

INHALATIONAL THERAPY, ADVANCES AND RETREATS

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ANESTHESIOLOGISTS have made valuable contributions to the field of inhalational therapy, including studies on pressure breathing, respiratory and metabolic acidosis, ambulatory oxygen therapy and resuscitation. This communication will be a critical appraisal of some serious problems that have developed concerning the principles and techniques of administering oxygen and the application of pressure to the respiratory passageway, and, in addition, to describe new concepts that require investigation. Some dangerous ground will be covered, but encouragement may be found in the remark of La Rochefoucauld, "He who lives without folly is not as wise as he thinks."

HYPERVENTILATION VERSUS OXYGEN IN THE TREATMENT OF HYPOXIC CLINICAL DISEASE

Intermittent hyperventilation has been substituted for the administration of oxygen in many clinics in the United States, especially in patients with chronic pulmonary disease; this practice has resulted in neglect of the treatment of severe hypoxia, even in patients with an associated cardiac failure. Physiologically directed therapy should include the continuous inhalation of oxygen to improve cardio-respiratory function and to induce a clinical remission.

In patients with pulmonary emphysema in whom the clinical symptom of irrationality or coma accompanies an *abrupt* elevation of arterial carbon dioxide tension and an acid shift in pH , the use of mechanically induced hyperventilation is valuable. However, the level of arterial CO_2 tension in itself is not an indication for hyperventilation. Unfortunately, the employment of hyperventilation procedures has been extended to rational, conscious individuals in whom the pH of the blood is normal with an elevated carbon dioxide and bicarbonate content. In these cases, over-

ventilation has been used in such a way as to impair a previously developed homeostatic equilibrium by which the patient had been able to eliminate carbon dioxide in an increased concentration in the alveolar and expired air.

The harmful consequences of hyperventilation in individuals who have a normal blood pH were originally reported to be tissue hypoxia, because of the Bohr effect of an alkaline pH on tissue cells; more recently the lowered brain oxygen tension induced by a decreased cerebral blood flow was found even when hyperventilation was conducted with oxygen.¹ Among other serious effects of hyperventilation-induced alkalosis are the electrocardiographic signs of coronary insufficiency,^{2, 3} as well as induction of laryngospasm and bronchospasm.

This routine substitution of hyperventilation for the therapeutic use of oxygen in advanced pulmonary emphysema has also been applied in cases in which a true respiratory acidosis is present; the employment of such pharmacologic agents as caffeine to stimulate the respiration, alkaline agents such as sodium bicarbonate and sodium lactate, steroids and diuretics, has often been neglected. Furthermore, in patients with mild stupor, high CO_2 and slight acid change of pH , the use of mechanically induced hyperventilation by intermittent pressure breathing applied to the face or trachea, is frequently less effective and more cumbersome than the program of treatment outlined above, including also the controlled, regulated continuous administration of oxygen. To restore the function of the brain, and other vital organs requires the employment of measures which lessen or eliminate hypoxia.

Carbon dioxide retention as a cause of the symptoms of cardiac and pulmonary insufficiency is frequently over-emphasized because of an inadequate realization of the beneficial homeostatic effects of the increase in CO_2 eliminated per unit of ventilation, as well as

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the consequent decrease in the metabolic energy requirement for dyspneic breathing. The value of hypercapnia as an integral part of oxygen therapy, in cases of cardiac insufficiency secondary to pulmonary disease as well as in primary heart disease, was stated in 1931, as follows: "The marked rise in carbon dioxide content of the arterial blood was constantly associated with clinical improvement . . . an adaptive change which favors the elimination of carbon dioxide."⁴ The technique of preventing an oxygen-induced coma by administering oxygen in gradually increasing concentrations was proposed in 1938.⁵ Following the demonstration that base was retained to compensate for the increase in carbonic acid,^{6, 7} the importance of sudden pH change toward acidity became more clear as the etiology of so-called CO₂ narcosis provoked by abrupt inhalation of high oxygen-enriched atmospheres.⁸⁻¹¹

A decade ago many investigators warned against the use of oxygen in chronic pulmonary emphysema.^{12, 13, 14} Unfortunately, it was not made clear in these reports that oxygen could be administered in a graduated program without adverse changes in mental or bodily function, nor, indeed, that hypercapnia could be used deliberately to provide the patient with a means of taking advantage of oxygen inhalation, *i.e.*, reducing the volume of breathing.^{4, 6-9, 15-17}

Although intermittent pressure breathing has at times been found ineffective¹⁸ and inferior to insufflation,¹⁹ as a method of inducing CO₂ elimination, many investigators have observed that its use is beneficial in carbon dioxide intoxication.^{20, 21, 22} The concept that hypercapnia in pulmonary emphysema was an un-mixed evil was additionally presented by investigators who apparently found that arbitrary lowering of the CO₂ tension, even in the absence of irrationality or coma, was beneficial.^{23, 24} In a recent study of kyphoscoliosis, the hazard of a progressive increase of arterial CO₂ tension was stated as follows: "It is clear that in kyphoscoliosis, as in chronic pulmonary disease, hypercapnia begets hypercapnia: once CO₂ retention exists, further increments in arterial blood CO₂ tension fail to elicit normal increments in minute ventilation. This deficient response is generally attributed to loss

of sensitivity of the respiratory center to CO₂."²⁵ However, in the same paper, our thesis was also agreeably presented: "An oxygen-enriched atmosphere may actually benefit such patients despite the hypercapnia it elicits. This proposition recognizes that a decrease in both the O₂ consumed and the CO₂ is produced by the respiratory muscles during breathing, that high levels of alveolar CO₂ tension are advantageous to the patient in that the CO₂ produced by metabolism may be eliminated by a smaller alveolar ventilation." Unfortunately, these authors then mention their "theoretical restraint that after enriched oxygen mixtures are discontinued, the patient is left with not only hypercapnia and depression of the respiratory center, but also with hypoxemia."

The latter statement assumes that the oxygen-treated patient has not been benefited by treatment, which may or may not be true in cases of kyphoscoliosis. However, when restoration of compensation, cardiac and respiratory, has been the result of regulated oxygen treatment and allied therapy, both hypoxia and hypercapnia are diminished proportionally to the improvement of lung function. Hypercapnia, therefore, is used as a tool just as oxygen is, in the accomplishment of a crucial therapeutic objective, which, when successful, is followed by a better oxygen and CO₂ diffusion in the lungs. Furthermore, a decreased ventilatory response to carbon dioxide does not appear to be simply related to hypercapnia but rather to increased pulmonary effort due to mechanically restrictive factors, such as the difficulty in moving the lungs.^{4, 26, 28, 30}

The respiratory center yields its priority to decrease of pulmonary effort; with decrease in pulmonary effort and ventilation, the organism consumes less oxygen, manufactures less CO₂ and progressively relieves itself of dyspnea and overwork, facilitating restoration of compensation, a restoration frequently incapable of achievement without deliberate abolishment of hypoxia and deliberate inauguration of hypercapnia, both of which are necessary to decrease the energy expenditure of the patient. These objectives have been obtained repeatedly in cases of cardiorespiratory insufficiency without coma and without so-called "carbon dioxide narcosis."

Despite an oxygen-induced rise of arterial P_{CO_2} to 60 to 120 mm. of mercury, carbon dioxide "intoxication" is not present as long as the pH has been normal during the evolution of carbon dioxide retention. When oxygen administration was increased gradually by the use of a nasal catheter or cannula (one liter per minute being progressively raised every day or two by one liter per minute) in our last series of 100 oxygen-treated cases, only one patient developed temporarily an irrational state, but some signs of acidosis were already present in this case. This characteristic favorable response has been noted previously in this country and by Simpson⁹ in England, who tried both the abrupt and the controlled technique of administering oxygen.

The decreased pulmonary ventilation which occurs when oxygen is administered in the regulated manner described has been emphasized as the critical aim of treatment in patients with pulmonary emphysema and allied syndromes, including the relief of hypoxia, and at times requires procedures which eliminate or neutralize increasing carbonic acid in the blood, if the pH becomes acid. The increase in CO_2 tension to graded oxygen therapy has, in my experience, been beneficial rather than the reverse.

In a survey of the literature of cases of pulmonary emphysema in which the CO_2 tension was elevated with a normal pH , there has been no evidence of harmful effect from the increase in CO_2 itself; the administration of sedatives together with oxygen therapy has in some cases induced an acid change in pH with clinical signs of acidosis, *i.e.*, irrationality and stupor, as a consequence of undue depression of respiration.

The patient with chronic pulmonary disease presents not only a remarkable priority accorded to oxygen-induced decrease of pulmonary effort over the respiratory center's response to CO_2 , but also manifests similar responses to other therapeutic procedures which decrease pulmonary effort and the energy needs of the body.²⁶ For example, the institution of diaphragmatic breathing results in a 20 to 30 per cent decrease in pulmonary ventilation without increase in P_{CO_2} or pH , when costal breathing ceases, as in the head-down position.¹⁵ The mechanical induction of

increased tidal air by means of abdominal compression may also result in decrease of the breathing volume, because of increased efficiency of alveolar ventilation. The lowering of the total pulmonary ventilation also takes place without increase of the previous hypercapnia.³¹ The patient, under these circumstances, selects a decreased minute volume of breathing in preference to diminishing the accustomed increased CO_2 tension in his blood and, in so doing, decreases the work of breathing itself. However, intermittent abdominal pressure breathing had been effectively used in respiratory acidosis in which the pH was lowered.⁸²

Reduction of CO_2 tension by over-breathing is followed by reduction of base; the consequences of dyspnea in normal individuals were illustrated by Houston and Riley³³ on individuals acclimatized to a 20,000-foot altitude for one month. Shortness of breath took place after the experiment was concluded, when the subjects walked *at a sea-level atmosphere*. For one week this exertional dyspnea continued because of the decreased neutralizing capacity of the lowered base in the blood. It was only when the base bicarbonate of the blood was elevated to the normal level that individuals could walk without shortness of breath. This interesting response took place in normal people when a 20 per cent lowering of the bicarbonate in the blood impaired its capacity to neutralize carbonic acid.

The use of air in intermittent pressure breathing devices as a gas to nebulize bronchodilator solutions reveals the extent of the fear of physiologic relief of hypoxia. A decrease of arterial oxygen unsaturation has been demonstrated when patients are hyperventilated with air, but the procedure is physiologically unsound when tissue hypoxia is induced, in proportion to the alkaline shift in pH . Conversely, intermittent pressure breathing can be carried out with 100 per cent oxygen without modifying the CO_2 tension of arterial blood.³⁴

The advantage of correcting the hypoxic state in patients with pulmonary and cardiac disease, has been revealed by studies in which it has been shown that pulmonary hypertension is promptly lowered by oxygen inhalation in cases of septal defect,³⁵ in the restoration of a more normal cardiac output by oxygen in-

halation³⁶ and by the favorable response of patients with coronary insufficiency to oxygen inhalation during exercise³⁷ or during acute coronary thrombosis.

AMBULATORY OXYGEN THERAPY

In the resuscitation of patients dead from coronary disease,^{38, 39} oxygen treatment has been described as a primary indication and should be used before and with cardiac massage.³⁹ Since hyperventilation, even with 100 per cent oxygen, produces electrocardiographic signs of coronary insufficiency³ as well as a lowering of brain oxygen tension,¹ it is contraindicated in conditions of cardiac failure. When pressure breathing, as in uncompensated respiratory acidosis and acute pulmonary edema, is indicated, the ventilation should be adapted to the metabolic requirement of the patient, and should include in most cases an oxygen-enriched atmosphere. Even in mouth-to-mouth insufflation it is preferable, whenever possible, for the operator to inhale oxygen before blowing his expired air into the mouth of the patient, since the oxygen concentration of expired air is 3 or 4 per cent less than that of the atmosphere and since in coronary insufficiency and other asphyxial states increase of the oxygen tension of the blood is of therapeutic value. The advantage of promptly instituting pressure ventilation for resuscitation is obvious, as well as the addition of oxygen when feasible, without delay.

Cardiac arrest in the operating room has recently been successfully treated by pressure breathing with oxygen without thoracotomy and cardiac massage; circulation of blood has also been successfully induced by rapidly pressing the heel of both hands against the sternum.⁴⁰ Miller⁴¹ had previously reported the lifesaving value of intermittent pressure breathing with 100 per cent oxygen in cases of pulmonary edema associated with coronary disease and shock, although in our introductory paper on the specific effect of pressure breathing in pulmonary edema, the presence of circulatory insufficiency was considered an indication for caution in the use of pressure.⁴²

The preceding discussion of oxygen inhalation may serve as a background for the recent development of the use of oxygen at home and on the street, a new and valuable extension of

inhalational therapy. Among the indications of ambulatory therapy, is the use of oxygen to facilitate an athletic training procedure in patients with chronic pulmonary disease^{16, 26, 43}; the restoration of increased exercise capacity after the use of an oxygen exercise regimen has also been observed by others.⁴⁴⁻⁴⁸ The mechanism of the immediate relief of dyspnea is based in part on the lowering of the pulmonary ventilation induced by breathing 40 per cent oxygen during exercise.^{44, 45} In addition, there are other advantageous effects of providing oxygen to patients during walking exercises, such as those known to take place as a result of oxygen inhalation at rest, *i.e.*, the beneficial effects on the coronary circulation, cardiac output and on pulmonary hypertension, referred to above. The inhalation of a bronchodilator aerosol immediately preceding the walking exercise, as well as manual compression of the chest and upper abdomen following the aerosol, often improves the capacity of patients with chronic pulmonary disease to carry out the prescribed exercise while breathing oxygen.

The beneficial effects of walking half a mile twice a day have been noted after 2 to 8 weeks, especially when the portable oxygen dispensers have been employed. The development of the increased capacity for exertion without oxygen is naturally the aim of therapy, although preservation of air-walking ability depends on maintenance of the oxygen exercise program. To ascribe the improvement in exertional dyspnea solely to the oxygen exercise program is difficult since often a variety of other procedures have been employed, such as training in the use of the diaphragm. Nevertheless, there have been approximately 35 individuals in my experience and 22 of 29 patients reported by Cotes and Gilson⁴⁴ in whom the oxygen exercise program appeared to engineer a specific athletic training response in less than 2 months, characterized especially by a decreased pulse rate on exercise, as well as by a lowering of the pulmonary ventilation at rest. In normal subjects performing severe exercise, oxygen results in lower ventilatory and cardiac rates^{49, 50}; the beneficial effect was more marked in individuals who had ingested alcohol prior to the test.^{51, 52}

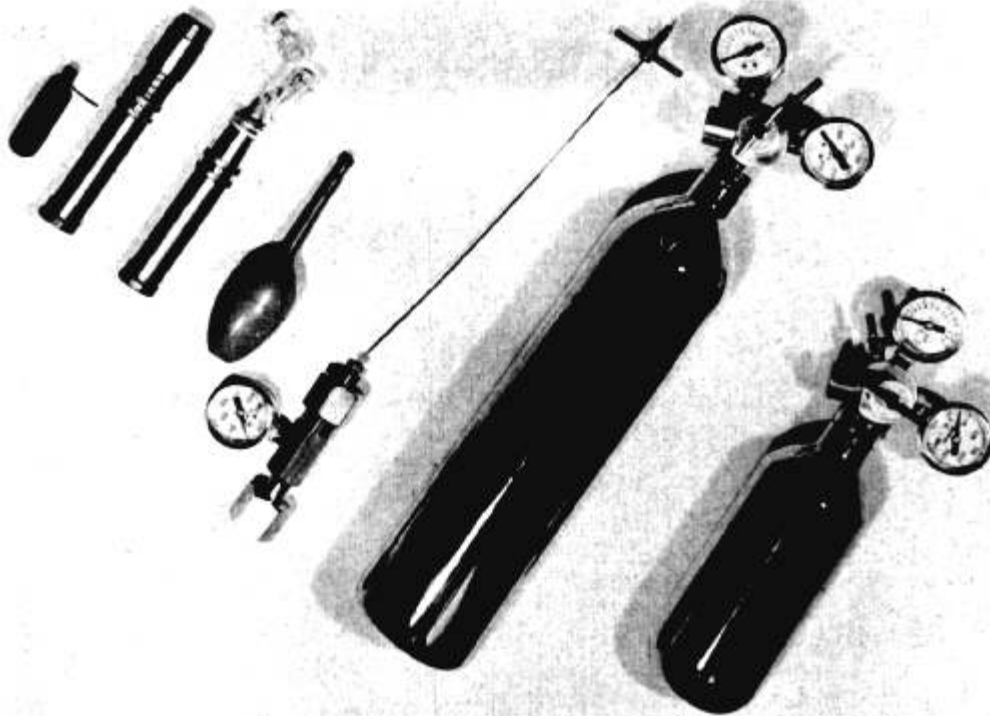


FIG. 1. Portable oxygen-dispensing devices. Two larger cylinders, one containing 75 and the other 150 liters of oxygen, equipped with a one-pound two-stage regulator and the device for filling these cylinders from a 244-cubic-foot tank are shown. The pocket oxygen dispenser is also illustrated with the small 5,000-p.s.i. cylinder alongside; its venturi provides various concentrations of oxygen from 26 to 50 per cent. A nebulizer may be used with oxygen, or CO₂ if desired, as the propellant. A rubber bulb is substituted when oxygen is not available.

The use of the pocket oxygen dispenser has made it possible for patients with chronic pulmonary disease and also some with coronary disease to extend their activities without the fear of a sudden attack of shortness of breath or cardiac pain.⁵³ The attempt to extend oxygen therapy to the street, the golf course, or at home, may be rewarding, provided the principles and standards of the therapeutic use of oxygen be as nearly as possible in conformity to those for hospital practice. The various devices employed are shown in figure 1. Reports of the lifesaving value of portable oxygen apparatus have appeared.⁵⁴

When portable oxygen therapy units are employed with fixed orifice outlets, *i.e.*, without a compensated regulator, the total volume of the cylinder cannot be used safely to the end of the period of oxygen flow.⁵⁵ In a 56-liter cylinder at 1,800 p.s.i., the oxygen flow decreased from 6 to 4 liters per minute the tenth minute, although the flow of oxygen con-

tinued for 25 minutes at a progressively smaller rate. These investigators mentioned the CO₂ accumulation when oxygen flows lower than 4 liters per minute are delivered

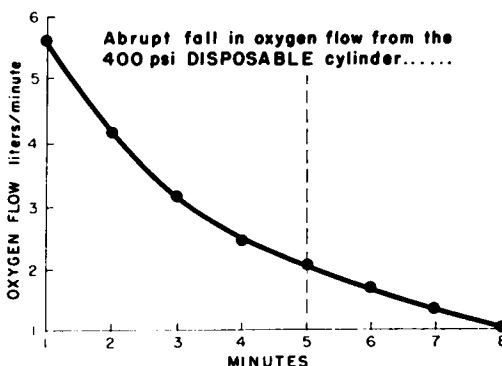


FIG. 2. Abrupt fall in oxygen flow rate during use of 400-p.s.i. disposable cylinder. During continuous administration of oxygen from 400-p.s.i. disposable cylinder, the oxygen flow rate falls abruptly below 2 liters per minute, after the first five minutes of use.

into a loosely fitting oxygen mask. In previous studies of the B.L.B. mask, in which partial rebreathing of expired air took place, the CO₂ percentage found in the inspired air was in excess of 2 per cent when 3 to 4 liters of oxygen were administered.^{56, 57}

A far more abrupt fall in oxygen flow rate takes place from a disposable oxygen cylinder with a pressure of 400 p.s.i. and a fixed orifice. (fig. 2.) About a decade after low pressure cylinders were abandoned and high pressure, 2,000-p.s.i. oxygen tanks were adopted, Benash and Carter⁵⁸ emphasized the importance and safety of providing a steady control of the rate of oxygen flow. "Oxygen must never be used without a suitable regulator or reducing valve to control the rate of flow." Unfortunately, the high standards of specialists in inhalational therapy have recently been flouted by the promotion of an emergency oxygen low pressure device that is both extraordinarily costly and inefficient.

This widely advertised product is said to contain 7 gallons of oxygen. However, the duration of its effective use is far below the oxygen content because of the progressive drop in oxygen delivered as the pressure in the

cylinder falls. At the end of 5 minutes the oxygen flow falls below 2 liters per minute, and after 8 minutes, below 1 liter per minute. When the device is used in inspiration, the volume of oxygen actually inhaled is from one half to one third that of continuous flow, depending on the length of inspiration.

Any device used for emergency oxygen treatment should, in the accompanying literature, describe the actual oxygen flow delivered per minute. If any mask is attached to the patient, with a rebreathing bag, partial or complete, the employment of continuous oxygen inhalation should be controlled by a compensated one-stage or a two-stage regulator capable of providing the rate of oxygen administration prescribed. Preservation of this standard of oxygen treatment will protect the patient from a progressive decline in the effectiveness of treatment of hypoxic emergencies.

It is clear that the oxygen concentration of the inspired air depends on the pulmonary ventilation of the patient as well as on the flow of oxygen into the apparatus, in devices applied to the nose or face. The more dyspneic the patient, the higher is the oxygen

TABLE 1
OXYGEN CONCENTRATION OF INSPIRED AIR DEPENDENT ON OXYGEN FLOW RATE,
PULMONARY VENTILATION OF INDIVIDUAL PATIENT, AND TECHNIQUE
OF ADMINISTRATION

Oxygen Flow Rate in Liters per Minute		Pulmonary Ventilation Liters per Minute					
Continuous Flow into Nonrebreathing Collecting Bag of Nasal or Mask Device	Administration During Inspiration. Flow Stopped in Expiration	6	8	10	12	16	20
		Percentage Oxygen Concentration Inspired Air					
1	2	34	31	29	28	26	25
2	4	47	41	37	34	31	28
3	6	61	51	45	41	36	33
4	8	77	61	53	47	41	37
5	10	87	70	60	54	46	41

The flow rate with inhalation of oxygen during inspiration is twice that of continuous administration, but the total volume inspired is the same when all oxygen delivered throughout the respiratory cycle is inspired. In nasal cannula administration, in which oxygen is lost during expiration, the volume of oxygen required to obtain similar oxygen percentages in the inspired air is two to three times higher, depending on the length of expiration.

The pulmonary ventilations of the ambulatory or dyspneic individual are frequently in the range of 10 to 16 liters per minute, at which 3 liters per minute will provide between 45 and 36 per cent oxygen in the inspired air, provided the oxygen delivered during expiration is utilized in the subsequent inspiration. When expiration is prolonged, in asthma and emphysema, considerable oxygen is wasted with current use of nasal cannula, 66 per cent or more; thus, 3 liters per minute provide 26 per cent oxygen when nasal oxygen is inspired during one-third of the respiratory cycle at a ventilation of 16 l./minute.

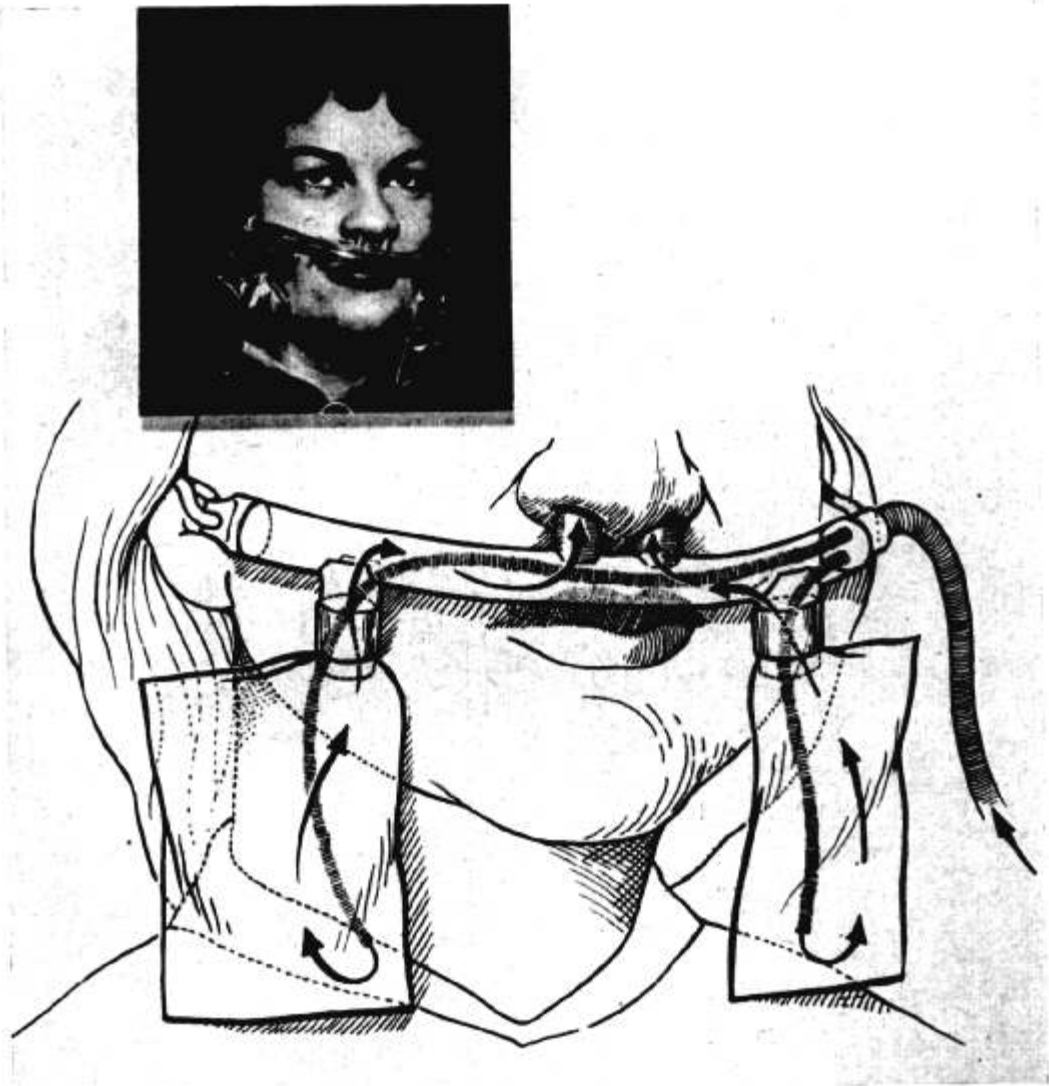


FIG. 3. Nasal cannula attached to 150-liter oxygen refillable cylinder. Inspiratory valve ahead of two 300-cc. collecting bags prevents rebreathing.

flow required (table 1). The danger of CO_2 accumulation in a mask is inevitably present with low oxygen flow rates when partial rebreathing occurs. The determination of the alveolar CO_2 cannot be used as a test of the efficiency of the mask employed or the actual CO_2 tension in the inspired air. Haldane's studies showed that for conscious subjects pulmonary ventilation increased to keep the alveolar CO_2 tension constant.⁵⁹ In normal subjects who inhaled 1 and 2 per cent CO_2 , an increase in pulmonary ventilation was

found, approximately 20 to 40 per cent respectively.⁵⁶

In respect to the principles of selection of apparatus used for oxygen inhalation, especially in chronic pulmonary disease or conditions characterized by hypoventilation, the rebreathing technique is generally contraindicated, *unless* oxygen is delivered at high enough flows to dilute the exhaled CO_2 or a venturi is used with high flows of mixed gas. The end-expiratory air, containing 4 or 5 per cent CO_2 , should not be inhaled

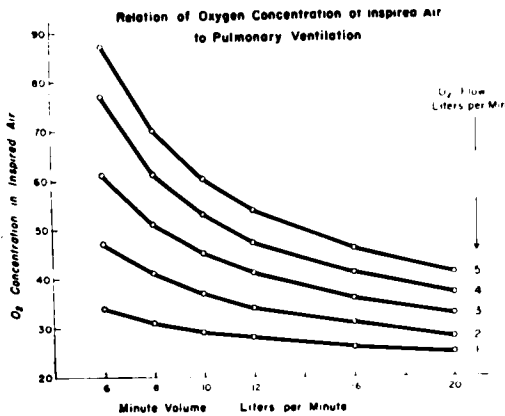


FIG. 4. In order to estimate the concentration of oxygen inhaled by a reservoir collecting mask or nasal apparatus, this chart may be employed by first selecting the oxygen flow used. By following the curve upward at the flow selected, from 1 to 5 liters per minute, the inspired oxygen percentage may be arrived at by determining the minute volume of breathing of the patient. The results shown on the chart apply only in those instances in which the oxygen flow is stored throughout the expiratory cycle and then inspired.

If the total oxygen flow were to be administered during inspiration only, the result would be the same. However, with the use of the plastic cannula in nares, the oxygen delivered during expiration is lost. Since in most patients with chronic pulmonary disease, inspiration is considerably shorter than expiration, the oxygen percentage obtained with the nasal cannula is less than half that provided when the nasal cannula with a collecting bag is used. Furthermore, high flows of oxygen from a cannula impinging on the nasal mucous membrane are uncomfortable. With the reservoir apparatus, 30 to 75 per cent oxygen may be provided through the enlarged plastic connections into the nares. Since the oxygen is first directed into the collecting bags, there is no limitation on flow.

during the succeeding inspiration. The various types of face masks and face tents originally suggested years ago are no longer recommended, but instead, masks or nasal apparatus that have an inspiratory valve ahead of the collecting bag.

The development of a nasal cannula with reservoir plastic collecting bags was recently reported;⁶⁰ this method of administering 30 to 75 per cent oxygen may supersede many other techniques in a variety of clinical entities in which acute or chronic hypoxia is encountered (fig. 3).

The actual concentration of oxygen in the inspired air can be estimated or calculated from the accompanying table 1 and figure 4,

provided the oxygen flow rate and minute volume of breathing are known, and these assumptions are made: (1) that all oxygen delivered during expiration is stored in a non-breathing collecting bag, and (2) that the oxygen inspired represents the total oxygen from the cylinders during inspiration and expiration. The plastic cannula inserted just within the nares contributes oxygen during inspiration only. Since in many patients with severe asthma and emphysema the length of expiration constitutes two thirds of the respiratory cycle, a flow of 9 liters per minute would add only 3 liters to inspiration in these cases. The cannula is not suited for administration of moderately high oxygen-enriched atmospheres to dyspneic subjects. The oxygen tent with a flow maintained at 15 liters per minute is the most effective and comfortable method of administering 45 to 50 per cent oxygen.

The convenience of portable light cylinders which the patient can carry, with two-stage regulators and safe refillable devices, has made the athletic training program more pleasurable than when it was carried out with the patient attached to a 50-foot rubber tube from a large cylinder, and no more expensive in the cost of gas.*

My colleagues and I have studied certain procedures for the prevention of cor pulmonale



FIG. 5. The 15-liter Oxy-Hale provides varying concentrations of air and oxygen; thus, 106 liters of 52 per cent oxygen, or 60 liters of 40 per cent oxygen. Refilled in 30 seconds from 2,000-p.s.i. cylinder.

* Light-weight regulators and cylinders and pocket oxygen dispensers are manufactured by Controlled Pressure, Inc., Erie, Pennsylvania. The nasal reservoir cannula is made by Shampaine Industries, Rozelle, New Jersey.

and left ventricular failure in patients with pulmonary emphysema, and allied chronic respiratory diseases.^{15, 16, 26, 31, 37, 43, 53, 56} One of these measures, consisting of the inhalation of oxygen continuously during brisk exercise, made use of new light-weight cylinders and regulators. In addition to the oxygen dispenser, called the Oxy-Hale, a similar device is available, containing 15 liters of oxygen, with a mixing venturi and a refillable feature. A patient who has become short of breath at a pulmonary ventilation of 10 liters per minute may inhale a 40 to 32 per cent oxygen-enriched atmosphere during inspiration for 6 to 10 minutes, depending on the concentration of oxygen selected (fig. 5).

Although the walking exercise program has been carried on for about ten years in our clinic, the recent advances in apparatus have made for a psychologically better approach to this method of therapy.†

ABRUPT RELIEF OF TRACHEOBRONCHIAL OBSTRUCTION

The significance of pressure breathing in the treatment of long-standing obstructive dyspnea is frequently overlooked. Tracheostomy in patients with laryngeal obstruction and in those patients with chronic pulmonary disease who have manifested expiratory obstructive respiration often leads to an abrupt

†The cylinders contain 75 and 150 liters, weighing 2 and 5 pounds respectively. A filling device may be attached, connected by a wire of 2 to 3 feet in length to a 244-cubic foot cylinder. The element of safety in the use of a wire for transmitting the oxygen from the large to the small cylinder depends on the obviously small volume of oxygen which would be released with a break in the connection. Crucial to the safety factor are: (1) the small orifice into the small 75- and 150-liter cylinders, *i.e.*, approximately .003 square inch; (2) an escape valve that opens at 3,300 p.s.i. Patients can carry the small cylinder in their pockets. The large cylinder is suspended by shoulder straps which also permit them to walk freely, not only in the home, but out of doors for a period of time that varies with the actual flow of oxygen. The 15-liter Oxy-Hale device is refillable by the same mechanism. The flow of oxygen with the use of a nasal cannula is 6 to 8 liters per minute, for the carrying out of the exercise program described. However, with the employment of the new nasal device in which the oxygen is stored during expiration, 3 to 4 liters per minute will provide the concentration of oxygen ordinarily obtained by 6 to 8 liters when the oxygen flow is lost during one-half of the respiratory cycle.

outpouring of fluid in the tracheobronchial tree, with congestion and edema of the mucous membrane. This preventable complication is due to the pulmonary capillaries being suddenly deprived of a previously existent back pressure. It constitutes a hazard to life, and is not a beneficent form of bronchial drainage! It can be avoided by the prompt use of an apparatus providing expiratory positive pressure after tracheotomy, with a gradual decrease of pressure, as has been employed in the treatment of pulmonary edema of cardiac origin.⁴² Similarly, a prolonged use of the negative phase of respiration in intermittent pressure breathing apparatus must be utilized with this precaution in mind, in those clinical entities in which the patient has previously pursued expiratory pressure breathing, as in the emphysema patient with pursed-lip breathing. However, when the negative pressure cycle exists only for one to two seconds, as in the exsufflator for expulsion of retained secretions, there are no adverse effects of this kind.

THE FUTURE

Cases of High Molecular Weight, and Miscellaneous Subjects. The study of heavy gases, such as SF₆ (sulphahexafluoride), C₃F₈ (octofluoropropane), and C₄F₁₀ (octofluorocyclobutane), has been instituted in our clinic with a view to their use as possible mechanical expectorants. When it is realized that C₄F₁₀ has a molecular weight of 238, air being 28.8, it may be surmised that profound differences in respiratory function were found during its respiration by patients with asthma or pulmonary emphysema. The expulsive effect on mucus after coughing with gases of increased density, such as C₄F₁₀, appears to be enhanced despite the lowered respiratory velocity of mixtures of 20 per cent oxygen and 80 per cent C₃F₈ and C₄F₁₀. Studies of Beck and the author have shown a 20 to 30 per cent decrease in maximal breathing capacity in cases of chronic bronchospastic disease, and at the same time minimal trapping of the inhaled atmosphere. The velocity of gas movement could be better maintained by the simultaneous use of the exsufflator.

Inhalational therapy is being extended to other fields, such as: the relation of decreased

oxygen uptake of the brain to the aging process, heated hypertonic and isotonic aerosols for diagnosis and therapy, intermittent abdominal pressure breathing, newer exsufflator mechanical cough apparatus and sensing devices for patient regulation of breathing. Increasing evidence is appearing, supporting an older concept, *i.e.*, the priority of pulmonary effort over chemical equilibria in certain dyspneic states, with modification of the concept of insensitivity of the respiratory center to CO₂ to include the pulmonary effort of the patient and his work of breathing.

SUMMARY

An appraisal of inhalational therapy has led to the following conclusions:

(1) An unwarranted increasing tendency to substitute intermittent hyperventilation procedures for continuous oxygen therapy has been observed in the medical clinic.

(2) A misconception of the role that oxygen plays in the homeostatic development of hypercapnia has resulted in unjustifiable warnings against the continuous administration of oxygen. The regulated regimen of oxygen inhalation may be safely and effectively employed in the treatment of cardiorespiratory insufficiency.

(3) The abrupt termination of obstructive dyspnea by tracheotomy often provokes increased serous secretions in the respiratory passageway. This complication should not be regarded as a new method of bronchial drainage. It can be prevented and treated by the application of expiratory positive pressure.

(4) The development of portable apparatus has been instrumental in opening a new field of oxygen treatment for the ambulatory patient with chronic coronary and pulmonary disease.

(5). The technical advance in portable oxygen therapy requires the use of compensated regulators that provide an unvarying flow of oxygen; needle valves or push-button valves from a cylinder in which the flow falls progressively are not suitable for continuous reliable oxygen therapy at home. The refillable feature of portable oxygen therapy increases the feasibility of oxygen therapy at home and out of doors. The use of low pressure cyl-

inders with fixed orifices is not recommended since an abrupt decrease of oxygen flow rate takes place.

(6) Recent studies on the employment of high-density gases, such as octofluoropropane and octofluorocyclobutane, are under investigation as mechanical expectorants and from the standpoint of appraisal of respiratory function.

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CARBON MONOXIDE POISONING

Two patients, one comatose and one semicomatose from the inhalation of carbon monoxide have been treated by pressurization of the patients to 2 atmospheres of oxygen. A pressure chamber was built sufficiently large to accommodate both patients and medical staff. In ten minutes of pressurization, the semicomatose patient could talk intelligently. In twenty minutes the comatose patient was able to breathe without the aid of a resuscitator, reflexes had returned, and spasticity had disappeared, and at forty minutes this patient was able to answer questions. Decompression of both patients was begun at the end of one hour, and they were removed from the chamber at one hour and thirty minutes. Recovery was complete in both cases. (Smith, G., and Sharp, G. R.: *Treatment of Carbon-Monoxide*

Poisoning with Oxygen under Pressure, Lancet **2**: 905 (Oct. 22) 1960.)

PNEUMOTHORAX A correct diagnosis of pneumothorax was made in 12 consecutive patients using the scratch sign. This sign is elicited by placing the Bowles stethoscope attachment over the thoracic spines or sternum. The skin is scratched on both lung fields with a finger or blunt object at equidistant points from the stethoscope. The equidistant scratch areas should be 7.5 cm. to 20 cm. from the head of the stethoscope. The examiner then compares the sounds from similar areas on the two sides. A positive sign consists of a considerably louder and harsher sound on the side of the pneumothorax. (Lawson, J. D.: *Scratch Sign—Valuable Aid in Diagnosis of Pneumothorax, New Engl. J. Med.* **264**: 88 (Jan. 12) 1961.)