Volume 29, Number 6
November–December 1968

THE JOURNAL OF Anesthesiology
THE AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC.

Editorial Views

An Adverse Effect of Respiratory Therapy

OXYGEN AND WATER are the fundamental drugs of respiratory therapy. Incorrect dosage of either may result in adverse physiologic effects.

In this issue of the Journal, Cheney and Butler report an increase in airway resistance resulting from inhalation of water and saline aerosols nebulized by ultrasound. This occurred in normal man as well as in patients with small-airway disease.

Aerosols of water or saline are in everyday clinical use. For humidification of dry gas, a water content of 53 mg/l at 37 C is required for full saturation. Denser mists will increase water content of thickened secretions and thereby decrease their viscosity. Addition of water to maintain secretions at normal or decreased viscosity is desirable in any patient with retained secretions. Specific indications include bronchitis, pneumonia, cystic fibrosis and burns of the tracheobronchial tree.[1] High-density ultrasonic mists are useful to promote coughing both for therapeutic mobilization of secretions and to obtain sputum samples. Cheney and Butler point out that water will provoke coughing more efficiently than saline, and that coughing *per se* is not associated with increased airway resistance.

Cheney and Butler recommend that when an ultrasonic nebulizer is used for therapy of patients with small airway disease, a bronchodilator should be administered as well. Our experience has been that the use of ultrasonic mist in conjunction with chest physical therapy has been especially valuable in managing patients with retained secretions.

Other physiologic changes associated with prolonged administration of aerosols have been described. In the newborn, continuous breathing of nebulized mist will eliminate insensible respiratory loss.[2] In the adult during prolonged mechanical ventilation, 300 to 500 ml per day may be added to water intake. This may aggravate water retention, which in patients with respiratory failure has been shown to decrease lung compliance and increase the alveolar–arterial oxygen tension gradient.[3]

In the normal lung, about a fourth of the total resistance to airflow is located in small bronchi (less than 2 mm in diameter).[4] In patients with small-airway disease, this peripheral airway resistance may be increased four to 40 times, secondary to mucus plugs and narrowing obliteration of bronchioles. This increase in peripheral airway resistance may result in changes in ventilation–perfusion relationships without significant decreases in expiratory flow rates. That is, patients with small-airway obstruction may have marked increases in peripheral resistance with normal total airway resistance.[4]

It is not known whether a simple measurement of expiratory flow (forced expiratory volume, maximum midexpiratory flow rate, peak expiratory flow rate) would reliably indicate changes in airway resistance during clinical use of aerosols. This information would be of value since measurement of total airway resistance is too cumbersome to be a practical clinical tool.

Further quantitative studies of this type are greatly needed for rational definition of indications, optimal methods of treatment, and evaluation of results for aerosol administration of water and drugs, intermittent positive-pressure breathing, chest physical therapy, and controlled ventilation—all aspects of respiratory therapy.

LEONARD S. BUSHNELL, M.D.
Director, Respiratory Therapy
Beth Israel Hospital
Instructor in Anaesthesia
Harvard Medical School
Boston, Massachusetts

References

1. Cushing, I. E., and Miller, W. F.: Nebulization therapy. *In* Safar, P. (ed.): Respiratory Therapy. Philadelphia, F. A. Davis Company, 1955, p. 169.
2. O'Brien, D., Hansen, J. D. L., and Smith, C. A.: Effect of supersaturated atmosphere on insensible water loss in the newborn infant, Pediatrics 13: 126, 1954.
3. Sladen, A., Laver, M. B., and Pontoppidan, H.: Pulmonary complications in prolonged mechanical ventilation, New Eng. J. Med. 279: 448, 1968.
4. Hogg, J. C., Macklem, P. T., and Thulbeck, W. M.: Site of airway obstruction in chronic obstructive lung disease, New Eng. J. Med. 278: 1355, 1968.

NOTE

The drawing on the cover of the symposium issue (July–August), depicting the vagus nerve and the cervical portion of the sympathetic trunk in man, was taken from Willis, T.: Cerebri anatome, *1644.*