MEDICAL GRAND ROUNDS
PARKLAND MEMORIAL HOSPITAL
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INTERMITTENT POSITIVE PRESSURE BREATHING

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REFERENCES

HISTORICAL INTEREST

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Although mouth to mouth artificial respiration has been used since biblical times, the first systematic use of intermittent positive pressure breathing was evidently by Vesalius in 1543. IPPB administered by bellows was used clinically in the 18th and 19th centuries, but the French Academy of Sciences declared in 1829 that the practice was dangerous, and the whole idea was dropped until the beginning of the 20th century. In 1904 Saurbruch introduced an entirely different principle of ventilation; this method was the administration of a gas under a continuous positive pressure. Although many investigators evidently realized that ventilation of an apneic patient could not be sustained by this technique, it continued to be used extensively until the 1940's. In 1929 Drinker introduced the tank respirator for the ventilation of apneic and paralyzed patients.

Continuing work by Swedish investigators headed by Giertz finally reintroduced intermittent positive pressure applied at the mouth during inspiration, and in 1938 Crafoord convincingly demonstrated the superiority of IPPB over continuous pressure breathing in the apneic patient. In the same year, however, Barach popularized in this country the inhalation of oxygen under continuous positive pressure (CPB) for the treatment of acute pulmonary edema and obstructive dyspnea. CPB assisted the patient during inspiration, but it caused a great increase in the work of expiration. The most striking deleterious effect, however, related to a decreased cardiac output tending to cause hypotension. In an attempt to obviate these difficulties, Barach then introduced the concept of intermittent positive pressure during expiration. He felt that this opposed filtration of edema into alveoli in cases of circulatory failure, and maintained airway patency in patients with obstructive lung disorders. However, prolonged use of expiratory positive pressure also decreased the cardiac output.

The impetus of military aviation during World War II caused extensive investigation of pressure breathing, and much of the basic physiology was defined by Barach, Richards, Cournand, Otis, Fenn, Rahn, and their various coworkers. Most clinical indications for intermittent positive pressure during inspiration (IPPB/I) were not delineated until the 1950's.

METHODS OF ADMINISTRATION

7. Mushin, William Woolf, Rendell-Baker, L. and Mapleson, W. W.: Automatic ventilation of the lungs. Charles C. Thomas, 1959.

This excellent book describes the mechanism of action of a very large number of ventilators. Unfortunately, it does not include the Bennett TV-2P, PR1 or PR2 or any more recent Bird Respirator than the Mark VII, which are the most common respirators used in this country. Nevertheless, the large number of respirators described give one a broad working knowledge of the principles involved.

The authors categorize ventilators by the mechanism the change-over from the inspiratory phase to the expiratory phase. This change-over may be: 1. Time cycled, occurring after a certain period of time. 2. Pressure-cycled, occurring when a pressure which is closely related, but not necessarily equal to that in the lungs reaches some critical value. 3. Volume cycled, occurring when a certain volume has been expelled from some chamber. 4. Mixed cycled. Examples of machines used frequently in this country include the Bennett PR1 and PR2 when used on automatic and the Air Shields in type 1; all Bennetts when not used on automatic, all Birds, and DOTCO in type 2; Emerson, Engstrom and Morch in type 3; and the Bennett volume ventilator in type 4.

PHYSIOLOGICAL EFFECTS

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There is a tendency to equate the negative intratank pressure of a body respirator to negative pleural pressure and hence regard this type of ventilation as being physiologically normal. This is clearly not correct, however, and the physiological effects of tank respirators are the same as the effects of positive pressure respirators.

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positive pressure breathing on the intrapulmonary distribution of inspired air. Am. J. Med. 29:946, 1960.

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The following section from the paper by Ayres, Kozam and Lukas (17) summarizes many of the important points concerning the pulmonary mechanics of positive pressure breathing:

"Intrathoracic pressure during intermittent positive pressure breathing cannot be predicted from the magnitude of changes in mask pressure because of its marked dependence on the degree of passivity of the subject. During inspiration this pressure may vary from a level more negative than normal to one more positive than the end-expiratory point throughout inspiration and expiration. The degree of positivity of intrathoracic pressure is not only related to subject relaxation, pressure, and flow rate, but also to the physical characteristics of the airways, lungs, and chest bellows. For any given setting of mask pressure, intrathoracic pressure is inversely related to chest wall compliance, airway resistance, and rate of air flow; in contrast, intrathoracic pressure varies directly with pulmonary compliance.

"Similarly, the amount of respiratory work performed by the subject while on IPPB is variable. If he actively leads the apparatus, he may perform more work in attaining a given tidal volume than he does in breathing spontaneously. This may be one of the factors responsible for the intolerance to the apparatus manifested by poorly instructed or anxious patients. In the completely relaxed subject with normal or diseased lungs, the active work of breathing approaches zero. In the patient with obstructive disease, all of the inspiratory work may be performed by the respirator and a considerable fraction of the active expiratory work may be eliminated."

The distribution of the inspired gas to the various pulmonary segments is probably dependent on the size of the tidal volume whether the tidal volume is due to spontaneous respiration or IPPB. Compliance (the amount of air by which the lung is distended for a change in pressure applied, Liters of air/cm. H20, and hence a measurement of the stiffness of the lung) during spontaneous breathing or IPPB is in part dependent on the rate of flow into the lungs; with slow flows compliance is probably the same with the two types of ventilation.

The work of Jones, MacNamara and Gaensler (13) is said to show that IPPB markedly

aggrevates expiratory resistance in patients with emphysema and increases the work of breathing when high pressures are used. The experimental model, however, does not simulate chronic obstructive lung disease in a reasonable way. The results obtained were due to this fallacious model, and there is no reasonable application of their results to clinical medicine.

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The circulatory effects of IPPB depend on the change of mean intrathoracic pressure from the negative pressure existing during voluntary respiration to the positive pressure existing during IPPB. In one of the classic papers on IPPB Cournand, Motley, Werko and Richards (24) demonstrated that the rise in mean intrathoracic pressure depends on the pattern by which the positive pressure is applied as well as the peak inflating pressure. To minimize the circulatory effects of IPPB the peak pressure should be as low as possible to achieve the desired ventilation, the mask pressure should fall as rapidly as possible to atmospheric pressure at the end of inspiration, and time of expiration should be as long as possible.

The increased mean intrathoracic pressure decreases venous return to the heart which causes a shift of blood from the thorax to the abdomen and extremities; this has been estimated to be 500 ml when IPPB is applied at 30 cm H20 pressure. In addition there is vasoconstriction. These changes result in an increased peripheral venous pressure which re-establishes the pressure gradient between peripheral and central veins and re-establishes the venous return to the heart. The increased venous pressure causes some transudation of fluid to the extravascular compartment, but the total drop in intravascular volume is insignificant. The net result of the entire process depends on the magnitude of rise of mean intrathoracic pressure, but in most normal subjects the cardiac output does not fall. In 88% of the patients with chronic lung disease the cardiac output does fall, but the decrease is usually insignificant (31). The blood pressure doesn't fall even if the cardiac output decreases because of the systemic vasoconstriction. Factors that tend to cause significant decreases in cardiac output and blood pressure are hypovolemia (particularly hemorrhagic shock) and paralysis of the nerves mediating peripheral vasoconstriction as in anesthesia. Clinically, hypotension is a very infrequent problem in the use of IPPB; more commonly it is the only way to oxygenate the patient and maintain the blood pressure.

It is only possible to speculate on the alterations in the dye dilution curves noted by Feinsilver to be caused by IPPB (30). However, these abnormally shaped curves were not noted by Cathcart, et al, (31) using the same technique. One man's fact is another's artifact.

CLINICAL STUDIES

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It has been well established that mechanical control of respiration by some form of IPPB maybe life saving in a variety of pulmonary problems whose common denominator is a stiff lung from bloody pulmonary edema and atelectasis. Such problems include patients with chest injuries including flail chest, post pulmonary resection, and post open heart surgery. In such patients controlled ventilation by means of a tracheostomy or naso-tracheal tube improves oxygenation, improves alveolar ventilation with an improvement in respiratory acidosis, decreases the work of breathing which improves metabolic acidosis, frequently improves cardiac output which further improves tissue perfusion and decreases metabolic acidosis.

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This study clearly demonstrates the usefullness of IPPB in the treatment of pulmonary edema.

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Becker, et al. (44) and Sands, et al. (45) report that routine post op IPPB is not

beneficial in preventing atelectasis, while Anderson, et al, (47) report that IPPB decreased the incidence of post op pulmonary complications from 19.5% to 2.5%. None of the studies adequately randomized all of the variables known to affect the incidence of post op atelectasis such as smoking, age, pre-existing lung disease, obesity, site of operation, length of operation, extent of mechanical sighing during anesthesia, narcotic dose post op, etc. Moreover, there is no indication as to the effectiveness of the inhalation therapy methods. The one difference that stands out between the author who got the good results and those that did not was that the former administered IPPB preop as well as post op. The only likely benefit of IPPB in these patients is the administration of deep breaths. It is unlikely that a post op patient already in pain and under the influence of narcotics would respond to IPPB by relaxing and allowing the machine to passively inflate his lungs unless he was already trained in its use. Thus, it is likely that the results are compatible. One would conclude that IPPB is probably beneficial in post op cases if the patient has been adequately instructed in its use preoperatively.

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No one disagrees with the use of assisted ventilation in patients with severe pulmonary disease and respiratory failure, but there is considerable disagreement about using IPPB to administer bronchodilators to chronic, "steady state" patients. It seems reasonable to believe that the effects of inhaled bronchodilators in patients with obstructive lung disease are maximal when the drugs are distributed widely throughout the lungs. Widespread distribution depends in part on the size of the tidal volume in which the drugs are contained. IPPB administered bronchodilators should be superior to bronchodilators administered by simple nebulization when IPPB augments the tidal volume during treatments. The physician must estimate each patient's physical capacity for deep breathing as well as the probability of the patient's performing such deep breathing during treatments on a continuing basis and weigh against this an appraisal of the patient's ability to consistently relax with IPPB allowing the machine to cause large tidal volumes. If IPPB tidal volumes are consistently larger than voluntary tidal volumes during treatment, IPPB is indicated for bronchodilator administration.

The article by Kamat, Dulfano and Segal (63) should be especially cited, since it is so frequently quoted as a condemnation of IPPB. These authors administered treatments to patients with severe chronic obstructive pulmonary disease at 5-10 cm H_2O mask pressure. Moderate increases in inspiratory airway resistance would cause such pressure to be inadequate and would lead to the stated results that patients resisted the respirator.

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Some patients with pulmonary failure retain a respiratory drive causing the respiratory rate to be adequate or fast, but each tidal volume is shallow and hence ineffective. It maybe impossible to assist the respiration in such patients with any type of IPPB device. Such patients must either be sedated, as first suggested by Sieker and Hickam (66, 67), or paralyzed to stop ineffectual respiration and allow adequate ventilation by IPPB. This step cannot be undertaken lightly, since the physician must then assume complete responsibility for adequate ventilation. Nevertheless, the need for patient relaxation must be realized to prevent the problems related by some authors (70).

Cullen, Brum and Reidt (68) reported that IPPB would not ventilate some of their patients with emphysema and CO_2 retention; they felt that the machine caused worse hypoventilation. In fact, their patients were breathing on a hypoxic drive which was blunted when their oxygen saturations were corrected by oxygen administered by IPPB. Problems in interpretation of results are common in the literature relating to IPPB.

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Considerable editorializing.

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