Intermittent Positive Pressure Breathing

Ellison C. Pierce, Jr., M.D., and Leroy D. Vandam, M.D.

INTERMITTENT positive pressure breathing (IPPB) or "controlled respiration" was described by Guedel [21] in 1934 after clinical observations on the apnea produced by hyperventilation during deep ether anesthesia. Since then its use during anesthesia has become almost routine, making possible adequate alveolar ventilation during partial or total respiratory paralysis. The development of mechanical devices for artificial respiration with IPPB followed the use during World War II of *continuous* positive pressure respiration as a means of increasing altitude tolerance in flight crews.[4, 45] In 1947, Motley *et al.*,[46] suggested the use of IPPB for certain clinical conditions other than apnea, such as the treatment of acute pulmonary edema and postoperative atelectasis. Since then IPPB has come to be used extensively in many abnormal respiratory conditions in some of which its use seems clearly indicated, perhaps life saving, but in others questionable.

Evaluation of the effects of IPPB produced by mechanical devices calls for considerable understanding of the normal mechanics of breathing, of the physical characteristics of automatic ventilators, and of the pressure-flow volume relationships in the ventilator and pulmonary systems. The relationships are complicated because of the varied functional characteristics of the many ventilators available and the diversity of pulmonary function in disease. It is not possible in the space allotted to discuss in detail the mechanics of breathing [9, 12, 19, 40, 50] or the physics of automatic ventilators.[15, 16, 48, 68] As this review is written there is in press an all inclusive volume on artificial respiration edited by Whittenberger.[67a] Rather this discussion of IPPB will

Dr. Pierce is Clinical Associate in Anaesthesia, Harvard Medical School, and Junior Associate in Surgery (Anesthesia), Peter Bent Brigham Hospital. Dr. Vandam is Clinical Professor of Anaesthesia, Harvard Medical School, and Director, Division of Anesthesia, Peter Bent Brigham Hospital, Boston, Massachusetts.

be limited to considerations of pertinent physiology and physics and of the practical application and indications for use of automatic ventilators primarily in situations other than during anesthesia. The plan of this discussion is as follows:

Physiological Aspects. Circulation: (1) positive pressure, and (2) negative pressure. Respiration: (1) mechanics of breathing, and (2) ventilation-perfusion relationships.

Characteristics of Automatic Ventilators.

Clinical Applications. Respiratory paralysis: (1) indications, and (2) practical aspects. Diseases of the cardiopulmonary system: (1) acute respiratory failure (acidosis) in chronic pulmonary or cardiopulmonary disease, (2) pulmonary edema, (3) acute bronchial asthma, (4) chronic obstructive lung disease—bronchodilator therapy, and (5) prevention of postoperative respiratory complications.

Physiological Aspects

CIRCULATION

Positive Pressure. A number of papers review the effects of positive pressure breathing on the circulation.[4, 32, 42, 47, 64, 65] It is important to note that the circulatory response may vary, depending upon physical status of the subject and the presence or absence of drugs with cardiovascular actions. In healthy, conscious man, increase in intrathoracic pressure results in elevation of right atrial pressure, impairment of venous return, and a reduction in cardiac output proportional to the mean positive pressure applied. Compensatory peripheral vasoconstriction occurs and systemic venous distensibility is probably reduced.[e] Peripheral venous pressure quickly rises with a consequent restoration of the venous gradient and cardiac output. Changes in systemic blood pressure are absent or minimal. There is a decrease in the volume of blood in the thorax, but, because of the increase in intrathoracic pressure, a net rise in venous

pressure is found despite the decrease in venous return. Compensatory mechanisms may fail to maintain the circulation, however, if mean pressure is excessive, as during application of continuous pressure above 20 cm. of water. When IPPB is properly performed, as will be discussed later, mean pressure should be considerably below peak inspiratory pressure.

Adverse circulatory effects of IPPB occur only rarely in previously fit patients who become apneic or who undergo general anesthesia requiring controlled respiration. They are more likely to occur in the patient with blood volume deficiency, actual or impending heart failure, or sympathetic blockade produced by antihypertensive drugs, spinal anesthesia, or surgical sympathectomy. They are most likely to occur if several of these factors are present.[47, 51, 65] When hypotension ensues it is generally proportional to the mean pressure applied and the degree of impairment of compensatory mechanisms.

Cournand et al.,[13] early realized the importance of the pattern of pressure applied. The pressure of significance that determines the degree of circulatory embarrassment is the mean pressure, defined by Mushin et al.,[47] as "not the arithmetical mean between the highest and lowest pressures in the respiratory cycle," but ". . . the mean of an infinite number of instantaneous readings of the pressure within the lung during one respiratory cycle." Thus five different pressure patterns can be obtained, all with the same peak pressure, rate of respiration, and the same tidal volume, but with mean pressures varying from -0.2 to $+7.5$ cm. of water (fig. 1). The following factors are conducive to the production of a low mean pressure:

(1) Inspiratory time equal to, or preferably shorter than, expiratory time.

(2) After attainment of peak pressure or volume of inflation, immediate release of positive pressure to avoid a "plateau."

(3) Rapid return to atmospheric pressure upon completion of inspiration by avoidance of expiratory resistance which causes positive pressure during expiration.

(4) Flow rates—a rapid flow rate (above 40 liters/minute) permits a short inspiration in normal individuals. In patients with obstruc-

tive lung disease the required low flow rates lengthen inspiration.

(5) Use of negative pressure during expiration.

Negative Pressure. Kilburn and Sieker [29] and Bader and Bader,[3] as a theoretical exercise, have recently reviewed the physiology of continuous positive and continuous negative pressure breathing, including effects on the kidney and blood volume. In the present discussion, however, consideration of negative pressures is limited to intermittent positive-negative pressure breathing, that is the addition to IPPB of a negative pressure during expiration. Despite the fact that expiratory negative pressure can increase venous return to the heart [25] and lower the peak positive pressure required for adequate ventilation,[47] the mean mask pressure usually remains above the atmospheric.[65] Negative pressure is recommended for use in patients requiring IPPB who present with circulatory disturbances.[35] Mushin, et al.,[47] describe the theoretical advantages of a negative pressure phase but also emphasize the disadvantages in patients with pulmonary emphysema or other obstructive lung disease because of the tendency to collapse the smaller air passages and hinder expiration. In ordinary usage it is seldom necessary to employ negative pressure during expiration. Instead, emphasis should be placed on the other factors contributing to maintenance of a low mean positive pressure and on restoration of cardiovascular adequacy with blood volume restitution and sympathomimetic drugs.

RESPIRATION

Mechanics of Breathing. Evaluation of the pressure-volume effects of IPPB [47, 53, 59, 65] calls for an understanding of the normal mechanics of breathing.[9, 12, 19, 40, 50]

During breathing the respiratory muscles supply the force (pressure) to overcome the elastic and nonelastic "resistances" offered by the lungs and thorax. During IPPB these pressures are applied externally. The elastic "cost" in a normal adult male at ordinary midposition is about 10 cm. of water per liter of volume change, divided nearly equally between lungs and thorax so that the change in pleural pressure is about 5 cm. of water per liter.
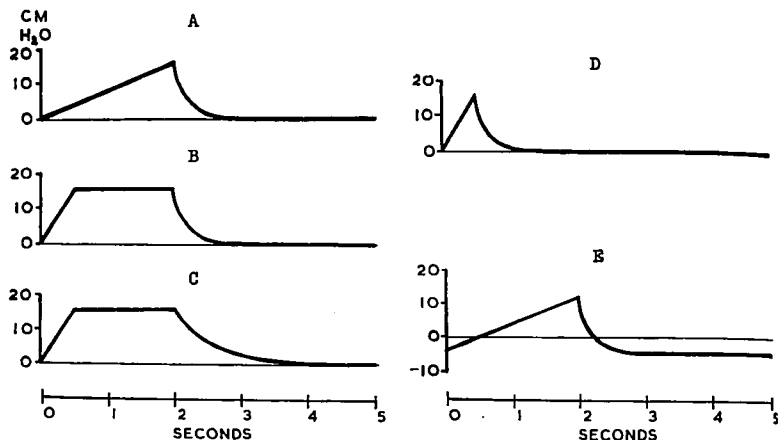
FIG. 1. Diagrams illustrating the meaning of the term 'mean pressure in the lungs' during artificial ventilation. In each case the total respiratory cycle lasts 5 seconds; the tidal volume is 800 ml. and the compliance is 0.05 liter/cm. of water. Hence the pressure range is 16 cm. of water. The airway resistance is taken as 2 cm. of water/(liter/second). However, possible changes in other variables produce a wide range of mean pressures.

A. Inflation is slow (flow rate of inflating gases is 24 liters/minute). Expiration is 'free.' The resistance of the apparatus is taken as 2 cm. of water/(liter/second). The mean pressure is 3.8 cm. of water.

B. Inflation occurs rapidly (flow rate of inflating gases is 96 liters/minute) at the beginning of the inspiratory phase. The lungs are then held inflated for some time so that the inspiratory phase lasts for 2 seconds, as in figure 1A. Expiration is again 'free.' The mean pressure is 6.2 cm. of water.

C. The inspiratory phase follows the same course as in spontaneous respiration, but expiration is impeded by an apparatus resistance of 10 cm. of water/(liter/second). The mean pressure is 7.5 cm. of water.

D. Inflation is rapid (flow rate of inflating gases is 96 liters/minute) and is followed immediately by the 'free' expiration. The mean pressure is 1.4 cm. of water.

E. Inflation is slow (flow rate of inflating gases is 24 liters/minute) and again occupies 2 seconds. During the expiratory phase a steady negative pressure (−4 cm. of water) is introduced. Expiration is 'free.' The mean pressure is −0.2 cm. of water.

(Mushin, W. W., Rendell-Baker, L., and Thompson, P. W.: Automatic Ventilation of the Lungs. Oxford, Blackwell Scientific Publications, 1959.)

(The compliance of the total respiratory system, which is the inverse of the *elastic resistance* or *elastance* is in this case 0.1 liter/cm. of water and of the lungs and thorax about 0.2 liter/cm. of water each.) The nonelastic resistance is principally that offered by the airways to the flow of gas. This amounts to about 2 cm. of water at a flow of one liter per second.

In quiet inspiration with a tidal volume of 0.6 liter and a maximum flow of about 0.5 liter/second, the elastic pressure swing would be 6 cm. of water and the flow-resistive pressure swing, 1 cm. of water. The total pressure swing would not be simply the sum of these, but rather would be somewhat less, because the flow and volume maxima do not correspond in time: maximal flow occurs at approximately mid-inspiration, and the maximum of the sum of elastic and flow-resistive pressures occurs at some point beyond that volume but before the end of inspiration when the elastic-pressure is maximal. In quiet expiration the "stored" elastic pressure is more than sufficient to overcome nonelastic resistance and to complete expiration in time, and the only energy expended normally during expiration is by inspiratory muscles as they "let up"

and allow the elastic forces to perform. During the complete breathing cycle the muscles accomplish the equivalent of a pressure swing of something between 6 and 7 cm. of water in this example, and the same result would be accomplished in the absence of muscle contraction by raising mask pressure from atmospheric by the same amount and with the same timing.

Values for compliance and airway resistance vary not only in normals but considerably in patients with pulmonary disease, the importance of which cannot be overemphasized. For example, in severe bronchial asthma airway resistance may increase to 18 cm. of water/liter/second or more,[1, 63] which, at a mid-volume flow rate of 30 liters/minute (0.5 liter/second), would result in a pressure difference of 9 cm. of water between peak mask and alveolar pressures. If compliance were the same as in the simplified pressure-volume relationships cited above, this increase in airway resistance would require a peak mask pressure of about 15 cm. of water to provide the same tidal volume. Moreover, depending upon accumulation of secretions, alteration of body position, administration of drugs, and upon several other factors, compliance and airway resistance may change from moment to moment. This is especially true in anesthetized patients or those with pulmonary disease.[16, 40, 47] Spencer[59] has discussed the contribution of the several variables. For example, increase in the cycling rate of a respirator may reduce the volume of air moved for a given peak pressure for these reasons: the duration of mean applied pressure is reduced and causes an increase in the pressure necessary to overcome airway resistance.

Ventilation-Perfusion Relationships. Fowler,[17] and Bouhuys and Lundin[7] have examined ventilation blood flow relationships in general but data are meager concerning these factors during IPPB. In 1957 Spencer[59] stated, "It is reasonable to assume that there are alterations in the spatial distribution of air introduced into the lungs by applied pressure on the one hand, or by diaphragmatic action on the other," but he also noted, "that these considerations may be of more theoretical than practical importance is suggested by Whittenberger. . . ."[65] The problem has re-

cently been investigated by Campbell, Nunn, and Peckett[11] who noted that earlier reports had suggested the need for greater degrees of ventilation to maintain acid-base homeostasis and that distribution of air is abnormal during IPPB.[10, 24, 34] On the whole there is little evidence that air distribution is abnormal but much evidence[16a] that alterations in the ventilation-perfusion ratio are largely due to changes in perfusion. Campbell's data[11] suggest that IPPB, during anesthesia, produces a pattern of inflation different from that produced by the respiratory muscles and that various abnormal ventilation-blood flow patterns may occur. Three and possibly another of the six subjects studied showed an increased "dead space effect" (relative overventilation of parts of the lung with poor blood flow), and one or two subjects demonstrated a "shunt effect" (continued perfusion of parts of the lung with decreased ventilation).

If this be the case in normals, then in patients with pulmonary disease who undergo anesthesia and operation with the addition of an open thorax it is possible that maldistribution of circulation and possibly ventilation may be great enough to be of clinical significance, resulting in development of respiratory acidosis even though external (alveolar) ventilation is normal. Hypoxemia is less likely to occur simply because IPPB is usually carried out with greater than normal oxygen concentrations. Ventilation with ambient air would, of course, result in concurrent hypoxemia. Certainly investigation of ventilation-perfusion relationships under these circumstances needs to be performed.

Characteristics of Automatic Ventilators

The physical characteristics of respirators have been discussed elsewhere in this symposium. It is convenient, however, at this point to consider briefly a few fundamental principles of automatic ventilators in order better to comprehend their clinical application. The following description is abridged from Mapleson.[48]

The majority of IPPB devices may be classified either as flow generators or pressure generators. Flow generators deliver either a constant flow rate throughout inspiration (fig. 2) or a preset pattern of flow rate (fig. 3).
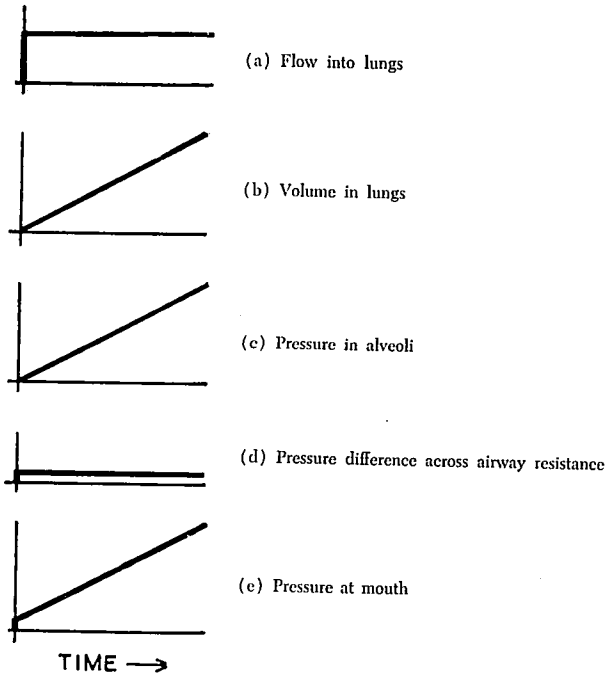
FIG. 2. Inspiratory phase: flow, volume, and pressure patterns with a constant-flow generator. (Mushin, W. W., Rendell-Baker, L., and Thompson, P. W.: Automatic Ventilation of the Lungs. Oxford, Blackwell Scientific Publications, 1959.)

An example of the latter is to be found in the sine-wave flow pattern, a gradually increasing flow, to a maximum rate near mid-inspiration followed by a gradual decrease to end-inspiration. In either case the volume and pressure in the lungs increase continuously throughout inspiration. The rate at which pressure at the mouth increases depends upon the flow rate and airway resistance and also on the other components of mechanical impedance.

Pressure generators, on the other hand, deliver a constant pressure throughout inspiration. Depending, however, on the magnitude of *source* pressure (within the device) and the apparatus resistance (resistance developed within the apparatus between the source and the patient), different patterns of flow rate, volume and lung pressure may result. Two examples of this are as follows:

(1) Relatively low source pressure and low apparatus resistance produce a high initial flow rate followed by steadily decreasing rate approaching zero, with a coincident rapid increase in lung volume and rapid rise in mouth and alveolar pressure (fig. 4).

(2) Relatively high source pressure and high apparatus resistance produce a moderate initial flow rate followed by only a slight decrease in flow rate, with a nearly linear rise in lung volume and mouth and lung pressure (fig. 5).

The characteristics of ventilators can be further subdivided according to whether the end-inspiration is time-cycled (T), pressure-cycled (mouth pressure) (P), or volume-cycled (V). Other details include expiratory characteristics and mode of cycling at end-expiration. The most complex respirators at-

tempt to combine most of these variables. Several of the ventilators commonly used in the United States are listed in table 1 according to the classification of Mapleson.[48]

From this brief description of ventilator characteristics it is apparent that the many variables amongst them may readily play a roll in the conflicting results of studies that will be cited concerning the effectiveness of IPPB. Obviously persons employing IPPB devices must learn to think in terms of flow rate and pressure characteristics as they affect alveolar ventilation.

Clinical Applications

RESPIRATORY PARALYSIS

The following conditions classified by Comroe[12] are included in this category: (1) respiratory center depression; (2) deficiency in

TABLE 1. Classification of Commonly Used
Ventilators According to Mapleson's
Criteria (see text).

A. Flow generators

   1. Constant flow
      a. Bird, without air entrainment (P)

   2. Sine-wave flow
      a. Engström (T)

B. Pressure generators

   1. Low pressure source, low apparatus
      resistance
      a. Bennett (TP)
      b. Burns (Pneophore) (P)

   2. High pressure source, high apparatus
      resistance
      a. Bird, with air entrainment (P)
      b. Emerson (P)

P = Pressure end-inspiratory cycled.
T = Time end-inspiratory cycled.

(a) Flow into lungs

(b) Volume in lungs

(c) Pressure in alveoli

(d) Pressure difference across airway resistance
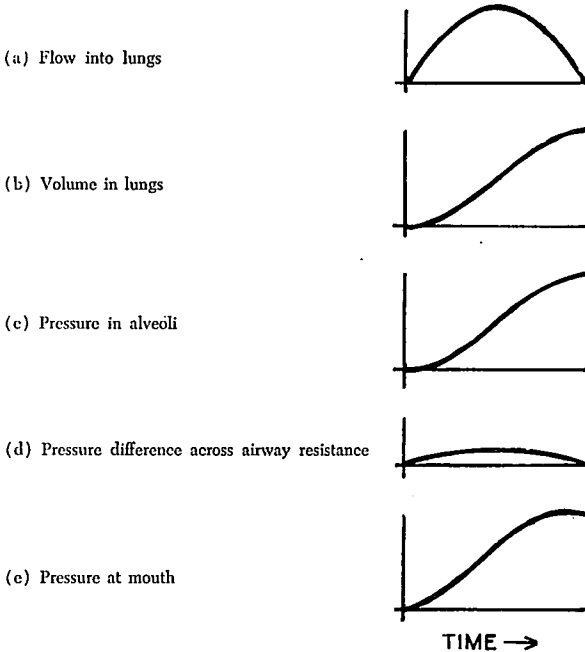
(e) Pressure at mouth

TIME →

FIG. 3. Inspiratory phase: flow, volume and pressure patterns with a sine-wave-flow generator. (Mushin, W. W., Rendell-Baker, L., and Thompson, P. W.: Automatic Ventilation of the Lungs. Oxford, Blackwell Scientific Publications, 1959.)

(a) Pressure generated by ventilator

(b) Volume in lungs

(c) Pressure in alveoli

(d) Pressure difference across sum of airway and apparatus resistance

(e) Flow into lungs
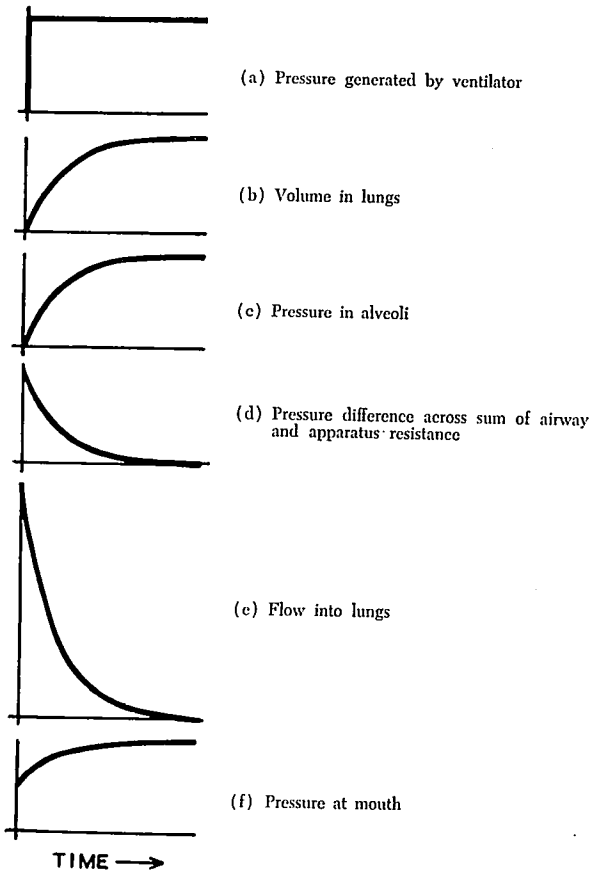
(f) Pressure at mouth

TIME →

FIG. 4. Inspiratory phase: flow, volume, and pressure patterns with a constant-pressure generator, with apparatus resistance equal to patient's airway resistance. (Mushin, W. W., Rendell-Baker, L., and Thompson, P. W.: Automatic Ventilation of the Lungs. Oxford, Blackwell Scientific Publications, 1959.)

neural conduction or neuromuscular transmission; (3) muscular diseases; (4) limitation of movement of thorax.

Indications. It is unfortunate that many otherwise well-trained physicians are deficient in a knowledge of how and when to perform respiratory resuscitation and that this deficiency is compounded when efforts are made to employ IPPB devices for this purpose. The use of automatic ventilators to provide artificial respiration during periods of respiratory paralysis is not questioned. Their use, however, to initiate ventilation in acute respiratory arrest is certainly unnecessary. Mouth-to-mouth (or mouth-to-nose) respiration is the method of choice for the inexperienced.[11] Use of a reservoir bag and mask is likewise satisfactory but requires greater skill in order

to maintain a mask fit and a patent airway. An automatic ventilator should not be employed until adequate pulmonary exchange is assured by one of the simpler methods. The reasons for this are obvious. First, it is necessary to make several adjustments on a ventilator before ventilatory requirements for the patient can be met. During this time ventilation will continue to be inadequate resulting in progression of hypoxia and hypercarbia. Secondly, to be effective, automatic ventilators require an air-tight fit and an un-

obstructed airway. By and large these can be assured only with a cuffed tracheostomy or endotracheal tube. Intubation, however, is secondary to immediate restoration of alveolar ventilation. Thirdly, persons who lack experience in respiratory resuscitation have difficulty enough in maintaining satisfactory ventilation with simple methods and are more likely to fail if attention to airway and chest movement be compromised by efforts to operate a ventilator. Whenever the situation is under control, however, attention may

(a) Pressure generated by ventilator

(b) Volume in lungs

(c) Pressure in alveoli

(d) Pressure difference across sum of airway and apparatus resistance

(e) Flow into lungs
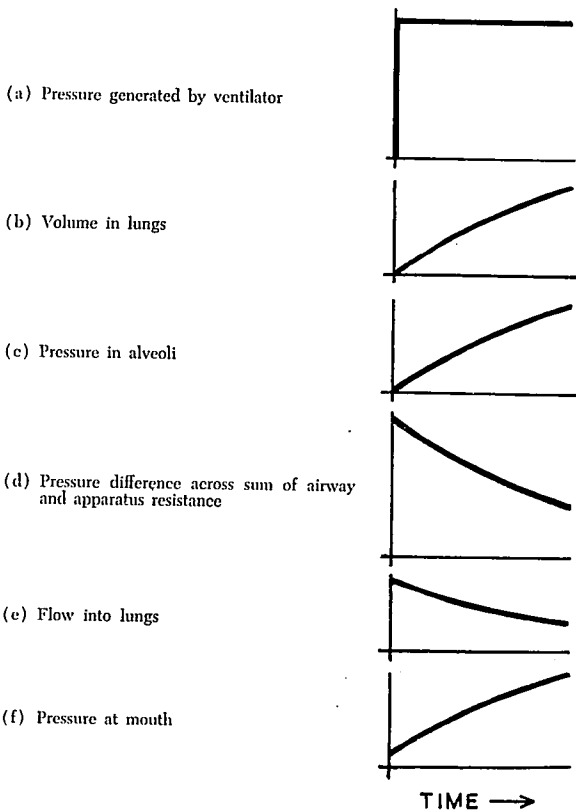
(f) Pressure at mouth

TIME ⟶

FIG. 5. Inspiratory phase: flow, volume and pressure patterns with a constant-pressure generator, with apparatus resistance equal to nine times the patient's airway resistance. (Mushin, W. W., Rendell-Baker, L., and Thompson, P. W.: Automatic Ventilation of the Lungs. Oxford, Blackwell Scientific Publications, 1959.)

be directed toward changing from the initial resuscitative method to IPPB. It is beyond the scope of this article to review further the physiology and treatment of respiratory failure, detailed discussions of which may be found elsewhere.[6, 14, 47, 52, 53, 65, 66, 67]

Practical Aspects. The adequacy of ventilation during IPPB for respiratory depression cannot be judged by the manometric readings on an IPP device. As previously noted, increased airway resistance due to bronchiolar spasm, mucus, or other obstruction will result in a large gradient between mask and intraalveolar pressures.

The adequacy of ventilation in patients with normal lungs may be determined grossly by astute observation of chest wall movement or by measurement of tidal or minute ventilation. It should be realized that tidal volume obtained may be much less than the measured gas volume delivered from a ventilator containing distensible components such as bellows or connecting tubes.[55] When a respirator is used during anesthesia in the circuit of a carbon dioxide absorption system, measurement of tidal volume may be inaccurate because of the addition of a constant flow of fresh gases to the system. Moreover, in the use of any respirator the development of a gas leak between the machine and the alveoli may result in inadequate ventilation. For these reasons any of several ventilation meters, now clinically applicable for measurement of tidal and minute ventilation, are more certain of providing accurate determinations when used in the expiratory phase of respiration.[47]

Intubation of the trachea or tracheostomy is usually necessary to insure a patent, gas tight airway and to facilitate removal of secretions, especially in patients with pulmonary disease. It has been our experience, as well as that of Hickam,[22] that care of a tracheostomy is frequently improperly performed. Often suctioning is inadequate or is carried out without regard for sterile technique or production of hypoxia. Moreover, air is allowed to leak around the tube because a cuff, if used at all, may not be properly inflated.

Measurement of arterial pH and blood gases, P_{CO_2} and P_{O_2}, (or percentage hemoglobin saturation) is the best method of assaying the adequacy of alveolar ventilation. This is essential in patients requiring assisted or controlled respiration for treatment of pulmonary insufficiency. It must be realized that a patient with depressed respiration who is receiving additional oxygen by nasal catheter, oxygen tent, or mask can maintain normal oxygen saturation while developing severe or even fatal respiratory acidosis.[15, 47, 53, 55] Frumin *et al.*,[18] have recently shown that in apnea the average rate of rise in arterial P_{CO_2} is approximately 3 mm. of mercury per minute.

An apneic patient receiving IPPB requires someone in continual attendance who has an understanding of pulmonary function, artificial respiration, and the apparatus used. This is perhaps the greatest need in long term respiratory resuscitation. Elsewhere in this symposium this aspect will be enlarged upon in the discussion of the Respiratory Care Unit.

DISEASES OF THE CARDIOPULMONARY SYSTEM

In contrast to the clear indications for use of IPPB devices in "restrictive" ventilatory failure its value in the therapy of pulmonary abnormalities remains controversial. Although a thorough understanding of the pathophysiology and overall management of pulmonary and cardiopulmonary disease is essential to evaluation of IPPB as adjunctive therapy, these aspects are again beyond the scope of this article. Moreover the almost total absence of well-controlled data necessitates discussion fo IPPB largely in terms of clinical concepts.

Acute Respiratory Failure (Acidosis) in Chronic Pulmonary or Cardiopulmonary Disease. Sieker and Hickman[58] reviewed the clinical syndrome of carbon dioxide intoxication and noted that it occurs most often in patients with chronic lung disease with or without cor pulmonale who develop further impairment of alveolar ventilation because of one or more acute processes: (1) infection, acute asthma, lung collapse, or thoracic operation; (2) heart failure; (3) respiratory depression from drugs; (4) elimination of hypoxia as a stimulant to breathing. They re-emphasized earlier suggestions[6, 33] of the importance of mechanical respirators in reducing the severe acidosis of carbon dioxide narcosis by improvement of alveolar hypoventilation.

In patients with bronchiolar narrowing and increased airway resistance, a respirator that

delivers a sudden inspiratory blast may be totally ineffective. High rates of flow increase the already elevated airway resistance since resistance is enhanced with increase in flow rate and development of turbulence. With a pressure cycled device this may result in too short an inspiratory phase and an inadequate tidal volume.[40] Respirators with preset slow flow rates or with adjustable flow rates may produce less increase in airway resistance and provide larger tidal volumes.[15, 40]

Mead and colleagues [36] noted the advantages of a slower flow rate in patients with high airway resistance and Hickam *et al.*,[23] examined the use of a respirator that permitted inspiratory flow rate to be altered independently of mask pressure. These studies showed that in patients with high airway resistance and diminished compliance, more effective ventilation was obtained if the mask pressure was high (25–30 cm. of water) and the flow rate low with a range from 10 to 30 liters/minute, depending on degree of airway resistance. In practice the maximum effective ventilation could be obtained by institution at a very low flow rate with a progressive increase until the maximal ventilation was achieved. Beyond that further increase in flow rate led to a diminution in effective ventilation.

More recently Jones, Macnamara, and Gaensler,[27] having witnessed failure of IPPB to correct severe respiratory acidosis, studied the effects of IPPB on simulated pulmonary obstruction in normal man. Under these circumstances expiratory flow resistance increased markedly during IPPB, due to development of high inspiratory alveolar pressure "compounded by machine and premature expiratory effort during insufflation." This resulted in airway closure, greatly increased work of breathing, and little or no improvement in pulmonary aeration. Their data suggest "design of a patient-cycled respirator capable of delivering very high flow rate at pressures which are decreasing rather than increasing during the inspiratory cycle." Gaensler and Lindgren [19] reasoned that IPPB "may not be helpful . . . partly because insufficient time may be allowed for the difficult and unassisted expiratory phase, partly because the transpulmonary pressure differential at the beginning of expiration may be raised even further and

thus accentuate the 'dynamic' expiratory obstruction, and partly because the pressure breathing device may not deliver the required large inspiratory flow rates during the short inspiratory phase." Examination of pressure-flow characteristics of their IPPB device showed sharp reduction in the peak flow rate during inspiration through the obstructed airway. It seems logical to the present reviewers that an IPPB machine with an independently adjustable, *constant* flow rate such as the Bird Mark VII may produce better results under similar circumstances. A thorough coverage of the relationship between pressure, volume, and gas flow in the lungs of normal and diseased human subjects may be found in the review of Fry and Hyatt.[18a]

Most other investigators [30, 44, 69] reporting improvement with IPPB have neglected to measure changes in arterial P_{CO_2} in confirmation of their impressions. One exception is the recent work of Jameson *et al.*[25] In studies, without bronchodilators, using a Bennett device with room air on patients with a low arterial P_{O_2}, a high arterial P_{CO_2}, or both, this group was able to show an increase in arterial oxygen saturation in 50 per cent and decrease in arterial P_{CO_2} in 83 per cent of the patients. With a Pneophore and room air arterial oxygen saturation rose in 70 per cent and P_{CO_2} fell in only 35 per cent of the patients studied. Again .it would be interesting to see if more patients would improve using a device with independent control of flow rate.

In summary then many patients with chronic pulmonary disease who develop moderate to severe respiratory acidosis may undergo improvement in arterial P_{CO_2} and hydrogen ion concentration with IPPB therapy. Others show further deterioration. The complexity of the disease, the role of other therapy directed at the precipitating cause, and the variance among IPPB devices all serve to confuse evaluation of therapy. Certainly tracheostomy should be performed in the severely acidotic. It seems reasonable, however, to give IPPB a thorough trial in all of these patients. It is essential that arterial P_{CO_2} and *p*H values be followed. Some patients who develop adverse cardiovascular reactions from IPPB may require vasopressor therapy and correction of diminished blood volume.

TABLE 2.   Gas Exchange Studies in Patient with Pulmonary Edema*

Arterial Blood	Room Air†	100% O₂ 40 Minutes	IPPB-O₂‡ 15 Minutes	IPPB-O₂‡ 30 Minutes	100% O₂ 20 Minutes
O₂ Saturation (per cent)	60	99	100	100	100
Po₂ (mm. Hg)	36	124	188	275	140
Pco₂ (mm. Hg)	36	38	28	29	—
pH	7.42	7.42	7.52	7.51	—
Alveolar Po₂ (mm. Hg)	108	664	674	674	—
A-a Po₂ difference (mm. Hg)	72	540	486	399	—
Ventilation (liters/minute)	32.4	17.8	22.8	22.0	20.3
Respiratory rate	70	40	31	22	36
Tidal volume (ml.) (BTPS)	459	445	719	998	563
CO₂ production (ml.) (STPD)	401	261	137	109	208
O₂ consumption (ml.) (STPD)	469	349	320	309	330
Blood pressure (mm. Hg) (systolic/diastolic)	60/40	65/40	80/50	95/60	90/50

* Miller, W. F., and Sproule, B. J.: Studies on the role of intermittent inspiratory positive pressure oxygen breathing (IPPB/I-O₂) in the treatment of pulmonary edema, Dis. Chest **35**: 469, 1959.

† On room air breathing for only a few minutes owing to profound distress and severe hypoxia produced.

‡ At no time during this or subsequent studies was anything but water aerosol used.

Pulmonary Edema.   Continuous positive pressure breathing for treatment of pulmonary edema was first advocated by Barach, Martin and Eckman in 1938.[5]   Much later in a critical review of the therapy of pulmonary edema Aviado and Schmidt[2] still recommended the use of positive pressure.   IPPB is probably more commonly used now than continuous positive pressure, and expiration at atmospheric pressure rather than at positive pressure has been found to be less tiring to the patient since generally less respiratory work is involved.

In studies of gas exchange in patients breathing room air, 100 per cent oxygen by mask, or 100 per cent oxygen by IPPB, in random sequence, Miller[41] has shown that IPPB is helpful in the treatment of acute pulmonary edema from a variety of causes. Data from a typical patient are shown in table 2. These studies demonstrated large "shunt effects" (continued perfusion of parts of the lung with decreased ventilation), further evidence that airway obstruction *per se* contributed to the anoxia.   Dyspnea was not relieved by 100 per cent oxygen at atmospheric pressure even though arterial oxygen saturation returned to normal.   However, dyspnea was relieved when the oxygen was administered by IPPB suggesting that significant decrease in the work of breathing occurred.   It was believed that IPPB not only decreased transcapillary pressure gradients and cardiac filling but

that it also improved ventilation-perfusion ratios.   In the opinion of the reviewers IPPB deserves trial in acute pulmonary edema, especially if response to more routine measures is not immediately evident.   It should be remembered, however, that an already failing cardiovascular system may be further impaired by the high positive pressures applied.

Acute Bronchial Asthma.   Segal[57] found IPPB of value as adjunctive therapy in acute bronchial asthma, although in general it has been reserved for patients with recurrent attacks and carbon dioxide retention.   Wells[63] believes it helpful because in inspiration it assists in the deliverance of high concentrations of oxygen and nebulized bronchodilator drugs both during acute attacks and prophylactically.   He stresses the skill necessary in the application of IPPB to an apprehensive asthmatic patient and notes as an important advance the development of automatic devices in which flow rate may be altered independently of pressure.   Koelsche,[29] however, has not found IPPB devices helpful in the treatment of status asthmaticus.   The devices he used produce high initial flow rates.

It appears logical to give IPPB a trial in severe asthmatics, with due attention to proper administration.   Considerable ingenuity may be required in order to obtain a satisfactory fit of mask or mouth piece.   Humidification of the inspired gas and addition of bronchodilat-

ing drugs are essential components of the therapy.

Chronic Obstructive Lung Disease—Bronchodilator Therapy. One of the most controversial issues lies in the use of IPPB for administration of bronchodilating drugs. The advantages initially claimed included: improved bronchial drainage because of higher peak expiratory flow rates, better distribution of bronchodilator drugs, and more uniform alveolar ventilation.[20, 43, 60] Whether any or all of these effects actually occur has been questioned.[30, 38] It must be emphasized that administration of bronchodilators by *nebulization* frequently is improperly performed, but if done correctly, little or no difference can be found when IPPB is added.[30, 38, 61] Others have found a greater improvement in timed vital capacity and maximum breathing capacity and more prolonged effects with a combination of IPPB and bronchodilators than with nebulized bronchodilators alone.[69] However, in none of these papers have changes in P_{CO_2} been reported. Levine,[31] a proponent of IPPB, emphasizes that it must be administered with care and that inexpert use may result in patient fatigue. His outline of principles in its use is most appropriate. If IPPB is to be used, the following seem to be logical recommendations:

(1) Administration only by trained therapists.
(2) Inhalation periods of 15–20 minutes, four times daily.
(3) Adequate humidification of inspired gas.
(4) Use of bronchodilators in weakest effective concentration.
(5) Inspiratory pressure settings between 10 to 20 cm. of water.
(6) Attention to mask or mouth piece fit.
(7) Slow inspiration and expiration with inadequate pause between.

Well-controlled data in the long term management of patients with chronic obstructive lung disease have not been reported. The use of IPPB devices with controllable flow rates permitting low initial and sustained inspiratory flow rates must be further evaluated before regarded as ineffective or harmful, the conten-

tion of many who have employed devices using only preset high initial flow rates.

Prevention of Postoperative Respiratory Complications. The possibility of development of acute respiratory insufficiency in the postoperative period must be recognized. The exact incidence of this complication is unknown, but it certainly occurs with frequency in patients with chronic pulmonary disease undergoing any type of major operation and in patients with prior good pulmonary function following cardiopulmonary bypass procedures. Among the factors associated with its development are respiratory center depression due to the use of narcotics; fever with increased metabolic needs; rapid, shallow respirations in the presence of sub-diaphragmatic abscess, acute gastric dilatation or intestinal obstruction associated with elevation of the diaphragm; and finally, circulatory inadequacy from any cause. Without question artificial respiration must be initiated immediately and is most conveniently continued using an IPPB device. Endotracheal intubation or tracheostomy should be performed when indicated. Futher discussion of postoperative respiratory complications may be found elsewhere.[37, 39]

The indications for the prophylactic use of IPPB to decrease the incidence of postoperative chest complications are not clearly defined. On the basis of clinical impression it is enthusiastically suggested,[49, 54] although recognized that the results may be due merely to increased attention to respiration.

In patients without prior cardiopulmonary disease who underwent abdominal surgery, Sands *et al.*,[56] have evaluated the use of IPPB without bronchodilators. Alternate patients received IPPB prophylactically. Effectiveness was based on auscultation of the chest, roentgenograms, performance of maximum breathing capacity, vital capacity, and three second timed vital capacity. In the control group, 15 of 42 patients developed chest complications (five major, ten minor); in the IPPB treated group, 17 of 42 showed complications (six major, 11 minor). Thus there was no difference in incidence of respiratory sequelae. In conclusion the authors stressed the necessity for the continuance of well-established postoperative therapeutic regimens.

It is not possible at present to decide whether prophylactic IPPB is useful in surgical patients with chronic cardiopulmonary disease. Careful trial, however, seems indicated because it is not an inconvenient procedure and is an excellent method of drawing attention of the nursing staff to ventilatory care.

Summary and Conclusions

Intermittent positive pressure breathing (IPPB) was defined and the history of its development presented. Physiological effects of IPPB on the circulation and respiration were noted. The mechanics of breathing and the physics of automatic ventilators were briefly discussed only insofar as relates to the use of IPPB in the clinical situations examined. An attempt was made to evaluate critically the reported work in these areas.

The usefulness of IPPB in respiratory paralysis or restrictive pulmonary disease was not questioned, but the indications for its use and certain practical aspects were emphasized. It was found that there were few well controlled data to support the efficacy of IPPB in the therapy of cardiopulmonary disease. Many factors serve to confuse the evaluation of treatment in patients with chronic pulmonary disease who develop severe respiratory acidosis. IPPB deserves a trial in the treatment of acute pulmonary edema particularly when simpler methods fail. Likewise IPPB seems indicated in the therapy of status asthmaticus. On the other hand the indications for application of IPPB with or without bronchodilator drugs in chronic obstructive lung disease are less well defined. A similar uncertainty prevails in the selection of IPPB for prevention of postoperative pulmonary complications.

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