Physiologic Factors in the Use of the Body Respirator for Impaired Respiratory Function*

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It is of interest that the history of body-enclosing chambers for the treatment of respiratory diseases goes back to at least 1885, when H. F. Williams, an American physician, aided by an engineer friend, used this principle in administering aerosol therapy to patients with pulmonary infections.1 A year later Williams reported that cyclic changes in pressure around the body could be used to produce passive respiration and to assist breathing in diseased conditions.2 This experience was forgotten except in a few physiology laboratories where body plethysmographs were used to study respiration and produce ventilation in experimental animals.3 The idea was rediscovered in 1927 by Drinker and Shaw, and following their successful application of a respirator in treating poliomyelitis patients4 its use has spread to the treatment of other types of respiratory failure.

Before considering physiologic aspects of the respirator, it is pertinent to review certain characteristics of the normal mechanics of breathing, gas exchange, and pulmonary circulation, since all of these components of external respiration are inseparably related to the interchange of carbon dioxide and oxygen between the body and the atmosphere. Likewise, all three components of external respiration are involved to various degrees in pulmonary diseases and are affected adversely or beneficially by the methods of therapy. Since the object of this report is consideration of the use of the respirator, most attention will be directed to the mechanics of breathing, but it must be kept in mind that mechanical ventilation of the lungs has little purpose except the regulation of alveolar gas concentrations, and the pressure changes in the thorax associated with ventilation, whether due to muscular activity or to a mechanical device, have inevitable effects on the circulation of blood through the thorax.

Lung ventilation in the normal organism is so regulated that the alveolar partial pressure of carbon dioxide seldom varies

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more than 3 mm. from the mean value of 38 mm. Hg., at sea level. At higher altitudes the alveolar pressure of carbon dioxide is stabilized at a lower level. When the carbon dioxide partial pressure varies beyond these narrow limits, alkalosis or acidosis occurs. If the abnormal value continues for many hours or days, adjustments in the bicarbonate levels of the body occur so that the pH is returned toward normal. After a few days of the abnormal carbon dioxide level, the respiratory center undergoes a change in sensitivity so that it tends to maintain ventilation at the new level. Of most practical importance is this sort of change in the emphysema patient whose chronic underventilation leads to accumulation of high concentrations of carbon dioxide in the body.

The fine control of carbon dioxide concentration in the body is not imitated in the case of oxygen. Because of the shape of the oxygen-hemoglobin dissociation curve an almost normal load of oxygen can be picked up by the blood passing through the lungs, although lung ventilation, and hence oxygen concentration, may be considerably less than normal. High concentrations of inspired oxygen, up to 60 per cent at one atmosphere, also have little effect on the body (other than to overcome an increased diffusion barrier). In fact, lung ventilation is not essential for supplying oxygen to the body, if the organism is under basal conditions, pure oxygen is administered, and the nitrogen is first flushed out of the system. This phenomenon has come to be known as "diffusion respiration." Although adequate for oxygenation, the method is not practical because in the absence of pulmonary ventilation carbon dioxide concentration soon reaches a narcotic level.

It is apparent, then, that mechanical treatment of respiratory failure is primarily to regulate carbon dioxide exchange, and that this exchange is ordinarily controlled within very narrow limits by the respiratory regulatory mechanisms. If carbon dioxide exchange is properly regulated and the patient continues to show signs of hypoxia, the concentration of inspired oxygen must be raised.

The normal active breathing movements are, like most integrated activities of the body, exceedingly complex in detail but simple enough in broad outlines. When all respiratory muscles are relaxed at the end of an expiration, the lungs have a certain volume, known usually as the functional residual volume, and in terms of the mechanics of respiration as the relaxation volume. This volume represents an important base line and is the volume at which the tendency of the lungs to contract is exactly balanced by the tendency of the thorax to expand. These opposing forces
result in the static negative interpleural pressure. The major forces involved are due to elastic properties of lungs and chest, plus the action of gravity on abdominal contents and diaphragm. It is worth emphasizing that the chest has a natural tendency to expand to a volume somewhere between 50 and 70 per cent of the normal vital capacity scale. This degree of expansion is usually reached in advanced emphysema when the counterforce of an elastic lung is largely lost.

In normal breathing the thorax is actively expanded by muscular action, thereby further reducing interpleural pressure. The associated reduction of pressure in the alveolar spaces results in a pressure gradient in the tracheobronchial tree, and air flows into the lungs until alveolar pressure approaches atmospheric pressure. The transpulmonic pressure difference at this point is greater than before since the lung is more stretched; in other words, the interpleural pressure is more negative. Usually, muscular relaxation starts before inspiratory air flow stops, interpleural pressure becomes less negative, and expiration quickly proceeds, assisted greatly by the elastic recoil of the lungs. In quiet unobstructed breathing, expiration ends when the opposing elastic and gravitational forces come into equilibrium.

Ventilatory failure may result from a variety of causes, including muscular paralysis, fatigue, skeletal deformities, tracheobronchial obstruction, and increased resistance to lung tissue deformation. Often there is a combination of causes. The aim of therapy is to replace or supplement the inadequate motor mechanism; the method is basically simple—the rhythmic creation of a pressure difference between the alveolar spaces and the outside of the thorax, sufficient to distend the lungs by the desired amount. The pressure difference upon which lung inflation depends may be created either by increasing pulmonary pressure, as in ordinary pressure breathing, or by reducing pressure outside the body, as in the respirator. The only significant difference between the two methods is that the gas density changes in the first method account for approximately 10 per cent more tidal exchange in the case of conventional pressure breathing. The importance of recognizing the essential similarity of the two methods is that the large body of evidence on the physiologic effects of pressure breathing can be applied to the therapeutic use of the body respirator. The circulatory effects of pressure breathing, as summarized by Barach, Fenn, Ferris, and Schmidt are primarily the reduction of venous return to the right heart (usually temporary), rise in systemic venous pressure, increased systemic capillary filtration, reduction in blood volume, systemic arteriolar constriction, and elevation of cerebrospinal fluid pressure. Barach, Martin,
and Eckman recognized the importance of these factors in the treatment of pulmonary edema. In addition, the increase in functional residual volume and widening of the bronchial channels associated with certain types of pressure breathing have led to beneficial results in the treatment of asthma.

**Comparative Advantages of Different Methods of Pressure Breathing.** Both methods of creating a pressure difference have been used extensively in the treatment of respiratory paralysis of central or peripheral origin. In general the body respirator has been adopted in treating prolonged respiratory paralysis, as in poliomyelitis and other neuromuscular defects of the respiratory motor apparatus. The most significant advantages of the body respirator are its safety and the fact that the head is free of mechanical appliances. Thus eating, drinking, talking, and aspiration procedures can be done without interruption of artificial respiration. It is difficult to gain these advantages with any form of mask, hood, or dome used independently of a respirator. On the other hand the body respirator handicaps nursing and other therapeutic procedures, requires at least a semirecumbent position, has a set pattern of cycling pressures, and has a limited range of cycling rates.

For short term respiratory paralysis or as an accessory to the body respirator, various means of applying pressure at the face are widely used. The intermittent positive pressure and alternating positive and negative pressure resuscitators apply pressure by means of a face mask; they adequately breathe the patient when properly used but are not suitable for round-the-clock dependability. Other devices have been used to supplement the pressure differential created by the body respirator, or to substitute for the respirator when free access to the body is needed. The former type includes bellows assemblies operating in phase with the respirator, supplying air or high oxygen mixtures to the airway through tracheotomy tube adapters, face masks, hoods, or plastic domes. The principles are the same for all types of pressure breathing.

**Effects on Ventilation**

**A. Tidal exchange when air channels are unobstructed.** The patterns of pressure change in most respirators are such that air flow proceeds almost to completion during the inspiratory phase of the cycle. Under these circumstances tidal exchange is maximal for a given pressure setting, since alveolar pressure approximates atmospheric when the respirator is at its negative peak. However, the effectiveness of the transpulmonic pressure difference is modified by the visco-elastic properties of lungs and
thorax, and by reflex resistance if the muscles are not paralyzed. Unfortunately, the variability of these properties in patients with emphysema, poliomyelitis, and other diseases makes it impossible to predict with accuracy what tidal ventilation will result from a given pressure setting. In the absence of ventilation measurements or blood gas data, physicians have had to rely upon the subjective reactions of the patient or the empiricism of experience. It is presumptuous to expect clinical judgment to be a perfect substitute for the body's complex neurochemical regulation of ventilation.

B. Tidal exchange in the presence of airway obstruction. With increased tracheobronchial resistance the flow of air into the lungs is slowed and the alveolar spaces do not have time to fill to atmospheric pressure before the peak negative respirator pressure is passed. The transpulmonic pressure difference is less than the cycling pressure range of the respirator, and the ventilation is less than would be expected from the respirator gage setting. The remedy is to alleviate the obstruction if possible, or to increase the pressure range until adequate ventilation is obtained.

As in asthmatic patients, achieving adequate ventilation in obstructed patients is not without cost. The respirator produces active inspiration, as in normal breathing, and expiration must occur by elastic recoil of the lungs, supplemented by active expiratory effort if the patient is able. In the presence of obstruction, there may not be time for expiration of a normal tidal volume, so that inspiration starts from a higher point on the lung volume scale. This process of increasing functional residual volume will continue until there is sufficient elastic recoil in the overstretched lungs to expel in the time available an amount equal to the inspired volume. If a further analogy to asthma is permissible, one would expect this situation to result eventually in loss of lung elasticity. The increase in functional residual volume can be prevented by augmenting the positive pressure phase inside the respirator, to assist expiration.

C. Effects of the respirator on pulmonary secretions and transudates. It is not appropriate to discuss here the theory of treating lung edema by pressure breathing, except to say that there is sound evidence for desirable effects on the mechanics of breathing, on gas exchange, and on the circulation, when suitable applications of pressure are made. As a means of giving pressure breathing to treat pulmonary edema, the respirator excels, in that a wide variety of pressure combinations is possible. It has the disadvantages of not responding to the patient’s breathing desires, if present, and of requiring the recumbent position. The first disadvantage is eliminated in a respirator to be described in a
later paragraph; the second handicap would be technically easy to eliminate.

Some individuals believe that the body respirator may cause aspiration of secretions into bronchi and alveolar spaces, resulting in alveolar edema and atelectasis, brought about in some way by the negative pressure. Some of these statements arise from a misconception about the nature of respirator operation, namely, failure to realize that the respirator has the same effects on tracheo-bronchial diameter and lung volume as normal breathing. Normal breathing with the same tidal volume should have the same effects on movement of secretions. If pressure breathing is useful in treating atelectasis, the respirator is likewise useful, although higher than average pressure gradients may have to be used.

Effects of the respirator on circulatory function. That the respirator has the same effects on the circulation as other forms of pressure breathing has been experimentally demonstrated. Elevation of airway pressure above the ambient pressure of the rest of the body impedes venous return and cardiac output, at least during the period of elevated airway pressure. This effect is of little or no consequence if the high airway pressure phase is short enough or if compensatory circulatory mechanisms produce a rise in systemic venous pressure. Failure of the compensatory response occurs in the presence of vasomotor paralysis or shock accompanied by low blood volume. In these circumstances the body respirator has been found to depress the circulation in man and in experimental animals. When expiratory positive pressure in the respirator is used to reduce the required degree of negative pressure, giving a mean pressure near atmospheric, the circulation is improved.

Another mechanism by which circulatory impairment can occur is that of gastric distention by air pressure. When lung ventilation is brought about by anesthesia bag compression, it is not rare to find that air has been forced into the stomach as well as the lungs; this complication is likely to occur when airway resistance is increased. Increase in gastric air has been observed in respirator patients, brought about by the same mechanism.

A Patient-Cycled Body Respirator. In addition to its use in treating respiratory paralysis, the body respirator has given excellent results, on a limited scale, in the treatment of cardiac pulmonary edema and the impaired respiratory function of emphysema. These conditions have been treated on a large scale by apparatuses applying pressure to the face. The latter have the advantage of cycling in response to the patient's breathing desires, but are difficult to maintain in use for long. It has been
considered desirable to combine the advantages of the two methods, by having a body respirator cycle in response to the patient's desires, and at the same time having the face free. Such a respirator would have potential usefulness not only in treating patients with asthma, emphysema, and pulmonary edema, but in treating victims of bulbar poliomyelitis, in whom breathing impulses are present but erratic, conflicting with regular pulsations of the conventional respirator.

In response to this need the J. H. Emerson Company has developed a respirator attachment which permits a patient to cycle the body respirator at almost any desired rate with very little effort. Any type of body-enclosing chamber may be used, the pressure fluctuations being obtained by connecting the chamber to a vacuum blower.* The effectiveness of the suction is determined by a solenoid-operated valve which in turn is controlled by the cycling mechanism attached to the patient. When the valve is closed, air is evacuated from the chamber, resulting in a steady fall of pressure until the valve opens. When the valve is open, air rushes back into the chamber, raising the pressure to atmospheric. The cycling mechanism consists essentially of a pressure-sensitive switch, which is opened or closed by the breath-

*Spencer Turbo-Compressor, capacity 50 cu. ft. per minute.

FIGURE 1: Time relationships of respiratory air flow rate and intratank pressure. Upper tracing, spontaneous breathing, tank not operating; lower tracing shows increase in ventilation when tank is operating.
ing movements. A pressure change of only a fraction of a millimeter of water is sufficient to trip the switch. This pressure change is readily obtainable from an ordinary pneumograph cuff around the chest or abdomen, or it may be obtained by holding the open end of the transmitting tube in the air stream at the mouth or one of the nares. The switch is set so that the solenoid-operated valve is open at the rest position. With this setting slight inspiratory effort causes closure of the solenoid-operated valve and the intratank pressure starts to fall, thereby assisting inspiration. When lung inflation is completed and air flow stops, the solenoid-controlled valve automatically opens and expiration occurs essentially at atmospheric pressure.

The time relationships of respiratory air flow rate and intratank pressure in a normal subject are shown in Figure 1. The top tracing indicates that in quiet spontaneous breathing there is a sustained inspiratory flow of 25 to 30 liters per minute. The lower tracing was made with the mechanism operating; it is evident that tank pressure starts to fall when inspiratory flow rate reaches a level between 10 and 20 liters per minute, at a time when only a small fraction of a normal tidal volume has been inspired. Inspiration then continues at higher than normal flow rates, producing hyperventilation similar to that seen in the first few moments of any type of pressure breathing.

Subjectively, the application of pressure in this way is comfortable. There is not only freedom from the discomfort of a tightly-fitted mask, but there is no added dead space effect to be overcome by increased tidal volume. The patient-cycled respirator has been used on two patients convalescing from poliomyelitis and on one patient in status asthmaticus. Data on these and other patients will be reported subsequently.

**SUMMARY**

In normal breathing the respiratory muscles change the configuration of the thorax in such a way that air inflates the lungs on each inspiration and is expelled by elastic recoil of the lungs on each expiration. These movements not only effect gas exchange but affect the circulation of blood through the thorax. If the respiratory muscles are for any reason inadequate, their efforts must be assisted or replaced by mechanical means. The pulmonary effects of normal breathing can be very nearly simulated by intermittent elevation of airway pressure (obtained by raising pulmonary pressure with the body at atmospheric pressure, or by lowering pressure around the body with the lungs remaining at atmospheric pressure). The effects of intermittent positive pressure breathing on the circulation are very different from those
of normal breathing; the impeding effect on cardiac output is beneficial in some conditions (pulmonary edema) and detrimental in others (shock). The advantages of alternative methods of pressure breathing are presented. A device which permits the patient to cycle a body respirator is described.

RESUMEN

En la respiración normal los músculos respiratorios cambian la configuración del tórax de tal manera que el aire infla los pulmones en cada inspiración y es expelido por retracción elástica de los pulmones en cada expiración. Estos movimientos no solo afectan el intercambio de gas sino también la circulación de la sangre a través del tórax. Si los músculos respiratorios son inadecuados por cualquier motivo sus esfuerzos deben ser ayudados o reemplazados por medio mecánicos. Los efectos pulmonares de la respiración normal pueden ser casi simulados por la elevación intermitente en la presión de las vías aéreas (obtenida por la elevación de la presión pulmonar con el cuerpo a presión atmosférica, o por descenso de la presión alrededor del cuerpo con los pulmones permaneciendo a presión atmosférica). Los efectos de la respiración a presión intermitente positiva sobre la circulación son muy diferentes de los de respiración normal; el impedimento del rendimiento cardíaco es benéfico en ciertas condiciones (edema pulmonar) y deprimente en otras (shock). Las ventajas de los métodos alternantes de la respiración a presión son presentadas. Se describe una innovación que permite al paciente arreglar los ciclos de un respirador corporal.

RESUME

Dans la respiration normale les muscles respiratoires modifient la configuration thoracique. Ainsi l’air gonfle les poumons à chaque inspiration et est chassé par la rétraction élastique des poumons à chaque expiration. Ces mouvements ne concernent pas seulement les échanges gazeux, mais ont également leur incidence sur la circulation du sang à travers le thorax. Si les muscles respiratoires sont incapables, pour une raison ou une autre, de remplir leur mission, il faut les aider ou les remplacer à l’aide de moyens mécaniques. L’action de la respiration normale sur le poumon peut être artificiellement obtenus de façon très proche de la normale, par l’élévation intermittente de la pression de l’air. Les effets sur la circulation de la respiration en pression positive intermittente sont très différents de ceux de la respiration normale. L’action sur l’insuffisance cardiaque, très heureuse dans certains cas (œdème pulmonaire) est fâcheuse dans d’autres (choc). Les auteurs montrent les avantages de la respiration en pression alternative.
REFERENCES


Discussion

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The studies of Whittenberger and Maloney clearly indicated that mechanical ventilation of the lungs produced by increasing the negative pressure within the respirator produced physiologic effects that were quite similar to pressure breathing applied to the upper respiratory passage-way by means of a mask or hood. This contribution has been of considerable importance since artificial ventilation in the tank respirator may now be understood to have the consequences, both advantageous and disadvantageous, which result from so-called positive pressure respiration. (I have just tried the device which allows the patient to initiate the ventilation by a slight chest or diaphragmatic movement and it seemed comfortable and effective).
In our own studies 15 years ago, pressure breathing was found to produce a compensatory rise in venous pressure of approximately 40 per cent of the applied mean extra-pulmonary pressure in normal subjects and 70 per cent in patients with congestive heart failure; the circulation time was also markedly prolonged in the cases of cardiac insufficiency. The study of pressure breathing during the war, used in the development of methods to increase altitude tolerance of the individual, likewise revealed a consistent compensatory rise in venous pressure, with a decrease in cardiac output when high mean pressures were employed.

During the past two years Dr. Beck, Dr. Seanor and I have made comparative studies on the venous pressure response in human subjects to pressure breathing carried out by increasing the negative pressure within the tank and to pressure applied to the upper respiratory passage-way. Since the venous pressure response varies, as indicated above, observations on the same subjects were made with the two methods. No differences were found in these two methods of mechanical ventilation of the lungs in respect to this aspect of circulatory stress. Our studies, therefore, confirm the hypothesis of Whittenberger and Maloney. The consequences of increased air density in upper respiratory pressure create several changes that are of little or no clinical significance.

In studies which we have recently made on mechanically induced coughing, a swift opening valve was employed in order to allow a sudden outlet of air from the lungs, simulating the volume flow of air during a normal cough after a full inspiration. In the slide which I will show, the decrease in the mean pressure applied to the lungs is revealed when the swift opening valve is employed, namely, a decrease from 8 to 5 cm. In addition, the venous pressure rise is shown to be approximately one-half that which takes place with the conventional operation of the respirator, even when the same peak pressure is obtained. Since an adequate pulmonary ventilation is maintained with this type of mechanical respiration which simulates, to some extent, the human cough, its advantage in patients with impairment of the peripheral circulation is evident, namely, a decreased interference with the return of blood from the right heart. In conditions of pulmonary edema, a higher mean pressure may be desired, but in patients with peripheral circulatory failure, mechanical respiration with a sudden opening valve is to be preferred, either attached to the conventional respirator or to a pressure hood.

Dr. Whittenberger and Dr. Maloney have presented so many interesting contributions that I have had to select only one aspect of their valuable paper for discussion.