# DISABILITY APPRAISAL OF MINERS

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# PART ONE

# INTRODUCTION

An accurate determination of the degree of systemic disability is difficult. This is especially true in relation to the respiratory system. Before one can proceed to determine whether or not disability exists, it is necessary to define the term disability. Disability is present when an individual can no longer perform his usual work. Because an individual no longer possesses the capacity to do his job does not imply that he is incapable of earning a livelihood in an occupation that requires less effort. In the determination of whether or not disability exists two questions must be answered. One, what capacity is necessary for the person to perform the work, and two, is the capacity that remains more or less than that required? If the capacity that remains is less than that which is necessary the individual is disabled. From this it follows that the essential pre-requisites for those engaged in the determination of the existence and the severity of disability which results from occupational diseases of the chest are familiarity with the job requirements and knowledge of the pulmonary function tests, their normal values and limitations. The tests used commonly are discussed in part II.

Mere determination of the function that is present, however, is insufficient, because some individuals may have little or no impairment of function; yet, they are unable to work! These persons are in no sense malingerers but may be affected by psychic disturbances which may be equally as incapacitating as organic disease processes. Psychic disturbances, however, are over-looked frequently

by physicians as etiologic factors in the production of disability. Many investigators have observed abnormal patterns of breathing during periods of stress. They are accompanied often by symptoms and physical findings; asthma, precipitated or aggravated by emotional disturbances, is an excellent example. Some of the changes in the pattern of breathing produced by psychic disturbances are discussed in part III.

All persons are subjected during their lives to episodes of psychic trauma of various degrees. Not infrequently these occur at work. Since mining is a hazardous and arduous occupation, it would not be unreasonable to suspect that miners are subjected to an unusual number of these episodes. It is not surprising, therefore, that the incidence ofpsychiatric illnesses in miners has been found by British observers to be greater than that found in persons engaged in less hazardous occupations. Not only the hazardous nature of the work, but the physical environment of the work-place, and the socio-economic conditions peculiar to mining communities have been suggested as factors which contribute to a high incidence of psychiatric disorders. These factors are discussed in part IV.

Fifty-one miners were studied during the period from August 20, 1951, to January 5, 1953. Analysis of the case records for the most significant diagnosis in each case gave the following results:

I. Pneumoconiosis. Sixteen cases (31.4%) were present. This group was comprised of four cases of silicosis, eight cases of anthracosis, two cases of anthracosilicosis, one case of pneumoconiosis, $P_2$, and one case of pneumoconiosis, $P_1$.

II. Psychoneurosis. Thirteen cases (25.5%) had a psychoneurosis as the most significant diagnosis.

III. Emphysema, pulmonary, etiology undetermined. This was the major diagnosis in eight cases (15.6%). Emphysema, of varying degrees of sewerity, was present in sixteen additional cases.

IV. Bronchial asthma. This was the most significant diagnosis in six cases(11.8%).

V. Arthritis, hypertrophic (various anatomical sites). This was the most significant diagnosis in three cases (5.9%).

VI. Miscellaneous diagnoses. Five cases (9.8%) were in this group which consisted of one case of each of the following diagnoses: (a) heart disease, etiology undetermined, (b) trachoma, acute, inactive, (c) papilloma, benign, left vocal cord, (d) bronchitis and bronchiolitis, (e) hyper-mobile semi-lunar cartilage, left knee.

In regard to disability, the patients were placed in one of the following groups:

I. Patients with no disability. Two patients (3.9%) were in this group.

II. Patients with mild psychiatric disturbances with no physical disability. Seven patients (13.7%) were in this group.

III. Patients with marked psychiatric disturbances with

no physical disability. Eight patients (15.7%) were in this group.

IV. Patients with organic disease and psychiatric disturbances with various degrees of disability. Twenty-four patients (47.1%) were in this group.

V. Patients with organic disease without significant psychiatric disturbances. Ten patients (19.5%) were in this group.

Ten patients were believed to be able to return to their occupation of mining. Rehabilitation with employment other than mining was considered possible for thirty-two additional patients, questionable for seven, and not possible for two.

The results of this study are discussed in part V.

# PART TWO

# PULMONARY FUNCTION TESTS

## GENERAL CONSIDERATIONS

Pulmonary function tests are physiologic measurements designed to provide a means for appraising the ability of the lungs to function in their capacity of gas exchange. These tests cannot provide a specific diagnosis, and normal results may be obtained in the presence of pulmonary disease. They demonstrate alterations of specific lung function which include the ventilation and distribution of gases in the lungs and the diffusion of gases across the alveolar structures.

These tests may be used to estimate the amount of pulmonary function that remains in persons with occupational and non-occupational diseases, to distinguish functional from organic diseases, to differentiate cardiac from pulmonary diseases, to study patients prior to thoracic surgery and various types of collapse therapy, and for the early detection of impaired function in individuals working in dusty environments.

Unfortunately, there is no single test which explores all of the functions performed by the lungs; therefore, multiple tests must be carried out on every individual being studied.

The interpretation of the results of these tests is often difficult because of the lack of sufficient numbers of normal values in large groups of healthy persons of different ages, sex, and stature. In addition there are differences in the types of equipment utilized and in the experience of the operators. Full cooperation of the patient is necessary. In this regard, the experienced operator is invaluable for obtaining the best possible response from each patient and for

estimating the degree of cooperation. The results of pulmonary function tests should be interpreted cautiously because of these factors.

Pulmonary function tests measure only the amount of function present at the time the tests are carried out. The exact loss or gain of function cannot be determined unless previous results on the individual are available for comparison. Unless these data are available, the wide normal ranges peculiar to most of these tests preclude all but qualified generalizations pertaining to the loss of pulmonary function, except, in moderately advanced or advanced cases.

## THE TOTAL LUNG CAPACITY AND ITS SUBDIVISIONS

The total lung capacity is the sum of the vital capacity and the residual volume; it is the maximum amount of gas that can be contained in the lung when it is fully expanded (87).

The vital capacity is the maximum volume of gas that can be expelled from the lungs by forceful effort following a maximum inspiration (87).

The inspiratory capacity (complemental or complementary air) is the maximum amount of gas that can be inspired from the end-expiratory level(87).

The inspiratory reserve volume (complemental or complementary air, complemental air minus tidal air, inspiratory capacity minus tidal volume) is the maximum amount of gas that can be inspired from the end-inspiratory position (87).

The expiratory reserve volume (reserve or supplemental air) is the maximum volume of gas that can be expired from the resting end-expiratory position. In function studies the resting, end-expiratory position is always used (87).

The functional residual capacity (functional residual air, subtidal volume, equilibrum capacity, normal capacity, mid-capacity) is the volume of gas remaining in the lungs in the resting, end-expiratory position (87).

The residual volume (residual capacity, residual air) is the volume of gas remaining in the lungs at the end of a maximum expiration (87).

A diagram illustrating the total lung volume and its subdivisions is presented in Figure 1, page /2f, in the appendix.

## THE VITAL CAPACITY

The vital capacity has been used for a long period of time to determine the loss of function in cardio-respiratory diseases. Some of the inadequacies of this procedure as performed commonly will be cited later in the discussion.

The affect on the vital capacity of various factors such as age, sex, height, physical training, and position are well known. Kaltreider et al (61), after a review of the literature, concluded that the vital capacity is only slightly decreased during the age span between 40 to 50 years; however, there is a more rapid decrease in subsequent years. Baldwin et al (6) found that the vital capacity decreases with an increase in age, increases with height, and does not correlate with weight. Rahn et al (95) found small changes when the afternoon and morning determinations were compared. An average standard deviation of 111 cc. was noted when comparisons were made over a period of two to three months. Stephen (102) observed a decrease in volume when subjects changed from a supine to a prone position and a slight increase from a recumbent to a sitting position. Mills (76) is of the opinion that the decrease in vital capacity in the supine position compared to the standing position is related to an increase in the pulmonary blood volume. Normal values for the vital capacity are listed in Table 1, page /03, in the appendix.

The vital capacity has a wide normal range. Moersch (77), cited by Bloomer (14), in a study of 1000 consecutive patients admitted to the Mayo Clinic for surgical treatment, found that a sufficient

number of the group had vital capacities below 85 per cent of the normal to warrant the conclusion that this finding could not be considered as a definite indication of disease in all cases. In the evaluation of the results obtained in lung volume studies, Hurtado and Boller (54) concluded that an impairment in the alveolar ventilation must be suspected if the vital capacity is less than 65 per cent of the total volume.

Baldwin et al (6) have developed regression formulae which include the factors of height, weight, and age. They are:

1. For males

$$\text{vital capacity} = \left[27.63 - (0.112 \times \text{age in years})\right] \times \text{height in cm.}$$

2. For females

$$\text{vital capacity} = \left[21.78 - (0.101 \times \text{age in years})\right] \times \text{height in cm.}$$

These investigators state that if the combined vital capacity (the total of the separate determinations of the inspiratory capacity and expiratory reserve volume) is smaller than the single measurement of vital capacity, the subject is not cooperating fully. This "two-stage" value may exceed the "one-stage value by as much as a liter in patients with asthma or emphysema.

Hurtado and Fray (55) developed regression formulae for predicting the vital capacity. They used the surface area of the lung fields from the posterior-anterior x-ray film of the chest in full inspiration and the anterior-posterior diameter of the chest. The predicted values obtained by this method showed relatively good correlation with the absolute values in healthy adult males. As a result of observations in which this method was used, they concluded that deviations in

the observed vital capacity (as compared to those predicted) are significant when there is a difference (decreased) greater than 15 per cent. Kaltreider et al (61) included the factor of age in the regression formula utilizing this method ("radiological chest volume").

Mechanisms which reduce the vital capacity are: (1) the available air space is encroached upon by some space-occupying lesion; (2) the mechanics of the chest wall or diaphragm are altered by some deformity or by paralysis; (3) the elasticity of the lungs is decreased, as in emphysema, inflammatory conditions, fibrosis, etc.; (4) the pleural surfaces have become adherent following some pleuritic process; (5) the distensibility of the lungs has been impaired, as in cardiac decompensation, edema, bronchiolar spasm, fibrosis etc. (14). In addition, the unwillingness or inability of the patient to comprehend or cooperate is a very important factor. Matheson et al (73) found that the introduction of an artificial air-flow resistance depressed profoundly the ventilation capacity and the capacity ratio (ventilation capacity/vital capacity) without altering the vital capacity. Experimental reduction in the vital capacity, which was accomplished by the means of a canvass vest, reduced the ventilation capacity proportionately with no change in the capacity ratio (73).

Goggio (46) demonstrated the inadequacy of the vital capacity determination without reference to time when it is used to measure impairment of function in patients with alterations in the elasticity or distensibility of the lungs. The importance of understanding this concept merits the quotation of portions of his discussion.

"It is at once evident that these three cases, which have in common extreme restriction in exercise tolerance, vary markedly in history and in various physical factors, Case 1, with a vital capacity of 1000 cc. and an arterial oxygne saturation of 60 per cent, was orthopneic, but not extremely so when not excited. Case 2, with a vital capacity of only 1800 cc. and an arterial oxygen saturation of 87 per cent, was not much more restricted in activity than case 3, who had a vital capacity of 4200 cc. and an oxygen saturation of 89 per cent.

In considering cases 2 and 3, with vital capacities of 1800 cc. and 4200 cc., respectively, yet similarly restricted exercise tolerance, one might conclude at first glance that the patient in Case 3, must have had a much greater impermeability of the respiratory epithelium or some much more marked defect of the pulmonary capillary network than did the patient in Case 2 to account for this startling incongruity. Such a conclusion is not justified. By making a kymographic tracing of the vital-capacity measurement a serious fault in the usual method of vital capacity measurement was exposed.

In Figure 2*, three of the solid lines show the vital capacity measurements of a normal subject and of Cases 2 and 3 as recorded kymographically. It is strikingly apparent that whereas the patient in Case 3 had a normal vital capacity, it required 20 seconds for him to exhale the same amount of air as was exhaled by the normal subject in one and a half seconds with ease. To illustrate the significance of this time factor, let it be supposed that the respiratory rate of both patients was 20, with three seconds per complete respiratory cycle

* Page 12? in the Appendix

and only one and a half seconds for the expiratory phase. It then becomes apparent that in this one and a half seconds the normal subject could exhale (and inhale) 4 liters of air if necessary, whereas the patient in Case 3 could exhale only some 900 cc. in the same period of time, and this with maximum effort. Although the latter had a vital capacity of 4200 cc., only a small fraction of this was available in the carrying out of any respiratory function, the cause of his marked limitation of exercise tolerance in spite of a quantitatively normal vital capacity can be logically explained on the basis of mechanical inability to ventilate the lungs".

Other workers have recognized this inadequacy (73, 16, 43, 79, 33). Recently, equipment made commercially has become available for the determination of the "timed-vital capacity" (23).

Baldwin et al (8) studied 39 cases of pulmonary fibrosis. The patients were divided into two groups as follows: Group 1. Those in whom the symptoms and abnormal physical findings were limited and not correlated in any way with the extent of the disability as demonstrated by x-ray. Of the twenty-five patients included in this group, there were eight cases of silicosis, six with chronic pulmonary infection, five with Boeck's Sarcoid, two with irradiation fibrosis and four of undetermined etiology. The mean vital capacity was reduced to 75 per cent of the predicted value in this group. Group 2. Those in whom the severity of the clinical symptoms far outweighed the relatively limited physical findings and frequently unimpressive x-ray findings. Of the fourteen patients in this group there were three cases of pulmonary

fibrosis associated with scleroderma, one with a history of exposure to the inhalation of sulfur-dioxide, one with a history of exposure by inhalation to asbestos fibers, two of lymphangitic carcinoma, and seven cases of pulmonary fibrosis following a mild influenza-like respiratory infection. The mean vital capacity in this group was 46% of the predicted value.

Hurtado and Fray (59) in a study of fifty-eight patients with pulmonary fibrosis found a mean value of 2.86 liters for the vital capacity compared to a mean predicted value of 4.55 liters. Both components, the inspiratory capacity and the expiratory reserve volume, were affected equally. Roelsen and Bay (96) found the vital capacity "very distinctly reduced in silicotics of Stage III". Stage III was defined as, "Characterized by strong, diffuse veiling and eventual appearance of larger nodules, which sometimes fuse into larger masses". In six cases of pneumoconiosis, Hurtado and Fray (57) found the vital capacity reduced in proportion to the total capacity; however, the expiratory reserve volume and the inspiratory capacity showed a normal relationship to each other.

In a study of patients with chronic pulmonary emphysema, Baldwin et al (7) did not find any close correlation between the variations of the vital capacity and those of the maximum breathing capacity. Motley et al (81) found good correlation between the increase of residual volume and the decrease of the vital and maximum breathing capacities as the degree of emphysema increased. The maximum breathing capacity, however, decreased more in proportion to the increase of emphysema than

did the vital capacity. In nine cases of pulmonary emphysema Hurtado and Fray (57) found that the observed vital capacity varied from 30.8 to 92.8 per cent of the predicted volume. The reduction of the vital capacity occurred chiefly because of a reduction of the inspiratory capacity.

Baldwin et al (9) observed the vital capacities in 16 cases of large pulmonary cysts or bullae. These patients were grouped as follows: Group I. Three cases with large air cysts communicating freely with the apparently normal remaining lungs. The vital capacities observed were 112, 89, and 77 per cent of the predicted normals. Group II. Three cases with large air cysts with poor or intermittent bronchial communication and associated with apparently normal remaining lungs. The vital capacities observed were 24, 66, and 51 per cent of the predicted normals. Group III. Four cases with air cysts with poor or intermittent bronchial communication associated with chronic diffuse emphysema in the remaining lung. The vital capacities varied between 52 and 77 per cent of the predicted values. Group IV. Six cases with air cysts with poor or intermittent bronchial communication associated with chronic emphysema. The vital capacities varied between 17 and 56 per cent of the predicted values.

Lindskog (68) frequently observed increases in the vital capacities of patients who had been subjected to resections of the lung for bronchiectasis.

## THE INSPIRATORY CAPACITY

The inspiratory capacity is determined usually by the use of spirographic techniques. Normally, it comprises approximately 75 to 80 per cent of the vital capacity (27). Thedetermination of this portion of the lung volume requires a maximum voluntary inepiratory effort by the patient. Normal values determined by various workers are presented in Table 2, page 104, in the Appendix.

This value may be decreased by: (1) an unwillingness to cooperate, (2) failure to comprehend, (3) physical deformities, (4) malingering, (5) age, (6) posture, (7) fatigue, (8) respiratory resistance and (9) disease states.

In patients with emphysema, the inspiratory capacity is smaller, and the residual volume is larger (81, 57, 82). Hurtado et al (59) noted that the decrease in the inspiratory capacity in patients with pulmonary fibrosis was not as large as that which occurred in emphysema.

Kaltreider et al (61) observed a slight to marked increase in the inspiratory volume in changing from a sitting to a recumbent position, while the remainder of the subdivisions of the total lung volume decreased.

## THE EXPIRATORY RESERVE VOLUME

The expiratory reserve volume is usually determined from a spirographic tracing. The determination of this subdivision of the total lung capacity requires a maximum voluntary expiratory effort from the resting end-expiratory position. This volume is influenced by many of the factors which affect the inspiratory capacity. Christie (20) noted variations greater than 300 cc. in normal and diseased subjects after excluding many of these factors and concluded the variations were due to "fortuitous changes in muscular effort rather than changes in the resting respiratory level". Rahn et al (95) demonstrated daily variations in reserve air in five subjects, the standard deviation ranging from 109 to 169 cc.. Aslett (4) showed that the relative expiratory reserve volume (expressed as percentage of total lung volume) is substantially decreased in obese persons, but this is associated with a compensatory increase in the inspiratory capacity. The effect of age on reduction of the expiratory reserve volume has been observed by many workers (61, 4, 50, 6). Normal values reported by ravious workers are presented in Table 3, page 165 , in the Appendix.

In severe cases of emphysema, Hurtado et al (57) found the inspiratory capacity made up a smaller portion of the vital capacity than in normal cases. In two cases in which this relationship was found, the expiratory reserve volume was normal. They concluded that when the vital capacity is composed chiefly of reserve air, or when the latter volume is not so markedly reduced as is the inspiratory capacity, there is probably a further handicap to effective alveolar ventilation. In miners, Motley et al (82) found that the average proportion of total

lung volume occupied by the expiratory reserve volume and tidal air remained fairly constant with only a slight tendency toward a reduction in these measurements in cases with far advanced emphysema.

Hurtado et al (59) observed that there was approximately an equal reduction in the inspiratory capacity and the expiratory reserve volume in relation to the total capacity of patients with pulmonary fibrosis.

## THE FUNCTIONAL RESIDUAL CAPACITY

The functional residual capacity is the total of the expiratory reserve and the residual volumes. Hurtado and Fray (55) found that this volume constituted approximately 38 per cent of the total lung volume. Normal values for this subdivision of the lung volume are presented in Table 5, page 107, in the appendix.

This portion of the lung volume is usually increased when the residual volume is elevated. Occasionally the residual volume may be increased without an increase in the functional residual capacity because of a reduction in the expiratory reserve volume.

Correlation between the thoracic measurements and this subdivision was obtained by Lundsgaard and Van Slyke (70). Hurtado and Fray (55) were unable to demonstrate any such relationship to external chest measurements. The best correlation observed by them existed between the area of the lung fields, on a film taken at the end of a normal expiration, and the functional residual capacity.

In patients with pulmonary fibrosis, Hurtado et al (59) found that the functional residual capacity was elevated significantly only in patients with chronic bronchial asthma and in those with large dense shadows in the upper lobes and evidence of emphysema in the bases.

The determined functional residual capacity was much larger than the value predicted from the radiographic measurements in patients with pulmonary emphysema (57).

## THE RESIDUAL VOLUME

Normal values for the residual volume are presented in Table 4, page 106, in the Appendix.

Daily variations of this volume have been observed by Rahn et al (95). Christie (20) observed differences under carefully controlled conditions and thought that they were caused by changes in the expiratory reserve volume which were "fortuitous in nature". Hurtado and Fray (56) observed a slight decrease in the residual volume in subjects who changed from a sitting to a recumbent position. Greifenstein et al (50) obtained high values in normal elderly persons. They postulated that the increase with advancing age may be due to: (1) the accumulated effect of a variety of pulmonary stresses or infections over a period of many years, (2) an aging process which leads to a diminution in pulmonary elasticity, and (3) postural changes in the thoracic spine.

When the residual volume is increased, hyperinflation of the lungs exists. This may occur as a result of: (1) structural changes such as diminution in the elastic fibers of the lung, tearing of alveolar septa, and reduction in the pulmonary vascular bed (true emphysema), (2) obstruction in the airway (asthma, etc.), (3) compensatory overinflation of the lung following surgical removal of lung tissue, (4) deformity of the thorax, (5) decrease in the elasticity of the lung such as appears to be a natural accompaniment of old age, and (6) a variety of pulmonary disorders including congestive heart failure and certain types of pulmonary fibrosis (27).

Christie and Meakins (75) believed that the efficiency of pulmonary ventilation was related to the ratio of the residual volume to the total capacity. This view has since gained the support of many investigators. Hurtado and Boller (54) found that the residual volume expressed as per cent of the total capacity in normal subjects varied from 14 to 31 per cent, with a mean of 22.0 $\pm$ .41 per cent. A greater variation was observed in the residual volume than in the vital capacity.

Hurtado and Fray (55) found little correlation of the residual volume with radiological measurements and nor correlation with external chest measurements. Some correlation was found between the degree of chest expansion (from the ratio of the radiological area at maximum expiration to the radiological area at maximum inspiration) and the relative proportions of subdivisions of the pulmonary capacity. Deficient expansion tends to be accompanied by a higher percentage of residual volume in relation to the total capacity. An increase in the ratio of the $\frac{\text{residual volume}}{\text{total capacity}}$ x 100 with advancing age has been observed (4, 50). Griefenstein et al (50) found the mean of this ratio in elderly individuals with no pulmonary incapacity and normal lung volumes to be 40.9 per cent.

An increase in the ratio does not necessarily imply that the absolute value for the residual volume is also greater than normal. Similarly, with a decrease in lung volume caused by changes in the chest wall or pleura, fibrosis or congestion, there may be a proportional decrease from the predicted values of the vital capacity and the residual volume with no change in the ratio of the $\frac{\text{residual volume}}{\text{total capacity}}$ x 100. Baldwin et al (6) believe that the finding of a normal value for this

ratio may be compatible with the existence of small localized areas of emphysema. They state that if this ratio is significantly increased "to a value over 35 per cent, an over all relative state of hyperinflation or emphysema probably exists. There is also the condition of diminished expansibility of the lung with a relatively normal residual volume, which may be seen in some patients with congestive failure, resulting in a small elevation in the ratio $\frac{\text{residual volume}}{\text{total lung volume}} \times 100$". Kaltreider et al (61) also state that this ratio rarely exceeds 35 per cent in normal individuals.

Hurtado and Fray (57), in a study of nine cases of pulmonary emphysema, observed a large increase in residual volume which correlated with the severity of the symptoms. In six cases of pneumoconiosis the residual volume was elevated, and because of this, emphysema was thought to be associated with fibrotic changes (57). The functional residual capacity was found to be normal in most instances, but the ratio of the $\frac{\text{residual volume}}{\text{total capacity}} \times 100$ was elevated, primarily because of a decrease in the vital capacity.

Fifty-eight cases of pulmonary fibrosis were studied by Hurtado et al (59). The patients were classified according to roentgenologic criteria as follows: Group I. This group consisted of 23 cases with increased linear markings in the lung fields; very slight feathering and beading were observed in a few instances. Group II. Seven cases with a history of chronic bronchial asthma are included in this group. The roentgenographic findings were similar to Group I, but in addition the shadow of the diaphragm appeared low and flat, and the intercostal

spaces were widened. Group III. Seventeen patients in this group showed nodular shadows. Group IV. In this group of four cases, the nodular shadows showed a tendency to agglomerate giving a mottled appearance. Group V. Four patients in this group presented large dense shadows, chiefly in the upper portions of the lung, and in addition showed marked emphysema at the bases of the lungs. Group VI. A fine and diffuse reticular fibrosis involving the entire lung field was present in 3 cases in this group.

The occupations listed for the patients were: (1) sandblasting, 10 patients, (2) iron moulding and foundry work, 15 patients, (3) mixed occupations (probably exposed to silica dust), 20 patients, (4) coal mining, 8 patients, (5) stone cutting, 1 patient, and (6) apparently irrelevant occupations (questionably infectious etiology), 4 patients.

The persons engaged in sand-blasting gave the shortest histories of exposure, had the shortest intervals between the exposure and the development of symptoms, and exhibited the most marked alterations in its subdivisions. Data from t his study are presented in Table 8, page 109, in the Appendix.

In this study a correlation was shown to exist between the ratio of the residual volume to the total capacity and the degree of respiratory disability in association with pulmonary fibrosis. Dyspnea on slight exertion was noted by fourteen patients in whom the mean value for the ratio of the residual volume to the total capacity times 100 was 49.1 per cent. The authors concluded that the ratio of residual volume to total capacity is possibly abnormal when greater than 30 per

cent, is definitely beyond the limits of normal if over 35 per cent, and is moderately abnormal if not higher than 45 per cent. When greater than 45 per cent there was a severe degree of respiratory disability, accompanied usually by a decrease in the saturation of the arterial blood with oxygen. Baldwin et al (7) found a similar correlation in most of the cases of emphysema studied by them. Roelson and Bay (96) observed an increase of this ratio in the more advanced stages of silicosis.

Thirty-nine cases of pulmonary fibrosis were studied by Baldwin and associates (8). The patients were placed in one of two groups: Group I. Those with pulmonary fibrosis with moderate ventilatory insufficiency without obvious disturbance of either the distribution or the diffusion factors. The range of the residual volumes was 36 to 110 per cent, the mean value being 76 per cent of the predicted value. The mean value for the ratio $\frac{\text{residual volume}}{\text{total capacity}} \times 100$ was 27 per cent. Group II. Those with pulmonary fibrosis in association with alveolo-respiratory insufficiency and only minimal impairment of the ventilatory function. The range of the residual volumes was 37 to 99 per cent of the predicted values. The mean value for the ratio was 33 per cent.

The value for this ratio was determined by Cournand (30) in 158 patients. In 17 normal persons it was approximately 22 per cent; and in 22 chronic cardiac patients it was elevated to 30 per cent. Thirty-six patients with pulmonary fibrosis or emphysema had the highest mean value, which was approximately 52 per cent. Forty-six patients with chronic pulmonary tuberculosis, including patients with pneumothorax

or thorocoplasty, had a mean value of 30 per cent. Twenty-two patients with miscellaneous diagnoses consisting of bronchiectasis, carcinoma of the bronchus, chest deformities and pneumonectomy had a mean value of the ratio of approximately 30 per cent.

Motley (80) has formulated a classification for estimating the degree of emphysema based on this ratio. This classification is as follows:

| Group | Degree of Emphysema | Residual Volume Percentage of Total Lung Volume | |
|---|---|---|---|
| I | None | 25% or less | Degree insignificant |
| II | Slight | 25 - 35% | |
| III | Moderate | 35 - 45% | Degree significant |
| IV | Advanced | 45 - 55% | |
| V | Far advanced | 55% or above | |

In a review of one hundred anthracite coal miners with pulmonary complaints, the above classification correlated well with other pulmonary function measurements (79). No correlation, however, could be established between the roentgenologic stage of silicosis and the degree of emphysema based on the above classification (80). These data are presented in Table 9, page 110, in the Appendix.

Baldwin et al (9) noted a minimal increase of this ratio in patients with large air cysts communicating freely with the bronchial tree and associated with apparently normal remaining lungs. In patients with air cysts communicating poorly or intermittently with bronchi, in association with chronic diffuse emphysema in the remaining lungs and disability due to ventilatory insufficiency, the ratio was larger than

normal. In a group of patients with air cysts communicating poorly or intermittently with bronchi, in association with chronic diffuse emphysema in the remaining lungs and disability due to both ventilatory and alveolar respiratory insufficiency, the ratio was high.

## THE TOTAL LUNG CAPACITY

Normal values observed by various workers are presented in Table 6, page 108, in the Appendix. The determined volume may vary ± 15 to 20 per cent from the predicted normal value in healthy subjects (6).

The predicted volume may be calculated from one of the following formulae (6):

15 - 34 years $\frac{\text{vital capacity}}{80.0} \times 100$

35 - 49 years $\frac{\text{vital capacity}}{76.6} \times 100$

above 50 years $\frac{\text{vital capacity}}{69.2} \times 100$

Hurtado and Fray (55) found good correlation of the total lung capacity with the "radiological chest volume" at maximum inspiration. They were unable to demonstrate a correlation between the total capacity and its main subdivisions, and the "chest volume" calculated from external measurements. A relationship was shown to exist between the general shape of the chest and total capacity of the lungs. Individuals with broad, muscular chests and high diaphragms tend to have lower total capacities than those with long, narrow chests.

Birath (12) observed a tendency toward diminution of the total capacity in patients with chronic tuberculosis. Lindskog (68) found that lung resections were often followed by actual increases in the total lung volume in patients with brochiectasis. This was attributed to the improved ventilatory mechanics exerted by a combination of several factors: (1) the removal of a poorly functioning, space-occupying portion of diseased lung with subsequent compensatory expansion of

the remaining lung tissue; (2) increased distensibility and elasticity of adjacent areas of lung after subsidence of the inflammatory response which had also involved them because of their proximity to the original lesion; and (3) absence of exudate in the trachea and bronchial tree.

In patients with pulmonary emphysema (57), the total capacity of the lungs corresponded with the value calculated from measurements of radiographs of the chest. Baldwin et al (7) also found that the determined values agreed well with the predicted values in patients with emphysema.

In patients with pneumoconioses, the determined total capacity was generally lower than the predicted value to an extent that was comparable to the severity of the disease as determined radiologically and clinically (57). Motley (82) observed that as the degree of emphysema increased, determined by an increase in the residual air volume, the total lung capacity decreased progressively from essentially the normal predicted value in the group with "slight" emphysema to 16.1 per cent in the "far advanced group".

The decrease in total capacity correlated with the nature and extent of the pulmonary lesions seen in the roentgenographic films in patients with pulmonary fibrosis (59). This correlation, however, was not obtained in patients with chronic bronchial asthma. In those cases the total capacity approximated the predicted normal values.

Patients with large air cysts which communicated freely with the bronchial tree and normal remaining lung tissue had relatively normal total lung capacities (9); whereas, the total lung capacities were greatly

and uniformly restricted in all patients with air cysts which communicated poorly or intermittently with bronchi. Marked reductions in the total capacity were observed in patients with air cysts which communicated poorly or intermittently with bronchi, in association with varying degrees of emphysema (9).

## THE MAXIMUM BREATHING CAPACITY AND RELATED TESTS

The maximum breathing capacity (M.B.C., voluntary ventilation capacity, maximum voluntary ventilation, maximum minute ventilation) is the maximum volume of gas that can be breathed per minute.

Wright (110) has reported values ranging from 90 to 210 liters per minute, the mean value of these determinations was 147 liters per minute, and the standard deviation was 25.81 ± 1.231 liters. From this it may be seen that a normal man may vary from his predicted normal figure by as much as ± 35 per cent. Other investigators have observed wide variations of the M.B.C. in normal persons (6, 48).

The maximum breathing capacity decreases with an increase in the body surface (4, 49) and age (4, 50, 110) of normal persons. Greifenstein et al (50) observed that the reduction of the maximum breathing capacity was proportionately greater than that of the vital capacity in normal elderly subjects. They believe that this may be due to loss of pulmonary elasticity, to bronchial obstruction, or to changes in the thorax and the diaphragm. Baldwin et al (6) have developed regression formulae which are based on age and body surface area, for predicting the maximum breathing capacity. They are as follows:

For males

$[86.5 - (0.522 \times \text{age in years})] \times$ square meters of body surface

For females

$[71.3 - (0.474 \times \text{age in years})] \times$ square meters of body surface

Dripps and Comroe (36) compa red the maximum breathing capacities of persons subjected to the inhalation of 7.6 and 10.4 per cent carbon

dioxide, to exhausting muscular exercise, and to maximum voluntary hyperventilation. The average values were 48.9, 71.4, 109.6 and 166 liters per minute respectively. From these data, it is apparent that maximum ventilation is obtained by voluntary effort. Performance is not usually improved after the first practice trial (48). Fatigue produced by successive determinations does not cause a significant reduction (4 to 5 per cent) in the maximum breathing capacity (48).

The factors (31) which are necessary to maintain an adequate maximum breathing capacity are: (1) patency of the tracheo-bronchial airways, (2) good elasticity and distensibility in the lungs, and (3) an effective bellows-like action of the thoracic cage.

Reduction of the maximum capacity may be produced by one or more of the following mechanisms: (1) from an abnormality of the chest wall, or a disturbance of the neuromuscular apparatus of breathing, such as occurs in advanced kyphoscoliosis, or as the result of diaphragmatic paralysis; (2) from constriction or obstruction in the air passages, such as occurs in asthma or obstructive emphysema; or (3) from limitation in pulmonary elasticity, such as occurs in fibrosis or pulmonary congestion (31). Matheson et al (73) reduced the maximum breathing capacity by reducing the vital capacity (by the use of a canvass vest) and by the introduction of artificial resistances to air-flow in the air-way. With the latter method, the maximum breathing capacity was reduced without altering the vital capacity. On the other hand, in some patients with diffuse pulmonary pathology, such as is seen in beryllosis, the maximum breathing capacity may remain high despite a fairly marked re-

duction in the vital capacity (109). This is easily understood because the last increment of the vital capacity is attained with great expenditure of time and effort. Because of this, Cournand et al (32) found the maximum rapid breathing capacity larger than the maximum deep breathing capacity in every instance. A normal individual may, therefore, employ only fifty per cent of his vital capacity (e.g. 4000 cc.) in each complete respiratory excursion while engaging in a test of his maximum breathing capacity. If a patient with diffuse pulmonary infiltration or fibrosis has a vital capacity of 3000 cc. and he is capable of utilizing two thirds of this with each breath during a determination of his breathing capacity, the resultant maximum breathing capacity may, therefore, equal that of a normal person.

Baldwin (8), in a study of twenty-five cases of pulmonary fibrosis, found that the maximum breathing capacity did not correlate well with the physical findings or the extent of the disease as demonstrated by x-ray examination.

Wright and Woodruff (108), cited by Waring (105), observed that temporary phrenicectomy without thoracoplasty has no material affect upon the maximum breathing capacity. Likewise, the maximum breathing capacity was not reduced materially by collapse therapy, provided good functioning lung parenchyma was not compressed.

In patients with emphysema, the decrease in maximum breathing capacity correlated with the increase in the severity of the disease (7, 81, 32). In patients with anthracosilicosis, Motley (81) observed that whenever the maximum breathing capacity was less than 40 liters per

minute, a significant degree of emphysema was present; and, when the maximum breathing capacity was greater than 100 liters per minute, the degree of emphysema was insignificant. The maximum breathing capacity was reduced consistently farther below the predicted value than was the vital capacity. During graphic recording of the maximum breathing capacity of emphysematous patients, the chest was maintained in an extreme inspiratory position (32). Similar findings, but of a lesser degree, were observed in pulmonary fibrosis (8).

Ratios involving the maximum breathing capacity are utilized in pulmonary function appraisal. Gaensler (44) reasoned, "since the vital capacity is a measure of air moved irrespective of time, and the maximum breathing capacity is a measure of air moved during a unit of time, the relationship between the two must therefore be related to time, or the velocity of air flow". This relationship was designated the air velocity index and is expressed in the following manner:

$$\frac{\% \text{ of the predicted M.B.C.}}{\% \text{ of the predicted vital capacity}} = \text{air velocity index (A.V.I.)}$$

Since a low maximum breathing capacity may be due to both loss of functioning lung tissue and obstruction to the flow of air, while the vital capacity is reduced significantly only in the former instance (unless a major bronchus is completely occluded), a comparison of the two may give some insight relative to the cause of ventilatory insufficiency. Equal impairment of the maximum breathing capacity and the vital capacity will result in an air velocity index of 1.0 regardless of the degree of impairment. An index less than 1.0 is found whenever there is a relatively greater impairment of the maximum breath-

ing capacity than of the vital capacity. This relationship is seen in cases of bronchial asthma. An index greater than 1.0 is found whenever there is a relatively greater impairment of the vital capacity than of the maximum breathing capacity. This is observed in patients with chronic ventilatory insufficiency due to reduced parenchymal lung tissue. The air velocity index for 34 patients with bronchial asthma ranged from 0.19 to 0.67 and averaged 0.36. In patients with ventilatory insufficiency due to a loss of aerated lung tissue it averaged 1.23 and ranged from 0.90 to 1.75. Good correlation between the air velocity index and the severity of the asthma was observed.

Warring (105) utilized the ratio of the walking ventilation to the maximum breathing capacity as an index of pulmonary function. The walking ventilation is defined as the number of liters of air ventilated while walking on a level surface at a slow, uniform pace. The values obtained remained fairly constant for an one patient over long periods of observation. A constant value is maintained with improvement or spread of disease, before and after induction and re-expansion of a pneumothorax, and before and after thoracoplasty.

A definite correlation was found between the ratio $\frac{\text{walking ventilation}}{\text{maximum breathing capacity}}$ and the degree of dyspnea. Whenever the ratio in a given patient was below 0.30, the patient was usually not dyspneic when walking on the level. Slight dyspnea was present at 0.35, moderate dyspnea at 0.45, and severe dyspnea at 0.50. In the consideration of candidates for collapse therapy, it is recommended

that the contemplated therapy should not reduce the maximum breathing capacity below twice the value of the patient's walking ventilation. In other words, the ratio of the walking ventilation to the maximum breathing capacity must not exceed 0.50, if the patient is to avoid becoming a respiratory invalid.

In a modification of this ratio, the numerator (walking ventilation) is replaced by the minute volume observed (at rest or during exercise). The ratio multiplied by 100, is termed the dyspnea index. Dyspnea is usually experienced by an individual when the dyspnea index is greater than thirty per cent.

The breathing reserve is the difference between the values for the maximum breathing capacity and the minute volume in any given physical state. Baldwin et al (6) found that the ratio $\frac{\text{breathing reserve}}{\text{maximum breathing capacity}} \times 100$ varies between 91 and 95 per cent in normal persons to the age of fifty, and reaches values as low as 88 per cent in older individuals. The mean value of 98.9 per cent was reported by Matheson and Gray (74).

Reduction of the breathing reserve may result from an increase in the minute ventilation or from a decrease in the maximum breathing capacity. A larger reduction in the resting breathing reserve is usually related to a decrease in the maximum breathing capacity, because the minute volume of resting patients with pulmonary disease rarely exceeds 20 liters per minute (27).

Cournand and Richards (31) believe that the onset of dyspnea occurs in the majority of cases when the breathing reserve is between

60 and 70 per cent. Correlation was shown to exist between the ratio $\frac{\text{breathing reserve}}{\text{M.B.C.}}$ x 100 and the presence and severity of dyspnea during the recovery period following exercise. No subjects experienced dyspnea when the ratio was above 93 at rest; when the values fell between 92 and 87 after exercise, half of the subjects were dyspneic, and the remainder were dyspneic for less than two minutes; when the values fell between 85 and 81 after more strenous exercise, all of the subjects complained of dyspnea in varying degrees; when the value was below 80, all of the subjects were dyspneic for two minutes or longer.

Baldwin et al (7), in a study of patients with emphysema, observed that the threshold of dyspnea occurred at lower values (39 to 56%) than previously reported. This discrepancy was thought to be related to psychological factors relative to the subjective complaint of dyspnea. Motley et al (79) found the breathing reserve, expressed as percent of the maximum breathing capacity, in anthracite miners to be 89.6, 85.0, 79.6, 73.8 and 67.5 respectively in five groups, classified according to increased severity of emphysema based on an increase of the ratio of the residual capacity to the total capacity. They observed a significant degree of emphysema when the breathing reserve was 70 per cent or less of the maximum breathing capacity; the degree of emphysema was insignificant when the breathing reserve was 90 per cent or more of the maximum breathing capacity.

In patients with pulmonary fibrosis, in whom the oxygen saturation was above 92 per cent following exercise, dyspnea was experienced when the ratio of the breathing reserve to the M.B.C. x 100 was between

61 and 71 per cent (8).

When reviewing the results of the ratio $\frac{\text{breathing reserve}}{\text{M.B.C.}}$ and the air velocity index, it is essential to note the absolute volumes, because two individuals may have the same values for either of these ratios but greatly different absolute volumes. Finally, it should be noted that the value of the ratio $\frac{\text{walking ventilation}}{\text{M.B.C.}}$ is in inverse proportion to that of the ratio $\frac{\text{breathing reserve}}{\text{M.B.C.}} \times 100$ under the same conditions; therefore, a patient for whom the ratio $\frac{\text{walking ventilation}}{\text{M.B.C.}}$ is 0.35, has a ratio $\frac{\text{breathing reserve}}{\text{M.B.C.}} \times 100$ of 65 per cent (105).

## INTRAPULMONARY GAS MIXING (DISTRIBUTION OF INSPIRED AIR)

If it is assumed that alveolar ventilation is uniform, then the inspired air should be distributed uniformly to all alveoli and should be diluted instantaneously with the residual air. When an individual without pulmonary disease breathes oxygen continously, the nitrogen in the lungs is diluted progressively and washed out in the expired air. In persons with pulmonary disease in which certain areas of the lung are poorly ventilated, the nitrogen from these areas is washed out at a slower rate than that from normal, well-ventilated areas. Because of this, the amount of nitrogen which is present in a sample of alveolar air, after a subject has breathed oxygen for a definite period of time, is used as an index to estimate the efficiency of the distribution of the inspired air (30).

The composition of the alveolar air is influenced by: (1) the number of breaths per minute, (2) the volume of the tidal air, (3) the volume of the respiratory dead space, (4) the volume of the functional residual capacity, (5) the rates at which oxygen is absorbed and carbon dioxide removed from the blood, and (6) the composition and pressure of the atmosphere. The nitrogen content of the alveolar gas in normal subjects is approximately 81 per cent when room air is breathed (30). Bateman (10) has pointed out that the composition of alveolar air is not constant in normal persons, and that no sample taken at a given instant can be regarded as the "average" alveolar air. It is possible that during inspiration carbon dioxide could be added and oxygen removed at a rate which would prevent a change in composition; however,

during expiration there would be a depletion of oxygen and an increase in the carbon dioxide content. The increase of carbon dioxide in the alveolar air reduces the diffusion gradient between the venous blood and the alveolar air, consequently, the loss of carbon dioxide from the blood will be slowed until the beginning of the next inspiration, when a high gradient is re-established. The above mechanism produces a brief decrease in the alveolar respiratory quotient. In the case of oxygen, the pressure gradient and the quantity of oxygen available in the alveolar air are greater; therefore, for the short expiratory time intervals, the rate of oxygen uptake remains fairly constant. Krogh and Lindhard (64), cited by Bateman (10), found, however, that the rate of uptake of oxygen was not constant during the respiratory cycle. It dropped sharply during the first stage of expiration and rose sharply during inspiration. These changes were attributed to variations in the rate of flow of blood through the lungs during the respiratory cycle. Roelsen (97), cited by Bateman (10), found that the last sample of alveolar air collected at the end of a deep expiration often contained more oxygen and less carbon dioxide than its predecessors. Because of these continuous changes, Bateman (10) suggests that the best average alveolar air may be calculated by correcting the composition of the total expired air for the effect of the dead space.

Uneven alveolar ventilation may be due to: (1) sequential distribution of inspired air (i.e. gas from the respiratory dead space is drawn preferentially or predominantly into those alveoli that fill early), (2) the unequal distribution of incoming air among various alveoli in

relation to their pre-inspiratory volumes, and (3) failure of intra-alveolar mixing (layering of gases in alveoli) (26). Uneven alveolar ventilation occurs in normal as well as in diseased lungs (40). The results of analyses of alveolar gases collected at the end of a deep expiration suggest that the samples of gas came partially from a region of the lung in which the uptake of oxygen was lower than elsewhere. Fowler (40), as a result of continuous analysis of nitrogen in the expired air of normal individuals, concluded that oxygen that is inspired is not distributed evenly throughout the functional residual gas; and that relatively poorly ventilated areas of the lung empty predominantly later in expiration.

Morphological changes which affect the manner in which inspired air is distributed are: (1) a decrease in the distensibility of certain areas of the lungs, (2) regional obstruction of the air passages, and (3) the loss of elasticity in certain areas of the lungs (27). In addition to pulmonary emphysema, abnormal distribution of gas occurs in bronchiectasis, asthma, sarcoid, pulmonary carcinoma, a nd congestive heart failure (26). Comroe et al (26) studied a group of patients over fifty years of age with no clinical or radiological evidence of pulmonary disease; they found that half of them showed "abnormalities" in the distribution of inspired air in relation to the findings in healthy subjects in the twenty to thirty-five year group.

Cournand et al (30) used the percentage of nitrogen found in alveolar air samples after a subject breathed oxygen for seven minutes as an index for measuring intropulmonary mixing of the inspired gas.

In a large series of determinations, it was found that the concentration of nitrogen was below 2.5 per cent in 94 per cent of normal subjects and in all patients with cardiac disease. On the other hand, it was elevated above 2.5 per cent in 17.7 per cent of the tuberculous patients and in most of those with pulmonary emphysema (30, 7). This test may be falsely positive if the functional residual capacity is small and the lungs are hypoventilated; or it may be falsely negative if the functional residual capacity is large and the lungs are hyperventilated.

The mean value for this index was not increased in a group of patients with pulmonary fibrosis (8). In patients with large air cysts communicating freely with the bronchial tree, the mean index was normal. Similar findings were observed in the majority of patients with large air cysts communicating poorly or intermittently with bronchi but with normal lungs otherwise. Patients with large air cysts communicating poorly or intermittently with bronchi in association with emphysema, had high indices of intrapulmonary mixing.

THE VENTILATION EQUIVALENTS AND THE RATIO OF OXYGEN REMOVAL

The ventilation equivalent for oxygen is the number of liters of air breathed per 1000cc. of oxygen absorbed. The ventilation equivalent for carbon dioxide is the number of liters of air breathed per 1000 cc. of carbon dioxide produced.

Normal values for oxygen ventilation equivalents reported by various investigators are presented in Table 7, page /09, in the Appendix.

The ratio of oxygen removal is the number of cubic centimeters of oxygen absorbed per liter of air breathed.

Knipping and Moncrieff (63) did not find differences in the ventilation equivalents of the sexes or in any appreciable relationship with age. Christie and McIntosh (21) have shown that the ventilation equivalents may be calculated without measuring the minute volumes. This is shown by the following mathematical relationships:*

$$\text{Ventilation equivalent for } O_2 = \frac{\text{min. vol. respired x 100}}{O_2 \text{ absorption/ minute}} =$$

$$\frac{\text{Min. Vol. respired x 100}}{\text{Min. Vol. respired x } O_2\% \text{ absorbed}} = \frac{100}{O_2\% \text{ absorbed}}$$

The ventilation equivalent for oxygen may vary in any given individual with changes in the respiratory quotient. Because of this, comparison of ventilation equivalents is not satisfactory unless the respiratory quotient is reasonably constant. McMichael (72) has suggested that the ventilation equivalent for carbon dioxide would be more constant than that for oxygen in the presence of varying respiratory

* These equations and other values reported in this discussion are based upon the number of liters of air breathed per 100 cc. of oxygen absorbed.

quotients. This suggestion is based on the fact that when a person hyperventilates, the rate of oxygen consumption remains essentially unchanged with the result that the ventilation equivalent for oxygen increases. In contrast, the volume of the expired carbon dioxide parallels the increase in ventilation. Consequently, large fluctuations in the ventilation equivalent for carbon dioxide are prevented.

McMichael (72) found a mean value of 3.06 liters with a standard deviation of 0.44 liters for the ventilation equivalent for carbon dioxide.

These ratios help to determine some of the factors involved in producing hyperventilation. In hyperpneas associated with a primary increase in tissue metabolism such as occurs in exercise, fever and hyperthyroidism, a rise in oxygen consumption parallels the increased ventilation and the ratio of oxygen removal remains more or less normal. A decrease in the ventilation equivalent during exercise, when compared to the resting v alue, may be due to increased pulmonary efficiency. In pulmonary disease, tissue metabolism and oxygen consumption remain approximately normal but ventilation may be increased by a decrease in arterial oxygen tension, increased carbon dioxide tension, or a decreased pH of the blood. In this event, the ventilation equivalent increases or the ratio of oxygen removal decreases.

Reduction in the ratio of oxygen removal at rest indicates that a state of hyperventilation exists. If a low ratio of oxygen removal is observed during exercise, factors responsible for an inadequate increase in cardiac output must be considered in addition to those which produce

hyperventilation. Some patients are unable to increase their ventilation during exercise; however, they are capable of maintaining a relatively high ratio of oxygen removal in the presence of severe physiological pulmonary and cardiac disturbances. For these reasons, changes in the ventilation equivalent for oxygen or the ratio of oxygen removal during exercise are not sufficient criteria for the differentiation of circulatory from pulmonary insufficiency (6).

Kaltreider and McCann (62) found an increase in the oxygen ventilation equivalent in all types of cardio-respiratory disease. In patients with pulmonary fibrosis the mean value was 3.15 liters, with extremes of 2.23 to 4.40 liters; and in patients with emphysema the mean value was 2.98 liters. Equally high values were found in a group of five patients with heart disease. It is interesting to note that during moderate physical activity, the ventilation equivalent for oxygen fell in all groups except one, the patients with heart disease, in which the values increased from 3.0 to 3.34 liters. In these observations, these investigators found only fair correlation of the ventilation equivalent for oxygen with the degree of dyspnea.

A unique method for estimating the degree of cardio-respiratory disability by utilizing the oxygen and carbon dioxide ventilation equivalents has been developed by Pelnar (88). The ventilation equivalents as used in Pelnar's paper refer to the percentage decrease in oxygen content and the percentage increase in carbon dioxide content of the expired air in comparison with the inspired air. The ventilation equivalent for oxygen decreases during hyperventilation and pathological conditions.

Similarly, the carbon dioxide ventilation equivalent decreases during hyperventilation, but to a lesser degree, with a resultant rise in the respiratory quotient. The oxygen equivalent is plotted against the simultaneous carbon dioxide equivalent on a graph which contains lines (corrected for the difference in volume of the inspired and expired air) that represent the respiratory quotients. When values obtained on a resting subject fall below the line representing an R.Q. of 1, it means that a relative hyperventilation occurred. In normal persons during exercise, the ventilation equivalents increase sharply; and when plotted on the graph, rise and move to t he right in a semi-circle. The points stabilize in the new area after a steady state is reached and the respiratory quotient is higher than at rest. During the recovery period after exercise, the ventilation equivalents drop to the original values in a semicircular pattern that is roughly inverse to that formed during exercise. The position of the curve at the start shows the function at rest. The size of the circle indicates the capability of the pulmonary reserves for increasing the equivalents during exercise and thus for sparing the lung ventilation. The larger the circle the less the ventilatory reserves are being exhausted during exercise. Some patients are able to produce normal results at rest by means of reserve mechanisms that prove insufficient under working conditions. Others, if studied at rest, produce results which are impaired by various factors such as bronchospasm, which may decrease during exertion, permitting greater ventilatory efficiency. For this reason, determinations obtained at rest, during exercise, and recovery from exercise, are de-

sirable. The various degrees of disability exhibit characteristic patterns with this technique.

Good correlation of the ventilation equivalents with the degree of dyspnea and loss of function was obtained in fifty-six patients with pathological conditions of the chest and circulation other than silicosis. Similar correlation was obtained in a group of thirty patients with pneumoconioses; however, correlation with the roentgenograms of the chest was poor.

## THE ARTERIAL CARBON DIOXIDE CONTENT AND TENSION

The normal value for arterial carbon dioxide content varies between 45 to 55 volumes per cent (18). The normal value for arterial carbon dioxide tension is 40 mm. Hg (11).

The arterial carbon dioxide content may be increased or decreased in either acidosis or alkalosis. Acidosis which is due to primary $H_2CO_3$ excess (increased $CO_2$ tension, increased carbonic acid) may result from either of two general situations: (1) from rebreathing the same air, or breathing air with a high carbon dioxide content, and (2) from impaired elimination of carbon dioxide by the lungs. The latter may be produced by any disease process which impairs sufficiently the ventilatory mechanism (the bellows action) or the blood-gas transfer of carbon dioxide. Mechanisms which tend to lower the elevated carbon dioxide tension of the blood are: (1) increased ventilation, (2) an increase in the alkali reserve of the blood (blood becarbonate), (3) an increase in the formation of ammonia by the kidneys, and (4) increase in the acidity of the urine.

Acidosis may be produced also when there is a primary alkali deficit (decrease in blood bicarbonate, $BHCO_3$). This occurs in: (1) diabetes mellitus (advanced), (2) renal failure, (3) starvation, (4) dehydration, (5) after ingestion of acids, (6) the late stages of pregnancy, and (7) during anesthesia induced by certain agents (18). Counteracting mechanism are: (1) an increase in the elimination of carbon dioxide by hyperventilation, (2) an increase in ammonia formation by the kidneys, and (3) an increase in acid excretion by the kidneys.

The state of alkalosis, likewise, may be present with either a high or low carbon dioxide content of the arterial blood. A primary carbonic acid ($H_2CO_3$) deficit may be produced by hyperventilation which may be secondary to an hysteria, fever, high external temperatures, anoxic anoxemia or encephalitis (18). Mechanisms which tend to combat this acid-base imbalance are: (1) an increase in the renal excretion of alkali, (2) a decrease in the formation of acids by the kidneys, (3) a decrease in ammonia formation by the kidneys, and (4) the retention of the acid metabolic products acetone, diacetic and beta-hydroxybutyric acid.

A primary alkali excess (increased blood bicarbonate $HBCO_3$) may result from: (1) a protracted period of vomiting with a loss of large amounts of HCL, (2) excessive administration of alkali, and (3) following deep x-ray and radium therapy and prolonged ultraviolet irradiation (18).

Because of the fact that the carbon dioxide content of the blood may be increased or decreased in alkalosis or acidosis, determinations of the hydrogen ion concentration, the $CO_2$ arterial tension, and blood bicarbonate are necessary for an accurate analysis of the acid-base relationship.

Patients who have both a high carbon dioxide content and tension with a decrease in the blood hydrogen ion concentration have acidosis which is due to pulmonary insufficiency. These conditions, however, are found only in patients with advanced pulmonary disease and are due usually to an extremely impaired ventilatory capacity (35). An increase

in the oxygen partial pressure gradient between alveolar gases and blood in alveolar capillaries occurs earlier than an increased carbon dioxide partial pressure gradient, because of the more rapid rate of diffusion of carbon dioxide. Because of this, pulmonary insufficiency for the oxygenating function of the lungs does not necessarily imply that there is insufficiency of the carbon dioxide eliminating function. The converse, however, is true unle ss the alveolar oxygen tension is increased by breathing air enriched with oxygen (27).

In 37 patients with pulmonary fibrosis, Hurtado et al (60) found a mean arterial carbon dioxide content of 44.89 volumes per cent in a range of 39.35 to 55.21 volumes per cent. Baldwin et al (8), in a study of patients with pulmonary fibrosis and moderate ventilatory insufficiency without obvious disturbances of either the distribution or the diffusion factors, found the mean arterial carbon dioxide tension to be lower than normal in all but one case in the resting state and in all but two cases following exercise.

In 24 patients with pulmonary emphysema, the mean value was 47.67 volumes per cent in a range of 40.70 to 58.16 volumes per cent (60). Correlation of the carbon dioxide content with the ratio of the residual capacity to the total capacity showed a definite tendency for the carbon dioxide content to vary directly with the ratio (60). From these observations, it was concluded that the critical values of this ratio at which the average values of carbon dioxide in the blood become abnormal fall in the range between 40 and 50 per cent. Similar results have been noted by other observers (81, 66, 7). Baldwin et al (7)

found the arterial carbon dioxide tension above 48 mm. Hg after exercise in 24 of 68 patients with emphysema.

## THE ARTERIAL OXYGEN SATURATION AND TENSION

The mean normal arterial oxygen saturation has been found to be 97.4 per cent with a standard deviation of ± 2.1 per cent (27). Greifenstein et al (50) found the mean value to be 96.1 per cent in elderly persons without evidence of cardio-pulmonary disease. Comroe et al (24) found the mean arterial oxygen tension to be 97.1 mm. Hg in young adults and the mean oxygen tension to be 97.4 mm. Hg in end-expiratory samples of alveolar air.

Arterial oxygen saturation does not decrease, but often increases, during exercise in normal subjects and in some patients with mild impairment of pulmonary function (62, 27, 80). Measurement of the arterial oxygen saturation before and after exercise, therefore, provides much useful information for ascertaining the degree of pulmonary insufficiency which may exist.

In pulmonary disease the a rterial oxygen tension and saturation depend primarily upon: (1) the ventilatory efficiency of the lungs, (2) the nature and amount of diffusing surface present, and (3) the perfusion of the alveoli with blood. Because of the nature of the oxygen dissociation curve, there may be a greater decrease in the arterial oxygen tension than in the oxygen saturation when slight degrees of impairment are present.

In patients with pulmonary fibrosis, the ventilation and perfusion of the lungs are impaired (81). The movement of air in and out of the lungs may be less efficient because of a decrease in the elasticity of the lungs. Concomitantly, there is impairment of the alveolar

blood flow which results in alveoli that are not perfused and aerated incompletely or not at all. This produces an increase in the dead space volume which contributes additionally to a decrease in ventilatory efficiency. In addition to these factors, there is decreased permeability per unit area of the diffusing surface. Motley et al (81, 83), in a study of anthracite coal miners with pulmonary fibrosis, believe that an abnormality of the distribution factor (unequal alveolar aeration and perfusion) is the most important cause for an increased oxygen gradient between alveoli and the arterial blood.

Hyperventilation is the mechanism which permits some patients, with minimal amounts of pulmonary fibrosis, to maintain normal arterial oxygen saturation at rest and after exercise. In patients with more extensive pulmonary fibrosis in whom the maximum breathing capacity is not markedly reduced, a decrease in arterial oxygen saturation after exercise is thought to be due primarily to the inability of the lungs to expand the effective pulmonary capillary bed (35). Compensatory hyperventilation, in these cases, cannot increase the alveolar oxygen tension sufficiently to provide the large diffusion gradient (transfer gradient) which is required to transfer oxygen across the damaged pulmonary membrane. The latter is accomplished, to a large degree, by an increase in the diffusion gradient created by a decrease in alveolar capillary blood oxygen tension with a subsequent drop in arterial oxygen saturation. The factors of venous admixture and an increase in the speed of blood flow in the capillaries are thought to be of lesser importance (35).

In 37 cases of pulmonary fibrosis, Hurtado et al (60) found the

mean value for oxygen saturation to be 92 per cent in a range of 80.6 to 98.5 per cent. Their findings in relation to the roentgenologic appearances of the lungs are presented in Table 10, page /// , in the Appendix. Baldwin et al (8), in a study of patients with pulmonary fibrosis whose patterns were characteristic of alveolar respiratory insufficiency associated with a relatively normal ventilatory function, found low arterial oxygen saturations.

In patients with emphysema the distribution of the inspired air is impaired. In addition to this there is probably an impairment of the distribution of blood (35, 81). This produces alveoli that are perfused adequately but hypoventilated; therefore, there is an increase in the venous component of the mixed arterial blood. This constitutes a functional shunt. If there is impairment or obliteration of the alveolar blood flow, the alveoli may be ventilated but not perfused. The dead space volume is increased thereby and the ventilatory efficiency is decreased (81, 35). In addition to unequal aeration and perfusion of the alveoli, degeneration of the walls of the alveoli decreases the size of the effective blood gas interface. The high mean alveolo-capillary oxygen tension gradient and low diffusion coefficient (oxygen uptake in milliliters per minute divided by the mean alveolo-capillary oxygen tension gradient) (35), observed in patients with emphysema is, therefore, related to alterations in aeration and perfusion, and to changes in the walls of the alveoli.

Hurtado et al (60) found the mean arterial oxygen saturation was 88.2 per cent in a range of 72.9 to 97.7 per cent in 24 patients with

pulmonary emphysema. They concluded from their observations that the oxygen saturation of arterial blood is affected less in patients with pulmonary fibrosis than in those with emphysema. Correlation of the arterial oxygen saturation with the ratio of residual volume to the total capacity showed a tendency for the saturation to decrease as this ratio increased; however, the relation was not linear. In their experience, it was usual to find some degree of anoxia of the arterial blood when the residual volume was 45 per cent or more of the total lung capacity.

The resting oxygen saturation of most of the anthracite coal miners studied by Motley et al (78) varied between 90 and 94 per cent. After exercise there was a decrease in saturation which correlated inversely with the severity of the emphysema determined by an increase in the ratio of the residual capacity to the total capacity. The arterial oxygen saturation was less than 92 per cent after exercise in forty-three patients with chronic pulmonary emphysema studied by Baldwin et al (7).

## THE SPIROGRAM

A spirogram is a graph of the respiratory movements from which the following observations may be made: (1) the forms of quiet and deep breathing, (2) the volume of the vital capacity and its component parts, (3) the maximum breathing capacity, (4) the minute ventilation, (5) the amount of oxygen consumed, and (6) the duration of any phase of the respiratory cycle.

In normal individuals, the amplitude of the inspiration and expiration curves, the rates, and the minute volumes may vary from person to person; however, the ratio of the periods of time involved in the inspiratory and the expiratory phases remain approximately the same. A maximum expiration requires about 1.2 times as long as a maximum inspiration (27). It makes little difference, in the relationship, whether the subject begins the performance with the inspiratory or the expiratory phase. The slope of the inspiratory curve is characterized by a uniform steepness of practically the entire span and the chest position returns to the resting end-expiratory level immediately after a maximum inspiratory or expiratory effort (19).

In an analysis of the curves obtained from maximum breathing capacity tests in normal individuals, Cournand et al (32) found that the average amplitude of each breath was 79.1 per cent of the vital capacity in males and 78.6 per cent in females, when the subjects were breathing as deeply as possible. Following an increase in the rate of breathing, the average amplitude of each breath was 50.6 per cent of the vital capacity for males and 49.7 per cent for females. If one observes the

curves recorded during the performance of the maximum breathing capacity test, it is often possible to establish the optimum relationship of these factors that will produce the highest value.

The spirograms of patients with pulmonary emphysema have many characteristics. Whenever a subject with pulmonary emphysema takes a deep breath, the lungs become over-stretched; and, because there is a decrease of elasticity, they fail to return immediately to the resting end-expiratory level after a normal expiration. Christie (19) referred to this as a "set". Because of this, there is a gradual "step-down" of the tidal air curves to the resting end-expiratory level, which often requires twelve or more respirations. The same phenomenon occurs after a maximum expiration; however, the tidal air curves move in a step-like manner in the opposite direction. This feature is illustrated more dramatically when a patient with emphysema is instructed to take a deep breath followed by a maximum expiratory effort. The reserve air volume portion of the vital capacity curve, under these circumstances, will be much smaller than that which is determined sepatately; it may be absent! In the performance of a maximum breathing capacity test, a patient with emphysema will tend, because of a series of "sets", to breathe with the chest fixed at a high inspiratory level (19). This characteristic pattern of breathing may be used to differentiate a decrease in the vital capacity due to emphysema from that which occurs in congestive failure. In the latter, the elasticity of the lungs is decreased to a much less degree. The factor of time, in relation to the vital capacity in patients with emphysema, has been discussed. The tidal air, when observed graph-

ically, fluctuates from minute to minute, and from day to day, with corresponding fluctuations in the inspiratory capacity, the expiratory reserve volume, and the functional residual capacity (19). The ratio of the residual volume to the total capacity is increased; this requires the subject to breathe at a higher inspiratory level than does a normal subject. As a consequence of this, the inspiratory capacity is decreased. In addition to these changes, there is a marked retardation of the entire respiratory cycle which is more pronounced near the end of inspiration and results in the formation of "plateaux" (32). The expiratory curve is affected similarly; and, in extreme cases, there is a rounding of this portion of the curve (7, 32). In advanced cases of emphysema, the curves are irregular and jerky (32).

The spirograms of patients with pulmonary fibrosis show retardation of both the inspiratory and expiratory portions of the curves with a proportionately greater reduction of the inspiratory reserve volume than of the expiratory reserve volume (32). Patients with pulmonary fibrosis and with respiratory patterns characteristic of alveolar respiratory insufficiency associated with normal ventilatory function (included in this group were cases of scleroderma, interstitial pneumonitis, asbestosis, lymphangitic carcinoma and exposure to sulfur dioxide), produced spirograms in which no evidence of ventilatory slowing, obstruction, or air trapping was observed (8). In patients with pulmonary fibrosis and with respiratory patterns characteristic of ventilatory insufficiency without obvious disturbances of either the distribution or the diffusion of respiratory gases (this group included cases

of silicosis, bronchiectasis, chronic fibroid tuberculosis, Boeck's Sarcoid, and radiation fibrosis), the maximum breathing capacity was performed in the normal mid-position in most instances and occasionally in the inspiratory position in the most advanced cases (8).

Patients with large air cysts which communicated poorly or intermittently with bronchi and with apparently normal lungs otherwise, showed evidence of obstruction, air trapping, irregularity, and asynergy of the respiratory movements in the performance of the maximum breathing capacity test (9).

Patients with congestive heart failure showed a retardation in their inspiration and expiration of the last 100 or 200 cc. of air, in addition to a decrease in the vital capacity which was often associated with a proportionately greater decrease of the expiratory reserve volume (32).

The lung volume was decreased uniformly without significant retardation of either the inspiratory or expiratory phases of respiration, in patients with chest deformities (32). At rest, the pattern of breathing showed evidence of hyperventilation. The maximum breathing capacity was reduced. No changes were observed in these findings after the patients had used bronchodilators.

Cournand (32) demonstrated the usefulness of the spirogram in a case of malingering. A test of maximum breathing capacity was recorded; then the carbon dioxide absorbent was removed from the circuit without the knowledge of the patient, and the test repeated. A significant increase in the maximum breathing capacity occurred due to the accumulation

of carbon dioxide in the respired gas.

Many observations have been made in regard to changes in the breathing patterns produced by various sensory stimuli and by emotional disturbances. Variations in the rate, amplitude, and shape of the tidal air curves have been observed. Some of these changes will be discussed more fully in Part Three. Extensive bibliographies are presented in papers by Coughey (29) and others (28, 17, 38, 3, 107).

## BRONCHOSPIROMETRY

Bronchospirometry is a method by which the functional capacity of each lung may be determined simultaneously. This is accomplished by the introduction of a double-lumen catheter with a curved tip into the left main-stem bronchus which is sealed by an inflated balloon proximal to the opening of the distal lumen. The other lumen is the airway to the right lung. A second balloon when inflated creates an airtight seal between the trachea and the catheter.

The results of the pulmonary function studies as discussed previously, represent the total function of both lungs. Although these results are adequate in many instances, there are occasions when it is necessary or advantageous to know the functional capacity of each lung. It is an accepted fact that the ventilatory function of a lung, which on an x-ray film appears well aerated and clear, cannot be determined accurately by roentgenologic techniques. Pleural or hilar fixations, not disclosed by radiological techniques, may be responsible for a marked reduction in the function of a lung (86, 90). Conversely, a lung which yields abnormal densities on x-ray films may have adequate function.

The indications for bronchospirometry are:

(1) To aid in the proper selection of patients for irreversible collapse therapy. In these cases it is not only necessary to determine what proportion of the total function is provided by the diseased lung on which collapse therapy is contemplated, but it is of the utmost importance to determine the amount of function that is present in the

contralateral lung and its ability to compensate for a decrease in function of the collapsed lung.

(2) To predict the effect upon pulmonary function of lung resection. In the study of patients prior to surgery, it should be emphasized that bronchospirometry evaluates the function of the better lung in the presence of the lung to be removed and not that of the better lung existing alone (27).

(3) To study disease processes and the effects of therapeutic procedures.

(4) To determine the contralateral lung function and compensatory ability in patients with limited function prior to pneumothorax therapy. This is necessary because lungs that are allowed to re-expand after the induction of therapeutic pneumothorax frequently have impaired function due to pleural abnormalities caused by this therapy.

Bronchospirometry is contra-indicated for certain patients (111). They are: (1) those who have had a pulmonary hemmorrhage within two weeks prior to the study; (2) those with acute ulcerations of the trachea or left main stem bronchus; (3) those in whom there has been recent spread of disease; and (4) those suffering from acute laryngitis or acute pyogenic disease of the tracheo-bronchial tree. Norris (85) states that dyspnea of a moderate degree is a contraindication.

Absolute normal values or standards are difficult to establish because of factors such as physical strain, differences in local anesthesia, apprehension, and stenosis of the airways, which vary in degree from person to person (89). Bjorkman (13), cited by Pinner et

al (89), found that the left lung contributed 46 to 47 per cent of the total oxygen uptake under normal conditions. Gaensler and Watson (42) found that the right lung was responsible for 51 to 62 per cent of the total function. Gaensler and Cugell (45) found that the absolute volumes of the various subdivisions of the total lung volume differed considerably in each of four normal subjects. However, the ratios of each subdivision to the total lung volume X 100 were almost identical for all subjects for the corresponding lung. The results obtained from four normal persons studied by them are presented in Table 11, page //2, in the Appendix.

The results of bronchospirographic observations must be interpreted with caution because the procedure induces an abnormal physiologic response. Because the lumens in the catheter are small, stenoses are present which create an increased and unequal resistance to the flow of air (85, 86, 89). The increased resistance to breathing created by a stenosis causes a shift in the mid-position of the lung. Because of this, the absolute values for the expiratory reserve volume and the inspiratory capacity are distorted; however, the distribution of the values as between the left and right lung is valid (86). Similarly, because of increased and unequal reistance to air exchange, the values for the maximum breathing capacities of the two lungs, if read directly from the tracings, are questionable (86). Norris (86) states that if it can be assumed that the ratio of the separate vital capacities represents the relative ability of the two lungs to increase their ventilation, the values for the individual vital capacities and the maximum breathing

capacities obtained on an external spirogram (the combined function of both lungs on a single tracing) may be used to estimate the separate maximum breathing capacities. This may be done by the following calculations:

$$\text{M.B.C., right} = \frac{\text{Vit. Cap., right}}{\text{Vit. Cap., right} + \text{Vit. Cap., left}} \times \text{M.B.C. external}$$

The ventilation reserves for each lung, calculated in the usual manner, are probably incorrect because of the increased minute volumes produced by the procedure. To arrive at a more correct value, the following calculations are made if it can be assumed that the separate vital capacities indicate the relative ability of the two lungs to increase ventilation (86):

$$\left(1 - \frac{\text{M.V. \% right} \times \text{M.V. external}}{\text{V.C. \% right} \times \text{M.B.C. external}}\right) \times 100 \text{ where}$$

$$\text{M.V. \% right is } \frac{\text{M.V. right}}{\text{M.V. right} + \text{M.V. left}} \times 100 \text{ and}$$

$$\text{V.C. \% right is } \frac{\text{V.C. right}}{\text{V.C. right} + \text{V.C. left}} \times 100$$

These equations are not valid in cases of pulmonary emphysema, because the vital capacity does not determine the ability of the lungs to increase their ventilation.

The total oxygen uptake in bronchospirometry is usually increased from that determined in the usual manner, because of the physical stress that is involved (89). This increase varies from person to person because of the differences in physical stresses, apprehension, etc.. The most important factor, in regard to oxygen uptake, is the extent of the

distribution between the two lungs. The amount of oxygen transferred by each lung depends on the partial pressure of oxygen in the alveoli, the permeability and unit area of the alveolar membranes, the oxygen tension of the pulmonary arterial blood, the time available for equilibration, and the amount of blood delivered to each lung from the right ventricle. In seven patients with diseased lungs, Gaensler and Cugell (41), found that the mean oxygen uptake on the affected side was three per cent of the total while breathing room air, and twelve per cent of the total while breathing oxygen enriched air. No difference in the per cent of oxygen uptake while breathing room air or oxygen enriched air was noted in normal persons.

The effect of posture on individual lung function has been investigated (104, 98, 90). Vaccarezza et al (104) found that the lung on the side on which the patient is lying has a higher oxygen intake, minute volume, vital capacity and inspiratory capacity than the upper lung. Rothstein et al (98), noted a decrease in the ventilation equivalent, which indicated increased efficiency on the part of the under lung. In the majority of patients studied, there was a simultaneous decrease in the oxygen absorption in the upper lung. Rothstein et al (98) also found that the oxygen consumption was increased in patients, not undergoing spirometry, in the lateral decubitus position when compared to the supine position.

The effect of strapping (unilateral) the chest was studied by bronchospirometric methods. No reduction of pulmonary motion could be observed (90). The same observers found that a weight placed on one

side of the chest did not result in a decrease of ventilation on the side under the weight and a corresponding increase of ventilation on the contralateral side; on the contrary, the ventilation increased on both sides, because of the increased work load on the chest.

Pinner et al (90) found that a pneumothorax decreases the uptake of oxygen by the collapsed lung by means of a decrease in the minute volume and less efficient use of the ventilated air. The latter was indicated by an increase in the ventilation equivalent. The reduced oxygen intake on the affected side was compensated by an increase of the oxygen intake in the contralateral lung. The latter was accomplished by an increased minute volume and more efficient utilization of the ventilated air. In a large proportion of the cases with a unilateral pneumothorax, the contralateral lung was found to have a reduced vital capacity (90). Compensation was thought to be accomplished not only by an increase in the ventilatory work of the contralateral lung but also by a cardiovascular factor. Collapse by thoracoplasty causes a reduction in all subdivisions of the lung volume (90, 45). Comparison before and after thoracoplasty showed that the oxygen uptake, the ventilation equivalents and minute volumes were not significantly changed; however, there was a decrease in the maximum breathing capacity (90). Long (69) and associates found that reduction in the oxygen uptake occurred more often than did reduction in the volume of air moved, in bronchiectatic lungs. In patients with localized unilateral parenchymal tuberculosis, the intrapulmonary mixing was normal on both sides, the vital capacity of the diseased lung was slightly reduced, and the residual

volume was affected little or even increased (45). Patients with extensive unilateral parenchymal tuberculosis had a marked decrease in the vital capacity and a moderate reduction in the residual volume, and an occasional increase in the index of pulmonary mixing was observed (45).

## THE PNEUMOTACHOGRAM

The pneumotachogram is a record of the respiratory air flow. The velocity of the respiratory flow, the tidal volume, and the respiratory rate can be measured from the record. The configuration of the curve is altered characteristically in certain abnormal pulmonary states.

The normal inspiratory curve begins with an air flow which accelerates rapidly, quickly reaches a maximum, remains constant for a brief time, then decelerates rapidly to zero as the inspiratory phase is completed. Termination of the inspiratory phase occurs quickly without a detectable pause before expiration is begun. The expiratory flow accelerates rapidly; then, it appears to decelerate in a more or less linear pattern to zero. Pneumotachographic patterns vary from person to person; and, they may vary in the same person on successive breaths (93, 101). Normal variations of the expiratory phase are: rapid deceleration near the termination of expiration; and, an expiratory phase without sudden changes in velocity, associated with a similar inspiratory pattern (93). The first variation is probably due to failure to obtain a true resting record, or to the subjects habitual breathing at a volume slightly above the normal resting point of the chest (93). The significance of the second variation has not been determined. Post-expiratory pauses may be present in normal tracings (93). Small undulations are characteristic, and it has been postulated that they are related to variations in the laryngeal aperture or to irregular relaxation of the respiratory muscles (101). Moderate changes in the rate of respiration do not change the pattern (101). No significant changes in the patterns have been ob-

served in older subjects (100). Characteristic patterns of pulmonary disease could not be reproduced by normal persons (101); however, some patients with pulmonary disease may produce patterns which lie within the limits of normal variability (93).

The normal pneumotachogram may be altered by: (1) a loss of lung tissue, (2) a decrease in the elasticity of the lung, (3) an increase in the resistance to air flow, and (4) an increase of "tissue viscance" which refers to "the viscous resistance to deformation of all or any of the structures contained within the chest (excluding air)" (94).

Diseases which impair the elasticity of the lung cause certain characteristic alterations in the expiratory flow patterns (101). They are: (1) a loss of the small undulations on the record during expiration, (2) stereotypy of successive breaths, and (3) peaking of the expiratory flow early in expiration with a concave or straight decline in flow to a rather sharp termination of the expiratory phase. These changes may be present during the inspiratory phase but are not as marked or consistent.

Silverman (99) has studied the pneumotachograms in patients with various pulmonary diseases. In asthma, the air flow curves are markedly damped, especially in the expiratory phase, there being a sharp return to zero at the end of the latter phase. Only three of eight patients with active tuberculosis showed abnormalities; these consisted of curves which suggested some resistance in the expiratory phase of respiration. Silicosis in the early stages did not usually produce positive changes, but in advanced second and third stage silicosis, a

marked damping and evidence of loss of elasticity were present. Three cases of emphysema were studied; and in two, the curves were almost rectangular, considerably damped, and had a rapid return to zero which is characteristic of inelastic tissue or of bronchial resistance.

# PART THREE

## THE CONTROL OF RESPIRATION

## THE EFFECTS OF EMOTIONS ON THE PATTERNS OF BREATHING

## THE CONTROL OF RESPIRATION

The respiratory center, which is located in the reticular substance of the medulla, is a group of cells that are capable of discharging rhythmical bursts of impulses to the respiratory muscles. The rhythmicity and frequency of these discharges may be modified by nervous and chemical mechanisms. The nervous modifications are effected by reflexes, voluntary control, and emotional influences from the higher central nervous system centers.

Reflexes involved in the regulation of respiration are:

(1) The Hering-Breuer reflexes. These reflexes, which are activated during inspiration and transmitted by the afferent fibers of the pulmonary vagi, have an inhibitory action on the inspiratory activity of the respiratory center. There are corresponding deflation reflexes; however, it is thought that they do not function during quiet breathing (11).

(2) Proprioceptive impulses. These impulses arise in the muscles of respiration, and the muscles and joints of the limbs (11).

(3) The carotid and aortic reflexes. These reflexes originate from bodies located within the walls of the carotid sinus and the aortic arch. Two types of receptors are present in each body viz. chemoreceptors and pressoreceptors. The latter evidently do not serve any useful respiratory function under normal physiologic conditions in man (11). The chemoreceptors cause reflex stimulation of the respiratory center in man when the arterial oxygen tension is reduced a little below 70 mm. Hg (11). They are much less sensitive to changes in the arterial

carbon dioxide tension (11).

(4) Reflexes from other parts of the body. These include reflexes from painful stimuli, manipulation of abdominal viscera, and stimulation of afferent nerve fibers which supply the respiratory tract (11).

Voluntary control of respiration supervenes frequently in acts such as laughing, coughing, blowing etc.. Much work has been done in relation to changes in the patterns of breathing produced by normal and abnormal states, neuroses and psychoses. These changes will be discussed later.

The chemical control of the respiratory center is dependent primarily upon the carbon dioxide tension of the blood. A slight decrease in the tension will produce a state of hypoventilation; a slight increase will cause hyperventilation. Changes of the pH concentration of the blood are of lesser significance in the control of respiration than changes in the carbon dioxide tension (11).

The reflex changes in the lungs are mediated through the autonomic nervous system. The bronchi and lungs are supplied mainly by neurons from the pulmonary ganglia and the pulmonary plexuses which are made up of parasympathetic and sympathetic components (65). The nerves in the walls of the respiratory tract include various sizes of myelinated and unmyelinated fibers and numerous ganglia (65). The ganglion cells are parasympathetic and constitute the ganglionic components of vagus efferent chains (65). Nerve fibers from this system extend into the walls of the atria (65). Afferent fibers extend distally as far as the proximal ends of the alveolar ducts(65).

The tonus of the bronchial musculature is influenced by impulses transmitted by the parasympathetic and sympathetic nerve fibers. Stimulation of the parasympathetic fibers causes broncho-constriction, and stimulation of the sympathetic fibers causes broncho-dilation. Changes in the size of bronchi are related to variations of nervous stimuli transmitted by the autonomic nervous system. Broncho-constriction, not unlike that observed in bronchial asthma in allergic states, may be produced by reflex stimuli created by emotional disturbances.

## THE EFFECTS OF EMOTIONS ON THE PATTERNS OF BREATHING

The fact that the emotions can give rise to symptoms which are similar to those produced by organic disease is well known. The effects of experimentally produced neuroses on some bodily functions in animals may be observed in the laboratory. This is discussed by Liddell (67). In reference to an investigator he states, "By means of Pavlov's method, he can bring under experimental control the emotional reactions of his animal subjects and can bring about, at will, chronic disorders of their emotional reactions involving specific respiratory dysfunctions". In experiments with well conditioned sheep and goats, bleating was suppressed by conditioned reflexes; however, when neuroses produced by experimental conditions supervened, the animals became vocal again. By changes in the methods of conditioning in neurotic sheep and goats, various types of breathing were produced viz. increases in rate, variations in depth, gasps, periods of marked apnea, and audible labored respirations (67).

It is common knowledge that certain emotional states can vary the pattern of respiration. This is seen in every day life situations viz. a gasp or shriek in a frightened person, sobbing respirations in crying, and the rapid, shallow respirations which are evident in tense situations.

In individuals at rest, differences in the patterns of breathing have been observed (47, 84). Golla and Antonovitch (47) observed two types of respiratory patterns under these conditions. In the first type, the rhythm and the amplitude of the respirations remained absolutely

regular. This pattern was found in subjects with predominantly "auditory imagery". In the second type, the rhythm and amplitude were exceedingly irregular, in subjects with predominantly "auditory imagery". The characteristics of the pattern of breathing for a particular individual remained constant over a long period of time, and in this respect are similar to an individual's handwriting (3, 47). Patterns of breathing have been observed in normal subjects engaged in various types of mental activities such as adding, solving visual puzzles, and recollecting poetry (47). Subjects who had regular patterns at rest continued to have regular patterns while preoccupied with thought; however, in those who had irregular patterns of breathing at rest, the patterns became regular.

Variations in respiration produced by painful stimuli have been observed in normal subjects and in patients with neuroses. In the majority of normal individuals studied, Finesinger et al (38) observed an increase in the depth of the respirations, an increase in the minute volume, and more acute expiratory-inspiratory angles, while the metabolic rates were unchanged. In a group of patients with anxiety neuroses, hysterias, and phobias, painful stimuli produced increases in the rate of respirations, the minute volumes and the metabolic rates, and the expiratory-inspiratory angles became more acute. A third group of patients with diagnoses of hypochondriasis, reactive depression, compulsion neurosis, and questionable schizophrenia showed little or no change in the rate, an increase in the depth of respirations, and a small increase in the minute volume. Changes in the metabolic rate and the ex-

piratory-inspiratory angles were not consistent.

By spirographic tracings, Finesinger (37) has shown that variations in the patterns of breathing are associated with pleasant and unpleasant thoughts. Changes in the rate, depth, minute volume, and expiratory-inspiratory angles of tidal air curves were more pronounced in both a control group and a group consisting of patients with diagnoses of hysterias, phobias, and anxiety neuroses, than they were in patients with manifestations of hypochondriasis, reactive depression, compulsion neurosis, and questionable schizophrenia. Changes in the patterns of breathing occurred in subjects while "day-dreaming" (28). The changes, called "respiratory plateaux", consisted of slight periods of apnea which had a tendency to occur at the end of each respiration. Similar changes have been observed in patients with schizophrenia (28). Several college students with this type of pattern displayed evidence of maladjustment (28).

In patients with bronchial asthma and anxiety states, Stephenson and Ripley (103) observed changes in the respiratory patterns produced by emotions of anxiety, anger, depression and guilt. The pattern became irregular in most of the patients when an unpleasant emotion was experienced. Many of the patients with bronchial asthma had an increase in the duration of expiration; wheezing became audible in three patients. McDermott et al (71) in a study of fifty unselected cases of asthma reported that twenty patients stated that their first attack was precipitated by emotional disturbances. Thirty-one patients stated that subsequent asthmatic attacks were often precipitated by like causes.

Other changes in the breathing patterns have been described in

neuroses. Patients with anxiety neuroses often showed irregular levels of respiration with varying tidal air volumes; however, the respiratory rate was affected less (22). Deep, rapid breathing, with a greater increase in the depth than in the rate, has been described in conversion hysteria (22). Frequent sighing is commonly seen in many types of neurotic disturbances (5, 22, 39). Christie (22) analyzed 1500 B.M.R. tracings and found evidence suggestive of a neurosis in 104. A review of the case histories of these patients revealed that neuroses were diagnosed or indicated as probable in 55, 35 were discharged without a diagnosis, and in the remaining 14 there were no indications of neuroses.

It is evident from the work of numerous investigators that emotional disturbances may cause marked changes in the patterns of breathing. Frequently, many of these changes are accompanied by a large variety of symptoms referable to the circulatory and respiratory systems. Spirography may be useful in the determination of the origin of these symptoms. The role of spirography in this relationship has been well stated by Christie (22) who wrote, "It must be emphasized that we in no way claim that all who have a respiratory neurosis will give a typical respiratory tracing. We do claim that these respiratory irregularities, if sufficiently pronounced, are diagnostic of a respiratory neurosis, and that the incidence of these cases is much greater than is generally supposed."

# PART FOUR

# PSYCHIATRIC ILLNESS IN COAL MINERS

## PSYCHIATRIC ILLNESS IN COAL MINERS

Relatively few papers have been published pertaining to psychiatric illness in coal miners. The most extensive of these have been published by British observers.

Halliday (51) observed a rising incidence of psychosomatic illness in the insured general population of Scotland after World War I. This observation was based upon three factors which were: (1) increased duration of incapacity, (2) increase in the labels known to cover a significant proportion of psychosomatic illness, and (3) youth (younger age groups were affected to a relatively greater extent). A decline in the "social health" of the population, despite progressive improvement in the indices of its "physical health", was associated with the rising incidence of psychosomatic illness (52).

As a medical referee under the National Health Insurance Act, Halliday (52) was impressed by the fact that psychoneurotic and psychosomatic factors were very common causes for incapacity in underground miners. This impression was shared also by Dickson (34). It was confirmed by Halliday (53) when he compared the findings obtained from consecutive examinations of 200 miners and 200 non-miners who received sick benefits. One hundred and twenty-eight miners (64%) were disabled because of psychoneurotic and psychosomatic affections as compared to seventy-six (38%) non-miners in the same age groups. The increase in mental illnesses was associated with an increase in the incidence of accidents and other diseases among miners when compared to non-mining workers. Disorders with a high rate of incidence are listed in Table XV, page 117, in the appendix.

These data were compiled by Halliday (53) from morbidity statistics for the insured population of Scotland for the year 1935. In his opinion, the higher rate of incapacitating disorders in miners is due primarily to environmental factors, physical and psychological, peculiar to the occupation of mining. In contrast to the diseases with a higher incidence, certain diseases had a lower incidence in miners than in non-mining workers. The diseases and the ratios of the incidences are listed in Table XVI, page 118, in the Appendix. A recent study by Alastair and Brathwaite (2) also indicated that underground workers are not as stable emotionally as above-ground workers.

Bodily disturbances varied with the types of psychoneuroses, which were classified by Halliday (52,53) into three groups viz., anxiety states, anxiety-hysteria, and hysteria. Miners with anxiety states, which were the most numerous, had facial expressions which were strained, worried and wearied, and often had pale or blotched faces. Tachycardia, tremors, brisk knee jerks, visceroptosis, weakness and pallor were observed commonly. The symptoms were numerous and varied. These patients were usually certified by their practitioners as cases of anemia, anemia and debility, or debility.

In the group labeled anxiety-hysteria, the above signs and symptoms were present together with symptoms referrable to the respiratory, gastro-intestinal and locomotor systems. Included among these were rapid breathing, air-swallowing, pain in the lower abdomen, loss of power in the limbs, numbness, tingling, inability to move joints, and excruciating pains in the limbs. Patients with these signs and symptoms were usually diagnosed as sciatica, lumbago, fibrositis, neuritis or rheumatism by

their local physicians.

Patients in the hysterical group usually had facial expressions "devoid of anxiety and could be described by such terms as detached, calm, pleasant, carefree — in short — pathologically cheerful". Severe pain was the commonest symptom, the site being painful to touch as long as the patient's attention was not diverted.

In a series of one hundred cases of Scottish miners with psychiatric illnesses, Post (91) reported the distribution of syndromes as follows: (1) anxious-depressive psychoneurosis, 30 patients; (2) depressive psychosis, 18 patients; (3) persistent psychoneurosis (including psychopaths), 14 patients; (4) organic psychiatric syndrome, 12 patients; (5) schizophrenic psychosis, 11 patients; (6) conversion hysteria, 10 patients; and (7) physical disease accompanied by psychiatric symptoms, 5 patients.

In contrast to these types of mental disturbances in Scottish miners, Wiesel (106) noted "complete inhibition of anxiety" in the older miners from the eastern Kentucky area in the United States. Passivity and dependency were prominent features in these men. An example of the multiplicity of complaints and symptoms presented by this type of miner is given in a typical case history, a portion of which is worthy of quotation. "The patient stated that he quit work 6 years ago because 'his nerves gave away'. He would become extremely weak and have to rest. Four years ago he had a smothering sensation with numbness over the body and was told that he had had a heart attack. Since then he had seen numerous physicians and had been given various conflicting opinions and diagnoses, among them, low blood pressure, hardening of the arteries, gall bladder trouble, etc. Attacks were periodic and characterized by

burning in the chest like a ball of fire, pains around the heart, jerking of the eyes, smothering and shaking. Upon waking his legs hurt and muscles quivered. If he lifted anything he would 'go to pieces' for 24 hours and have to remain in bed. Dizzy spells and headaches were frequent. He mentioned that at one time he had shoved a coal car, which may have 'bust a nerve'". In the younger miners from the same area, however, Weisel observed, "a conscious awareness of anxiety and tension, similar to anxiety states seen in urban practice". These men also had passive dependent attitudes. A representative history of the complaints in this type of patient follows: "On admission he complained of 'nervousness'. In June 1950, while working in the garden, he had the sudden feeling of electricity running over him and blacked out. He remained off work for a few weeks and improved. On the day he was to return to work he had 2 spells during which everything seemed blurry and a peculiar feeling passed over him. He was afraid to return to work for fear of precipitating another attack, which aroused considerable anxiety. He also complained of urinary frequency, palpitation, indigestion, restlessness, a vague feeling of dread, and frightening dreams".

The psychosomatic illnesses in the Scottish miners affected primarily the respiratory, gastro-intestinal, cardiovascular and locomotor systems (53). The most common respiratory illnesses were asthma and chronic bronchitis. It is a well accepted fact that psychogenic factors are known to precipitate initial attacks of asthma (71, 1). The "Effort Syndrome" was the most frequent cardiovascular manifestation, and peptic ulcer was a frequently noted gastrointestinal illness (53). The locomotor system was implicated during periods of

emotional stress by symptoms referable to fibrositic nodules (53). Miner's nystagmus became a problem of considerable proportions (15).

Predisposing or contributing causes for an increased incidence of psychiatric illness in miners arise primarily from the mining environment, the social, and the economic conditions of the mining community (53).

Mining, even under the most ideal conditions, is hazardous and extremely arduous work which must be seen to be appreciated fully. The environment at the mine "face" has variable conditions pertaining to the degree of dampness, which may be dry to very wet with water dripping from the "roof", darkness, noise, dust, smoke, fumes and gases, and the vertical work space may be only twenty-seven inches. The bodily attitudes frequently assumed in the presence of these conditions are indeed remarkable. The mechanization of mines, which reduced the miner's work to "mere shovelling of coal" and kept them working at full capacity, was thought to be a factor which contributed to emotional disturbances (34). Post (91) found that the work conditions were closely related to the main etiological factor in twenty-eight out of one hundred miners with psychiatric disturbances. An accident to the miner himself or to a fellow worker is frequently a precipitating factor (53,91). The fear of accidents may be conscious or unconscious and the latter type was frequently revealed by dreams of mine accidents which, in some men, commenced after an accident had occurred (53). Fear of disease, likewise, is a very important factor (53, 92). Post (91) states that 41 of 100 patients admitted persistent fear, which "was the emotional background in 26 of 40 miners suffering from psychoneurotic illness of recent origin".

In contrast to this, Wiesel (106) noted that most of the older miners from the southeastern Kentucky areas denied fear of their jobs and even liked their work. On the other hand, younger miners from the same area had definite fears pertaining to their work in the mines.

A review of the social background of the southeastern Kentucky mining communities is presented in a paper by Wiesel and Arny (106). Prior to the development of the mining and lumbering industry in that area, the people supported themselves by farming. Family ties were very close and there was much personal interdependence. The standard of living was low and all had to work hard to exist. Large scale mining began in 1911, and with it there was a great influx of people. With the advent of this social and economic revolution, family solidarity began to deteriorate. Gradually the population became more and more dependent upon the mining companies, which became extremely powerful as individual self-reliance and independence dwindled. In the meantime, coal production continued to increase and the mining industry attracted large numbers of workers from other occupations, thereby creating new community social problems. In 1922 a sudden drop in the demand for coal produced unemployment, which caused many hardships. Out of desperation and necessity the miners were gradually attracted to unions, and as the latter gained power conflicts between the operators and unions developed. A period of violence occured in the early 1930's, which resulted in a retardation of social progress. Concomitantly, the depression of 1929 increased the suffering, with the result that many of the people began to farm as a means of earning a livelihood. During these turbulent times large numbers of miners became affiliated with the union and transferred their dependence from the mine

operators to the unions. Many miners in the older age groups have witnessed and experienced these marked socio-economic changes in these communities.

In some respects, the families in the Scottish mining communities were not unlike those in the Kentucky mine areas. Family ties were strong, and sons followed in their father's footsteps (52). Fathers, sons, relatives and close friends worked together harmoniously in small groups in the mine. They enjoyed their work and were proud of their occupation. The advent of mechanization, however, brought about a great change in the nature of the work (34). The miner's task was reduced to shovelling the coal which had been cut for them during the night into the conveyers. The extreme noise of the machinery made it impossible for them to hear the warning signs of cracking strata. The close work relationships of one miner to another were altered, and, according to Dickson, the men stated that they no longer had the same personal interest in their work, and felt that they were like cogs in a wheel (34). In addition the men complained that the machines kept them working at full capacity, and in their opinion they were chased at their work. Because of these changes in the work pattern, Dickson believed that a new attitude had evolved which he described as follows: "that feeling is not one of active discontent with the conditions of work, but what may, from our point of view, be worse, passive dissatisfaction. It almost goes as far as being a repression". Greater contact with the outside world, made possible by automobiles, papers, and movies, helped to disrupt the established social patterns in the mining communities of both Kentucky and Scotland (52,106). The younger generations began to show dissatisfaction with the existing

social and cultural conditions.

The reaction to unemployment has been described by Brock (15) as follows: "Whenever large numbers of men were discharged from a pit there was at once a rush to be certified. Many of these men declared quite frankly that they could work if only they had a chance to do so, but, as they often added, a man must live. Yet having once been certified - the die having been cast - many of them proceeded to develop chronic neuroses and a depressed and melancholy state of mind". In addition to unemployment, defective or poorly administered sick benefit funds fostered dependency and encouraged the development or prolongation of psychiatric illness (52,106). Often, the men were satisfied with the money received from a source of this type and preferred to stay at home rather than return to hazardous work (106).

# PART FIVE

# SUMMARY

## SUMMARY

Fifty-one coal miners were studied consecutively during the period from August 20, 1951 to January 5, 1953. The patients included in this study were sent to us by officials of the United Mine Workers of America Welfare and Retirement Fund, because they presented problems with respect to either diagnosis, the extent of physical incapacity, or both. Most of the patients were studied on an out-patient basis except for short periods of hospitalization which were required for diagnostic and minor surgical procedures. One patient (No. 25, L.S.) required hospitalization upon arrival, a second patient (No. 28, S.D.) had to be hospitalized shortly after his arrival, and a third patient (No. 44, S.S.) had to be admitted to a closed psychiatric ward because of the development of an acute psychotic episode.

The geographic distribution of these patients was as follows: (1) thirty-six from southeastern Kentucky, (2) twelve from mid and southwestern Indiana, (3) one from each of the states Ohio, West Virginia, and Michigan. The patient from Michigan had worked in mines in Illinois and Kentucky.

The ages of the patients ranged from 36 to 66 years, and averaged 51.3 years.

The number of years worked in underground mining could be approximated only, because of memory defects, frequent changes of employment in various mines, and time lost because of strikes, idleness, and miscellaneous causes. Two of the patients did not give a history of underground mining. The approximate number of years worked underground ranged from 4 to 50

years, and averaged 27 years. The great majority of men performed various types of work, which included drilling, blasting, hand loading, timbering, laying track, maintenance, driving mules, supervisory positions, and operating cutting machines.

The patients were plain, sincere, honest, humble, grateful and very cooperative, except in isolated instances.

Tables giving the age, geographical area, number of years of underground mining, admission diagnosis, psychiatric diagnosis if psychiatric illness was present, and discharge diagnosis for each patient, are presented in the appendix (pages 119-127 ).

Most of the patients were observed and studied over a period of three to six weeks, the average stay being 29 days. Each day(except Sunday) was spent in the pulmonary function laboratory. This provided an opportunity for constant observation and for frequent conversation between each patient and physicians, nurses, and technicians. The close relationships thus established were found to be invaluable for allaying fears pertaining to procedures, for casual, objective observation of the patients during rest and social activities, for providing competition whenever voluntary efforts were required, and for becoming familiar with the personality of each patient. Much insight into any emotional components which contributed to the individual's "disability" was gained in this manner and, in our opinion, aided in the accurate appraisal of the physical and mental status of each patient. The cost of studying patients on this type of out-patient basis is relatively small, and an equal expenditure would permit only a short-term study if hospital facilities were utilized.

On admission, detailed general and occupational histories were obtained, and a physical examination was performed by a physician. Upon the completion of the history and physical examination, the following studies were done. Initially, not all of these studies were possible, because of limitations in facilities and personnel.

A. Hematology

(1) Complete blood count

(2) Hematocrit

(3) Sedimentation rate

B. Blood chemistry

(1) Non-protein nitrogen

(2) Fasting blood sugar

C. Serology

(1) V.D.R.L.

D. Complete urine analysis

E. Sputum examination. Three twenty-four hour specimens were obtained and examined for the following:

(1) Volume

(2) Appearance

(3) Smears and cultures for:

(a) Acid fast bacilli

(b) Curschmann's spirals

(c) Cellular material

(d) Sensitivity of the organisms cultured to penicillin, streptomycin, Aureomycin, Terramycin, erythromycin, and Chloromycetin.

F. X-rays

(1) P-A inspiratory and expiratory films

(2) Lateral film

G. Fluoroscopic examination of the chest

(1) Anatomical abnormalities

(2) Movements during normal respiration

(3) Movements during forced respiration

(4) Lateral view during normal and forced respirations

H. Cardiovascular studies

(1) Electrocardiogram

(2) Circulation time

(a) Arm to tongue

(b) Arm to lung

(3) Venous pressure

(4) Fluoroscopic examination of the heart

I. Pulmonary function studies

(1) Maximum breathing capacity

(2) Exercise tolerance studies at rest and after exercise

(a) Minute ventilation

(b) Oxygen consumption

(c) Carbon dioxide production

(d) Respiratory quotient

(e) Oxygen ventilation equivalents

(f) Carbon dioxide ventilation equivalents

(g) Dyspnea indices

(3) Arterial blood studies at rest and after exercise

(a) Oxygen content

(b) Oxygen capacity

(c) Oxygen saturation

(d) Carbon dioxide content

J. Lung volume determination

(1) Vital capacity

(2) Expiratory reserve volume

(3) Functional residual capacity

(4) Residual volume

K. Spirogram

L. Pneumotachogram

M. Direct observation of the patient while walking up four flights of stairs (if this effort could be tolerated).

Most of the patients (46 or 90.2%) were examined by a cardiologist to determine whether cardiovascular disease contributed to the symptomatology, objective signs, or respiratory insufficiency when present.

Initially only those patients with obvious psychosomatic factors were referred to a psychiatrist. It soon became apparent that psychosomatic illness was one of the major factors responsible for the "disability" of a significant number of patients. Because of this finding, psychiatric consultation was included as one of the routine diagnostic examinations for most of the patients. A definite pattern of interviewing was used in the examinations. This consisted of questions related to: (1) childhood factors, (2) adult adjustment before the illness, (3) accidents to the patient, (4) accidents to others, (5) relevant deaths,

(6) other major events, (7) the relation of the symptoms to events, and (8) the medical care which had been received.

Forty patients (78.4%) were seen in consultation by psychiatrists. Of these, thirty-five were believed to have psychiatric disturbances. Five patients, in the opinion of the psychiatrists, appeared to be fairly well adjusted to their physical incapacities. Four additional patients (case nos. 2, 13, 18, 27), not examined by psychiatrists, had, in our opinion, emotional disturbances which contributed to their disability.

On admission, sixteen patients had diagnoses of silicosis, two were diagnosed as probable silicosis, and one was diagnosed as a pneumoconiosis. Other diagnoses included arthritis, asthma, bronchiectasis, bronchitis, emphysema, pulmonary fibrosis, spontaneous pneumothorax, and tuberculosis.

Analysis of the case records for the most significant diagnosis in each case gave the following results:

1. Pneumoconiosis. Sixteen cases (31.4%) were present. This group was comprised of four cases of silicosis, eight cases of anthracosis, two cases of anthracosilicosis, one case of pneumoconiosis, $P_2$, and one case of pneumoconiosis, $P_1$. The diagnosis of pneumoconiosis was made from the history of exposure and x-ray interpretations.
2. Psychoneurosis. Thirteen cases (25.5%) had a psychoneurosis as the major diagnosis.
3. Emphysema, pulmonary, etiology undetermined. This was the major diagnosis in eight cases (15.6%). Emphysema, of varying degrees of severity, was present in sixteen additional cases. The diagnosis of emphysema was based upon: (a) abnormal fluoroscopic findings, which consisted of limited diaphragmatic motion and decreased ability to empty

the lungs on forced expiration; (b) abnormal x-ray findings, which were detected when the full inspiratory and full expiratory films of the chest were compared and consisted of limited changes in the positions of the diaphragm and bony structures of the thoracic cage; (c) decreased values for the pulmonary function tests which measure the ventilatory capacity of the lungs.

4. Bronchial asthma. This was the major diagnosis in six cases (11.8%).

5. Arthritis, hypertrophic (various anatomical sites). This was the major diagnosis in 3 cases (5.9%).

6. Miscellaneous diagnoses. Five cases (9.8%) were present in this group which consisted of one case of each of the following diagnoses: (a) heart disease, etiology undetermined, (b) trachoma, acute, inactive, (c) bronchitis and bronchiolitis, (d) papilloma, benign, left vocal cord, (e) hyper-mobile semi-lunar cartilage, left knee.

These data are presented in Table 12, page 114, in the appendix.

The patients in this study have been placed in one of the following groups:

Group I. Patients with no disability. Two patients (3.9%) were in this group.

Group II. Patients with mild psychiatric disturbances and no physical disability. Seven patients (13.7%) were in this group.

Group III. Patients with marked psychiatric disturbances and no physical disability. Eight patients (15.7%) were in this group.

Group IV. Patients with organic disease and psychiatric findings and varying degrees of disability. Twenty-four patients

(47.1%) were in this group.

Group V. Patients with organic disease without significant psychiatric findings. Ten patients (19.5%) were in this group.

Seventeen patients (33.3%) in groups I, II, and III did not have physical incapacities, in our opinion, which would prevent them from mining. The patients in Group II were thought to be capable of returning to mining, provided they were given psychotherapy in the form of reassurance and support by a physician. The marked psychiatric disturbances present in the patients of Group III made the probability and desirability of a return to mining unlikely. None of the twenty-four patients in Group IV, were, in our opinion, capable of mining because of physical incapacities associated with psychiatric disturbances. The severity of the organic disease present in nine of the ten patients in Group V precluded the occupation of mining. One patient in this group was believed capable of mining, if the recommended eye surgery was successful. These data are summarized in Table 13, page 115, in the appendix.

Rehabilitation and employment in some type of productive work was thought to be possible for forty-two patients (82.3%). All of the patients in Groups I and II were believed to be capable of mining. Work in an occupation other than mining was considered possible for all the patients in Group III. Employment of some type was believed possible for eighteen patients, questionable for four patients, and improbable for two patients in Group IV. Seven patients in Group V were considered able to engage in various types of productive work other than mining. These data are summarized in Table 14, page 116, in the appendix.

The results of this study emphasize clearly the extreme importance

of investigating the psychic as well as the physical factors which contribute to the "disability" present in every case. The "disability" incurred from emotional disturbances is as important as that which results from organic disease processes, in respect to productivity and economic loss to the individual and society.

The loss of function which results from organic disease may be estimated only after a careful review of the general and occupational histories, the physical examination, roentgenologic studies, the indicated clinical laboratory studies, and pulmonary function tests. The evaluation of "disability" which results from psychic disturbances must take into consideration family relationships and environmental factors with respect to the community and conditions of work. The latter must include the type of work, the hazardous nature of the work, the physical conditions of the work environment, and the effects on the individual produced by accidents at work, which involve the patient, relatives or friends.

Not infrequently emotional disturbances and psychoneuroses have been precipitated, or existing feelings and fears exaggerated, after the patient has sought medical attention for complaints referable to either the pulmonary or other systems. Many of the patients in this series have been told by physicians that their symptoms are related directly to work in the mines and that they should not return to mining. Statements to this effect are justifiable in many instances; however, in many cases they were completely unfounded and were made on the basis of inadequate histories and cursory physical examinations, and usually without recourse to physiologic measurements. Frequently the symptomotology cannot be explained and the physician may be compelled by the patient to express an

opinion in regard to the causal factors. Frankness, of course, is the most desirable approach; however, situations occasionally may arise where this is not desirable. Under these circumstances, implication of the patient's occupation or occupational environment is dangerous. Explanations associating the work as the etiologic agent are accepted readily by the patients and in many instances become fixed, so that, at some future time when the actual etiologic factors are known, the patient has become a psychological problem of such magnitude that successful rehabilitation is difficult, prolonged and even unlikely. The social and economic problems created under these conditions, not only for the patient but for society, are indeed serious.

Physical incapacities may be measured often in a relatively short time. In contrast, however, an understanding of the emotional components, which may be responsible in part or entirely for the existing "disability", are time-consuming and require an awareness by the physician of the role which they play in contributing to the inability or reduced capacity for work.

Rehabilitation (excluding those patients in Groups I and II who were thought capable of returning to mining), with a return to some type of productive work other than underground mining, was considered possible in 64.7% of the patients. Unless this is undertaken, it is quite possible that many of these men will show progressive mental regression followed by a more rapid physical deterioration than would otherwise occur if they were engaged in some type of employment. Rehabilitation with employment in an industry other than mining in the southeastern Kentucky area has limited possibilities at the present time and presents a problem which

deserves much consideration. It is believed, however, that some of the patients with less disabling organic diseases and mild psychiatric disturbances can be aided in their rehabilitation by frequent reassurance, support, and guidance by their private physicians.

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# APPENDIX

TABLE I

VITAL CAPACITY

| Investigator | Sex | Mean age | Number of cases | Mean (liters) | S. D. (liters) | Range (liters) | Position |
|---|---|---|---|---|---|---|---|
| (1) | M | 38.3 | 66 | 3.80 | 0.55 | ____ | sitting |
| (2) | M | 29.3 | 16 | 5.08 | 0.48 | 4.37-5.89 | " |
| (3) | M | 22.9 | 50 | 4.78 | 0.59 | 3.40-5.85 | recumbent |
| (4) | M | 22.9 | 50 | 4.78 | 0.59 | 3.40-5.85 | " |
| (4) | M | 48.2 | 50 | 4.07 | 0.62 | 2.64-5.85 | " |
| (5) | M | 25.5 | 17 | 4.012 | 0.616 | 2.792-4.950 | ____ |
| (5) | M | 42.7 | 15 | 4.160 | 0.480 | 3.300-5.240 | ____ |
| (5) | M | 54.6 | 18 | 3.417 | 0.825 | 2.184-5.429 | ____ |
| (6) | M | ____ | 194 | 5.13 | 0.66 | ____ | ____ |
| (7) | M | 61.5 | 11 | 3.48 | 0.48 | ____ | semi-recumb. |
| (5) | F | 25.1 | 16 | 3.057 | 0.551 | 2.312-4.150 | ____ |
| (5) | F | 43.3 | 10 | 2.830 | 0.397 | 2.212-3.435 | ____ |
| (5) | F | 59.8 | 13 | 2.431 | 0.532 | 1.570-3.525 | ____ |
| (2) | F | 29.4 | 19 | 3.57 | 0.44 | 2.86-4.47 | sitting |
| (6) | F | ____ | 40 | 3.58 | 0.53 | ____ | ____ |
| (7) | F | 60.4 | 15 | 2.34 | 0.45 | ____ | semi-recumb. |

(1) Aslett (4)
(2) Birath (12)
(3) Hurtado & Boller (54)
(4) Kaltreider et al (61)
(5) Baldwin et al (6)
(6) Matheson et al (73)
(7) Greifenstein et al (50)

TABLE II

THE INSPIRATORY CAPACITY

| Investigator | Sex | Number of Cases | Average Age | Mean (Liters) | S.D. (Liters) | Range (Liters) |
|---|---|---|---|---|---|---|
| (1) | M | 50 | 22.9 | 3.79 ± .05 | .52 ± .04 | 2.41 - 4.93 |
| (1) | M | 50 | 48.2 | 3.37 ± .05 | .57 ± .04 | 2.04 - 4.75 |
| (2) | M | 16 | 29.3 | 3.39 | _____ | 2.68 - 4.24 |
| (3) | M | 50 | 22.9 | 3.79 | .52 ± .04 | 2.41 - 4.93 |
| (4) | M | 11 | 61.5 | 2.61 | 0.61 | _____ |
| (2) | F | 19 | 29.9 | 2.56 | _____ | 1.93 - 3.34 |
| (4) | F | 14 | 60.9 | 1.90 | 0.34 | _____ |

(1) Kaltreider et al (61)

(2) Birath (12)

(3) Hurtado and Boller (54)

(4) Greifenstein et al (50)

TABLE III

THE EXPIRATORY RESERVE VOLUME

| Investigator | Sex | Average Age | Number of Cases | Mean (Liters) | S.D. (Liters) | Range (Liters) |
|---|---|---|---|---|---|---|
| (1) | M | 39 and under | 38 | 1.39 | _____ | _____ |
| (1) | M | 40 and over | 26 | 1.22 | _____ | _____ |
| (2) | M | 23 | 50 | .98 ± .02 | 0.26 ± .02 | 0.26 - 1.58 |
| (2) | M | 48.2 | 50 | .69 ± .03 | 0.31 ± .02 | 0.14 - 1.76 |
| (3) | M | 22.9 | 50 | .98 ± 0.02 | .26 ± 0.02 | 0.26 - 1.58 |
| (4) | M | 29.3 | 16 | 1.69 | _____ | 1.01 - 2.21 |
| (5) | M | 61.5 | 11 | 1.02 | 0.61 | _____ |
| (2) | F | 23.1 | 50 | .73 ± 0.02 | 0.19 ± 0.01 | 0.28 - 1.42 |
| (4) | F | 29.9 | 19 | 1.02 | _____ | 0.51 - 1.59 |
| (5) | F | 60.9 | 14 | 0.44 | 0.25 | _____ |

(1) Aslett (4)

(2) Kaltreider et al (61)

(3) Hurtado and Boller (54)

(4) Birath (12)

(5) Greifenstein et al (50)

TABLE IV

THE RESIDUAL VOLUME

| Investigator | Sex | Average Age | Number of Cases | Mean (Liters) | S.D. (Liters) | Range (Liters) |
|---|---|---|---|---|---|---|
| (1) | M | 22.9 | 50 | 1.19 | 0.35 | 0.56 - 1.98 |
| (1) | M | 48.2 | 50 | 1.30 | 0.41 | 0.54 - 2.29 |
| (2) | M | 29.3 | 16 | 1.49 | 0.23 | 0.96 - 1.80 |
| (2) | F | 29.9 | 19 | 1.22 | 0.25 | 0.81 - 1.61 |
| (1) | F | 23.1 | 50 | 1.10 | 0.30 | 0.53 - 2.26 |
| (3) | F | 22.9 | 50 | 1.36 | 0.38 | .81 - 2.16 |
| (4) | F | 39 & under | 38 | 1.52 | ——— | ——— |
| (4) | F | 40 & over | 26 | 1.70 | ——— | ——— |

(1) Kaltreider et al (61)

(2) Birath (12)

(3) Hurtado and Boller (54)

(4) Aslett (4)

TABLE V

THE FUNCTIONAL RESIDUAL CAPACITY

| Investigator | Sex | Number of Cases | Average Age | Mean (Liters) | S.D. (Liters) | Range (Liters) |
|---|---|---|---|---|---|---|
| (1) | M | 50 | 22.9 | 2.18 | 0.50 | 0.96 - 3.09 |
| (1) | M | 50 | 48.2 | 2.00 | 0.50 | 1.09 - 3.05 |
| (2) | M | 16 | 29.3 | 3.18 | 0.45 | 2.60 - 3.90 |
| (2) | F | 19 | 29.9 | 2.22 | 0.49 | 1.32 - 3.20 |
| (1) | F | 50 | 23.1 | 1.82 | 0.39 | 1.15 - 3.10 |
| (3) | F | 50 | 22.9 | 2.34 | 0.49 | 1.09 - 3.38 |
| (4) | F | 38 | 39 & under | 2.92 | ——— | ——— |
| (4) | F | 26 | 40 & over | 2.92 | ——— | ——— |

(1) Kaltreider et al (61)

(2) Birath (12)

(3) Hurtado and Boller (54)

(4) Aslett (4)

TABLE VI

THE TOTAL LUNG CAPACITY

| Investigator | Sex | Number of Cases | Average Age | Mean (Liters) | S.D. (Liters) | Range (Liters) |
|---|---|---|---|---|---|---|
| (1) | M | 50 | 22.9 | 6.13 | 0.82 | 4.42 - 7.86 |
| (2) | M | 50 | 22.9 | 5.97 | 0.81 | 4.25 - 7.68 |
| (2) | M | 50 | 48.2 | 5.37 | 0.84 | 3.62 - 7.61 |
| (3) | M | 16 | 29.3 | 6.57 | 0.61 | 5.56 - 7.69 |
| (4) | M | 66 | 38.3 | 5.42 | 0.84 | ——— |
| (3) | F | 19 | 29.3 | 4.79 | 0.63 | 3.74 - 6.08 |

(1) Hurtado and Boller (54)
(2) Kaltreider et al (61)
(3) Birath (12)
(4) Aslett (4)

TABLE VII

VENTILATION EQUIVALENTS FOR OXYGEN

| Investigators | Mean (Liters)* | Range (Liters)* |
|---|---|---|
| Knipping and Moncrieff (63) | 2.37 | 1.68 - 2.89 |
| Hurtado and Boller (54) | 2.66 | 1.47 - 4.04 |
| Kaltreider et al (62) | 2.40 | 1.83 - 3.98 |
| McMichael (72) | 2.49 | ——— |

* Liters breathed per 100 cc. $O_2$ consumed.

TABLE VIII

FINDINGS IN 58 CASES OF PULMONARY FIBROSIS

| Group | Total Capacity | | Functional Residual Capacity | |
|---|---|---|---|---|
| | Determined Volume in Liters | Percentage of Difference from Predicted | Determined Volume in Liters | Percentage of Difference from Predicted |
| I | 4.90 | -16.0 | 2.19 | -1.4 |
| II | 5.72 | -5.4 | 3.60 | +55.4 |
| III | 4.28 | -24.5 | 2.26 | 3.6 |
| IV | 4.09 | -21.9 | 2.10 | +4.1 |
| V | 5.00 | -23.4 | 3.19 | +27.7 |
| VI | 3.22 | -39.6 | 2.53 | +7.4 |

| Group | Residual Volume | | $\frac{\text{Residual Volume X 100}}{\text{Total Capacity}}$ | |
|---|---|---|---|---|
| | Determined Volume in Liters | Percentage of Difference From Predicted | Determined Volume in Liters | Percentage of Difference from Predicted |
| I | 1.61 | +24.9 | 44.8 | 18.2 |
| II | 3.16 | +134.8 | 53.9 | 145.0 |
| III | 1.66 | +31.0 | 39.2 | +78.1 |
| IV | 1.55 | +29.2 | 37.5 | 70.4 |
| V | 2.55 | +76.1 | 50.3 | +128.6 |
| VI | 1.74 | +23.0 | 44.4 | 101.8 |

TABLE IX

COMPARISON OF THE ROENTGENOLOGIC FINDINGS WITH THE DEGREE OF EMPHYSEMA BASED ON THE RESIDUAL VOLUME PER CENT OF TOTAL LUNG VOLUME (80)

| Group | Residual Volume Per Cent of Total Lung Volume | Number of Cases | Normal or Borderline Cases | Stage of Silicosis | | |
|---|---|---|---|---|---|---|
| | | | | First | Second | Third |
| I | 25 or Less | 30 | 5 | 1 | 10 | 14 |
| II | 25 - 35 | 62 | 6 | 6 | 14 | 36 |
| III | 35 - 45 | 74 | 7 | 9 | 5 | 53 |
| IV | 45 - 55 | 60 | 4 | 12 | 11 | 33 |
| V | 55 and above | 50 | 5 | 8 | 9 | 28 |
| Total number | | 276 | 27 | 36 | 49 | 164 |

TABLE X

CORRELATION OF THE ROENTGENOLOGIC APPEARANCE OF THE LUNGS WITH ARTERIAL OXYGEN SATURATION IN PATIENTS WITH PULMONARY FIBROSIS (60)

| | | $CO_2$ Content | | $O_2$ Saturation | |
|---|---|---|---|---|---|
| Group | No. of Cases | Average Vol. % | Variations in Vol. % | Average % Sat. | Variations % Sat. |
| I | 17 | 43.72 | 39.35 - 47.80 | 94.1 | 91.4 - 98.5 |
| II | 12 | 45.08 | 39.62 - 55.21 | 90.1 | 80.6 - 96.3 |
| III | 3 | 44.13 | 42.00 - 46.54 | 93.8 | 92.2 - 95.7 |
| IV | 3 | 48.91 | 47.39 - 51.83 | 91.2 | 87.3 - 93.7 |
| V | 2 | 47.61 | 46.86 - 48.37 | 89.3 | 86.7 - 91.9 |

Group I. Cases which showed increased linear markings in the lung fields.

Group II. Patients in this group showed moderate shadows.

Group III. The nodular shadows showed a tendency to agglomerate, giving a mottled appearance.

Group IV. Large dence shadows chiefly in the upper portions of the lung and in addition showed marked emphysema at the bases of the lungs.

Group V. A fine and diffuse reticular fibrosis involving the whole of the lung fields was present.

TABLE XI

| | | Vital Capacity | | | | Residual Volume | | | | Total Lung Capacity | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Right Side | | Left Side | | Right Side | | Left Side | | Right Side | | Left Side | |
| Patient Number | Sex | CC. | Per Cent of Total | CC. | Per Cent of Total | CC. | Per Cent of Total | CC. | Per Cent of Total | CC. | Per Cent of Total | CC. | Per Cent of Total |
| 1 | M | 2355 | 50 | 2335 | 50 | 490 | 51 | 470 | 49 | 2845 | 50 | 2805 | 50 |
| 2 | M | 2320 | 60 | 1580 | 40 | 680 | 55 | 550 | 45 | 3000 | 59 | 2130 | 41 |
| 3 | M | 2390 | 53 | 2070 | 47 | 610 | 54 | 530 | 46 | 3000 | 54 | 2600 | 46 |
| 4 | M | 2070 | 54 | 1770 | 46 | 490 | 54 | 410 | 46 | 2560 | 54 | 2180 | 46 |
| Mean | | 2284 | 54 | 1939 | 46 | 568 | 54 | 490 | 46 | 2851 | 54 | 2429 | 46 |

TABLE XI (CONTINUED)

| Patient Number | Sex | $\frac{\text{Residual Volume}}{\text{Total Lung Capacity}} \times 100$ | | | Alveolar Mixing Index | | |
|---|---|---|---|---|---|---|---|
| | | Right Side (Per Cent) | Left Side (Per Cent) | Combined Spirometry (Per Cent) | Right Side Per Cent Nitrogen | Left Side Per Cent Nitrogen | Combined Spirometry Per Cent Nitrogen |
| 1 | M | 17 | 17 | 17 | 1.16 | 1.46 | 1.56 |
| 2 | M | 23 | 26 | 24 | 1.46 | 1.66 | 1.62 |
| 3 | M | 20 | 20 | 20 | 0.96 | 0.82 | 1.10 |
| 4 | M | 10 | 19 | 19 | 1.18 | 1.26 | 1.34 |
| Mean | | 20 | 21 | 20 | 1.19 | 1.30 | 1.41 |

TABLE XII

DIAGNOSES CONSIDERED MOST SIGNIFICANT IN EACH CASE

| Diagnoses | Case Numbers |
|---|---|
| Pneumoconiosis | 2,7,11,12,13,17,20,22,23, 24,25,29,43,44,49,50 |
| Psychoneurosis | 1,3,6,10,14,16,26,30,35, 36,41,45,48 |
| Emphysema, pulmonary | 5,8,19,21,31,40,42,51 |
| Asthma, bronchial | 15,28,32,37,38,46 |
| Arthritis, hypertrophic | 4,9,39 |
| Heart disease, etiology not established | 33 |
| Trachoma, acute, inactive | 34 |
| Bronchitis and bronchiolitis | 47 |
| Papilloma, benign, left vocal cord | 18 |
| Hyper-mobile semi-lunar cartilage, left knee | 27 |

TABLE XIII

CAPABILITY OF PATIENTS FOR MINING

| Group * | Patients thought capable of a return to mining. | | Patients thought incapable of a return to mining. | | Patients thought capable of a return to mining after corrective procedures. | |
|---|---|---|---|---|---|---|
| Group | Number of cases | % of Total | Number of Cases | % of Total | Number of cases | % of Total |
| I | 2 | 3.9 | | | | |
| II | 7 | 13.7 | | | | |
| III | | | 8 | 15.7 | | |
| IV | | | 24 | 47.1 | | |
| V | | | 9 | 17.6 | 1 | 1.9 |
| Total | 9 | 17.6 | 41 | 80.4 | 1 | 1.9 |

Cases:

Group I: 4,12

Group II: 1,3,18,35,36,41,49

Group III: 6,10,14,16,26,30,45,48

Group IV: 2,5,11,13,15,17,20,21,25,27,29,31,32, 33,37,38,40,42,43,44,46,47,50,51

Group V: 7,8,9,19,22,23,24,28,34,39

* These groups are described in Part V, page 9.

TABLE IV

REHABILITATION STATISTICS

| Group* | Rehabilitation with a return to mining or other work thought possible | | Rehabilitation questionable | | Rehabilitation thought not probable | |
|---|---|---|---|---|---|---|
| Group | Number of cases | % of Total | Number of Cases | % of Total | Number of cases | % of Total |
| I | 2 | 3.9 | | | | |
| II | 7 | 13.7 | | | | |
| III | 8 | 15.7 | | | | |
| IV | 18 | 35.3 | 4 (case nos.32, 44,47,51) | 7.8 | 2 (case nos. 20,25) | 3.9 |
| V | 7 | 13.7 | 3 (case nos.7, 8,19) | 5.9 | | |
| Total | 42 | 82.3 | 7 | 13.7 | 2 | 3.9 |

* These groups are described in Part V, page 7.

TABLE XV

DISORDERS WITH A HIGH RATE OF INCIDENCE (53)

This table shows the "disease" having a rate of incidence higher in miners than in working Scottish males who were not miners and indicates the ratio of excess in respect of each disorder.

| 2½ - 3 times | | 2 - 2½ times | | 1½ - 2 times | | 1 - 1½ times | |
|---|---|---|---|---|---|---|---|
| Accidents | (2.7) | Rheumatism | (2.2) | Cardiac debility | (1.8) | Upper Respiratory Affections | (1.3) |
| Sepsis and inflammation of the skin | (2.6) | Influenza | (2.2) | Bronchitis and pneumonia* | (1.8) | Gastric and Duodenal Ulcer | (1.2) |
| | | Anemia | (2) | Diseases of the veins | (1.7) | Appendicitis | (1.2) |
| | | Gastritis | (2) | Defined Skin Affections | (1.6) | Hernia | (1.1) |
| | | D.A.H. and tachycardia | (2) | Debility | (1.5) | Neurasthenia | (1.1) |

*Attention is called to the fact that "Bronchitis and pneumonia" were not separately treated in the original report from which the figures were taken, and that "anemia" is known to cover many examples of anxiety states.

TABLE XVI

DISEASES WITH A LOWER INCIDENCE IN MINERS THAN NON-MINING WORKERS (53)

| Disease | Ratio |
|---|---|
| Tuberculosis | .9 |
| Nervous debility* | .8 |
| Cerebral hemorrhage | .8 |
| Diabetes | .7 |
| Malignancy | .8 |
| Infectious Diseases | .5 |

* It is noted that the term "nervous debility", which has a low incidence, is one which is inacceptable to miners and therefore seldom appears in medical certificates.

TABLE XVII

CASE SUMMARIES

| Case | Age | Area | Years of Underground Mining | Admission Diagnosis | Psychiatric Diagnosis | Other Diagnoses |
|---|---|---|---|---|---|---|
| (1) P.M. | 44 | Auxier, Ky. | 27 | 1. Granular stage of silicosis | Psychoneurosis, anxiety, conversion and cardiac organ neurotic features | 1. Postural strain of upper back and neck<br>2. Osteo-arthritis of upper spine, mild<br>3. Dental caries |
| (2) L.C. | 53 | Millstone, Ky. | 27 | 1. Silicosis<br>2. Chronic bronchitis | Psychoneurosis, anxiety reaction | 1. Silicosis, second degree, not incapacitating<br>2. Ankylosis of the carpo-phalangeal joint of the right index finger |
| (3) D.H. | 45 | Carver, Ky. | 29 | 1. Bronchiectasis<br>2. Moderate arteriosclerosis | Psychoneurosis, anxiety and reactive depression type | 1. Carious teeth |
| (4) H.W. | 47 | Whitesburg, Ky. | 18 | 1. Pulmonary fibrosis<br>2. Silicosis | ——— | 1. Juvenile kyphosis, moderate, with secondary hypertrophic arthritis of the thoracic spine, moderate, non-disabling<br>2. Carious teeth |
| (5) W.W. | 63 | Flint, Mich. (Mined in Ill. and Ky. | 31 | 1. Pulmonary fibrosis<br>2. Emphysema | Psychoneurosis, anxiety (N.C.A.) and depressive reaction | 1. Emphysema, severe, non-obstructive<br>2. Heart disease, arteriosclerotic, with right bundle branch block |
| (6) J.A. | 54 | Cromona, Ky. | 29 | 1. Silicosis<br>2. Chronic Bronchitis<br>3. Arthritis, moderately severe | Psychoneurosis, depression and hypochondriacal features | 1. Arthritis, hypertrophic, cervical spine, moderate, not incapacitating<br>2. Mal posture (adult round back) |
| (7) E.S. | 66 | Terra Haute, Ind. | 20 | 1. Bullous emphysema, wide-spread | ——— | 1. Heart disease, right failure, etiology not established<br>2. Anthraco-silicosis |

TABLE XVII CONTINUED

| Case | Age | Area | Years of Underground Mining | Admission Diagnosis | Psychiatric Diagnosis | Other Diagnoses |
|---|---|---|---|---|---|---|
| (8) O.Mc. | 48 | Praise, Ky. | 25 | 1. Bronchiectasis, chronic, bilateral | _____ | 1. Emphysema, chronic, severe, non-obstructive, etiology undetermined<br>2. Bronchiectasis, minimal, right lower lobe |
| (9) G.B. | 64 | Brazil, Ind. | 35 | 1. Miner's asthma, marked dyspnea on exertion | _____ | 1. Arthritis, hypertrophic, lumber spine, severe, with mild sciatica<br>2. Arthritis, hypertrophic, right knee<br>3. Circulatory deficiency of feet probably due to inactivity<br>4. Dental caries |
| (10) B.B. | 50 | Melvin, Ky. | 27 | 1. Bronchial asthma<br>2. Bronchiectasis | Psychoneurosis, depressive and hypochondriacal features | 1. Arthritis, hypertrophic, lumber spine, advanced<br>2. Ethmoiditis, chronic, severe, improved<br>3. Ichthyosis and dermatophytosis<br>4. Dental caries |
| (11) C.T. | 49 | Kite, Ky. | 18 | 1. Bronchial asthma | Psychoneurosis, anxiety, aggravating pneumoconiosis | 1. Pneumonoconiosis, $P_2$<br>2. Bronchial asthma, psychogenic, severe<br>3. Emphysema, pulmonary, secondary to (2)<br>4. Arteriosclerosis, generalized, asymptomatic |
| (12) J.C. | 49 | Praise, Ky. | 33 | 1. Silicosis | _____ | 1. Anthraco-silicosis, first degree, non-disabling<br>2. Eosinophilia, etiology undetermined |
| (13) G.W. | 52 | Morton, Ky. | 36 | 1. Chronic fibrous pathology of the upper ½ of the right lung, prob. tuberculous | Psychoneurosis, anxiety depressive reaction | 1. Anthracosis with focal emphysema<br>2. Cystic disease of the lungs<br>3. Bronchiectasis |

TABLE XVII CONTINUED

| Case | Age | Area | Years of Underground Mining | Admission Diagnosis | Psychiatric Diagnosis | Other Diagnoses |
|---|---|---|---|---|---|---|
| (14) W.R. | 39 | Catletts-burg, Ky. | 20 | 1. Silicosis<br>2. Hypochlorhydria<br>3. Inadequacy, physical and personal | Psychoneurosis, anxiety and conversion features | 1. Carious teeth |
| (15) B.S. | 57 | Ulvah, Ky. | 25 | 1. Bronchial asthma<br>2. Pulmonary fibrosis | Psychoneurosis, depressive reaction, organ neurosis (bronchial asthma and hypochondriacal belief in tuberculosis) | 1. Bronchial asthma<br>2. Emphysema, pulmonary, moderately severe<br>3. Hydrocoele, left<br>4. Carious teeth, severe |
| (16) J.L.W. | 59 | Shelborn, Ind. | 26 | 1. Silicosis<br>2. Asthma | Psychoneurosis, reactive depressive (N.C.A.) | 1. Emphysema, pulmonary, mild<br>2. Carious teeth<br>3. Obesity |
| (17) O.J. | 55 | Pleasant-ville, Ind. | 35 | 1. Bronchial asthma | Psychoneurosis, depression and organ neurosis (N.C.A.) factors | 1. Anthracosis<br>2. Emphysema, pulmonary, secondary to (1)<br>3. Arteriosclerosis, generalized, moderate<br>4. Hypertension |
| (18) O.R. | 41 | Virgie, Ky. | 28 | 1. Pulmonary fibrosis, cause unknown | Psychoneurosis, anxiety reaction | 1. Papilloma, benign, of the left vocal cord<br>2. Fracture, sixth rib, healed<br>3. Probable pleural adhesions adjacent to the old rib fracture on the right, not disabling |
| (19) L.W. | 52 | Greenville, Ky. | 22 | 1. Emphysema, pulmonary | ——— | 1. Emphysema, pulmonary, bilateral, severe, with probable cystic disease of the lung<br>2. Pulmonary hypertension secondary to (1)<br>3. Arthritis, hypertrophic, of the spine<br>4. Hernia, inguinal, right<br>5. Tinea corporis |

TABLE XVII CONTINUED

| Case | Age | Area | Years of Underground Mining | Admission Diagnosis | Psychiatric Diagnosis | Other Diagnoses |
|---|---|---|---|---|---|---|
| (20) W.K. | 52 | Terra Haute, Ind. | 37 | 1. Silicosis | Psychoneurosis, conversion— depressive reaction | 1. Bronchiectasis, bilateral<br>2. Anthracosis, disabling<br>3. Emphysema, pulmonary, moderate<br>4. Seborrheic dermatitis |
| (21) E.R. | 47 | Busy, Ky. | 30 | 1. Bronchial asthma<br>2. Bronchiectasis | Psychoneurosis, anxiety depressive reaction | 1. Emphysema, pulmonary, marked, with severe disability<br>2. Bronchitis, chronic<br>3. Adhesions, pleural |
| (22) J.C. | 57 | Lothair, Ky. | 38 | 1. Tuberculosis, pulmonary, bilateral, far-advanced | ——— | 1. Anthracosis, with conglomeration, but without nodulation<br>2. Emphysema, bullous, probably secondary to (1)<br>3. Perforated nasal septum, traumatic |
| (23) R.W. | 55 | Allais, Ky. | 35 | 1. Silicosis | Regressive emotional reaction aggrevating organic disease symptom picture | 1. Silicosis, advanced,(probably with old tuberculous cavitation)<br>2. Ethmoiditis, chronic, suppurative<br>3. Otitis media, healed, old |
| (24) W.Y. | 44 | Blue Diamond, Ky. | 29 | 1. Silicosis | Organic disease without aprreciable emotional components | 1. Silicosis, far-advanced, not disabling at the present time<br>2. Pyorrhea and partial edentia<br>3. Pruritis, secondary to lubricating oils (on thighs) |
| (25) L.S. | 56 | Hardburley, Ky. | 11 | Bronchial Asthma, severe | Psychoneurosis, depressive reaction and organ neurosis(bronchial asthma) | 1. Pneumonoconiosis, $P_1$<br>2. Bronchopneumonia, left lung<br>3. Emphysema, pulmonary, advanced, secondary to (1)<br>4. Diverticulosis of colon<br>5. Inverted stomach<br>6. Heart disease, arteriosclerotic, compensated<br>7. Femoral hernia, bilateral<br>8. Sinusitis, maxillary, bilateral |

TABLE XVII CONTINUED

| Case | Age | Area | Years of Underground Mining | Admission Diagnosis | Psychiatric Diagnosis | Other Diagnoses |
|---|---|---|---|---|---|---|
| (26) K.C. | 45 | St. Helens, Ky. | 20 | 1. Asthmatic attacks, or 2. Bronchiectasis | Psychoneurosis, anxiety reaction | 1. Sinusitis, paranasal, chronic 2. Intestinal parasites (Trichuris trichiura) 3. Eosinophilia, secondary to: (a) intestinal parasites, (b) respiratory hypersensitivity |
| (27) R.S.W. | 51 | Beattyville, Ky. | 28 | 1. Bronchial asthma 2. Arthritis, hypertrophic, lumber spine | Psychoneurosis, hypochondriacal features | 1. Hyper-mobile semi-lunar cartilage, left knee 2. Moderately tight ilio-tibial band over the right hip 3. Seborrhea capitis, severe, and seborrheic dermatitis of the face |
| (28) S.D. | 52 | Wooten, Ky. | 17 | 1. Emphysema, pulmonary 2. Bronchial asthma | Satisfactory adjustment to illness | 1. Bronchial asthma 2. Emphysema, pulmonary, advanced, secondary to (1) 3. Heart disease, arteriosclerotic, compensated 4. Nasal septum perforation |
| (29) E.R. | 64 | Terra Haute, Ind. | 50 | 1. Silicosis, advanced | Psychoneurosis, reactive depression (prolonged grief) | 1. Silicosis, advanced 2. Hypertension, mild 3. Inguinal hernia, bilateral, recurrent 4. Carious teeth |
| (30) H.B. | 60 | Rosedale, Ind. | 44 | 1. Silicosis | Psychoneurosis, hypochondriacal features | 1. Edentia |

TABLE XVII CONTINUED

| Case | Age | Area | Years of Underground Mining | Admission Diagnosis | Psychiatric Diagnosis | Other Diagnoses |
|---|---|---|---|---|---|---|
| (31) L.O.L. | 58 | Terra Haute, Ind. | 43 | 1. Silicosis | Psychoneurosis, reactive depression | 1. Emphysema, pulmonary, bilateral, diffuse, severe<br>2. Bronchitis, chronic, low-grade<br>3. Lipoma, fourth finger, right hand<br>4. Possible renal pathology (patient left without permission) |
| (32) C.D.V. | 44 | McCarr, Ky. | 12 | 1. Asthma | Organ neurosis related to bronchial asthma | 1. Bronchial asthma<br>2. Emphysema, pulmonary, secondary to (1)<br>3. Inguinal hernia, direct, bilateral<br>4. Arthritis, hypertrophic, cervical and lumbar spine with narrowing of I.V. disc at C3 |
| (33) G.Mc. | 50 | Racoon, Ky. | 30 | 1. Silicosis | Psychotic character reaction, schizoid and hypochondriacal features | 1. Heart disease, etiology undetermined, compensated<br>2. Arthritis, hypertrophic, lumbosacral, minimal |
| (34) C.O. | 55 | Francisco, Ind. | None | 1. Severe dyspnea | ——— | 1. Trachoma, acute, inactive<br>2. Disseminated pulmonary calcification, etiology undetermined<br>3. Deformity of the left hand involving the index finger, 2nd metacarpal bone and thumb, old, traumatic |
| (35) K.C. | 45 | Leburn, Ky. | 4 | 1. Possible tuberculosis | Psychoneurosis, depressive reaction and hypochondrical features | 1. Intestinal parasites, Strongyloides stercoralis<br>2. Chronic, multiple dietary deficiency |
| (36) E.D. | 46 | Chesapeake, Ohio | None | 1. Episode of spontaneous pneumothorax | Psychic traumatic neurosis, anxiety, inhibition and anhedonic features | 1. Arteriosclerosis, generalized, with possible coronary insufficiency<br>2. Pneumothorax, spontaneous, old, recovered<br>3. Headache, post-traumatic |

TABLE XVII CONTINUED

| Case | Age | Area | Years of Underground Mining | Admission Diagnosis | Psychiatric Diagnosis | Other Diagnoses |
|---|---|---|---|---|---|---|
| (37) H.D. | 59 | Bicknell, Ind. | 18 | 1. Asthma, bronchial, severe with pulmonary emphysema | Psychoneurosis, reactive and organ neurosis (bronchial asthma) | 1. Bronchial constriction, chronic, allergic in type<br>2. Emphysema, pulmonary, minimal<br>3. Benign prostatic hypertrophy<br>4. Arteriosclerosis, generalized, moderately advanced |
| (38) O.W. | 52 | Van, W. Va. | 34 | 1. Asthmatic bronchitis | Psychoneurosis, traumatic | 1. Bronchial asthma<br>2. Emphysema, pulmonary, moderate, secondary to (1)<br>3. Arthritis, hypertrophic, dorso-lumbar spine, secondary to an ancient compression fracture of the 1st lumbar vertebra<br>4. Congenital bifid-type kidney<br>5. Obesity |
| (39) C.M. | 53 | Hymera, Ind. | 36 | 1. Arthritis | No significant psychiatric findings | 1. Ostero-arthritis, severe, of the dorsal and lumbar intervertebral bodies and involving both sacro-iliac joints<br>2. Degeneration of the fifth intervertebral disc<br>3. Arteriosclerosis, generalized, moderate |
| (40) N.T. | 50 | Beaver, Ky. | 29 | 1. Tuberculosis | Psychoneurosis, depressive reaction | 1. Emphysema, pulmonary, mild<br>2. Pyorrhea, severe, with dental caries<br>3. Perforated tympanic membrane, right, old<br>4. Amputation of second finger, right hand at the distal phalangeal joint, old |
| (41) R.D. | 42 | Anco, Ky. | 27 | 1. Emphysema, pulmonary<br>2. Otitis media, chronic, bilateral | Psychoneurosis, anxiety reaction | 1. Otitis, external, chronic<br>2. Tonsilitis, chronic<br>3. Pharyngitis, chronic<br>4. Obesity, moderate |

TABLE XVII CONTINUED

| Case | Age | Area | Years of Underground Mining | Admission Diagnosis | Psychiatric Diagnosis | Other Diagnoses |
|---|---|---|---|---|---|---|
| (42) A.V. | 37 | Topmost, Ky. | 13 | 1. Bronchial asthma | Psychophysiologic reaction, anxiety attacks (N.C.A.) or bronchial asthma | 1. Emphysema, pulmonary, mild<br>2. Pyorrhea and dental caries |
| (43) S.S. | 49 | Weeksbury, Ky. | 30 | 1. Silicosis | Hysterical psycosis, acute | 1. Emphysema, pulmonary, moderate<br>2. Anthracosis, pulmonary function efficiency undetermined |
| (44) R.R. | 49 | Barridge, Ky. | 27 | 1. Emphysema<br>2. Bronchiectasis | Psychic traumatic neurosis | 1. Anthracosis<br>2. Emphysema, pulmonary, moderate |
| (45) E.H. | 36 | Hardshell, Ky. | 11 | 1. Bronchiectasis, right lower lobe | Psychic traumatic neurosis, anxiety (N.C.A.) and conversion features | 1. Schuster-Brust chest deformity<br>2. Pyorrhea and dental caries |
| (46) I.B. | 56 | Robinson Creek, Ky. | 19 | 1. Asthmatic bronchitis<br>2. Possible silicosis | Psychoneurosis, anxiety and repressive (grief) reactions with organ neurosis (bronchial asthma) | 1. Bronchial asthma<br>2. Emphysema, pulmonary, slight, secondary to (1)<br>3. Arteriosclerosis, generalized<br>4. Mastoiditis, right, chronic |
| (47) A.D. | 59 | Terra Haute, Ind. | 40 | 1. Asthma<br>2. Emphysema, fibrosis<br>3. Chronic bronchitis | Psychic traumatic neurosis, masked depression and paranoid trends | 1. Bronchitis and bronchiolitis<br>2. Emphysema, pulmonary, secondary to (1)<br>3. Gynecomastia, right<br>4. Arthritis, hypertrophic, of the spine, severe<br>5. Fracture, compression, old, of 1st lumbar and lower thoracic vertebrae with residual deformity of spine |
| (48) F.H. | 49 | Whiteburg, Ky. | 16 | 1. Emphysema | Psychoneurosis, anxiety reaction | 1. Diverticulosis of the colon<br>2. Duodenal ulcer |
| (49) L.T. | 51 | Betsy Lane, Ky. | 27 | 1. Silicosis | Psychoneurosis, anxiety reaction | 1. Anthracosis<br>2. Tuberculosis, healed, of the third lumbar vertebra |

TABLE XVII CONTINUED

| Case | Age | Area | Years of Underground Mining | Admission Diagnosis | Psychiatric Diagnosis | Other Diagnoses |
|---|---|---|---|---|---|---|
| (50) W.W. | 53 | Allais, Ky. | 36 | 1. Possible tuberculosis<br>2. Possible pneumonoconiosis | Psychoneurosis, masked depressive reaction | 1. Anthracosis, advanced, mildly disabling at the present time but progressive<br>2. Probable ureteral disease which requires further investigation |
| (51) D.S. | 46 | Jackson, Ky. | 25 | 1. Possible emphysema | Psychoneurosis, post-thyroidectomy, neurasthenia and masked depression | 1. Emphysema, focal, with pulmonary fibrosis, mostly in the upper lobe |

FIGURE I

SUBDIVISIONS OF THE TOTAL LUNG VOLUME (87)

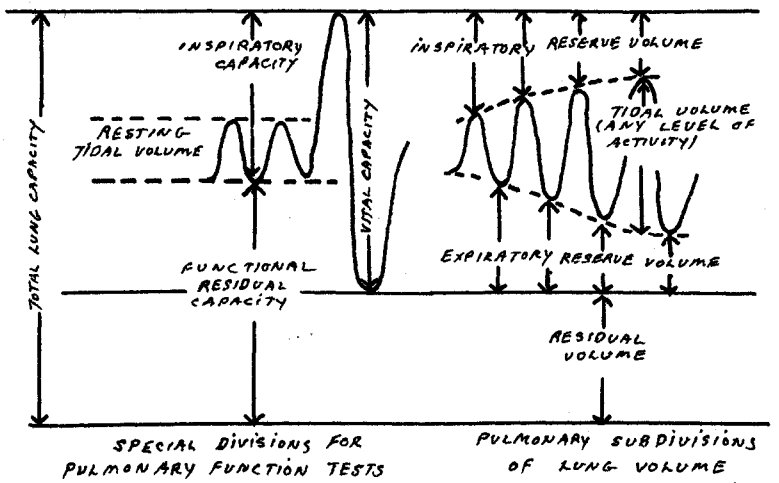
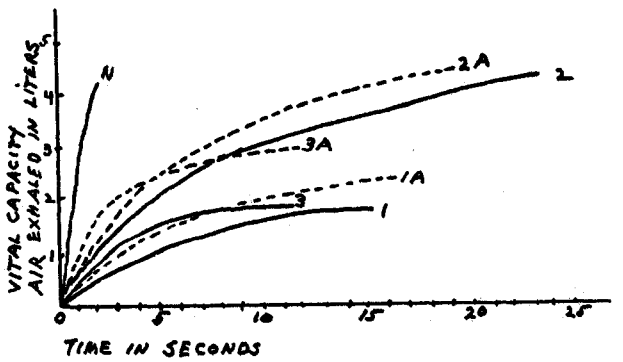



FIGURE II

VITAL-CAPACITY EXHALATION TIME CURVES




N tracing obtained from a normal person
1 and 1A tracings obtained in Case 2
2 and 2A tracings obtained in Case 3