Physical Exercise in Breathless Subjects with Pulmonary Emphysema, Including a Discussion of Cigarette Smoking*

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A training program of graded exercise while breathing oxygen is employed in patients with pulmonary emphysema in whom physical unfitness has been induced by excessive rest. Dyspnea on exertion naturally leads to inactivity which in itself promotes weakness, cardiovascular insufficiency and certain adverse biochemical changes, such as loss of nitrogen, calcium and potassium. Although walking ability is uniquely enhanced by inhaling oxygen, the aid of procedures which increase the efficiency of breathing is of crucial importance.

In patients in whom the capacity for physical activity has been increased, the imposition of questionable restrictions deserves renewed consideration. Cigarette smoking is discussed and a technique of non-inhaling smoking will be described that does not produce perceptible irritant effects on the lungs. These are the topics of this paper.

An increase in "excess deaths" attributed to cigarette smoking has been described for pulmonary emphysema, as well as a variety of diseases except the deaths due to suicide and violence; however, the relative death rate of the smokers who said they did not inhale was sharply lower than that of the inhaling smoker; thus, if 1 represents the death rate of the non-smoker, it is 1.3 for those who said they did not inhale and 2.3 for the regular smoker.1 A technique which did not in fact permit smoke to enter the lungs would naturally prevent those diseases said to be caused by inhaled cigarette smoke; this simple concept deserves emphasis.

The aim of this paper is not only to describe the methods which enhance the efficiency of the breathing mechanism, and restore exercise capacity, but also to question the validity of curtailing such pleasures as non-inhaling smoking. Although enjoyment cannot be measured in molecular equivalents, research in this fourth dimension of medicine warrants some consideration by the physician, even if it cannot be brought into the circle of laboratory medicine. The total well-being of breathless pulmonary disease patients is a legitimate therapeutic purpose.

RESTORATION OF PHYSICAL EXERCISE BY OXYGEN INHALATION

Oxygen has been used for many years to support ambulatory activity, i.e., to enable dyspneic subjects to walk to the bathroom. A deliberate graded exercise program during inhalation of oxygen was undertaken in 1952 in which the patient's walking distance was increased progressively; in two controlled hospitalized subjects, a significant increase in their capacity for physical activity took place after several weeks' exercise, accompanied by a decreased pulse rate and lessened dyspnea after exertion.

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carried out in an air atmosphere. The restoration of exercise capacity induced by an oxygen-supported walking program has been confirmed by clinical and physiologic studies.14

The beneficial effects of exercise include a reversal of the process of muscular atrophy that generally takes place in cases obliged to be inactive because of dyspnea. It is of interest to note that the cardiovascular deterioration and the loss of nitrogen and potassium induced by bed rest were found to occur very rapidly as a specific consequence of the weightlessness of simulated space flight.14 However, other factors are involved in inducing the adverse changes of excessive rest; for example, in the extreme type of immobilization of the body and chest achieved by the equalizing pressure chamber there was no clinical evidence of muscular atrophy after four months' residence in this apparatus and there was a decrease in urinary excretion of potassium, rather than the loss of potassium characteristic of prolonged inactivity.14

In some emphysema patients who did not manifest physical unfitness, the increased ability to walk appeared to result from alterations in function characteristic of an athletic training process; this adjustment to progressive exercise in normal subjects includes an increased cardiac output and enhanced diffusion of oxygen through the lungs.14 In six cases in which the lactic acid content of venous blood was determined before and after exercise, no change was found as a result of inhaling oxygen, indicating that lactic acid accumulation in muscles did not limit exercise in our emphysematous patients.† The protection provided by oxygen inhalation against excessive dyspnea was related, at least in part, to enhancement of the patient's breathing reserve, manifested by a lower minute volume of ventilation when oxygen was substituted for air.14 In some cases, androgenic steroids were employed to enhance muscle function since testosterone propionate and physical exercise improve retention of nitrogen with the use of an adequate diet.

Patients with an advancing type of pulmonary fibrosis were not permanently benefitted by oxygen supported exercise, nor was their disease arrested.14 In a few of our cases of pulmonary emphysema with progressive bullous disease, increased activity was made possible for a while and then a vanishing lung syndrome and cardiac failure took place with an eventual fatal outcome. In the majority of patients with diffuse obstructive pulmonary emphysema, the benefit obtained by the exercise program was especially marked when combined with appropriate breathing training.

Patients with chronic pulmonary emphysema do not necessarily get steadily worse, year by year. The prognosis is better in those with a prominent bronchial or bronchiolar factor, so-called centrilobular emphysema.14 Thus, a patient with diffuse obstructive pulmonary emphysema who has been under my care for 18 years is still ambulatory 19 years after onset of the disease was demonstrated by x-ray examination. Whether prolongation of life is achieved through this kind of rehabilitation program will require a carefully documented follow-up; nevertheless, most patients can expect some restoration of physical activity, and, if needless restrictions are not imposed on their personal habits, an enhanced capacity for enjoyment.

The techniques of gradually increasing the exercise performed while inhaling oxygen and the use of training in diaphragmatic breathing have been described.1415 The portable oxygen apparatus employed are shown in Fig. 1.††

In our earlier studies, the patient walked 50 feet back and forth inhaling oxygen from a nasal cannula connected to a 244 cu. ft. cylinder. The walking distance was gradually increased from 100 steps to one-

†The lactic acid determinations were done in the laboratory of Dr. Robert Darling in the course of a study by Barach, A. L. and Pons, E. R.

††Oxygen cylinder apparatus with a small aperture for transfiling from a 244 cu. ft. tank are distributed by Controlled Pressure Division, J. H. Emerson Company, Cambridge, Massachusetts.
half mile twice daily. Although this technique is simpler for some older people, it does not provide the sense of psychologic freedom of a small cylinder which may be concealed in a man’s pants’ pocket or overcoat or a handbag. The latter, weighing 3 lb. including a two-stage or compensated one-stage regulator, provides oxygen at 6 liters per minute continuously for approximately 12 minutes. The 180 liter cylinder with regulator weighs 6 lb., and is still feasible for most patients.

The cost of the treatment is that of one large cylinder every three or four weeks. The expense is small, but can be lowered if the patient uses a push-button valve in his hand to turn the oxygen on in inspiration and off in expiration when using the cannula. If the disposable Venturi oxygen mask is used, a flow of 5 liters per minute provides 40 to 50 per cent oxygen, and the off-on valve is not used since the collecting bag stores oxygen.

The pace of the walking exercise is of interest. In athletic training, exercise to the point of dyspnea enhances the ultimate peak of performance. In chronic pulmonary disease, the patient may gain more exercise capacity by extending his efforts to the point of mild breathlessness, provided he does not relapse into the use of the accessory muscles of the neck and shoulder girdle. At the end of his route, manual compression of the upper abdomen may be employed to relieve trapping of air; in addition oxygen can be inhaled for three to five minutes at rest, or until breathing effort has become normal.

Since hypoxia is the critical source of impaired functioning in these cases, oxygen inhalation may be administered with benefit, and, when properly regulated, without
undue fear of the production of comatose states in ambulatory subjects. In some patients, intermittent administration of oxygen has been accomplished by a remote control device in which the large tank is stored in a closet and connected by rubber tubing to a mask, mouthpiece or cannula on a table at the bedside or the living room. A solenoid on the regulator is connected to a push-button also on the table in order for the patient to inhale oxygen during inspiration without the tank being alongside of him. The regulator is set at 6 liters per minute, or the prescribed flow. This device can also be used for inspiratory nebulization of bronchodilator and saline aerosols as well as a remote source of power for an intermittent pressure breathing valve.

Since the restoration of diaphragmatic function and allied measures facilitate walking, an outline of these procedures is presented to enhance the clinical effectiveness of the oxygen exercise program.

Breathing Training Procedures

A bronchodilator aerosol is inhaled by means of a hand bulb nebulizer prior to each walking exercise. Manual compression of the upper abdomen is carried out ten times, by the patient himself; the abdomen being pushed upward and inward during expiration. This important maneuver generally results in release of trapped air and an immediate increase in lung capacity.

The leaning forward posture is employed during and after walking. Patients should learn to adopt this posture when sitting in a chair. In many cases, effective diaphragmatic motion during walking is not possible unless the patient leans forward 10° to 20°.

The diaphragm muscle of chronically inactive subjects is presumably in a state of atrophy, but it can be strengthened, as well as stimulated into natural activity, by the application of buck-shot weighted pads to the abdomen, increased progressively from 15 to 30 lbs., for one-half hour or more, twice daily, the patient being in the supine position. The spring-controlled (Gordon-Barach) emphysema belt* is the main appliance of this kind that significantly facilitates expiratory ascent of the diaphragm. It is especially valuable for patients with a protuberant abdomen, since it decreases the heavy downward visceral pull on the diaphragm. In addition, a less forward leaning posture can be assumed when the belt

*The use of this belt is illustrated in a brochure provided by the Spencer Co., New Haven, Connecticut.

![Figure 2: Maximal lowering of the diaphragm was accomplished by forcible diaphragmatic contraction. The marked ascent of the diaphragm was achieved by use of vigorous contraction of the lower intercostal and the abdominal musculature.](image-url)
exerts a firm pressure on the lower abdomen.

The teaching of natural diaphragmatic respiration is essential to any program of rehabilitation, whether or not oxygen-supported exercise is used. The Hoffbauer technique of facilitating ascent of the diaphragm by contraction of the abdominal musculature should be abandoned, even by patients who have previously learned to carry it out. Although it can be performed skillfully at rest, the method does not generally become an automatic habit that is preserved when the patient is dyspneic. In our experience, the patient who has been trained in this unnatural technique is apt to revert to the burdensome and inefficient accessory muscle respiration on exertion, i.e., the use of the shoulders and the neck during inspiration. The diaphragm may then become functionless, or subject to paradoxic respiration, in which rebreathing takes place between the upper and lower lobes of the lungs, thus fomenting an extremely vicious cycle of impaired alveolar ventilation. The degree of diaphragmatic excursion possible in a physician trained in breathing exercises, age 67, is illustrated in Fig. 2: the lung volume at the end of a forced expiration was one-fourth that at the end of inspiration after maximal diaphragmatic contraction (Fig. 2).

That continuous pressure of the hand on the abdomen of the supine patient promptly induces diaphragmatic excursion can be observed by the protrusion of the abdomen during inspiration. A similar result was obtained by placing weights on the abdomen. The pressure of an emphysema belt in the sitting or erect position has long been known to facilitate diaphragmatic excursion. Pneumoperitoneum is an additional example of the effect of elevation of the position of the diaphragm into the chest as a technique of restoring its function in cases of pulmonary emphysema. The leaning forward posture, especially when combined with the emphysema belt, is always helpful in the restoration of diaphragmatic function and far more valuable than the attempt to synchronize expiration with contraction of the abdominal musculature.

The physiologic rewards of discarding upper costal for diaphragmatic breathing are plainly evident when appropriate respiratory function tests are selected for comparative data. The minute volume of ventilation of patients with pulmonary emphysema is markedly lowered in the head-down position; furthermore, the arterial oxygen saturation is not decreased nor is the CO₂ tension elevated in the presence of a 20 to 30 per cent fall in pulmonary ventilation; thus, alveolar ventilation induced by visceral diaphragmatic breathing is more efficient than that found with upper thoracic breathing in the erect sitting position. In addition, the mechanical work of breathing is conspicuously decreased, as is also readily seen by clinical observation.

The value of breathing of this kind is not revealed by pulmonary function tests which measure bronchial constriction, since static tests of lung function are irrelevant to the situation studied.

The reduction of the minute volume of breathing when physiologically directed procedures are employed, such as diaphragmatic breathing, constitutes a plain response to more efficient alveolar ventilation. The studies of the volume of lung ventilation illuminated the value of restoration of a functioning diaphragm. The maximal breathing capacity, or the 1st second forced vital capacity, commonly employed to measure bronchial constriction, are unsatisfactory when extended to an evaluation of these procedures.

The techniques of inducing a change-over of upper costal, accessory neck and shoulder muscle breathing to diaphragmatic respiration, illustrated in Fig. 3, include the weighted bag on the abdomen, application of an emphysema belt, the head-down and leaning forward positions.

**Pharmacologic Agents as an Aid to the Oxygen Exercise Regimen**

Pharmacologic agents are also used to aid the performance of physical exercise,
i.e., to facilitate exertion with the least possible dyspnea. The inhalation of a broncho-dilator aerosol just prior to walking results in some widening of the bronchial passageway, either through a transient shrinkage of the mucous membrane or reduction in muscle spasm. Oral administration of aminophylline on an empty stomach is employed before breakfast and at 3 p.m., at least one hour before exercise. Ephedrine is combined with aminophylline in patients who do not have prostatic difficulty; various preparations are available but enteric-coated tablets are ineffective.*

Elixirs of aminophylline or theophylline have been found especially helpful in the treatment of acute broncho-spasm, since a solution of 20 per cent alcohol results in a swifter absorption of these drugs. Unless the patient is bothered by asthmatic coughing at the time of exercise, these elixirs are not employed to facilitate exercise but rather for an acute attack or when the patient is at rest, such as a half-hour before dinner and on retiring.

A solution made of theophylline in 20 per cent glucose, 80 mg. per 15 mL., appears to facilitate the absorption of theophylline through the stomach. In a series of patients with bronchial asthma and the bronchospastic type of pulmonary emphysema, the administration of 45 mL., 3 tablespoons, has been found remarkably effective. At times, 2½ tablespoonsful are adequate to induce a prompt subjective relief with rare incidence of nausea. Our tentative conclusion is that 20 per cent glucose is similar to 20 per cent alcohol in respect to relief of bronchospasm with freedom from side-effects.** Larger doses of theophylline solution given by methods which induce quick absorption, including those just mentioned, as well as by the intravenous and rectal route, should only be prescribed when the

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*The Dainite Day tablets include aminophylline 0.2 and ephedrine 0.015, with aluminum hydroxide and benzocaine, the latter added to reduce local effects of aminophylline on the stomach.

**This study now includes 200 cases. To increase the palatability of the solution, compound spirits of orange is added, 6 mL. to 1 quart. The solution may be obtained from Feldhammer Pharmacy, New York City.
The patient is resting in bed. A considerable lowering of the intraspinal and peripheral venous pressure induced by 300 to 400 mg. of theophylline, or the equivalent dose of aminophylline, may result in fainting or headache in cases who stand erect or walk.

The administration of steroids is helpful in cases with demonstrable broncho-spasm not adequately controlled by conventional measures. In some patients with pulmonary fibrosis, prednisone may also have a favorable effect. The dosage is regulated according to the needs of the patient, a maintenance program of prednisone, for example, generally being between 10 mg. and 20 mg. daily. Provided an aluminum hydroxide antacid is given 1 1/2 to 2 hours after eating and at bedtime, no apprehension concerning the development of peptic ulcer is justified. This opinion has been validated by the experience of our clinic over a period of more than 10 years in which various steroids have been administered to more than 500 patients continuously for two to ten years. In addition, there has been no evidence that osteoporosis or excessive loss of potassium has taken place at this dosage level. Maintenance antibiotic therapy is not considered advisable or necessary. Undue weight gain has been controlled in patients who are able to diet, especially by elimination of fat.

In patients with obstructive pulmonary emphysema, the constricted bronchial pas sageway handicaps the flow of air and leads to air-trapping and alveolar over-distention. In those cases in which broncho-spasm can be demonstrated, steroids are definitely indicated unless freedom from asthmatic dyspnea can be adequately controlled by routine measures. In addition, there is a group of cases in which an abundant mucus production aggravates obstructive dyspnea. In these patients, the sero-mucoid secretions can often be eliminated by a larger dosage of steroids, i.e., 40 mg. daily for five days, with a gradual reduction to a maintenance dosage of 15 to 20 mg.

The possible side-effects are of a nature that can be counteracted in most cases, and the rewards of a carefully-followed program of steroid therapy as outlined are often considerable. Furthermore, this approach is far superior to creating a permanent tracheostomy with all the attendant disadvantages of that procedure. Elimination of the mucus obstructing the bronchi also facilitates exercise, both with and without oxygen inhalation.

Restrictions Imposed on Patients with Chronic Pulmonary Disease

The advice to conserve the energies of older people has quite naturally been applied to breathless subjects. However, the consequent atrophy of disuse is productive of still further impairment of bodily function; therefore, physical activity rather than rest was advocated even though oxygen inhalation was required to carry out exercise. Furthermore, the technique of reducing alveolar distention, previously induced by rapid breathing, can be readily learned by the patient; therefore, he can learn to expand the range of his interests without being handicapped by trapping of air within the lungs primarily caused by exertion, or by physical unfitness.

Let us look at a list of restrictions that have some theoretical validity. Should the older patient diet to overcome a middle-age spread? Restrict cream in his coffee, omit an egg for breakfast, because he might exacerbate a possible sclerosis of the coronary artery? Should he move to a mild, dust-free climate, away from friends and family, or avoid cocktail parties to avoid smoke in the room or excessive ingestion of alcohol? Does the smoking of five cigarettes daily constitute harmful over-indulgence? Should he give up smoking completely? Renounce other pleasurable activities that are supposedly more appropriate to men in better health, or of a younger age?

Levy is one of those who has warned against the imposition of needless restrictions on cardiac patients; except in the rare
case of nicotine sensitivity, he has not found that smoking up to ten cigarettes a day is harmful to the coronary patient. The viewpoint of moderation in the management of the personal habits of the emphysematous patient will be illustrated by a consideration of cigarette smoking. Let us suppose that an individual does not inhale, and that little or no smoke enters the lungs. What then is the harm of cigarette smoking to patients with pulmonary emphysema, the cardiac and other diseases that have been ascribed to absorption of nicotine, carbon monoxide and tars from the lungs? It seemed to me that methods of non-irritant smoking deserved study.

The effectiveness of a technique of non-inhaling cigarette smoking is susceptible to accurate testing, for example, by measurement of the arterial blood CO concentration in controlled experiments. The blood of the average smoker and of a person in a smoke filled room is said to contain between 5 and 6 per cent carbon monoxide. A similar average percentage, 5.7 per cent, was found in a series of subjects tested after supposedly inhaling the smoke from 20 cigarettes from 9 a.m. to 3 p.m.; however, the concentration of CO actually varied from a percentage of 2 to 12* per cent. A comparative study has been undertaken concerning the effects of inhaling and a special technique of non-irritant smoking.

The usual method of non-inhaling is to draw the smoke into the mouth and promptly blow it out again. There is so little satisfaction in this procedure that most smokers reject it. The technique which the author has employed provides considerable satisfaction, is more esthetic in appearance and does not induce cough in patients with pulmonary emphysema and bronchial asthma who have a cough sensitivity to tobacco smoke. The patient draws a puff of smoke into the mouth, closes the lips, then inhales deeply through the nose and finally expells the smoke retained in the mouth and the air previously inhaled from the nose. The buccal cavity is closed by the soft palate and back of the tongue so that smoke does not enter the lungs during the nasal inhalation of air. (If the individual opens his mouth after the double inhalation without breathing, the smoke will be seen to filter out of the mouth without any exhalation.) That irritation of the buccal mucosa would continue with this procedure is evident, but the incidence of

*Despite the fact that all subjects considered themselves inhaling smokers, the wide variation in the CO concentrations of the arterial blood revealed that some inhaled deeply and others to a minimal extent. It is evident that questionnaires are not very reliable for this kind of information.
buccal cancer is very low compared to all the other listed clinical entities in which a two-fold excess death rate is ascribed to regular (or inhaled) cigarette smoking. This technique is illustrated in Fig. 4.

The problem of dealing with cigarette smoking in the management of patients with chronic pulmonary disease has become acute since the release to the press, magazine publications and television of statistical and pathologic data regarding lung cancer, heart disease and pulmonary emphysema, and the increased death rate in the diseases ascribed to smoking. There are, however, a considerable number of physicians who are not convinced of the causal role of cigarette smoking in these illnesses. Even in the case of pulmonary cancer in which the statistical association between heavy smoking and cancer of the lung is higher than any other illness, Pelner concluded: "In the present state of our knowledge, it is premature to say that cigarette smoking is the cause or even an important cause of lung cancer. Cigarette smoking may be considered one of the vectors or vehicles carrying a carcinogen, perhaps 3:4-benzpyrene;" he subscribed to the opinion of those investigators who believe air pollutants are the more potent carcinogenic agents. Lawton and Goldman pointed out that whereas 83 per cent of cancer specialists believed cigarette smoking was the causal agent in the increase of lung cancer, only 63 per cent of physiologists had a similar conviction.

A chain of evidence has been reported in which cigarette smoking is said to cause heart failure in patients with chronic pulmonary disease; carbon monoxide in the blood of smokers is said to be one link because of its hypoxic effect in displacing oxyhemoglobin. However, there is no factual evidence that 5 to 6 per cent arterial carbon monoxide in the blood of the average smoker induces cardiac insufficiency or increases pulmonary artery pressure. Furthermore, if a technique of non-inhaling were adopted, cigarette smoking would not in fact result in accumulation of carbon monoxide in the arterial blood, since the gas is almost entirely absorbed in the alveoli. The same inference would apply to the irritant effects of the smoke of cigarettes to which alveolar rupture has been attributed.

Passey, accepting the thesis that heavy cigarette smoking increases the risk of lung cancer, remarks that it has never been claimed that the heavy smoker is stricken earlier than the light smoker; from his analysis of the smoking history of 499 men with lung cancer (four were non-smokers), it was found that the light smoker was afflicted at the same age as the heavy smoker, and that neither the total amount smoked nor the age at which smoking began influenced the age at which cancer developed. Since the same age phenomenon was found in lung cancers associated with the nickel industries, he concluded that lung cancer arises by some other agency than by carcinogens, arising as a "natural" form of cancer, the result of changed conditions in a damaged respiratory tract, such as the presence of chronic bronchitis, and perhaps, as a speculation, the excess of mucoid secretion as a factor. This thesis has impressed the writer, since the history of lung cancer patients so frequently includes that of chronic cough and expectoration. Since many cigarette smokers do not have chronic bronchitis, it is conceivable that these individuals may not develop pulmonary cancer. As Passey has emphasized, the death rates from cancer and chronic respiratory disease are both lower in Australia and New Zealand than in England even though the consumption of cigarettes is similar. In none of these studies is information available in respect to the amount of smoke inhaled. Since the days of John Hunter the role of disturbed emotions, es-
especially anger, has been acknowledged as a factor in causing coronary insufficiency, and at times death. Most people have assumed that smoking provides a solace, a lessening of tension; for this reason, doctors have assured coronary and other cardiac patients that moderate smoking, up to ten cigarettes daily, is not harmful.\textsuperscript{2,3} Although there are many contrary views, it is our belief that tests of smoking on pulmonary function reveal no convincing evidence of sustained bronchospasm.\textsuperscript{4,5,6}

The heavy cigarette smoker may be a more tense individual than the non-smoker;\textsuperscript{7} smoking is said to be an "oral pacifier."\textsuperscript{8} However, the non-smoker may manifest signs of neurosis not present in the moderate smoker.\textsuperscript{9} The so-called oral needs of the individual, originally connected with sucking milk, are represented in the psyche by an impulse for receptivity and yieldingness. In a Rorschach study of the emotional attitudes of cigarette smokers, the moderate smoker acknowledged these needs and used smoking as a partial substitute for them whereas the non-smoker tended to reject all oral needs, attempting to handle his anxiety by a dry, pedantic, intellectualized approach, apparently deeply insecure in relation to early mother and father images; the heavy smoker evinced more urgent oral needs and employed the habit as an excessive displacement of more mature emotional outlets.\textsuperscript{10}

Larson \textit{et al.}\textsuperscript{11} after extensive investigations on tobacco smoking, summarized their view as follows: "It is never a work of supererogation to repeat that tobacco smoking, as it is performed by the smoking millions, is a pharmacologic, and only very rarely indeed a toxicologic phenomenon."\textsuperscript{11}

There would thus appear to be some justification in preserving an open mind concerning the possible harmful effects of cigarette smoking, especially if the physician is permitted to evaluate the results of non-inhaling smoking. Furthermore, does the reported statistical relation between moderate smoking and excess death rates (1:1.3) represent a causal factor, i.e., that the smoking of less than ten cigarettes daily is itself the cause of death in cardiopulmonary diseases? Or may the association represent the kind of person who tends to smoke, either because of an hereditary or genotype disposition or an acquired characteristic? Or may it represent a collaborative action of factors admittedly associated with cigarette smoking such as increased drinking of coffee and alcohol, as well as other exposures of the smoker? Is there decisive evidence of damage to the lungs of the moderate smoker or to the non-inhaling smoker? I ask these questions to elucidate the therapeutic problem of the physician.

Regardless of the adverse effects of excessive inhaling of smoke, the management of patients with chronic pulmonary disease is not necessarily confined to the heavy smoker. If the non-inhaling smoker is not significantly harmed by a habit which brings solace, the physician need not be stampeded into demanding that all smoking be stopped. The practice of medicine should not involve a war against enjoyment. Moderation in drinking was finally accepted by most physicians rather than prohibition of alcohol, even in the face of the terrific cost of health and life to the heavy drinker.

In respect to the patient with chronic pulmonary emphysema, \textit{excessive inhalation of tobacco smoke} is admittedly irritating to the mucous membrane and it is possible that carbon monoxide displacement of oxyhemoglobin may be sufficient to increase exertional dyspnea. This statement, however, cannot be used as an indictment against all kinds of smoking. In my clinical practice, the use of the non-inhaling technique here described has not

\textsuperscript{1}Comroe\textsuperscript{8} stated: "Our study indicates that mild bronchoconstriction, insufficient to cause symptoms, occurs immediately in most individuals after inhalation of cigarette smoke, lasts 10 to 80 minutes, and re-occurs with a second cigarette. We suggest that there are insufficient and inadequate data at present to warrant relating the 'bronchoconstriction' of smoking causally to the initiation or aggravation of chronic pulmonary disease."
induced coughing in patients with chronic pulmonary disease, nor could this kind of moderation in the use of cigarettes induce alveolar rupture or right and left heart failure, eventualities that have not yet been conclusively proved to be the result of regular (or inhaling) smoking.

The writer has made the following selection from the voluminous literature on the effect of cigarette smoking on the bronchial glands and mucous membrane: Simpson" concluded it was reasonable at least to accept the statistical association between chronic bronchitis and cigarette smoking; he described the controlled study of Leese" in which it was found that 59 per cent of the bronchitics had smoked a total of 150,000 cigarettes (equal to about 20 per day for 20 years) compared to 32 per cent for the controls. It was of interest that 5 per cent of the controls had smoked a total of more than 300,000 cigarettes (equal to 40 per day for 20 years) without apparent damage. There appeared to be some significance to the association of smoking and bronchitis at the level of 20 cigarettes a day. He did not mention the effects of ten cigarettes a day, nor the topic of not inhaling the smoke.

The onset of bronchitis after respiratory infections is well known. In many instances, the patient has been a cigarette smoker without symptoms. Following influenza or a common cold, chronic productive cough may take place, suggesting a hypersensitive state, defined in some quarters as bacterial allergy. Air pollutants and cigarette smoke now result in an exacerbation of the condition. Long-term smoking has played a less critical role in pathogenesis in these cases than the sensitizing viral infection. Chronic bronchitis takes place similarly in non-smokers, initiated by, but not maintained by, acute infection.

I have discussed cigarette smoking as an illustration of one of man's solaces that may be used without demonstrable harm if moderation is practiced, i.e., by adopting a non-inhaling technique. Other needless restrictions may be counselled by physicians who permit no compromise between excess and abstinence. In a more liberal approach, the patient's problem is considered on an individual basis, and the treatment may then be more soundly documented, in respect to humanitarian as well as physiologic principles.

The relationship between the doctor and the patient is being scrutinized more closely as the total rehabilitation of the patient is considered a proper therapeutic aim.' It has been said that with the exception of the relation between the sexes, that which exists between doctor and patient is not approached anywhere in our society. The physician frequently possesses what Weber' has called the "charismatic authority," an extraordinary quality thought to provide a unique or magical power derived from superhuman or exceptional gifts. The use of an influence of this kind to curtail enjoyments that cannot be proved harmful to the individual patient must be weighed carefully, especially in the light of the present re-investigation of ethical systems. There are only a few who, like Thoreau, could live alone, but even of him it was said that "the life he held in such harsh control finally evaporated in his hands."

The case histories of two patients were selected to illustrate (a) the improvement in exercise capacity as a result of enhanced physical fitness and (b) the development of increased exercise capacity due to changes in physiologic conditioning suggestive of the athletic training process. In the latter case, cigarette abstinence had previously resulted in decreased coughing without improvement in dyspnea. It is of interest to mention here that a recent study of the acute effect of cigarette smoking revealed no significant differences in vital capacity, timed vital capacity, maximal breathing capacity, or functional residual capacity after cigarette smoking in either patients with pulmonary disease or the normal subjects.

**CASE 1**

*History:* Woman, aged 53. Dyspnea and unproductive cough were noted nine years before...
consultation with the writer at patient's home. Shortness of breath increased very gradually for seven years when an exploratory chest operation was performed; it was found impractical to remove the bullous right lower lobe. The pulmonary artery to the right lower lobe was ligated and severed. A progressive deterioration in her condition took place subsequently, with increasing anorexia, weight loss and dyspnea, partially relieved by oxygen, two to three liters per minute by nasal cannula. For one year she had been unable to walk, was helped or carried to a chair, felt hopeless and drank five to six martinis daily.

Various intermittent pressure breathing devices which had been tried previously had resulted in fatigue and no benefit. Phlebotomy was employed at varying intervals. Pulmonary function tests were not attempted.

Physical Examination: She was seen to be in constant respiratory distress, manifested by raising of the upper chest, use of the accessory neck muscles during inspiration, a despairing facial expression and cyanosis of the nailbeds. Lungs: breath sounds absent over both lower lobes with scattered crepitant rales. Liver was enlarged and tender.

X-ray film of chest: almost complete absence of lung markings over right lower lobe due to large bullous lesions; bulging of fissure indicative of increase in volume of right lower lobe. Hyperaeration also noted in left lower lobe. Streaky densities extended from hilar areas to upper lung fields.

Laboratory findings: During the succeeding several months, the following observations were made: total protein 63.8, albumin 3.8, globulin 2.5, serum potassium 4.7, sodium 141 (m.Eq./L) chlorides 600, sugar 88, urea 12, cholesterol 324, hematocrit 54 to 62, hemoglobin 16, electrocardiogram ST lowered in Leads 1, 2, V5, V4, V3 and V6.

Treatment: Walking exercise was begun with oxygen at 6 liters per minute by nasal cannula beginning with ten steps daily and gradually increasing to 200 or more steps twice daily; the patient was at first supported by a technician or her maid. She gradually learned to breathe with the diaphragm, but only when the leaning forward posture was adopted. Manual compression of the upper abdomen was accompanied by marked relief of dyspnea. This maneuver was utilized five to ten times daily. Martinis were voluntarily reduced to one daily, as the patient felt less short of breath.

Medication consisted of intramuscular injections of testosterone propionate 100 mg., weekly for six weeks; then once or twice a month; prednisone, 20 mg. daily, was prescribed for six weeks, reduced gradually to 5 mg. daily. Digitals was stopped and chlorothiazide (50 mg. hydrodiuril) administered two to three times weekly. Hand-bulb nebulizer with Dylephrin used three times daily with occasional slight relief of dyspnea.

Course: At the end of two months, she manifested a striking clinical improvement. Appetite was restored. She was cheerful and carried on walking exercises with oxygen. At times, out of doors, she dispensed with oxygen for one to two hours, walking 200 feet without oxygen. She entertained friends and resumed at home many of the activities she had not been able to perform previously. Her weight increased 10 lb. in five months, without edema, liver tenderness or enlargement.

Seven months after she was first seen by the writer, surgical removal of the right lower lobe was suggested to the patient since this part of the lung had no blood supply and obviously increased the dead space ventilation. The upper lobes of both lungs did not seem seriously involved since breath sounds were vesicular. Furthermore, there was evidence of increasing compression of the right upper lobe and the mediastinum as a result of progressive increase in the volume of the right lower lobe. Blood gas studies performed at this time are shown in Table 1.

Table 1—Arterial Blood Gases and pH Breathing Air and Oxygen

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<th>Air</th>
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<td>O2 vol.</td>
<td>10.5</td>
<td>14.1</td>
</tr>
<tr>
<td>O2 cap.</td>
<td>21.7</td>
<td>20.7</td>
</tr>
<tr>
<td>O2 satur</td>
<td>48.8</td>
<td>69.2</td>
</tr>
<tr>
<td>pH</td>
<td>7.33</td>
<td>7.31</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>47.3</td>
<td>48.7</td>
</tr>
<tr>
<td>CO2 tension mm.Hg</td>
<td>62.3</td>
<td>63.2</td>
</tr>
</tbody>
</table>

Arterial oxygen saturation without oxygen was 48.8 per cent, 69.2 per cent with oxygen at 10 liters per minute, the pCO2 62 mm.Hg breathing air and 63 mm.Hg breathing oxygen for five minutes. The arterial pH was 7.3.

At this time, the patient wrote me: "I hope you are not disappointed in me for refusing to submit to an operation. At all times my desire is to do whatever you think best. However, everything about me is so greatly improved I'm loath to test fate."

For three months more she had many good days, but there was evidence of further impairment of respiratory function. She needed three to four liters of oxygen continuously to be comfortable, although a higher flow resulted in still more benefit.

She died six months later of progressive respiratory and, finally, cardiac failure. During this last period, she manifested sudden attacks of

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twitching and muscular spasms of the extremities, accompanied by tachycardia and dyspnea; these seizures frequently came on when she wakened from sleep or as a result of sudden change of position. Electrolyte studies at that time revealed no explanation in respect to abnormalities of calcium, phosphorus, potassium or sodium. She showed no clinical signs of an uncompensated respiratory acidosis; i.e., irritation or stupor. Hypoxia is more pronounced during sleep, and these convulsive symptoms may have been hypoxic in nature; it was also thought that compression of the mediastinum may have initiated circulatory insufficiency.

Increasing dyspnea and tachycardia during the last two days indicated progressive circulatory and respiratory failure.

CASE 2

Man, age 56. Present Illness: Dyspnea on exertion and cough began two years prior to consultation in the writer's office. One year ago, he stopped smoking and cough almost entirely disappeared, but dyspnea increased gradually, being manifested on stairs, walking several blocks, especially marked on an up-grade or after eating. Very little mucoid expectoration.

Past history included bronchitis and three attacks of pneumonia. No asthma.

Physical and Laboratory Findings: Well-built man, 6 ft., 3 in., weight 220 lb. After walking exercises in office, upper chest was raised anteriorly with visible neck muscle contraction on inspiration. Little diaphragmatic respiration was evident when standing in erect posture. Lungs: Breath sounds distant over both sides. No rales. Blood pressure 120/70. Physical examination otherwise negative. X-ray examination of chest: increased radiability of both lungs; heart not enlarged.

The vital capacity was 4400 ml.; after inhalation of a bronchodilator aerosol, 4800 ml. These measurements were made on a McKesson bellows apparatus. The vital capacity later recorded on a Collins respirometer, was 4400 ml.; the forced expiratory volume 3000 ml.; with 25 per cent expired in the first second, 40 per cent expired in the first two seconds, and 50 per cent in the first three seconds. The pulmonary ventilation (P.V.), determined in the erect sitting position, was 10.8 breathing air and 8.0 liters per minute breathing 100 per cent oxygen.

Course: He gradually abandoned the use of the accessory muscles of respiration and developed an almost exclusive use of the diaphragm. The weighted buck-shot bag applied to the mid-abdomen was increased from 15 lb. to 30 lb. during the second year. It was used for one-half hour twice daily. The Gordon-Barach emphysema belt was worn constantly when up and around. The pad of the belt moved conspicuously inward during expiration and forward during inspiration. Walking with oxygen was accomplished with the 180 liter 5 lb. cylinder, about one-half mile twice daily, an oxygen flow of 5 to 7 liters per minute being generally employed with the nasal cannula. This apparatus was also used on a bicycle, which the patient rode on weekends, 20 to 25 miles, and at times for short periods in the morning before going to work. After the first year the patient was able to cycle up hills without oxygen that were formerly difficult with oxygen. Walking 30 to 40 blocks did not induce dyspnea, unless carried out after a heavy dinner. He also climbed mountains, 4000 feet, with intermittent use of oxygen.

Medication consisted of a day tablet before breakfast, aminophylline 0.2 gm. and ephedrine 0.015 gm., and a night tablet on retiring, aminophylline 0.35 gm.* Aerosol bronchodilator therapy included the use of mixtures of 2.50 per cent racemic epinephrine hydrochloride with 0.5 per cent atropine sulfate (Dylephrin) or 2.25 per cent racemic epinephrine hydrochloride (Vaponephrin), 5 to 6 drops, with 1 per cent phenylephrine hydrochloride (Neosynephrine) 6 to 8 drops and/or normal saline 6 to 10 drops, nebulized with oxygen, or, at times, the hand-bulb nebulizer with undiluted Dylephrin or Vaponephrin.

The initial improvement in the capacity for exercise, evident within two months, increased gradually during the following year and has been maintained now for three years. During this time he lost 40 lb., the diet having been changed to low fat with high protein. The initial cephalin flocculation test was positive, without other laboratory signs of disordered liver function, and now it is weakly positive or negative.

The blood lactic acid was determined after five minutes of walking with air and with oxygen but no change was evident in this measurement as a result of oxygen inhalation.

The pulmonary function data now reveal the same minute volume breathing air and 100 per cent oxygen; in the last test, October, 1962, the resting P.V. sitting erect was 9.6 liters breathing air and 9.6 liters breathing 100 per cent oxygen. The patient manifested diaphragmatic breathing with little or no chest motion. The vital capacity is generally 4700 ml., increasing to 5000 ml. after a bronchodilator aerosol. The maximal expiratory flow rate with the McKesson Vilator apparatus was 70 liters per minute.

DISCUSSION OF CASES

In Case 1, physical unfitness was pronounced, associated with inability to walk

*The Daint Tablets are manufactured by the Irwin, Nealer Co.
for the two previous years. The degree of walking with oxygen was sufficient to overcome weakness and to restore muscle tone, but the program was never increased to the point of inducing an athletic training response. This woman gained 10 lb. during her period of improvement, presumably with a reversal of a state of negative nitrogen balance, the latter aided by the administration of an anabolic steroid as well as exercise. In Case 2, a loss of 40 lb. was related to a change in diet, as well as to the remarkable increase in exercise, at first mainly with the aid of oxygen and later with both the continuance of oxygen-supported exercise plus the employment of the bicycle for the most part breathing air. There were no changes in the x-ray picture, but it is of interest that inhalation of 100 per cent oxygen for seven minutes no longer induced a fall in minute volume of respiration. This test of respiratory function was employed to determine the efficiency of alveolar ventilation.

The benefit that may be specifically traced to a new therapeutic procedure is often difficult to appraise in patients treated by various other measures. In our original report on the value of a deliberately planned graded exercise program in 1952, it was feasible to add oxygen inhalation during walking to the regimen previously employed for patients with pulmonary emphysema; the beneficial clinical response was mirrored by a striking drop in pulse rate subsequently when a similar degree of exertion was carried out in an air atmosphere; our program to eliminate as far as possible the disordered breathing pattern had already been employed in these cases, as well as other procedures that lessen the dyspnea of physical effort, such as inhalation of bronchodilator aerosols and manual compression of the abdomen. In the studies made in this clinic and others on the response of patients to exercise, the therapeutic measures found of value in the past have not been discarded in an attempt to isolate the precise importance of oxygen supported exercise.

Various physiologic responses to exercise with oxygen in patients with pulmonary emphysema, as well as coronary disease, have recently revealed changes in the direction of normal; the measurements made include those on pulmonary artery pressure, electrocardiogram and arterial oxygen saturation, as well as the previously published data on the pulmonary ventilation referred to above and the prevention of the electrocardiographic signs of coronary insufficiency in patients with angina pectoris.

I selected two case histories that illustrated the range of use of the rehabilitation program. Although the pulmonary function tests were not as extensive as they might have been in a hospital atmosphere, the clinical alterations were striking. Admittedly, training in diaphragmatic breathing was used to eliminate the inefficient and costly dyspnea induced by upper costal and accessory neck muscle respiration. Nevertheless, in Case 1, walking was not possible without the inhalation of oxygen, nor indeed without physical support. The writer's technician spent one hour daily with this woman for the first month, teaching her to walk bending forward, as well as the other measures outlined above that constitute the breathing and oxygen exercise program. Prednisone had little if any effect on the pulmonary physiology since she had no discernible bronchospasm, but may well have contributed at the start to the increase in appetite.

This patient was for a while able to do without oxygen out of doors, one of the conspicuous manifestations of improvement. The progressive enlargement of the bullous lower lobe may have taken place as part of the course of this disease, but it seemed also possible that the ligation of the pulmonary artery played a role in this unfortunate development. Unquestionably, the pathologic processes encountered in chronic pulmonary disease, such as progressive pulmonary fibrosis and vanishing lung or bullous change will be encountered. In other instances, however, the
symptomatic benefit induced by restoration of physical fitness may usher in a prolonged state of well-being.

In the second patient, physical unfitness was not present, at least, of the kind produced by inactivity. The increase in exercise capacity was marked indeed, gradual in onset over the first year, and preserved during the third year. Oxygen was employed in cycling and in walking, and this regimen was continued despite the enhanced capability for physical effort in an air atmosphere. Oxygen inhalation facilitated exercise performance even after breathing training had resulted in an almost exclusive use of the diaphragm. The emphysema belt was crucially helpful in this patient, and made it possible for him to walk in a relatively erect posture. When he was on the bicycle, the posture required of itself induced diaphragmatic respiration. This patient later on climbed a 4,000 foot mountain with the use of the small amount of oxygen in the 6 lb. cylinder apparatus, namely, 180 liters. The writer has encountered no other patient that has extended the exercise concept to the degree manifested in this case. Walking 40 blocks was no longer a problem. This patient represented, in my opinion, an athletic training response to progressive exercise.

**Summary**

Breathlessness on exertion limits the physical activity of patients with severe pulmonary emphysema to such an extent as to result in physical unfitness and muscle atrophy. Oxygen inhalation during a graded exercise program overcame this handicap in some cases. In many others, restoration of diaphragmatic function during walking is required even with the use of oxygen.

The technique of breathing training required for effective use of oxygen supported exercise is described. Elimination of a disordered breathing pattern in patients with obstructive pulmonary emphysema is shown to result in a more efficient alveolar ventilation, and a consequent increase in the capacity for exertion.

Case histories of two patients with pulmonary emphysema are presented: in one, the oxygen supported exercise program resulted in increased exercise capacity associated with the development of physical fitness; in the other, a marked enhancement of exercise capacity took place as a result of mechanisms apparently similar to an athletic training process.

A technique of non-irritant cigarette smoking is described. If tobacco smoke does not enter the lungs, the question is propounded: is there justification for an adamant position in respect to rejection of cigarette smoking? The study of methods of smoking without inhaling deserves more consideration than it has received.

**Resumen**

La disnea de esfuerzo limita la actividad física de los enfermos con enfisema pulmonar grave hasta tal grado que produce incapacidad física y ateria muscular. Las inhalaciones de oxígeno durante la realización de ejercicios graduales vence esta desventaja en algunos casos. En muchos otros se requiere la restauración de la función diafragmática durante la marcha aún con el uso de oxígeno.

Se describe la técnica del adiestramiento respiratorio por medio del uso efectivo del oxígeno. Se demuestra que la eliminación del trastorno del cuadro de la respiración en enfermos con enfisema obstructivo produce mayor ventilación alveolar y por consecuencia un aumento de la capacidad para el ejercicio.

Se presentan dos casos de enfermos con enfisema. En uno el plan de tratamiento con oxígeno como apoyo del ejercicio produjo un aumento de la capacidad al ejercicio asociado a desarrollo de mayor aptitud física. En otro se observó un marcado aumento de la capacidad de ejercicio como resultado de un plan de entrenamiento semejante al atlético.

Se describe una técnica de fumar cigarrillos que no es irritante. Si el humo del tabaco no entra a los pulmones se plantea la pregunta: ¿está justificada la actitud inflexible respecto del prohibir el fumar cigarrillos? El estudio de los métodos de fumar sin inhalar requiere más consideración de la hasta ahora recibida.

**Resumé**

La dyspnée d'effort limite l'activité physique des malades atteints d'emp lysème grave du pou-
mon à tel point qu’il en résulte une inadaptation physique et une atrophie musculaire. L’inhalation d’oxygène selon un programme d’exercice progressif surmonte ce handicap dans certains cas. Dans beaucoup d’autres, il est nécessaire de restaurer la fonction diaphragmatique pendant la marche, même avec l’emploi d’oxygène.

L’auteur décrit la technique de rééducation respiratoire nécessaire par l’utilisation effective d’oxygène pendant l’effort. L’élimination d’une perturbation dans le tracé respiratoire des malades atteints d’emphysème pulmonaire obstructif montre qu’il en résulte une ventilation alvéolaire plus efficace, et en conséquence une augmentation de la capacité à l’effort.

L’auteur présente l’histoire de deux malades atteints d’emphysème pulmonaire: chez l’un, le programme d’administration d’oxygène pendant l’effort se traduit par l’augmentation de la capacité d’effort associée au développement de l’adaptation physique; chez l’autre, l’augmentation nette de la capacité d’effort se comporte selon un mécanisme apparemment semblable à un processus d’entraînement athlétique.

L’auteur décrit un procédé pour fumer sans irritation. Si la fumée n’entre pas dans les poumons, la question est posée: est-il justifié de rechercher une position favorable en ce qui concerne le rejet de la fumée de cigarette? L’étude des méthodes permettant de fumer sans inhaler la fumée mérite plus de considération qu’elle n’en a reçu.

ZUSAMMENFASSUNG

Kurzatmigkeit unter Belastung begrenzt die körperliche Aktivität von Patienten mit schweren Lungenemphysem in einem solchen Grade, daß daraus eine physikalische Untauglichkeit und Muskelatrophie resultiert. Eine Sauerstoffinha-

FURTHER EXPERIENCES WITH METHYL DOPA

Twenty hypertensive patients were treated with methyl dopa for periods ranging from six weeks to nine months. The result was good in 15 cases. The daily dose varied between 0.5 gm. to 6 gm. with 13 patients requiring less than 3 gm. No toxic effects were observed. The side effects were mainly dryness of the mouth and drowsiness. In three cases, it was possible to reduce the dosage after some time. One patient may have developed tolerance. It would appear that methyl dopa is a valuable new hypotensive agent.


SWEAT ELECTROLYTES

Two groups of men were studied, a control group of hospital patients and a test group of patients with chronic lung disease. The subjects were exposed to a standardized thermal stimulus and sweat was collected from the forehead, chest, abdomen, and palm. Sweat was collected for six 20-minute periods during which the rate of sweating and the chloride and sodium concentrations increased logarithmically. When the sweat volumes from the two groups were compared, the rate of sweating was found to be higher on the forehead than on the abdomen or chest; these three latter rates were much higher than those for the palm. The chloride concentrations of the sweat collected from the abdomen, forehead, and chest were significantly higher in the test group with chronic pulmonary disease than in the control group.