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LABORATORY ASSESSMENT OF RESPIRATORY IMPAIRMENT FOR DISABILITY EVALUATION

Brian Boehlecke

INTRODUCTION

This chapter will consider the usefulness and limitations of various laboratory examinations in the assessment of impairment due to respiratory disease. Overall disability determinations must be based on socioeconomic and psychologic factors as well as medical examination and objective laboratory test results.

To appropriately screen large numbers of subjects, a laboratory test should meet certain requirements (79). The cost should be reasonable—relative to other tests giving the same or comparable information. The test must be safe, relatively simple, and acceptable to the subject population. Finally, the yield of useful information should be high. Several aspects must be considered to determine how well a test meets this last requirement: (a) The test should be objective, i.e., results should be independent of subject motivation and cooperation. Bias of the technician administering the test or the observer interpreting the results should also have little influence. Clearly no laboratory test of pulmonary function is completely objective under these criteria (15). If a subject is apprehensive while performing the test, physiologic responses may be altered. Thus, hyperventilation during an exercise test may be caused by anxiety or deliberate noncooperation as well as cardiopulmonary impairment. (b) The test should be reproducible. Variation in results, when multiple measurements are made on an individual, stems from both the true biologic variability of the function measured and measurement error induced by the equipment or the observed (15). Equipment and techniques must be calibrated and standardized so that results obtained in different individuals and in different laboratories can be directly compared. (c) The test must measure the biologic function of interest. Many medical tests provide only an indirect measure of the biologic function of interest. For example, the relationship between "disability" and measures of physiologic impairment is far from direct.

Impairment is generally accepted to mean reduction in function below that found in health. For objective tests to accurately quantitate impairment, the level of function prior to the onset of injury or illness must be known or an accurate prediction for "normal" function in health must be available. Often, neither condition is fulfilled. Disability may be considered to be present when an individual lacks the ability to perform a certain level of a specific task. Reduced efficiency for accomplishing a task may also constitute disability if the worker experiences undue distress or risk to well-being while performing the task. However, the severity of symptoms constituting undue distress is as much a social as a medical decision. The importance of distinguishing impairment from disability has been discussed by several authors (8)(14)(26)(33).

Laboratory tests of function may be used to assist disability evaluation in two ways. The measured level of function remaining may be compared to the demands of a given activity. Test results may also be correlated with independent measures of disability such as symptoms experienced while performing a given task. However, test results and symptoms may not be closely related for several reasons (14). In healthy individuals, pulmonary function capacity greatly exceeds daily activity performance requirements. A significant loss in function can occur before symptoms are experienced during usual levels of exertion (Figure I-22). Also, if more than one organ system is impaired, a test measuring the function of only one system will not correlate closely with the overall function of the individual. Individual variability in psychological response to illness and sensations of discomfort also contribute to the lack of direct correspon-

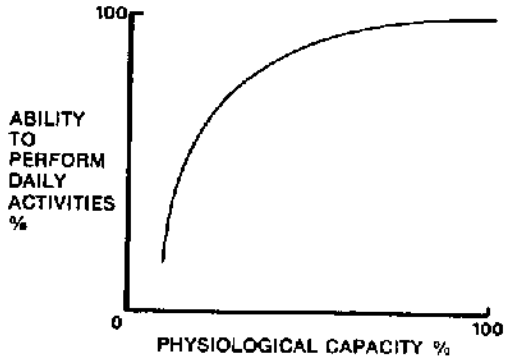

Figure I-22. Idealized relationship between physiologic capacity and ability to perform daily activities.

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dence of symptoms and objective tests of function. This is especially true when economic benefits are involved. Also, in conditions characterized by exacerbations and remissions (e.g., asthma), incapacity for work may be determined more by the frequency and severity of attacks than the level of function measured on a given day.

With these principles in mind, individual tests of pulmonary function and their use in disability assessment will be discussed.

SPIROMETRY

One of the most widely used objective tests of pulmonary function is the ability to move air as measured by spirometry. The technique requires the subject to make a full inspiration, to blow out as hard and rapidly as possible into the spirometer, and to continue exhaling until he has breathed out as much gas as possible. Measurements which can be made from this maneuver include the total volume of gas exhaled or the forced vital capacity (FVC), the volume of gas exhaled during the first second (FEV_1), and flow rates, i.e., the rate at which gas is being expelled at various points during the forced vital capacity maneuver. Results of this test clearly depend on a subject's ability to understand the maneuver, to completely fill his lungs prior to beginning the exhalation, to sustain a maximal effort during the exhalation, and to continue the effort until he has completely emptied his lungs. Results also depend on the number of maneuvers the subject performs, adequacy of coaching, and equipment characteristics—including resistance to air flow and inertia. The technique used for measuring the volumes and flows, including start-of-test and end-of-test criteria also influences results. Detailed reports of expert opinion on proper techniques and instrumentation standards have been presented (6)(27), and recommendations of standardized methodology have been prepared for the National Heart, Lung and Blood Institute (23). Spirometers accurate to within 3% of reading are currently available.

If an individual performs three maneuvers with values for FEV_1 within 5% of each other, further maneuvers add little information and do not significantly reduce the variability of results for the session. If a subject repeats spirometry several times over a single day or over several weeks, the coefficient of variation for this FVC and FEV_1, averages 2.5% to 3.0% (13)(46). Variability for flow rate at the mid-point of vital capacity is significantly greater, ranging from 6% to 8% when studied over a single day, to approximately 12% when studied over several weeks. Flow rates at lower lung volumes are even more variable. Thus, in terms of reproducibility, the standard forced vital capacity and FEV_1 measurements are most advantageous.

The ratio of FEV_1/FVC is a useful measure of the presence of obstruction. Many pathologic processes reduce the ability of the respiratory system to generate normal flow rates, especially on expiration. Although the FEV_1 may be reduced in absolute terms, because of the wide range of normal values and the usual lack of baseline data, small decrements in FEV_1 may not be easily detected. This is especially true if the subject's pulmonary function was significantly above the mean predicted for persons of the same sex, age, and physical characteristics, prior to the onset of his decrement. In obstructive lung disease, the FVC is often not reduced to the same extent as the FEV_1; therefore, the ratio of FEV_1 to FVC provides a more sensitive index of obstruction. Prediction equations for FEV_1/FVC ratio have been published. However, the variability of the FEV_1/FVC ratio has not been examined as thoroughly as each separate parameter. It is not appropriate to consider the ratio of FEV_1 to FVC alone, since restrictive impairment (which limits the total volume of air that can be expelled from the lungs, but not the rate at which it is expelled) would not be detected.

Spirometric values have an inherent vari-

ability in the same individual, even in the absence of changes in factors known to influence pulmonary function. Airway spasm induced by infection, allergic reaction, or inhalation of toxic substances can produce marked transient variations in ventilatory function measured by spirometry. Therefore, for impairment assessments, spirometry should always be done when a patient is as near to a "baseline" state as possible; i.e., free from infection or medication known to affect pulmonary function. If medication might reverse impairment, spirometry should be repeated after the administration of a bronchodilating drug. Based on the variability in normal subjects, an increase of at least 10% in FEV_1 is necessary to be considered a significant response to medication. Complete discussions of the clinical implications of spirometric findings can be found in standard textbooks of pulmonary diseases (7)(17).

Large groups of persons, in whom there is no reason to suspect pulmonary impairment, have been studied to establish the normal range for spirometric values. Studies of this kind have established that factors of age, height, sex, and race, all influence spirometric values in healthy individuals. Several recent studies using modern spirometric equipment and techniques have been published (39)(52)(53)(60). Prediction equations for spirometric values obtained in the most recent of these studies are shown in Figure I-23. For any given age and height, there is a range of values for FVC or FEV_1 even among healthy individuals. The lower limit of normal for a given age and height is often defined as that value above which 95% of healthy individuals in a population would fall. This is approximately the mean value minus 1.64 standard errors of the estimate. From the magnitude of the standard error of FEV_1, it can be shown that any value above approximately 80% of the mean predicted value is considered "normal" by this definition. This points out the wide variability in normal function and the inherent difficulty in assessing impairment of an individual subject when his previous pulmonary function values are not known.

For males ≥ 25 years of age:

FVC (liters) = 0.065 Height (cm) − 0.029 Age (years) − 5.459, S.E.E. = 0.601

FEV_1 (liters) = 0.052 Height (cm) = 0.027 Age (years) − 4.203, S.E.E. = 0.541

For females ≥ 20 years of age:

FVC (liters) = 0.037 Height (cm) − 0.022 Age (years) − 1.774, S.E.E. = 0.519

FEV_1 (liters) = 0.027 Height (cm) = 0.021 Age (years) − 0.794, S.E.E. = 0.434

S.E.E. = Standard Error of the Estimate

Figure I-23. Prediction equations for spirometry in healthy subjects.

Adapted from Knudson, R.J., Slatin, R.C., Lebowitz, M.D., Burrows, B. The maximal expiratory flow-volume curve: Normal standards, variability, and effects of age. Am Rev Respir Dis, 1976, *113*:587-600.

Even recent studies of healthy subjects have had limitations. Studied groups have generally been composed primarily or solely of Caucasians living in a specific region, and no effort has been made to assess possible influences of social class or occupation on respiratory function. Petersen et al. studied asymptomatic, nonsmoking, working coal miners (60); no significant differences were found between this group and others which were more heterogenous for occupation. Several studies have demonstrated the forced vital capacity and FEV_1 of black males are somewhat lower than those for white males of the same age and standing height (1)(19)(42)(58)(64). The FEV_1 of black males has been found to be approximately 85% to 90% of that for their white counterparts. Although less information is available, a similar relationship may apply in females. However, further study is necessary because application of a general scaling factor would not be totally accurate; only a separate prediction equation, based on a large study of healthy blacks, will produce accurate predictions (66). The ratio of FEV_1 to FVC does not seem to be affected by race. The FEV_1 and FVC are decreased proportionately so the ratio remains relatively unchanged.

Another spirometric measure which has been used extensively is maximum voluntary ventilation (MVV), or the maximum amount of air which can be breathed out in one minute. The test is usually performed for 12 seconds and the results extrapolated to one minute. The MVV is more difficult for the subject to perform than a single forced vital capacity maneuver, and, for that reason, results are more variable. In some subjects the maneuver produces dizziness, wheezing, or chest pain. Even with good cooperation, results are influenced not only by the state of lungs, but also by chest wall musculature, neurologic function, coordination, the presence of cardiac disease, and other factors. Although the learning effect for the MVV ma-

neuver is greater than that for the forced vital capacity, the second MVV was, on average, 98% of the best effort out of 3 in 425 persons (25). In healthy subjects there is a close relationship between the FEV_1 and the MVV, and a reasonable approximation of MVV can be obtained by multiplying the FEV_1 by 40. However, when an abnormality outside the lung limits the MVV, it may be significantly lower than 40 times the FEV_1. This comparison serves as a useful check on the validity of the MVV and aids in identifying an inability to understand directions; outright malingering; or the presence of other conditions such as heart disease or muscular weakness, which may affect the MVV more than the FEV_1. Because the MVV maneuver requires sustained effort, it might be expected to correlate better with overall capacity for work than a single expiratory maneuver. However, because of the arduousness of the maneuver, the greater variability, and the increasing availability and use of exercise tests which produce more information, the MVV has fallen out of favor. Many well-equipped pulmonary function laboratories no longer routinely perform this test. Although prediction equations for MVV in normal subjects exist (males (40); females(43)), none of the more recent surveys of pulmonary function in normals utilized the MVV maneuver. Standardized apparatus and methodology have been suggested for those who continue to use the test (23).

DIFFUSING CAPACITY OF THE LUNG (D_{LCO})

The diffusing capacity of the lung is operationally defined as the amount of gas transferred from the alveoli to the pulmonary capillary blood per unit of time per unit gradient of pressure difference. Although in practice we are most interested in the diffusing capacity of the lung for oxygen, it is difficult to measure this directly, and a good approximation can be obtained by using carbon monoxide. The test is useful because in many infiltrative diseases, predominantly affecting the parenchyma of the lungs rather than the airways, spirometric values may be relatively well preserved, even though the lung's efficiency for transferring gas is severely impaired. Although there are several variants of the procedure—including those where subjects breathe normally at rest or during exercise—the technique commonly used today involves a single breath: the subject exhales to empty his lungs completely; breathes in a mixture containing a low concentration of carbon monoxide and a relatively inert gas such as helium; holds his breath for approximately 10 seconds; and then begins to exhale rapidly. After gas which resided in the upper airways (the non-gas exchanging portions of the respiratory system) has been exhaled, a sample of end expiratory, or so-called "alveolar" gas, is collected and analyzed for carbon monoxide and helium concentrations. The amount of carbon monoxide absorbed during the breath holding period can be calculated, and the alveolar concentration of CO estimated from the measured concentration of helium in the expirate. From these values, the diffusing capacity for carbon monoxide is calculated. Standardized methodology has been suggested for this test (23).

Many technical and biologic factors, other than the condition of the lung's "diffusing surface," affect the results. Technical factors include the duration of breath-holding, the method used to measure this period, and the timing and volume of the alveolar gas sample collected. Also important is the calibration of the analytical instruments measuring gas concentrations. Biologic factors include the lung volume and alveolar pressure during breath-holding and the "back-pressure" of venous blood carbon monoxide content. The latter may be elevated in cigarette smokers. The volume of pulmonary capillary blood, the blood hemoglobin concentration, and to some extent the cardiac output, also affect results. Uneven distribution of ventilation, while having a more significant affect on steady state methods, does affect the diffusing capacity measured by the single breath technique.

Measurement of D_{LCO} is generally less reproducible than spirometry. The coefficient of variation for repeat tests on an individual in the same laboratory have been approximately 5%-6% over a single day and 10% over several months (9)(34)(57). When identical gas samples were sent to 11 different laboratories for analysis and calculation of a simulated D_{LCO}, results varied from 46% to 171% of the "true" D_{LCO} (16). Although predictions for normal values of D_{LCO} are available (17), they are less well documented than those for spirometry. The variation of values among healthy individuals is large, and the "normal range" includes values down to approximately 70% of mean predicted. Additionally, technical and biologic factors explained above may produce alterations in the value of the D_{LCO} independent from lung function alterations. However, because the test is non-invasive

and produces information supplemental to spirometry, it is still useful in certain conditions despite its imprecision.

ARTERIAL BLOOD GASES

Measurement of arterial blood gas tensions gives an indirect estimate of the adequacy of gas exchange. Sampling of arterial blood from a peripheral artery, such as the radial or brachial, is relatively simple and causes only minor discomfort. Risk of damage to the vessel and thrombosis; formation of a large hematoma at the site of puncture; inadvertent damage to an adjacent structure such as a nerve; or infection at the site where the indwelling catheter is placed, are minimal. A good description of techniques and calibration procedure is provided in Kanner and Morris (38). Basically, the technique consists of obtaining arterial blood under sterile and anaerobic conditions, and either analyzing it immediately for oxygen, carbon dioxide, and pH on standard electrodes, or storing it under iced conditions and performing the analysis within approximately one hour. It has been reported that the oxygen tension in properly iced arterial blood falls only 4 torr (mm of mercury) in 12 hours when the initial value is in the range of 85 to 100 torr (84). Samples should always be analyzed in duplicate and should agree for pH to within ± .01 pH unit and for PO_2 and PCO_2 to within ± 2 torr. The electrodes must be calibrated daily with precision gases; for some equipment, the membrane factor (which corrects for differences between measurements of gases and blood) must be determined by using tonometered blood. Although possibilities for technical error in the analysis of arterial blood gases are numerous, many of the problems arise at the time the blood is sampled or in its handling prior to laboratory analysis. Blood may be sampled from a vein rather than an artery, resulting in spuriously low results for oxygen tension. If blood is exposed to room air after sampling, the measured oxygen tension will be falsely elevated.

Assuming blood has been properly collected and analyzed, inherent biologic variability still occurs, both among healthy individuals and within a given individual sampled at different times and under different conditions. Changes in the pattern of a subject's respiration can alter the alveolar and thereby the arterial oxygen tension without any change in the intrinsic function of the lung. Altered patterns of respiration can also affect the distribution of ventilation-perfusion ratios and thereby alter arterial blood oxygen tension. A patient's posture at the time blood is sampled also affects results. Arterial oxygen tension is usually slightly lower with the subject supine, probably due to alterations in ventilation-perfusion ratios throughout the lungs. This effect can be marked in obese persons who do not have any apparent intrinsic lung pathology. Falls of arterial oxygen tension of up to 39 torr were seen in severely obese subjects when they assumed a recumbent position (65).

Table I-70

EFFECT OF ALTITUDE ON BAROMETRIC PRESSURE AND AMBIENT PARTIAL PRESSURE OF OXYGEN

Altitude Above Sea Level (feet)	Barometric Pressure (mm Hg)	Ambient PO_2 (mm Hg)
0	760.0	159.0
1,000	732.9	153.5
2,000	706.7	148.0
3,000	681.1	142.7
4,000	656.4	137.5
5,000	632.4	132.5
6,000	609.1	127.6
8,000	564.6	118.3
10,000	522.7	109.5
12,000	483.5	101.3

Adapted from Kanner, R. E., Morris, A. H. Clinical pulmonary function testing, Intermountain Thoracic Society, 1975.

The effect of altered inspired oxygen tension is complex, but must be considered because subjects studied at different altitudes inspire different partial pressures of oxygen. As barometric pressure falls with increasing altitude, the partial pressure of inspired oxygen also falls (Table I-70). The decrease in ambient oxygen pressure does not produce an identical change in arterial blood oxygen tension or tissue oxygen supply. Persons exposed to increased altitude exhibit several adaptive mechanisms to attempt to maintain adequate delivery of oxygen to the tissues. These include an increased cardiac output and, after a period of time, an increased concentration of red cells in the blood. These and other changes increase the delivery of oxygen to the

tissues for any given level of arterial blood oxygen tension. Ventilation is also increased, but nevertheless, arterial blood oxygen tension is lower at altitude than it is at sea level. Table I-71 provides estimates of normal blood gases at various altitudes.

Age must be considered when interpreting arterial blood gases. Arterial blood oxygen tension decreases in healthy adults at an average rate of approximately 0.27-0.33 torr/year, so that mean predicted values at sea level decrease from approximately 95 torr for ages 20-29 to 80-85 torr at ages 60-69 (45)(48). Lower limits of normal, defined by the mean minus 2 SD are approximately 85 torr at age 20 and 75 torr at age 60. Each laboratory generally establishes its own range of normal for healthy individuals.

Subtle impairments in gas exchange may be detected by measurement of the alveolar to arterial oxygen gradient abbreviated as (A-a)PO_2. However, it too is altered by many of the factors already discussed and increases with age, rising from a mean at rest of approximately 5 torr at age 15 to approximately 20 torr at age 75. Variation among individuals is large; the normal range includes values at least 10 torr greater than the mean (48). This measurement is also sensitive to cardiac output and may change without any change in lung function. A change in cardiac output can result in an increase in the relative fraction of blood being shunted through anatomic or physiologic shunts and/or a decrease in mixed venous oxygen content. Both cause widening of the (A-a)PO_2.

To estimate biologic variability, blood gases were measured on two separate occasions in individuals resting on a bed, breathing room air (37). The coefficient of variation for each variable was then calculated from the standard deviation of the difference between the first and second measurements. For arterial blood oxygen tension, the coefficient of variation was 3.6% and for Pa_{CO_2}, 3.4%. The alveolar to arterial oxygen pressure gradient had an even larger variability, with a coefficient of variation of almost 19%. Thus, although (A-a)PO_2 may be sensitive to changes in gas exchange within the lung, it is also highly variable.

In summary, analysis of arterial blood gas tensions and pH is a valuable tool for studying pulmonary system function. However, at the present time, methods for calibration and techniques are not fully standardized and numerous factors must be considered when interpreting results. When tests from different laboratories are to be compared, it is imperative that techniques be as nearly identical as possible. It is recommended that blood be drawn from patients in a sitting position, due to the large falls in arterial oxygen tension which may occur in the recumbent position, especially in overweight subjects. Although the alveolar to arterial oxygen gradient may be a sensitive indicator of gas exchange abnormalities within the lung, it is highly variable in normal individuals and is no more predictive of limitation than other parameters more easily measured.

EXERCISE TESTING

Exercise testing is useful in clinical medicine in several ways. Certain patterns of response to exercise aid the clinician in differentiating pulmonary from cardiac impairments, or in assessing relative contributions to overall limitation when combined impairments are present. Symptoms or subtle abnormalities in function may not be detectable under resting conditions because the pulmonary and cardiovascular systems normally have an excess of functional capacity above demands. With the increased stress of exercise, use of abnormal compensatory mechanisms or inability to achieve a normal level of performance may be detected.

The ability to perform sustained work is dependent upon adequate gas exchange with the atmosphere. Although brief periods of work can be performed without the use of oxidative metabolism by the tissues, this process is highly inefficient, rapidly depletes substrates, and results in buildup of metabolic products such as lactate. Several linked processes are necessary to supply oxygen to the tissues and to rid the body of carbon dioxide. These include proper mechanical functioning of the chest and lungs, effective matching of deoxygenated venous blood with fresh gas in the alveoli, adequate diffusion of gases across the alveolar membrane, and an adequate total volume of blood being pumped per minute. Also required is the proper neurochemical monitoring and control of the respiratory system to maintain ventilation at a level appropriate for metabolic demand. Finally, oxygenated blood must be appropriately distributed to match tissue requirements. Normal exercise response requires adequacy and coordination of all these functions. Several excellent

Table I-71

NORMAL ARTERIAL BLOOD GAS VALUES

	Sea Level	Salt Lake City (altitude 1400 m)	Denver, Colorado (altitude 1580 m)
Pa_{CO_2}(torr)	80-100	68-85	65-75
Pa_{CO_2}(torr)	35-45	34-40	34-38
pH	7.35-7.45	7.35-7.45	7.35-7.45

Adapted from Kanner, R.E., Morris, A.H. Clinical pulmonary function testing, Intermountain Thoracic Society, 1975.

reviews of exercise physiology have been produced recently (36)(70)(76).

Adequate exercise testing requires that major muscle groups be utilized so that sufficient stress is placed on the cardiovascular and pulmonary systems to detect abnormalities not seen with lesser demands. The amount of exercise, i.e., the amount of work being done, must be quantitated in a manner which allows comparison of results between individuals and between different laboratories. This is best done by using the rate of oxygen comsumption as the index of energy expenditure. The influence of body size on oxygen comsumption can be partially controlled by expressing oxygen consumption per unit body weight or as a multiple of resting requirements. Most of the useful information gleaned from exercise testing can be obtained with a few simple measurements. During exercise, measurements should be obtained allowing calculation of heart rate, respiratory rate, total minute ventilation, oxygen uptake, and carbon dioxide production. Constant electrocardiographic monitoring and periodic blood pressure measurements should be performed during prolonged exercise. Monitoring arterial blood gases and pH during exercise allow more sophisticated interpretation of results, which may be helpful when impairment is minimal, or when the underlying diagnosis is not known.

NORMAL PHYSIOLOGIC RESPONSES TO EXERCISE

As energy expenditure increases, exercising muscles utilize more oxygen. An increase in oxygen available to muscle can be achieved by: 1) an increase in the oxygen content of arterial blood (Ca_{O_2} entering the muscle; 2) a greater extraction of oxygen from each unit volume of blood passing through the muscle; or 3) an increase in total blood flow (Q) to the muscle. Because hemoglobin is almost completely saturated with oxygen at the normal arterial blood oxygen tension (Figure I-24), little increase in Ca_{O_2} can be attained. The extraction of oxygen does increase such that the saturation of hemoglobin in mixed venous blood may decrease from 75% at rest to 25-35% or lower at maximal exercise. This second mechanism for increasing oxygen delivery is limited by the inability of normal metabolic pathways to function below a critical level of tissue oxygen tension.

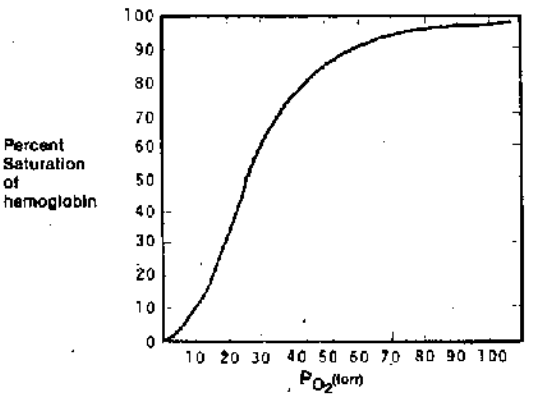

Figure I-24. Hemoglobin-oxygen dissociation curve at 37°C and pH = 7.40.

An increase in flow is accomplished by several mechanisms: a marked redistribution of blood flow occurs—the relative share to exercising muscles increases and that to certain organs e.g., gut and kidney, decreases. The total amount of blood pumped by the heart per minute (cardiac output) also increases. Both heart rate and amount of blood pumped with each heartbeat (stroke volume) increase. Stroke volume rises to near maximum at relatively low levels of exercise, so further gains in cardiac output are the result of increasing heart rate. Overall heart rate increases approximately linearly with oxygen consumption.

To supply the increased amount of oxygen necessary to re-oxygenate venous blood, ventilation must also rise. Minute ventilation rises

linearly with oxygen consumption as exercise level is increased (Figure 1-25), and no fall in arterial blood oxygen tension occurs during steady state exercise.

The response of heart rate and ventilation to exercise varies among healthy individuals; tables summarizing the "normal range" from several studies appear in Cotes (17) and Jones *et al.* (36). Differences between individuals depend on many factors including body size, total body hemoglobin content, and habitual level of physical activity. These factors account for most of the differences found between sexes and races. When comparisons are made, work rate must be quantitated by oxygen consumption, not by apparent external work achieved. Obese subjects expend more energy than lean persons for a given level of external activity (30). Even if weight is considered, the extra work done in accelerating the limbs is difficult to quantitate.

The response to exercise of a given individual also varies. Jones and co-authors reported that the oxygen consumption may vary by ±4%, the heart rate by ±3% and minute ventilation by ±4% when measured on successive days at the same level of exercise (36). Anxiety tends to raise heart rate and minute ventilation for a given level of exertion, as does a recent large meal. The heart rate response to exercise has a normal diurnal variation with lowest values occuring in the early morning. Many medications alter cardiovascular response to stress.

Additionally, the type of exercise performed may affect the relationship between heart rate and oxygen consumption. The energy expended walking on a level treadmill and walking on the floor at speeds of 1.75 to 3.5 miles per hour is the same (63). However, Jessup found heart rates to be higher when pedaling at 80 rather than 50 revolutions per minute on a bicycle ergometer at low levels of oxygen consumption (35). Although others disagree, Michael and co-workers found a higher heart rate at a given level of oxygen consumption when measured on a treadmill (49). To be strictly comparable, studies should utilize the same method of exercise.

MAXIMAL EXERCISE CAPACITY IN HEALTH

In healthy subjects, the maximal level of exertion is heralded by the onset of an intolerable sensation of shortness of breath. However, ventilation at maximal exertion seldom exceeds 60-80% of the resting maximal voluntary ventilation (17) and arterial blood oxygen tension does not fall from resting levels (76). Thus, neither mechanical limits to ventilation nor gas exchange capacity within the lungs determine the end point for exercise. The end point is reached when the cardiovascular system can no longer supply oxygen at a rate sufficient to meet aerobic energy requirements.

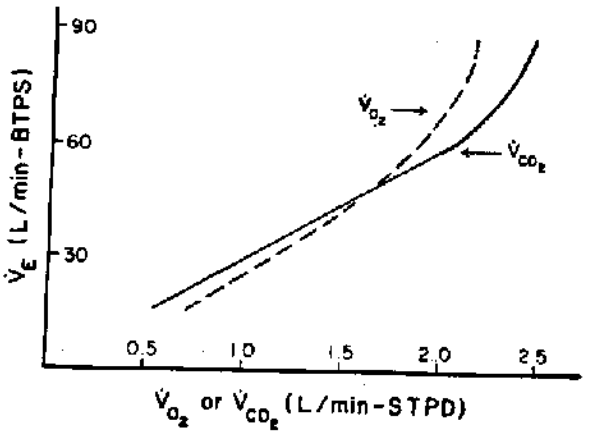

Figure 1-25. Relationship between ventilation (V_E) and oxygen consumption (V_{O_2}) or carbon dioxide production (V_{O_2}) during exercise.

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As oxygen demand increases, the mechanisms previously discussed increase the supply. At high levels of demand only an increase in cardiac output is effective in significantly increasing supply. When the heart rate reaches its maximum value, cardiac output no longer increases and oxygen supply to exercising muscles has reached its limit. Further increases in energy expenditure must be met with anaerobic metabolism. Lactic acid is produced and excess hydrogen ions are buffered by bicarbonate producing carbon dioxide. To maintain a normal blood pH, ventilation must increase to eliminate this excess CO_2. The increase in ventilation is perceived as "inappropriate" and the subject terminates the exercise. Thus, although the sensation of shortness of breath appears to limit exercise capacity, the underlying cause is cardiovascular not pulmonary.

Exactly why a certain level of ventilation is perceived as "inappropriate" is not known. The dyspnea index (D.I.) has been defined as the

minute ventilation divided by the resting maximal voluntary ventilation. Several authors have found a close correlation between the dyspnea index and shortness of breath during exercise (29)(44). If the dyspnea index is less than 35%, very few subjects complain of shortness of breath; however, if the D.I. is greater than 50%, virtually all do. Thus, the perceived "stress" of exercise seems related to how much of a subject's ventilatory capacity must be used to perform the exercise. Campbell and Howell have shown that dyspnea (the sensation of shortness of breath) is related to an imbalance between the amount of force exerted on the lung and the resulting displacement (10). A "length-tension inappropriateness" can result from stiffening of the lung or increase in resistance to airflow in the tracheobronchial tree. The increased respiratory rate during maximal exertion may somehow contribute to the perception of dyspnea by this mechanism. Clearly individuals differ in the perception of unpleasant sensations. Limitation of exercise by the subjective sensation of dyspnea is thus influenced by the subject's previous experience, his understanding of "normal" function, his mental attitude, and his familiarity with the type of exercise performed.

Maximum oxygen consumption reaches a peak in the late teens, remains relatively stable until the mid-twenties and then begins to decrease. Although other factors may play a role, a decrease in the maximum achievable heart rate is the major reason for this decline. Oga and coworkers found a decrease of 0.4 ml/kg/minute per year in maximum oxygen consumption in healthy men (56). All these factors, including some which are difficult to quantitate (e.g., habitual level of physical activity), result in a range of exercise capacities in healthy subjects. Maximal oxygen consumption can be measured directly or estimated by projecting the relationship between heart rate and oxygen consumption ($\dot{V}_{O_2}$) measured during submaximal exercise to a predicted maximum heart rate. Maximum heart rate can be predicted from age alone by a relationship derived from studies of normal subjects (4).

EXERCISE CAPACITY IN THE PRESENCE OF CARDIAC OR PULMONARY IMPAIRMENT

Subjects with significant heart disease often have diminished exercise tolerance. This may be due to an inability to raise cardiac output in the presence of a diminished stroke volume. Heart rate rises abnormally rapidly, relative to exertion levels, and reaches its predicted maximum at a lower than normal work load (76). However, subjects with pulmonary impairment often do not achieve heart rates approaching their predicted maximum when they feel constrained to terminate exercise. Instead they reach ventilatory limits due to a diminished capacity for ventilation; an increased demand for ventilation not directly related to circulatory factors; or both.

In patients with obstructive lung disease, a significant decrease in ventilatory capacity results in an inappropriately high dyspnea index for a given level of exertion. Thus, maximal exercise capacity may be diminished. An impairment in gas transfer capacity may further lower exercise capacity by requiring increased ventilation to maintain adequate gas exchange. However, the contribution of this latter impairment is difficult to predict from resting measurements because gas exchange often improves during exercise in patients with obstructive pulmonary impairment. This is probably due to improvement in the distribution of perfusion relative to ventilation in the lungs during exercise (69).

The capacity of the lung to exchange gas, as estimated by the diffusing capacity, does not contribute to exercise limitation in normal subjects (67). For subjects with severe diffusion impairment, limitation of exercise should theoretically appear rather abruptly at some critical level of oxygen consumption. This would be expected because oxygen saturation of end pulmonary capillary blood is well maintained until oxygen consumption reaches this critical level; then saturation drops rapidly. The critical level of $\dot{V}_{O_2}$ is reduced as diffusing capacity decreases. In persons with primarily obstructive impairment, mechanical ventilatory limits are reached before the critical level of $\dot{V}_{O_2}$ is attained. However, in subjects with severe diffusing capacity impairment, arterial blood oxygen tension often falls during exercise. This hypoxemia may cause an increased ventilatory response by acting on receptors in the carotid body, and the excessive ventilation results in dyspnea and limitation of exercise tolerance. The decrease in blood oxygen tension may not significantly affect tissue oxygenation because of the shape of the oxyhemoglobin dissociation curve (Figure I-24). A decrease in arterial blood oxygen tension from

95 torr to 55 torr decreases saturation only about 10%. If Pa_{O_2} falls below 55 torr, oxygen delivery may be only slightly diminished, but excessive ventilation relative to the level of exertion may diminish exercise tolerance.

ESTIMATION OF OVERALL FUNCTIONAL CAPACITY

A straightforward approach to estimating overall functional capacity is to measure maximum exercise tolerance directly. Theoretically, maximum exercise capacity could then be compared to the demands of any activity and the relative "stress" of that activity for the individual determined.

The direct measurement of maximum exercise tolerance has several disadvantages. The procedure is time consuming, extremely uncomfortable for the subject, and has some risk of precipitating a cardiac emergency. In a group of subjects over 60 years old, progressive exercise tests had to be terminated due to electrocardiographic signs of myocardial ischemia, or significant alterations in cardiac rhythm, in 70% of the males and 55% of the females (18). Therefore, most investigators have attempted to estimate maximum capacity for exercise from measurements made during submaximal exercise or at rest. Submaximal exercise tests have proven to be relatively safe, with only 16 deaths reported in 170,000 studies (70). However, even submaximal exercise testing requires the presence of a physician and is generally more expensive and less widely available than resting tests of pulmonary function. Therefore, considerable effort has been made to relate objective tests of function made at rest to symptoms experienced during exercise.

VALIDATION OF OBJECTIVE TESTS BY COMPARISON WITH SYMPTOMS AND EXERCISE TOLERANCE

Comparison of objective function tests with symptoms experienced during exertion is necessary to validate these tests as predictors of overall functional capacity. The correlation of symptoms with impairment—as measured by these tests—is often not close due to the complex interaction of compensatory mechanisms and the variation in individual perception and interpretation of "abnormal" sensations. This is especially true when more than one organ system is impaired.

Special problems may also be encountered when evaluating disability applicants. Cotes studied 125 miners applying for disability awards and 125 miners seen in a chest clinic for other reasons (15). Those who had applied for disability awards complained of more severe symptoms at a given functional level (as measured by FEV_1) than the control miners. Similar results were found in 50 consecutive cases evaluated for total disability due to pulmonary disease under the Social Security system (12). Clinical grade of dyspnea was based on whether shortness of breath occurred only when hurrying (grade 1), when walking at a normal pace on level ground (grade 2), or with ordinary activities including dressing (grade 3). Those classified grade 2 differed little in mean function from those classified grade 3; in fact, the grade 3 group had higher mean values for the MVV and the FEV_1/FVC ratio. A group of clinic patients with grade 1 dyspnea who were not applying for disability benefits had an average FEV_1 to FVC ratio of 37% while claimants reporting grade 1 dyspnea had an average ratio of 52%. Lindgren and coauthors studied 100 randomly selected claimants for total disability due to lung disease or shortness of breath syndromes and 100 patients matched for age, sex, and degree of pulmonary function impairment (44). A clinical history was taken and the subjects observed during a standard level walk. Claimants more often overestimated (26% vs. 9%) the severity of their dyspnea compared to that observed during the exercise test.

Patients may also be poor judges of their own ability to perform activities. Sixty-two patients, 44 of whom had obstructive lung disease, and 18 of whom had an infiltrative lung disease without obstruction, were asked to estimate the distance they could walk, at their own pace, before having to stop due to shortness of breath, and the distance they could walk in 12 minutes (47). They were then asked to walk at their own pace as far as possible in 12 minutes. Results showed no correlation between the distance walked and either of the estimates. Thus it is difficult to correlate symptoms with function, especially in disability claimants.

Numerous attempts have been made to predict disability due to respiratory impairment from resting spirometric measurements. Two hundred and sixteen patients with obstructive lung disease and no other disabling conditions,

were separated into six functional classes by taking a detailed history (81). Most closely correlated with the clinical degree of pulmonary disability were the FEV_1 and the MVV, with correlation coefficients of .93 and .96 respectively. The authors derived an equation to predict the clinical degree of pulmonary disability based upon the MVV, FEV_1, age, and vital capacity. The overall correlation coefficient of the prediction, from the equation to the actual clinical grade, was high (0.83); however, individual variability was also high. Individuals judged clinically as class 3 were placed in classes 2 through 5 by the prediction equation.

In subjects with obstructive lung disease, maximum voluntary ventilation measured at rest correlates closely with symptoms during exercise (29). This does not necessarily hold true for subjects with interstitial lung disease and definite abnormalities in gas exchange, but without significant obstruction. Although maximum voluntary ventilation may be within the predicted normal range, such subjects often hyperventilate during exercise due to impaired gas exchange and thus have a decreased exercise tolerance (24). In these subjects, significant errors in estimating impairment can be made if the diffusing capacity is not also considered (22).

In another study, 30 men with obstructive lung disease—all of whom had an FEV_1/FVC ratio less than .55—were exercised to tolerance on a treadmill (28). Most closely correlated with the ability to exercise was the FEV_1. However, exercise tolerance varied widely for a given level of FEV_1. The correlation was higher between exercise ability and absolute FEV_1, than for the FEV_1 expressed as a percent of predicted (28) as McGarvin and co-workers also found (47). This is not surprising since exercise capacity is determined by function remaining, not the amount which has been lost. The importance of basing assessment of disability on remaining function has been stressed by several authors (15) (26).

The dyspnea index (exercise ventilation/maximum voluntary ventilation) has been found to correlate highly with the clinical grade of breathlessness during exercise (29). The dyspnea index was also studied by Lindgren and coworkers in their examination of 100 disability claimants together with patient controls (44). Impairments were mostly the obstructive type. The dyspnea index for patients who had no shortness of breath during the standard exercise averaged 23%, with very few values above 35%. Those who complained of severe shortness of breath during the exercise had an average dyspnea index of 78% and all were above 50%. For a given severity of dyspnea during the exercise, the dyspnea index was 10% lower in the claimants than in the patients. Claimants expressed greater symptoms than patients at objectively comparable levels of stress, and the relationship between MVV (or FEV_1) and exercise capacity was different for the two groups. Thus, the FEV_1 and the MVV appear to be closely correlated with symptoms during exercise and exercise capacity for groups of subjects with obstructive lung disease. However, the relationship is highly variable for individuals and may differ between disability applicants and others. The ability to predict symptoms and exercise capacity from arterial blood gas studies has also been examined.

Teculescu and co-workers found that the FEV_1 correlated closely with resting arterial blood oxygen tension in 156 patients with symptoms of shortness of breath (75). A prediction equation for the Pa_{O_2} was developed, based on FEV_1 alone. However, the standard error of the estimate around the regression line was approximately 20%. Thus, the range of prediction for an individual was so large as to be clinically useless. The correlation was not improved when the FEV_1 was expressed as a percent of predicted.

Most workers have not demonstrated a direct relationship between arterial blood gas values and resting spirometry (68). Neukirch et al. did show some correspondence between the FEV_1 as a percent of predicted and blood gas abnormalities at a low level of exercise, but they could not accurately predict individual results due to wide scatter in the data (54).

Early studies of exercise tolerance showed no relationship between hypoxemia at rest or during exercise and the ability to exercise or the clinical degree of disability (50)(73). Studied subjects primarily had obstructive lung disease. Coates found no difference in resting Pa_{O_2} or Pa_{CO_2} between three groups of disability applicants with dyspnea ranging from grade one to grade three in severity (12). A study of patients with severe obstructive lung disease (maximum voluntary ventilation less than or equal to 35% of predicted) showed no correlation between arterial blood oxygen tension at rest or at

maximal exercise with the maximum work load tolerated (74). Subjects terminated exercise on the bicycle ergometer due to dyspnea; no subject experienced chest pain suggestive of angina. The alveolar to arterial oxygen tension gradient, when measured at rest or at the point of maximum exercise, also showed no correlation with the maximum work load tolerated. A similar study of subjects with less severe obstructive lung disease showed no correlation between the value of arterial blood oxygen saturation or carbon dioxide tension during exercise with the level of exercise (28).

Spiro and co-workers conducted a more detailed study of 20 subjects with moderately severe obstructive lung disease (mean FEV_1 equal to 49% of predicted); 20 very severely obstructed patients (mean FEV_1 to 24% of predicted); and 20 normals (71). Subjects performed progressive exercise on a bicycle ergometer, until having to stop due to shortness of breath or reaching 85% of maximal predicted heart rate. The normals all reached the heart rate end point; maximal exercise ventilation was, on average, 46% of the MVV predicted from the measured FEV_1. At the break point of exercise, moderately obstructed patients were found to be using 99% of their predicted maximum ventilation; severely obstructed patients were breathing at a level of 146% of the predicted maximum. Heart rate, at maximum exercise tolerated, was far below levels of predicted maximum for both obstructed groups. Thus, the major limitation to exercise in the obstructed subjects was ventilatory. The Pa_{O_2} and Pa_{CO_2} did not significantly change from rest to exercise in the moderately obstructed group. The severely obstructed group did show a fall in Pa_{O_2} from 69.7 torr to 60.9 torr, but this fall would not cause a significant change in arterial blood oxygen content, and so is unlikely to have been the cause of exercise limitation. The rise in lactate 5 to 10 minutes after maximal exertion (which is indicative of anaerobic metabolism) was less in the patient group than in the normals. None of the patients manifested a rise in ventilation relative to oxygen consumption, which indicates the anaerobic threshold has been reached. The authors concluded it was unlikely that the fall in arterial blood oxygen tension or anaerobic metabolism significantly influenced the end point of exercise for these patients.

In general, arterial blood oxygen tension is not increased by training even though tolerance for exercise is improved (11)(59). Under some circumstances, even normal subjects may show a fall in Pa_{O_2} with exercise (61)(82). Young and Woolcock had healthy, young, non-smoking subjects, with normal pulmonary function, walk up stairs at 9 meters per minute (84). The mean arterial blood oxygen tension fell from 92 torr at rest to a mean lowest value of 65 torr during the first minute of exercise. The maximum fall observed was 33 torr. Thus, significant decreases in Pa_{O_2} may occur transiently during exercise in completely healthy subjects. Therefore, it is important to allow subjects to reach a steady state prior to measuring arterial blood gases in disability evaluation exercise tests. If this is not possible because a subject is unable to maintain the [chosen] exercise level, the study should be repeated at a lower level of exercise.

Arterial blood gas tensions are not correlated with other tests of pulmonary function in a manner which allows prediction of results for individual subjects. Also, they are only indirectly related to exercise limitation in most subjects, even those with severe obstructive lung disease. They may be more valuable in subjects with diffusion impairment and are useful in allowing more sophisticated analysis of exercise results when diagnostic considerations require detection of minimal levels of impairment. As indicated by changes seen in normals working at altitudes, "abnormalities" in arterial blood gases cannot be automatically equated with disability (26).

When evaluating the relationship between diffusing capacity and symptoms or exercise tolerance, careful distinction must be made between subjects with interstitial lung disease and those primarily with obstructive impairments and associated defects in gas transfer. Coates found no significant difference in diffusing capacity among three groups of applicants for Social Security disability benefits who had symptoms of dyspnea ranging from grade 1 to grade 3 in severity (12). However, only 15% of these subjects had interstitial lung disease; the remainder primarily had obstructive impairment. Diffusing capacity also showed no correlation with exercise tolerance (28) or fall in Pa_{O_2} during progressive exercise to maximal tolerance (71) in subjects with obstructive lung disease. However, in patients with interstitial lung disease without obstruction, diffusing capacity was

closely correlated with the maximal distance walked in 12 minutes, and the patient's estimate of the stress of the exercise (47). Wilson was able to estimate incapacity in patients with primary gas exchange impairment—with moderate accuracy—from an equation based on D_{LCO} (80). Wehr & Johnson included D_{LCO} in a theoretical model predicting maximal oxygen uptake for persons with lung disease (78). Thus, diffusing capacity may be useful in estimating exercise capacity in subjects with interstitial lung disease. It is generally not helpful in those with primarily an obstructive impairment.

In summary, objective measures of pulmonary function and symptoms during exercise do not correspond closely in individual subjects. For groups of subjects, the FEV_1 and MVV correlate most closely with symptoms in those with obstructive lung disease; in those with interstitial (restrictive) lung disease, the diffusing capacity correlates best. Arterial blood gas studies are not helpful in predicting symptoms or exercise tolerance in patients with obstructive lung disease. They may be useful in patients with a predominant impairment in gas exchange.

PREDICTION OF MAXIMAL EXERCISE TOLERANCE

In persons with significant pulmonary impairment, exercise tolerance is most often determined by a ventilatory limit (71). When minute ventilation reaches a critical level relative to maximal ventilatory capacity, the subject experiences symptoms of dyspnea. Exercise tolerance may be reduced by a decrease in maximal ventilatory capacity; an increased demand for ventilation relative to energy expenditure; or a combination of both. An increased demand for ventilation may be caused by impaired gas exchange in the lungs. Wright developed an equation to predict maximal oxygen consumption from measures of ventilatory capacity (the MVV) and gas exchange (the ventilatory equivalent for oxygen or $\dot{V}E_{O_2}$) (83). The ventilatory equivalent is the minute ventilation divided by oxygen consumption and should be elevated if gas exchanged is impaired. Armstrong et al. applied this equation using MVV measured at rest and the ventilatory equivalent measured during submaximal exercise (2)(3). Maximal oxygen consumption estimated in this way correlated closely with that measured directly in 70 subjects with lung disease and 13 normal subjects (Figure I-26). These studies confirmed that arterial blood oxygen saturation and carbon dioxide content at rest or during exercise were not predictive of impairment levels (Figure I-27).

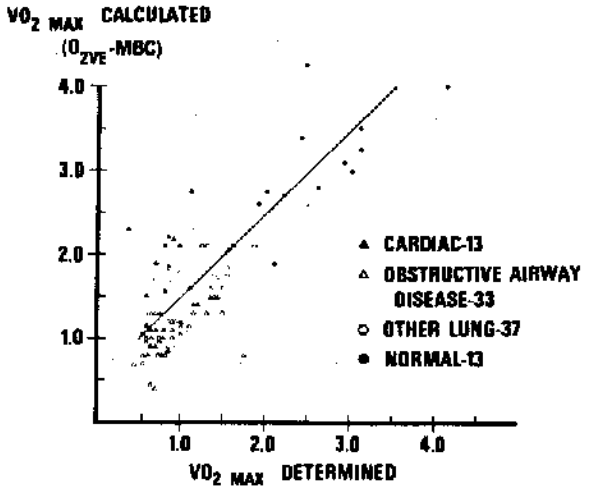

Figure I-26. Comparison of maximal oxygen consumption calculated from resting MVV and ventilatory equivalent during submaximal exercise to directly determined $\dot{V}_{O_2}$ max.

Adapted from Armstrong, B. W., Workman, J. N., Hurt, H. H. Roemmich, W. R. Clinico-physiologic evaluation of physical working capacity in persons with pulmonary disease, Part II, Am Rev Respir Dis, 1966, 90:223-233.

This approach provides a useful method of estimating overall impairment of exercise capacity. However, certain limitations must be recognized. The presence of significant cardiovascular impairment may cause the actual maximal oxygen consumption to be lower than that estimated from Wright's equation. This was confirmed by Armstrong in several patients. Also, the ventilatory equivalent depends on the level of exercise at which it is measured. If measured at too low an energy output, voluntary hyperventilation can falsely elevate the ventilatory equivalent. When the anaerobic threshold is reached, ventilation begins to increase more rapidly relative to oxygen uptake, thereby increasing the ventilatory equivalent. At levels of exercise between these extremes, ventilation ($\dot{V}$) and oxygen consumption ($\dot{V}_{O_2}$) are related by the equation $\dot{V} = A\dot{V}_{O_2} + B$ where A and B are constants. Thus, $\dot{V}/\dot{V}_{O_2}$ (ventilatory equivalent)

$$= A + \frac{B}{\dot{V}_{O_2}}$$

and the measured ventilatory equivalent decreases as exercise level ($\dot{V}_{O_2}$) increases. Finally, in patients with significant diffusion impairment, a sudden fall in end-pulmonary capillary blood

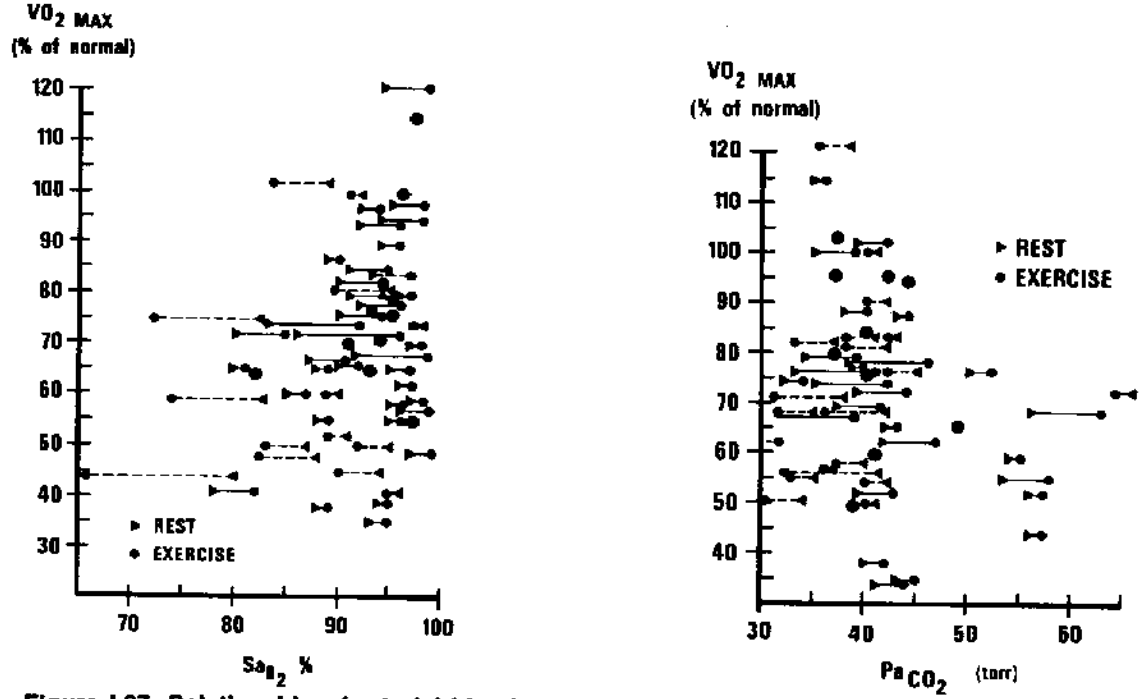

Figure I-27. Relationship of arterial blood oxygen saturation (Sa_{O_2}) and carbon dioxide tension (Pa_{CO_2}) to measured maximal oxygen uptake ($\dot{V}_{O_2}$ max).

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oxygen saturation may occur when a critical level of oxygen consumption is reached. This critical level is not necessarily predicted by the ventilatory equivalent at lower levels of exercise. Despite these limitations, the ventilatory equivalent has been found to be a useful measure in estimating impairment of exercise capacity (69).

RELATION OF EXERCISE CAPACITY TO WORKING ABILITY

If measured or predicted maximal oxygen consumpton could be compared to the demands of work, the stress produced by that work could be estimated. The relative stress could then be used to decide whether a subject is disabled for a given job. Directly measuring oxygen consumption during normal working activity is difficult. Most studies have measured the relationship between oxygen consumption and heart rate in the laboratory, and then—by telemetry—measured heart rate during normal working activities. Such studies reveal that in most jobs, the level of energy expenditure is highly variable during the working period; brief periods of intense activity are followed by periods of lesser activity. Walking mail carriers over 50-years-old and carrying 15 kg sacks of mail had an estimated oxygen consumption (averaged over the entire work period of approximately 2 hours) of 1.17 liters per minute. The most straining phase of their day's work required 1.45 liters per minute of oxygen consumption (56). The relative aerobic strain of the work (i.e., the oxygen consumption utilized, divided by the maximum oxygen consumption measured in the laboratory) was not significantly different between young and older men. The average relative aerobic strain for the working period was 54% for the men less than 35 years of age, and 55% for those greater than 50 years of age. However, the older men had a significantly lower maximal oxygen consumption than the younger men. This suggests that the older men were able to reduce the strain by pacing their work. The most straining phase of the work produced a stress of 68% for both the young and older men.

Among Columbian sugar cane loaders who load bundles weighing 1 to 2 kg on wagons, older workers showed a greater relative strain, using 35% of their maximum oxygen uptake averaged over an 8-hour period, compared to only 20%

in younger workers (72). However, productivity (as measured by the amount loaded) did not correlate with age, again suggesting that older workers are able to pace themselves and accomplish the same amount of work over an 8-hour day.

Astrand has shown that for high work rates, brief periods of work with brief rest periods produce very little elevation in blood lactate, whereas longer periods of work with longer rest periods produce high levels (Figure I-28)(5). This is probably attributable to utilization of muscle energy and oxygen stores to briefly achieve aerobic work rates above those usually carried out totally aerobically. When work stints are short, these stores are adequate to prevent anaerobic metabolism and lactate production. Stores are replenished during rest periods. When work stints are long, these stores are exhausted and lactate production ensues. The highest steady paced work, sustained by normal young men over a period of 8 hours, was approximately 35% of maximal oxygen consumption (49). Mail carriers over 50 years of age were able to tolerate short periods of oxygen consumption requiring 68% of maximal ability without undue fatigue. They sustained an average of 55% of maximal oxygen consumption for a 2-hour period. Clearly, work rate and pattern are crucial in determining the stress experienced by an individual with any limitation.

Several other factors are known to influence working capacity. Both physical size and level of habitual physical activity influence the total amount of work a subject can perform (77). The efficiency of transforming consumed oxygen into measurable external work performed varies between individuals and depends on work rate. As speed of walking was increased, net efficiency decreased in normal subjects (21). In older subjects, the net efficiency of work performed with the arms significantly decreased as the work rate was increased (55). For a given amount of external work, older subjects also consumed more oxygen than younger subjects, perhaps due to decreased coordination of movement. They also had a higher minute ventilation per unit of external work done (or oxygen consumed) than younger subjects (Figure I-29). A given level of external work may represent a greater physiologic strain for an older person, regardless of impairment.

Physiologic studies indicate that efficiency may increase with training (31). Eight patients with emphysema, all of whom were hypoxemic at rest, were studied on a bicycle ergometer and then given 21 days of training on a treadmill. Training consisted of five 10 minute sessions per day (59). They were then restudied on the bicycle ergometer at the same level of exercise they had performed prior to the training. Exercise ability on the treadmill improved significantly; maximum tolerated speed increased from 1.35 miles/hour prior to training to 2.4 miles/hour after training. However, oxygen consumption, minute ventilation, ventilatory equivalent for oxygen, and arterial blood gases did not change between the pre- and post- training bicycle tests (Table I-72). The percent of total energy requirements obtained from anaerobic metabolism, estimated both by oxygen debt measurement and lactate levels during exercise, showed no change after training. The subjects' stride length on the treadmill increased significantly during the training period. This study suggests that training effect is not transferable between tasks and may be due to increased efficiency, specifically for the task performed during training. A similar study of 21 patients with obstructive lung disease and 8 control patients, included even more detailed physiologic monitoring (11). These subjects were studied before and

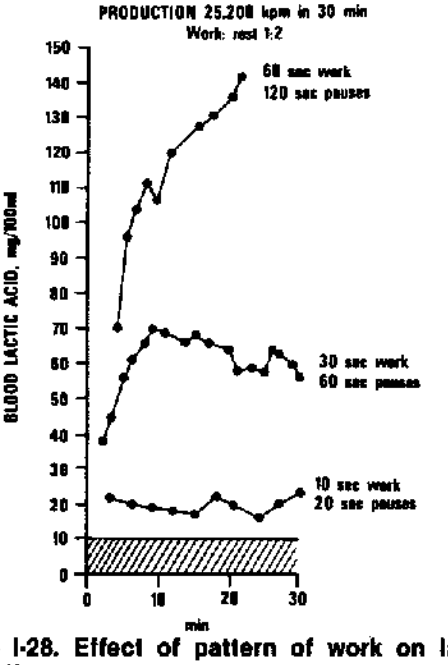

Figure I-28. Effect of pattern of work on lactate production.

From Textbook of Work Physiology by Astrand, P., Rodohl, K. Copyright 1970, McGraw-Hill Book Co. Used with permission of McGraw-Hill Book Company

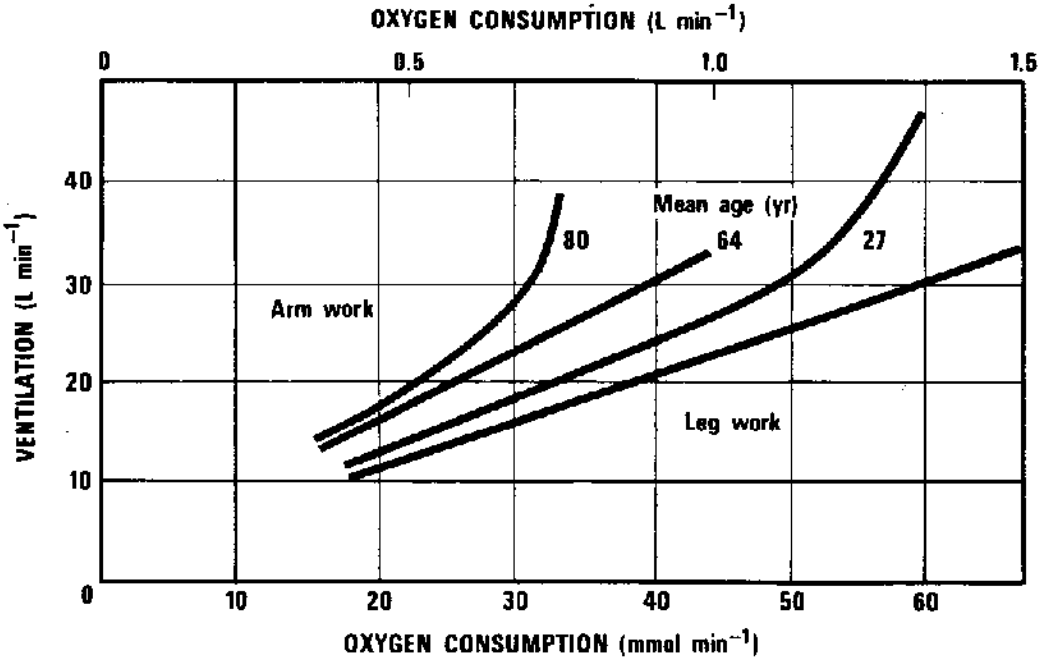

Figure I-29. Ventilation and oxygen consumption during work using the arms for subjects of various ages.

Adapted by Cotes (1975) from Norris, A. H., Shock, N. W., Yiengst, M. J. Age differences in ventilatory and gas exchange responses to graded exercise in males. J Gerontol, 1955*10*:145-155.

after an intensive 4 week rehabilitation training period consisting of daily exercise on a treadmill, rowing machine, bicycle and wall pulley; breathing exercises, postural drainage and medications; and psychological and vocational rehabilitation programs. After training, the FEV_1, peak flow, and forced expiratory flow over the mid portion of the vital capacity, maximum voluntary ventilation, residual volume, diffusing capacity, airway resistance, and dead space ventilation to tidal volume ratio did not change significantly in either the patient or control groups. Arterial blood gases, the alveolar-arterial oxygen gradient, the shunt fraction estimated by breathing 100% oxygen, and the ventilatory equivalent resting or during exercise, also showed no change. Likewise, no changes in cardiovascular function were noted. Heart rate, cardiac index, stroke volume, mean pulmonary artery pressure, and pulmonary vascular resistance were unchanged. However, the amount of total work performed on the treadmill by the patient group increased significantly. Oxygen consumption and minute ventilation at a given level of work on the treadmill decreased after training in the patient group but not in the controls. Neither the patients nor the controls showed any change in ventilation or oxygen consumption on the bicycle after training. The authors, therefore, concluded that while training produced an increased ability to work, cardiopulmonary function had not changed. The increased ability to perform work on the treadmill was probably due to increased neuromuscular coordination and perhaps a decrease in the subject's sensitivity to the sensation of dyspnea as familiarity with the task increased. Clearly these studies have serious implications for attempting to relate exercise capacity in the laboratory to working ability on the job. As pointed out by Gaensler and Wright, conditions of work cannot easily be simulated in the laboratory, and prior training for a task clearly affects performance regardless of cardiopulmonary function (26).

Armstrong and co-workers found that none of 59 subjects who were working at the time of their study had an estimated maximal oxygen consumption less than 50% of the predicted normal value (3). Roemmich and co-workers applied the approach used by Armstrong to disability evaluation (62). They confirmed the Armstrong method produced reasonable estimates of maximum oxygen consumption for coal miners without significant impairment of gas exchange. They also estimated that an energy expenditure

Table I-72

PHYSIOLOGIC MEASUREMENTS IN PATIENTS WITH LUNG DISEASE BEFORE AND AFTER 21 DAYS OF TRAINING ON A TREADMILL

	Treadmill		Cycle Ergometer				
	Max. Speed (MPH)	Pulse (L/min)	$\dot{V}_{O_2}$ (L/min) STPD	$\dot{V}_E$ (L/min) BTPS	V.E.	Pa_{O_2} (Torr)	$(a-\bar{v})O_2$ (Vol%)
Before training	1.35	91.3	.406	21.6	35	54	10.1
After training	2.40*	96.5	.412	20.5	34	57	11.7*

*$p<.05$

Adapted from Paez, P.N., Phillipson, E.A., Masangkay, M., Sproule, B.J. The physiologic basis of training patients with emphysema. Am Rev Resp Dis, 1967, 95:944-953.

$\dot{V}_{O_2}$ = oxygen consumption per 100 kg-m of work

$\dot{V}_E$ = ventilation

V.E. = ventilatory equivalent for oxygen

Pa_{O_2} = arterial blood oxygen tension

$(a-\bar{v})O_2$ = arterial-mixed venous blood oxygen content difference

level of 7.5 times basal requirements would exceed the demands of the vast majority of jobs in the general labor market. Indeed, Jones et al. indicate energy demands for "mining and heavy industry" are approximately 7 to 8 times basal or 1.75-2.00 L/min of oxygen consumption (36). On direct exercise testing, Roemmich and co-workers found 26% of working coal miners had a capacity less than or equal to 1.75 liters per minute of oxygen consumption—confirming that a worker with this level of capacity can perform relatively strenuous work. By making certain assumptions, the FEV_1 expected to correspond to a capacity for maximal oxygen consumption of 7 to 8 times basal can be calculated from the Armstrong equation. Details are explained in Appendix IV. Values of FEV_1, which Roemmich and co-workers suggested as disability indicators in coal miners, were based on estimates of maximal oxygen consumption. This logical approach of relating objective measurements to overall functional and work (job) ability is useful but limited in application.

Perhaps more important than any of the technical factors already discussed are certain socioeconomic considerations. Haber studied the relationship between functional limitations as determined by ability to perform specific tasks (walking, lifting, writing, etc.) and overall disability as determined by a subject's actual work history (32). At each level of functional limitation, a greater percentage of older subjects were disabled and a greater percentage of blue collar workers than white collar workers were disabled (Table I-73). Persons with a high school or college education were less disabled than those with lesser levels of education. Clearly, the type of work a subject is able to obtain has an important influence in determining disability levels associated with impairment.

Socioeconomic factors were also found to play a major role in influencing return to work following pneumonectomy for carcinoma (41). Fifty-seven percent of patients with severe lung disease (FEV_1 to FVC ratio of less than 50% or a vital capacity of less than 40% of predicted) returned to work compared to only 39% of those with less severe lung disease. When the type of work was considered, 26% of persons engaged in heavy labor activities or agriculture returned to work, whereas 73% of professionals were able to resume work. Diener and Burrows found that symptoms of dyspnea did not correlate with work status in 99 patients with obstructive lung disease who were followed for one year (20). However, a good prediction of work status could be obtained if job difficulty as well as cardiopulmonary function was taken into consideration. Gilbert and co-workers found no difference in MVV, FEV_1 or arterial blood gases

Table I-73

RELATIONSHIP BETWEEN FUNCTIONAL LIMITATION (FOR SPECIFIC TASKS SUCH AS LIFTING) AND DISABILITY DETERMINED FROM ACTUAL WORK HISTORY

	% Severely Disabled								
	Age			Education				Job	
Functional Limitation	18-44	45-54	55-64	<9 yr	9-11	12	College	Blue Collar	White Collar
None	14.6	17.5	23.4	29.5	13.7	15.2	8.5	27.6	14.7
Minor	25.6	23.9	40.0	} 39.0	} 30.9	} 22.3	} 24.3	} 49.9	} 31.5
Moderate	28.1	27.7	44.8						
Severe	43.2	39.3	52.9	} 64.6	} 59.4	} 41.6	} 43.1	} 70.8	} 52.6
Dependent	54.4	59.7	73.7						

Adapted from Haber, L.D. Disabling effects of chronic disease and impairment II. Functional capacity limitations. J Chronic Dis, 1973, *26*:127-151.

between working and nonworking subjects with symptomatic obstructive lung disease (Figure I-30). All subjects who had an FEV_1 greater than 2 liters were working, but values of FEV_1 less than 2 liters had no predictive value for work status (28).

It has been amply demonstrated that factors other than objective impairment are vitally important in determining disability. Laboratory studies can provide estimates of functional capacity and even job demands, but must never be used as the sole criterion for disability evaluation.

RELATIONSHIP OF IMPAIRMENT TO CHEST RADIOGRAPH

Although abnormalities on chest radiograph are often present in persons with lung diseases associated with significant impairment, correspondence between function and radiologic findings is generally poor. A normal chest radiograph in no way eliminates the possibility of significant functional impairment. Forty percent of applicants for pulmonary disability benefits with normal chest radiographs had abnormal pulmonary function (12). Lindgren and co-workers also found poor correlation between pulmonary function and chest radiographs in 100 randomly selected claimants for disability due to lung disease (44). Of those without any objective impairment of pulmonary function, 50% showed some type of radiographic abnormality, while 62% of those with slight to moderate impairment and 60% of those with severe pulmonary impairment had an abnormal chest radiograph. Radiographic abnormalities, including those suggestive of pulmonary hypertension, are not correlated with work status in patients with obstructive lung disease (20).

Lack of correlation between radiographic findings and function has also been demonstrated for occupational diseases such as simple coal workers' pneumoconiosis (51). Gaensler and co-workers demonstrated a significant correlation of restrictive impairment and radiographic abnormality in workers with exposure to asbestos but not in those with other dust exposures (24). For individuals, correspondence of function and radiologic findings was poor. Persons whose radiograph was classified as 0/0 or 0/1 by the UICC/Cincinnati classification system (i.e., showing little or no evidence of pneumoconiosis) had vital capacities ranging from 50% to 98% of predicted. The range for diffusing capacity in these same subjects was 49% to 128% of predicted. In persons with very abnormal chest radiographs (categories 3/3 or 3/4), the vital capacity ranged from 38% to 72% of predicted and the diffusing capacity from 18% to 86% of predicted. The authors concluded that chest radiographs are of no use in predicting impairment in individuals and thus of no use in this phase of disability evaluation. Chest radiographs are, however, important in establishing a diagnosis and may be helpful in relating pulmonary impairment to occupational exposure.

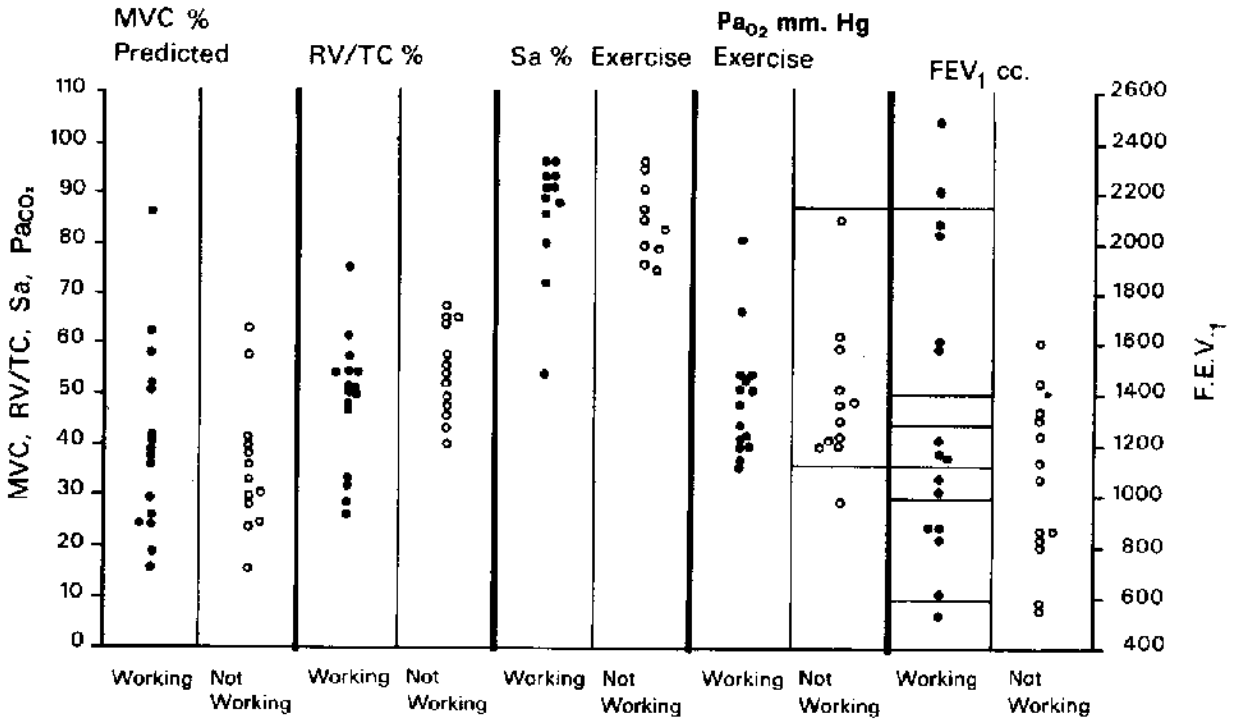

Figure I-30. Relationship of various parameters of pulmonary function and working status.

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SUMMARY AND RECOMMENDATIONS

Impairment is difficult to quantitate because of the wide range of "normal" function, and the [usual] lack of information about individual functional capacity prior to the onset of illness. Remaining functional capacity can be determined more accurately than amount of function lost. The physiologic stress caused by an activity is directly proportional to the fraction of an individual's remaining maximal capacity required by the demands of that activity. A given percentage reduction in function from "normal" is more incapacitating for an older individual. The remaining function is lower than that of a younger person with the same percentage loss in function, and the demand for function (e.g., ventilation) to perform at a given level of work is greater in older subjects. Thus, disability evaluation should focus on determining the remaining ability to function and its relation to the demands of the work to be performed.

Work or job demands are difficult to quantitate due to individual factors of work pattern and rate and prior training. Also, the ability to tolerate sensations of breathlessness varies between individuals. Nevertheless, an estimate of job demands is essential in determining whether an individual with a given level of function is disabled.

The final determination of disability must take into account socioeconomic and psychological factors such as education, past work experience, job availability, and motivation, as well as remaining pulmonary function. Because of the complex interaction of these factors, no level of function defined by medical testing can accurately separate those who are unable to perform a certain job from those who are. Appendix I contains a summary of criteria for disability evaluation currently in use or suggested by authorities. Appendix II provides specific values or ratings for Federal programs. More complex measurements than those indicated in Appendix I have not been shown to improve the accuracy of predicting the ability to work. Exercise testing

may add useful information when more than one organ system is impaired and in cases with borderline pulmonary impairment. It is probably not necessary as a screening procedure in disability evaluation if spirometry, resting blood gases, and in some cases, diffusing capacity are performed. It is valuable as a research tool.

Appendix III compares pulmonary function values, which define severe impairment or disability, for a male 70" tall under six schemes of evaluation. Values for the "degree of pulmonary disability" (DPD) from Wilson's equations (81) and estimated maximal oxygen consumption according to Armstrong and co-workers (3) have been calculated by making certain assumptions when all parameters in the prediction equations are not specified in the evaluation scheme. Use of these assumptions (noted in Appendix III), even if not completely valid, allows a useful comparison of the various schemes.

If a fixed percentage of normal predicted values is used to determine disability, older subjects are clearly disadvantaged. The estimated $\dot{V}O_2$ max declines and the DPD increases with age. With obstructive impairment, an FEV_1 of 1.8 L is associated with an estimated maximal oxygen consumption of approximately 1.8 L/min and a DPD of 250-275, depending on age. This is a level of impairment which may be disabling for work requiring moderate physical exertion. This FEV_1 is approximately 55% of predicted for a 60-year-old (70" tall) male and 49% for a 40-year-old. Thus, setting a guideline for disability due to obstructive impairment at 55% of the predicted FEV_1 for 60-year-olds, and applying this value to all younger applicants is reasonable. This assumes no severe "gas exchange" impairment is present. For restrictive (interstitial) impairment, values for FVC and $D_{L(SB)}$ of 55% of those predicted for a 60-year-old result in DPD scores of approximately 250-275, depending on age. Estimated $\dot{V}O_2$ max values are not as useful because of the arbitrary choice made for $\dot{V}E_{O_2}$ in the calculations, but do suggest that this level of impairment would be disabling for moderately strenuous work.

This author recommends that values of pulmonary function, equal to or less than 55% of the predicted level at age 60 years, be used as general guidelines for possible total disability due to pulmonary impairment. Predicted levels should be those of one of the recent surveys, and separate predictions for women and blacks should be used.

Arterial blood gas tensions are generally difficult to interpret as an index of impairment. However a Pa_{O_2} at or below 55 to 60 torr at sea level (with Pa_{CO_2} = 40 ± 2 torr) should be considered suggestive of disabling impairment. If not consistent with all other findings, arterial blood gases should be measured during steady state exercise of mild intensity (e.g., $\dot{V}_{O_2}$ = 0.75 - 1.0 L/Min). Any further drop in Pa_{CO_2} should be considered confirmation of severe impairment. A Pa_{CO_2} equal to or greater than 50 torr at sea level should also be considered evidence of severe impairment.

These recommendations are presented as guidelines for disability evaluation. They cannot be used to define disability nor substitute for the judgment of experienced physicians and claims adjudicators in determining the capability for work of a given individual.

RESEARCH NEEDS

It is clear that research is needed in several areas in disability evaluation. Better predicted values for normal levels of pulmonary function are needed, especially in non-Caucasians. The influence of subject cooperation on pulmonary function testing in disability applicants needs further study. More accurate predictions of the demands of work and the influence of work rate and training on the ability to perform a given task are needed. An area which has received essentially no study is that of psychological factors related to the perception of the sensation of dyspnea. Of these, the quantitation of the physical demands of contemporary jobs is probably the most urgently needed "technical" research. A better understanding of the interaction of psychological and social factors with physical impairment would probably have the most significant impact on the overall evaluation of disability.

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Appendix I

SUMMARY OF CRITERIA FOR SEVERE IMPAIRMENT OR TOTAL DISABILITY DUE TO PULMONARY DISEASE

	"Obstructive" Impairment	"Restrictive" (Interstitial) Impairment
AMA[2] (60-90% impairment)	FVC, FEV_1, and MVV < 55% predicted (at least 2 should be measured). Note: "obstructed" and "restricted" not distinguished, blood gas values placed under "restriction" for convenience	Arterial blood oxygen saturation usually less than 88% at rest and after exercise (Sa_{O_2} = 88% corresponds to approximately Pa_{O_2} = 54 torr at 37°C and pH = 7.40
Gaensler & Wright[3] (severe impairment)	$FEV_1/FVC < .40$ MVV ⩽ 45% predicted (if done)	FVC ⩽ 50% predicted or $D_{L(SB)}$ ⩽ 40% predicted Sa_{O_2} < 92% at rest and decreasing with exercise (Sa_{O_2} = 92% corresponds to approximately Pa_{O_2} = 63 torr at 37°C and pH = 7.40)
Wilson, et al.[4]	Degree of pulmonary disability (DPD) calculated from formula including MVV, FEV_1, age and FVC (DPD = 300 if dyspnea with slight exercise; DPD = 200 if mild dyspnea at rest)	DPD computed from formula including MVV, $D_{L(SB)}$, FEV_1, and age Same interpretation as "obstructive"
Veterans Administration[8]	Rating of impairment in earning capacity based on comparison of symptoms and examination results with rating schedule description. Total disability may be assigned even if rating less than 100% when person is unable to secure or follow a substantially gainful occupation provided that a single disability of ⩾ 60% rating is present (or combined disabilities of ⩾ 70% rating). See Appendix II(a))	
Social Security[1] (total disability)	FEV_1 and MVV values based on height only (see Appendix II(a))	FVC values based on height [see Appendix II(b)] and $D_{L(SB)}$ < 30% predicted or < 9 ml/mmHg/min or arterial blood oxygen saturation ⩽ 87 (adjusted up if arterial blood carbon dioxide tension is below 40 torr) Note: Sa_{O_2} ± 87 corresponds to approximately Pa_{O_2} = 52 torr at 37°C and pH = 7.40
Social Security Black Lung Benefits**[5]	FEV_1 and MVV values based or on height (see Appendix II(a))*	Pa_{CO_2} ⩽ 55 torr (adjusted up if Pa_{O_2} < 40 torr)

Appendix I

SUMMARY OF CRITERIA FOR SEVERE IMPAIRMENT OR TOTAL DISABILITY DUE TO PULMONARY DISEASE

	"Obstructive" Impairment	"Restrictive" (Interstitial) Impairment
Social Security Black Lung Interim***[6]	FEV_1 and MVV values based on height (see Appendix II(a))*	$Pa_{CO_2} \leqslant 55$ torr (adjusted upward if $Pa_{O_2} < 40$ torr)
Department of Labor Proposed Black Lung Benefits[2]	$FEV_1 \leqslant 60\%$ predicted for age, height, and sex (based on Knudson *et al.*, 1976) and MVV $\leqslant 60\%$ of the 40 × predicted FEV_1*	$Pa_{CO_2} \leqslant 60$ torr (Pa_{CO_2} = 40-45 torr Pa_{O_2} adjusted upward if $Pa_{CO_2} < 40$ torr) or $Pa_{CO_2} < 45$ torr with any Pa_{O_2}

*"Obstructive" and "restricted" not distinguished, blood gas values placed under "restriction" for convenience.
**These standards have also been used by Department of Labor to administer this program since 1973.
***These standards (with a revision of Pa_{O_2} to 60 torr and addition of Pa_{CO_2} 45 torr with any Pa_{O_2} are being used to administer the program until permanent revised standards (under the Black Lung Benefits Reform Act of 1977) are adopted.

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Appendix II(a)

SOCIAL SECURITY

OBSTRUCTIVE IMPAIRMENT

Chronic obstructive airway disease (chronic bronchitis, chronic asthmatic bronchitis or pulmonary emphysema with or without abnormal x-ray findings). With: Spirometric evidence of airway obstruction demonstrated by MVV and FEV_1 both equal to, or less than, the values specified in Table I-70, corresponding to the applicant's height.

Height (inches)	MVV (MBC) equal to or less than L./Min.	and	FEV_1 equal to or less than L.
57 or less	32		1.0
58	33		1.0
59	34		1.0
60	35		1.1
61	36		1.1
62	37		1.1
63	38		1.1
64	39		1.2
65	40		1.2
66	41		1.2
67	42		1.3
68	43		1.3
69	44		1.3
70	45		1.4
71	46		1.4
72	47		1.4
73 or more	48		1.4

Appendix II(a)

SOCIAL SECURITY

BLACK LUNG BENEFITS

Pneumoconiosis shall be found disabling if it is established that the miner has (or had) a respiratory impairment because of pneumoconiosis demonstrated on the basis of a ventilatory study in which the maximum voluntary ventilation (MVV) or maximum breathing capacity (MBC), and 1-second forced expiratory volume (FEV_1) are equal to or less than the values specified in the following table or by a medically equivalent test:

Height (inches)	MVV (MBC) equal to or less than L./Min.	and	FEV_1 equal to or less than L.
57 or less	52		1.4
58	53		1.4
59	54		1.4
60	55		1.5
61	56		1.5
62	57		1.5
63	58		1.5
64	59		1.6
65	60		1.6
66	61		1.6
67	62		1.7
68	63		1.7
69	64		1.8
70	65		1.8
71	66		1.8
72	67		1.9
73 or more	68		1.9

Arterial blood gas values are the same as those for "Interim" Social Security Black Lung Benefits.

Appendix II(a)

SOCIAL SECURITY

INTERIM BLACK LUNG BENEFITS

In the case of a miner employed for at least 15 years in underground or comparable coal mine employment, ventilatory studies establish the presence of a chronic respiratory or pulmonary disease as demonstrated by values which are equal to or less than the values specified in the following table:

	Equal to or less than		
Height (inches)	FEV_1	and	MVV
67" or less	2.3		92
68"	2.4		96
69"	2.4		96
70"	2.5		100
71"	2.6		104
72"	2.6		104
73" or more; and	2.7		108

Arterial oxygen tension at rest (sitting or standing) or during exercise and simultaneously determined arterial P_{CO_2} equal to, or less than, the values specified in the following table:

Arterial P_{CO_2} (mm. Hg)	and	Arterial P_{CO_2} equal to or less than (mm. Hg.)
30 or below		65
31		64
32		63
33		62
34		61
35		60
36		59
37		58
38		57
39		56
40 or above		55

Appendix II(a)

DEPARTMENT OF LABOR

INTERIM BLACK LUNG BENEFITS

Spirometric values are the same as Social Security Interim Black Lung Benefits.

Arterial pCO_2 (mm. Hg)	and	Arterial pO_2 equal to or less than (mm. Hg.)
30 or below		70
31		69
32		68
33		67
34		66
35		65
36		64
37		63
38		62
39		61
40-45		60
Above 45		Any value

Appendix II(a)

DEPARTMENT OF LABOR

BLACK LUNG BENEFIT STANDARDS

A miner who meets the following medical specifications shall be found to be totally disabled, in the absence of rebutting evidence, if the values specified in the following table are met.

For arterial blood-gas studies performed at test sites up to 4,000 feet above sea level:

Arterial pCO_2 (mm. Hg)	Arterial pO_2 equal to or less than (mm. Hg.)
25 or below	75
26	74
27	73
28	72
29	71
30	70
31	69
32	68
33	67
34	66
35	65
36	64
37	63
38	62
39	61
40-45	60
Above 45	Any value

For arterial blood-gas studies performed at test sites between 4,000 and 6,000 feet above sea level.

1. Any pO_2 value which is equal to or below 60 mm. Hg., or
2. Any pCO_2 value which is equal to or above 42 mm. Hg.

Appendix II(a)

VETERANS ADMINISTRATION

RATING SCHEDULE FOR THE RESPIRATORY SYSTEM

Selected Conditions

	Rating
Bronchitis, chronic	
Pronounced; with copious productive cough and dyspnea at rest; pulmonary dyspnea at rest; pulmonary function testing showing a severe degree of chronic airway obstruction; with symptoms of associated severe emphysema or cyanosis and findings of right-sided heart involvement	100
Severe; with severe productive cough and dyspnea on slight exertion and pulmonary function tests indicative of severe ventilatory impairment	60
Moderately severe; persistent cough at intervals throughout the day, considerable expectoration, considerable dyspnea on exercise, rales throughout chest, beginning chronic airway obstruction	30
Moderate; considerable night or morning cough, slight dyspnea on exercise, scattered bilateral rales	10
Mild; slight cough, no dyspnea, few rales	0
Emphysema, pulmonary	
Pronounced; intractable and totally incapacitating; with dyspnea at rest, or marked dyspnea and cyanosis on mild exertion; severity of emphysema confirmed by chest X-rays and pulmonary function tests	100
Severe; exertional dyspnea sufficient to prevent climbing one flight of steps or walking one block without stopping; ventilatory impairment of severe degree confirmed by pulmonary function tests with marked impairment of health	60
Moderate; with moderate dyspnea occurring after climbing one flight of steps or walking more than one block on level surface; pulmonary function tests consistent with findings of moderate emphysema	30
Mild; with evidence of ventilatory impairment on pulmonary function tests and/or definite dyspnea on prolonged exertion	10
Anthracosis (Black Lung Disease)	
Silicosis	
Pneumoconiosis, unspecified	
Pronounced; with extent of lesions comparable to far advanced pulmonary tuberculosis or pulmonary function tests confirming a markedly severe degree of ventilatory deficit; with dyspnea at rest and other evidence of severe impairment of bodily vigor producing total incapacity	100
Severe; extensive fibrosis, severe dyspnea on slight exertion with corresponding ventilatory deficit confirmed by pulmonary function tests with marked impairment of health	60
Moderate; with considerable pulmonary fibrosis and moderate dyspnea on slight exertion, confirmed by pulmonary function tests	30
Definitely symptomatic with pulmonary fibrosis and moderate dyspnea on extended exertion	10

Appendix II(b)

SOCIAL SECURITY

RESTRICTIVE (INTERSTITIAL) IMPAIRMENT

Diffuse pulmonary fibrosis (sarcoidosis, Hamman-Rich Syndrome, idiopathic interstitial fibrosis, and similar diffuse fibroses substantiated by chest x-ray or tissue diagnosis. This category does not include cases of bronchitis or emphysema with incidental scarring or scattered parenchymal fibrosis on x-ray). With:

A. Total vital capacity equal to, or less than, values specified in Table below corresponding to the applicant's height.

Height (inches)	V. C. equal to or less than (L.)
57 or less	1.2
58	1.3
59	1.3
60	1.4
61	1.4
62	1.5
63	1.5
64	1.6
65	1.6
66	1.7
67	1.7
68	1.8
69	1.8
70	1.9
71	1.9
72	2.0
73 or more	2.0

RESTRICTIVE (INTERSTITIAL) IMPAIRMENT (continued)

B. Diffusing capacity of the lungs for carbon monoxide less than 6 ml./mm. Hg./min. (steady-state methods) or less than 9 ml./mm. Hg./min. (single-breath methods) or less than 30 percent of predicted normal. (All methods—actual values and predicted normal for the method used should be reported);

C. Arterial oxygen saturation at rest and simultaneously determined arterial pCO_2 equal to, or less than, the values specified in Table below.

Arterial pCO_2	and	**Arterial O_2 saturation equal to or less than (%)**
30 mm. Hg. or below		93
31 mm. Hg.		93
32 mm. Hg.		92
33 mm. Hg.		92
34 mm. Hg.		91
35 mm. Hg.		91
36 mm. Hg.		90
37 mm. Hg.		89
38 mm. Hg.		88
39 mm. Hg.		88
40 mm. Hg. or above		87

Appendix III(a)

VALUES FOR SCHEMES DESCRIBED IN APPENDIX I

FOR MALE OF HEIGHT 70" (178 cm) WITH "OBSTRUCTIVE" IMPAIRMENT

	Age (yrs)	40	50	60
Social Security	FEV_1 (L)	1.4(35%)*	1.4(38%)	1.4(41%)
Estimated $\dot{V}_{O_2}$ max**	(L/min)	1.58	1.58	1.58
Estimated DPD***		240	220	200
AMA	FEV_1 (L)	2.18(55%)*	2.04(55%)	1.89(55%)
Estimated $\dot{V}_{O_2}$ max**	(L/min)	2.05	2.02	1.95
Estimated DPD***		315	290	275
Gaensler & Wright	FEV_1 + (L)	1.79(45%)*	1.67(45%)	1.54(45%)
Estimated $\dot{V}_{O_2}$ max**	(L/min)	1.90	1.84	1.78
Estimated DPD††		250	275	200
Black Lung Interim	FEV_1 (L)	2.5(63%)*	2.5(68%)	2.5(73%)
Estimated $\dot{V}_{O_2}$ max**	(L/min)	2.24	2.24	2.24
Estimated DPD***		360	340	325
Department of Labor Black Lung Standards	FEV_1 (L)	2.38(60%)*	2.22(60%)	2.06(60%)
Estimated $\dot{V}_{O_2}$ max**	(L/min)	2.19	2.11	2.03
Estimated DPD***		350	310	260

*( ) = % predicted based on Knudson, et al. (1976).

**From equation of Armstrong, et al. (1966) assuming VE_{O_2} = 25, MVV = 40 FEV_1 if not specified.

***From equation of Wilson, et al. (1964), if not specified assumed FVC = $\frac{FEV_1}{.55}$.

†Approximately equivalent to MVV<45% predicted.

††FVC assumed = $\frac{FEV_1}{.40}$.

Appendix III(b)

VALUES FOR SCHEMES DESCRIBED IN APPENDIX I

FOR MALE OF HEIGHT 70" (178 cm) WITH "RESTRICTIVE" (INTERSTITIAL) IMPAIRMENT

	Age (yrs)	40	50	60
Social Security	FVC (L)	1.9(38%)*	1.9(41%)	1.9(43%)
$D_{L(SB)}$ (ml/mmHg/min)		9.72(30%)*	9.12(30%)	8.52(30%)
Estimated $\dot{V}_{O_2}$ max**	(L/min)	1.11	1.11	1.11
Estimated DPD†		222	200	178
If $D_{L(SB)}$ reduced in proportion to FVC				
$D_{L(SB)}$ (ml/mmHg/min)		12.3(38%)*	12.5(41%)	12.2(43%)
Estimated $\dot{V}_{O_2}$ max***	(L/min)	1.11	1.11	1.11
Estimated DPD†		239	222	202
AMA	FVC (L)	2.72(55%)*	2.56(55%)	2.40(55%)
$D_{L(SB)}$ (ml/mmHg/min)		17.82(55%)*	16.72(55%)	15.62(55%)
Estimated $\dot{V}_{O_2}$ max**	(L/min)††	1.65	1.58	1.52
Estimated DPD		326	291	255
Gaensler & Wright	FVC (L)	2.48(50%)*	2.33(50%)	2.18(50%)
$D_{L(SB)}$ (ml/mmHg/min)		13.0(40%)**	12.2(40%)	11.4(40%)
Estimated $\dot{V}_{O_2}$ max***	(L/min)	1.33	1.27	1.21
Estimated DPD		279	246	214
If $D_{L(SB)}$ reduced only to 50% predicted				
$D_{L(SB)}$ (ml/mmHg/min)		16.2(50%)**	15.2(50%)	14.2(50%)
Estimated $\dot{V}_{O_2}$ max††	(L/min)	1.55	1.49	1.44
Estimated DPD		300	266	235

*( ) = % predicted based on Knudson, et al. (1976).

**Normal predictions from Cotes (1975).

***From equation of Armstrong, et al. (1966) VE_{O_2} assumed = 40.

†From equation of Wilson, et al. (1968), FEV_1 assumed = .8 FVC, MVV assumed = 40 FEV_1.

††Assumed to be reduced in proportion to FVC.