minute intervals.

The study patients were divided into four groups. Group I was ventilated with distilled water mist. Group II was ventilated with 0.45 per cent saline mist. Group III was ventilated with 0.9 per cent saline mist. Three of the patients in the water-mist group were also given three metered doses of isoproterenol mist (Medihaler, Isuprel, 4 mg/ml) at 2-minute intervals, and the determinations were repeated. Group IV consisted of six subjects who had control resistance values above 9 cm H$_2$O/l/sec at an expiratory flow rate of 0.5 l/sec. In a previous study this value of resistance was shown to be associated with abnormal pulmonary function in asymptomatic subjects.

Apparatus resistance, including the endotracheal tube, was determined using a 20-liter carboy as a resistance-free patient analog. Values for apparatus resistance were subtracted from those obtained in the patients to obtain individual respiratory resistance for each patient.

Statistical comparison of the data was by t test for paired data.

RESULTS

The results are summarized in figures 1–3. The resistance data are in cm H$_2$O/l/sec at a gas flow rate of 0.5 l/sec.

Twenty minutes of ventilation with water mist (Group I) increased respiratory resistance from 5.93 (SD = 1.62) to 7.25 (SD = 1.70) (P < 0.005, fig. 1). Similar ventilation with 0.45 per cent saline mist (Group II) increased respiratory resistance from 5.66 (SD = 1.66) to 7.72 (SD = 2.15) (0.01 > P > 0.005); while 20 minutes of ventilation with 0.9 per cent saline mist (Group III) increased respiratory resistance from 6.21 (SD = 1.33) to 7.05 (SD = 1.58) (0.025 > P > 0.01, fig. 1).

In the Group I patients given isoproterenol, resistance increased from 4.32 (SD = 0.77) to 7.07 (SD = 1.22) with water mist and then decreased to 4.64 (SD = 1.67) after isoproterenol (0.01 > P > 0.005, fig. 2).

Patients whose control resistance exceeded 9 cm H$_2$O/l/sec (Group IV) had a mean increase in resistance from 11.97 (SD = 1.11) before

Fig. 2. The effect of isoproterenol (Isuprel) on the increased resistance resulting from water mist.
mists to 15.28 (SD = 3.40)0.05 > P > 0.025) after mist (fig. 3).

Compliance did not change in any group as a result of ventilation with mist. There was no postoperative problem in these patients that could be attributed to the study procedure.

**DISCUSSION**

Ultrasonic aerosols of water, 0.45 per cent saline solution and 0.9 per cent saline solution significantly increase total respiratory resistance when delivered directly into the airways of healthy patients as well as those who have increased respiratory resistance. The magnitudes of increase were the same in both groups and were not related to the composition of the mist. However, exhalation was noticeably prolonged in the patients whose control resistances were increased. Audible wheezing did not occur in any patient.

Cheney and Butler reported that mist breathed from a face tent did not increase respiratory resistance in patients who did not have respiratory disease. It is probable that the mist in these patients was deposited in the upper airways and thus would not be expected to change resistance. Asmundsson and associates have reported that ultrasonic aerosols given to awake subjects breathing normally do not reach the distal airways. They found that of 6 ml nebulized, only 55 to 65 μl were deposited in the airways distal to the larynx.

The response of our patients to isoproterenol mist indicates that the increase in respiratory resistance was largely the result of increased bronchial tone. It is unlikely that accumulation of the water or saline solution occurred. Fluids are absorbed very rapidly from the airways, and the total amount of liquid nebulized rarely exceeded 50 ml given over a 35-minute period. Also, the increased resistance was not progressive during any study period.

The results of this study and that of Cheney and Butler should not be interpreted to indicate that dry anesthetic or ventilator gases delivered directly into the trachea should not be humidified. Respiratory gas is normally fully humidified in the upper airway and does not cause an increase in respiratory resistance. This emphasizes the problem of gas humidification when the upper airway is bypassed. The point at which humidification becomes mist therapy is not easily recognized.

The increased respiratory resistance caused by the ultrasonic mist was clinically evident only in patients in whom respiratory resistances were already increased. In view of this effect, patients with pre-existing airway disease who are given high-density mists directly into the trachea should be watched for evidence of increased airway resistance.

**References**