Respiratory Therapy Modalities in the Treatment of Acute Respiratory Failure

JAMES A. L. MATHERS, JR., M.D.

Assistant Professor of Medicine and Co-Medical Director, Respiratory Therapy, Medical College of Virginia, Health Sciences Division of Virginia Commonwealth University, Richmond, Virginia

Rapid advances have been made in the field of respiratory therapy in the past several years, resulting in an increasing sophistication and range of application. Properly applied, these modalities have led to significantly increased survival in patients with acute respiratory failure and a decreased morbidity among individuals with chronic pulmonary insufficiency. It is the purpose of this article to put into perspective respiratory therapy techniques and their application in the treatment of acute respiratory failure. To this end, we may divide respiratory therapy into five categories: 1) oxygen delivery, 2) airway hygiene, 3) expansion therapy (lung inflation), 4) artificial airways, and 5) mechanical ventilation.

While the symptoms are not specific, the onset of acute respiratory failure is not a subtle event. Abnormal arterial blood gases are invariably present. Indeed, the entity is usually defined in terms of the degree of alteration in the arterial blood gases. Acute respiratory failure results from one of three general abnormalities: 1) neuromuscular dysfunction, 2) increased airway resistance, and 3) reduction of pulmonary compliance. In pure form each of these categories produces a predictable pattern of arterial blood gas derangement. Rational management of acute respiratory failure requires an awareness of these alterations so that the respiratory therapy may be intelligently applied to stabilize or reverse the patient's pulmonary deterioration.

In the initial assessment of the patient in acute respiratory distress, it is important to determine the nature of the respiratory compromise. Patients with neuromuscular dysfunction such as occurs with drug overdose or Guillain-Barré syndrome may have normal lungs but do not have the ability to maintain an adequate minute ventilation. The arterial blood gas derangement consists of hypoxemia and relatively acute hypercarbia with a normal alveolar-arterial oxvgen tension gradient. Respiratory failure due to a sudden increase in airway resistance includes entities such as status asthmaticus, or bronchospasm with retained secretions in patients with underlying chronic airway disease. The arterial blood gas analysis will reveal hypoxemia with hypercarbia and a widened alveolar-arterial oxygen tension gradient. The functional residual capacity is elevated and, in many patients, there is actually an increase in pulmonary compliance. The gas exchange abnormality results from ventilation-perfusion mismatching. Patients whose respiratory failure results from an acute loss of pulmonary compliance are those with an overwhelming parenchymal insult such as occurs with extensive bacterial pneumonia, aspiration, or a systemic process such as pancreatitis. These disorders may be grouped under the heading of adult respiratory distress syndrome. The physiologic alterations are hypoxemia with hypocarbia and respiratory alkalosis because of right-to-left shunting of blood, a fall in the pulmonary compliance, and a marked reduction in the functional residual capacity.

This is a review of respiratory therapy modalities as they are currently used at the Medical College of Virginia.

Correspondence and reprint requests to Dr. James A.L. Mathers, Jr., Box 93, Medical College of Virginia, Richmond, VA 23298.

Hypoxemic Hypercapneic Respiratory Failure with Normal A-a O₂ Gradient

The Respiratory Therapy Department of an institution becomes involved with these patients when the decision has been made to institute mechanical ventilation. While the initial selection of artificial airways is not in the domain of this department, usually it is the respiratory therapists who are called upon when these airways do not function properly. Because of the experience gained in managing airway problems, we at the Medical College of Virginia have developed a number of recommendations for this aspect of respiratory care. When selecting an artificial airway, one must make an initial decision between "compliant" and "stiff" cuffs. We prefer to use the large volume compliant cuff tubes because a seal is obtained at a lower cuff pressure. Furthermore, the intracuff pressure measured by the respiratory therapist is a more accurate assessment of pressure exerted against the tracheal wall than pressures obtained from stiff cuffs.

While orotracheal tubes may be rapidly inserted during an emergency such as a drug overdose, these tubes are associated with the highest incidence of tracheal damage since they are difficult to stabilize and have a tendency to move about in the trachea when the patient is turned, moves or coughs. Nasotracheal tubes are more stable and, when a compliant cuffed tube is placed via this route, they may be left in place for as long as two to three weeks with a minimum of tracheal damage. The cross-sectional diameter of the nasotracheal tube is smaller than the orotracheal tube. The combination of longer length and the narrow diameter may make adequate suctioning difficult. Should the management of secretions be a problem and a prolonged intubation anticipated, elective tracheostomy offers many advantages such as the wide variety of tubes available, including talking tracheostomy tubes, fenestrated tubes, and cuffless tubes; patients may be fed via the oral route; vocal cords are not damaged; and there is no damage to the nasal septum. With the advent of the compliant cuff and better management of the artificial airway, the incidence of tracheal damage has decreased significantly. It does occur from time to time, however, and we stress the importance of periodic monitoring of intracuff pressure.

When the decision has been made to intubate patients with this form of respiratory failure, and the appropriate airway has been selected, mechanical ventilation is usually instituted with relative ease because of the normal physiology of the underlying lung.

Hypoxemic Hypercapneic Respiratory Failure with Abnormal A-a O₂ Gradient

This disturbance is most frequently found in patients with increased airway resistance due to bronchospasm, obstructing secretions, and mucosal swelling and edema. The immediate approach to the hypoxemic patient is to stabilize the arterial oxygen tension while treating the underlying defect in hopes of avoiding intubation. The goal is to maintain the Pao₂ in the range of 60 to 80 mm Hg which will insure hemoglobin saturation in excess of 90%. Further increases in the arterial oxygen tension do not significantly increase oxygen delivery to the tissues. The mode of oxygen administration is of great importance in the presence of hypercaphia. Oxygen therapy devices are divided into high-flow or low-flow systems. A high-flow apparatus mixes oxygen and room air prior to delivery to the patient. The gas flow delivered by the device is sufficient to provide the patient's total minute ventilation. Low-flow systems do not provide enough gas flow to supply the entire inspired atmosphere, and some part of the tidal volume will be supplied by the entrainment of room air. Such systems may provide any concentration of oxygen from 21% to 95%, but the fraction of inspired oxygen (F_1O_2) can be altered by the patient's respiratory pattern. Low-flow devices include the nasal cannula and mask oxygen and may be used when the tidal volume of the patient is between 300 and 700 cc, the ventilatory rate is less than 30/min, and there is relatively stable ventilatory pattern with time. Because of the potential danger of excess oxygen administration, we prefer to keep the F_1O_2 as low as is consistent with adequate oxygen delivery to the tissues. The unpredictability of the tidal volume and minute ventilation in an acutely ill patient are reasons why we generally recommend high-flow oxygen systems; the most commonly used of these are masks that work on the Venturi principle. The standard inspired O₂ fractions available are 24%, 28%, 35%, and 40%. Humidifiers are not required with these masks because the major portion of inspired gas is room air. To supply a high flow of oxygen in excess of 40%, special systems can be arranged by the Respiratory Therapy Department.

The importance of the mucociliary escalator in maintaining airway homeostasis is often underestimated. If the mucous blanket becomes dry or abnormally thick, secretions will not mobilize normally and cough will become ineffective, leading to dramatic increases in airway resistance. The most effective means of reestablishing the mucous blanket is to hydrate the tracheobronchial tree. Water may be delivered to the airway in molecular form as humidification or in particulate form as an aerosol. Simple bubble-type humidifiers are sufficient for patients with an intact upper airway. Should the nasopharynx be bypassed by an artificial airway, a heater is added to the humidifier to further increase water content. If one wishes to deliver particulate water or medications to aid bronchial hygiene, a nebulizing device should be used. Ultrasonic nebulizers produce a very heavy water mist with uniform particle size on the order of $.5\mu$ to 3μ . Jet nebulizers, which operate on the Bernoulli principle, may be used for aerosolization of both water and medications. Droplets are propelled against baffles which eliminate the larger and heavier particles since droplets of greater than 10μ in size are clinically useless as they do not deposit in the tracheobronchial tree. Aerosolized medications include mucolytics, bronchodilators, vasoconstrictors, and steroids. In addition to instillation of water into the airway, efforts have been made to change the viscosity of the secretions by nebulizing sodium bicarbonate or acetylcysteine. Usually these agents are no better at mobilizing dried secretions than good hydration; however, in circumstances where the mucous is abnormal, such as cystic fibrosis, they may be useful. They may also be successfully used in those patients where active oral or intravenous hydration is not practical or safe. Inhaled bronchodilators exert their effect following absorption into the circulation. Absorption from the pulmonary system is rapid and the degree of bronchodilation obtained will be equal to that of an intravenous dose with similar blood levels. Vasoconstrictors may be administered by aerosol primarily for their topical properties. Many acute pulmonary diseases are accompanied by an inflammatory reaction of the respiratory mucosa which includes profound capillary congestion and swelling. Topical application of a vasoconstrictor may have dramatic effects in decreasing the hyperemia and increasing lumen size. Racemic epinephrine is a very effective topical vasoconstrictor and a mild systemic bronchodilator. It may be of significant benefit as adjunctive therapy to intravenous theophylline. Choice of nebulizing devices for any of these medications should be made by assessing the patient's clinical status and the ability of the patient to

generate adequate tidal volumes. We prefer that a hand-held nebulizer be used since these devices are inexpensive and are powered from a readily available compressed air source. There is evidence of improved distribution of particles when spontaneous inspiration is used with these nebulizers in contrast to medication delivered via intermittent positive pressure breathing (IPPB). These attempts at reducing airway resistance may be applied in conjunction with "expansion" therapy. Incentive spirometry appears to be superior to IPPB for preventing and treating atelectasis.

Despite the above measures (controlled oxygen administration, aerosolized medications, aggressive management of secretions, and expansion therapy) a number of patients with acute respiratory failure due to increased airway resistance will require intubation and mechanical ventilation. A volume-cycled ventilator is the modality of choice in acute respiratory failure. We follow the usual guidelines of 15 cc tidal volume/kg of body weight, respiratory rate of 15 times per minute and F_1O_2 of 40% for initial setting. Twenty minutes to a half hour after beginning mechanical ventilation, the arterial blood gases are repeated and adjustments are made as necessary. Because the basic problem with these patients is varying degrees of airway resistance in different areas of the lung, it is advisable to use as low an inspiratory flow rate as is consistent with the patient's ventilatory pattern. This promotes gas delivery to poorly ventilated regions rather than over-expansion of well-ventilated regions. It is also important to provide for sufficient expiratory time. If too short a period is allowed for expiration, hyperinflation of the patient's lungs because of gas trapping may result. A most useful recent addition to our therapeutic techniques is intermittent mandatory ventilation (IMV). A circuit of constant gas flow is added in parallel to the patient's ventilator allowing the patient to breathe spontaneously at his or her own tidal volume between pre-set breaths delivered by the volume ventilator. This system minimizes the possibility of excessive mechanical ventilation and allows better acid-base control. As the patient's clinical condition improves, the respirator rate may be gradually reduced, promoting a smoother weaning period. Complications that may develop in patients on ventilators include atelectasis in poorly ventilated areas, pneumothorax, superinfection, or drying of secretions with mucous plugging if the inspired atmosphere is not properly humidified.

Hypoxemic Hypocapneic Respiratory Failure

This blood gas pattern is found in syndromes in which the pulmonary parenchyma has been damaged. The severity of the condition will be apparent when there is a minimal increase in arterial oxygen tension in response to an F_1O_2 of .4 or above. This documents the presence of right-to-left shunting of blood. The reduction of parenchymal compliance in these patients makes the selection of tidal volumes more complex than in those with obstructive problems. The pressure volume curve of the respiratory system may be determined at the time the patient is placed on mechanical ventilation. We are often able to identify a point in the curve above which a slight increase in tidal volume produces a dramatic increase in intrathoracic pressure. This may occur at a tidal volume as low as .7 liters. If one arbitrarily applies the rule of 15 cc/kg to these patients, the thoracic capacity may be exceeded and there will be an inappropriately high intrathoracic pressure relative to the effective tidal volume. An appropriate minute ventilation may be delivered by reducing the tidal volume and increasing the rate of ventilation. This will reduce mean intrathoracic pressure and reduce the incidence of barotrauma.

Most patients in this category who are gravely ill will remain hypoxemic despite the increased F_IO_2 and mechanical ventilation. These modalities do not

reverse the primary defect of reduced Functional Residual Capacity. This problem may be partially corrected by increasing the distending pressure within the thorax. This is done by applying positive end expiratory pressure (PEEP). Complications include a reduction in cardiac output and pneumothorax. It should be noted, however, that the occurrence of pneumothorax is related to the level of *mean* intrathoracic pressure during mechanical ventilation and not to the presence or absence of PEEP.

In summary, respiratory therapy should be thought of as a series of specific steps designed to treat the anatomic and physiologic alterations that occur with the various forms of respiratory failure. The scientific basis of the field is rapidly advancing as more is learned about the mechanisms of pulmonary disease. Respiratory therapy should no longer be equated solely with IPPB but should be regarded as a field encompassing a number of effective treatment modalities.

Suggestions for further reading

- 1. BURTON GG, GEE GN, HODGKIN JE: Respiratory Care: A guide to Clinical Practice. New York, JB Lippincott Company, 1977.
- 2. HEDLEY-WHITE J, BURGESS GE, FEELEY TW, ET AL: Applied Physiology of Respiratory Care. Boston, Little, Brown and Company, 1976.