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Testimony of

Hans Weill, M.D.
on Behalf of the
American Lung Association/American Thoracic Society

Public Responsibility in Asbestos-Related Diseases

Before the

Subcommittee on Labor Standards
of the
Committee on Education and Labor
U.S. House of Representatives

June 14, 1983

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Statement of Hans Weill, M.D.

Chairman and members of the Subcommittee:

My name is Hans Weill and I am Professor of Medicine and Chief of the Pulmonary Disease Division, Tulane Medical Center, New Orleans, Louisiana. I direct an inter-disciplinary unit engaged in the investigation of occupational lung disease with current research programs dealing with a wide spectrum of minerals; inorganic dusts; and chemical gases and vapors in the work-place. For the past 14 years I and my collegues have been active in the investigation of asbestos health effects encountered in workers engaged in the manufacturing of asbestos building products.

I have served as president of the American Thoracic Society, the medical section and scientific arm of the American Lung Association, and am currently the Chairman of the Pulmonary Disease Advisory Committee to the National Heart, Lung and Blood Institute.

As a chest physician, I am frequently asked to evaluate workers who have had asbestos exposure for the presence and extent of adverse respiratory effects of such exposure. These cases are referred from both plaintiffs and defendents as well as from government agencies.

The members of our unit have special interest and expertise in the use and standardization of chest radiographs and pulmonary function techniques and methods for both epidemiolgy and clinical purposes.

Our research program has been funded primarily by government agencies including the NIH (NHLBI) and NIOSH. A small portion of our research funding comes from industry and labor. I serve as a consultant to management, labor and federal agencies.

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With this background and interest, I am pleased to have an opportunity to provide these comments on the important question of compensating workers whose health has been effected by exposure to asbestos dust.

I appear before this Committee on behalf of the American Lung Association/ American Thoracic Society and as an independent biomedical investigator, physician and academician. I am employed soley by Tulane University. Beginning July 1, I will be at the Brookings Institute as a Fellow in Science and Public Policy investigating the effect of science on policy making in the area of asbestos health effects.

The American Thoracic Society and American Lung Association are pleased to have this opportunity to provide input in the deliberations of this Committee concerning one of the major occupational health issues faced by our society. We represent a professional society composed of chest physicians and biomedical scientists who investigate the causes of and work toward the prevention of lung diseases, as well as public volunteers committed to reducing the burden of lung disease in communities around the country.

At the outset, we would like to emphasize our conviction that, as with many other occupationally induced disorders, the asbestos-associated diseases are best dealt with by prevention through control of exposures to airborne concentrations of asbestos dust. Monetary awards can never compensate individuals for loss of health, or their relatives for loss of life, nor can any compensation scheme provide the adequate disincentive for industry which would lead to prevention of these conditions; this is due in large part to the long latency period between relevant exposure and clinical manifestations of these diseases. Recognizing, however, that past exposures have produced these conditions, we are committed to the provision

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of just resolution of claims by injured workers. We have testified previously that it is our belief that disease-specific approaches to federal compensation legislation are not optimal in reaching the objective of equitable and timely awards for all workers who develop diseases as a consequence of their employment. The inadequacies of the black lung legislation have received considerable attention and have been the subject of periodic controversy, often focusing on questions of cost effectiveness and equity. Thus we regret the extension of a fragmentary approach to the overall burden of occupationally-induced lung disease implicit in the passage of further legislation dealing with the conditions caused by exposure to one specific material. Nevertheless, we recognize that there may well be overwhelming public and economic pressure making it necessary to consider seriously legislation dealing specifically with the asbestos associated diseases. Therefore, in spite of our reservations, we are anxious to provide our views on how this problem might be approached.

Much has been written and said recently concerning the inadequacy of the present system in meeting the needs of individuals who have developed asbestos-associated diseases. We are in full agreement with this assessment. In this context, tort litigation has been shown to be inequitable, costly, and markedly unjust in the distribution of resources, a high proportion of awards going to individuals in whom evidence of disease cannot be demonstrated. Indeed one of the main deficiencies in this approach is that it distorts medical evidence, calling, for instance, for "proof" when in fact medical diagnosis is more often hypothesis, i.e., the best available

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explanation given the facts about a case . Other deficiencies are the failure to rely on objective medical evidence in individual cases, as in mass settlements; and the fact that in the assessment of individual cases medical experts may be chosen not for their expertise and objectivity, but because their testimony will support one side or the other. Given the nature of medical diagnosis, the facts about a case are frequently open to more than one interpretation. We are also mindful of the projections of the future burden of such litigation affecting all segments of our society. Similarly, we feel that the State compensation systems have often dealt poorly with asbestos and other workplace-associated diseases, and we have testified previously in regard to our view that a uniform federal system of occupational lung disease compensation should be developed. We have reviewed, with disappointment and considerable reservation, the draft bills that have been proposed, since it is our opinion that they have not dealt adequately with medical criteria for the conditions to be compensated.

Our testimony on an approach to the compensation of asbestos-associated diseases is predicated on the belief that public policy in this area should be based on scientific information. The known asbestos-associated diseases have all been shown to be dose-related, and we are persuaded by the evidence that there are differences in biological activity of asbestos related to fiber type and process. While this is not the appropriate forum for a comprehensive discussion of these or other related scientific issues, it seems reasonable, however, to suggest that the scientific data base can and should be used in the decision-making process, as the means of assessing the likelihood that the disease is due to exposure,

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particularly for conditions which also occur in the general population in the absence of asbestos exposure. While we readily recognize, however, that many of the decisions made in the clinical setting concerning individual cases lack precision, informed medical opinions most often lead to reasonable conclusions. So it is with many clinical decisions.

The following, in our view, are the necessary elements of a more equitable and workable system of compensating the injured worker who has been exposed to asbestos. In the individual case, the major medical issues to be decided are diagnosis, causation, and impairment. The process of reaching a diagnosis depends upon sound medical practice irrespective of the underlying cause of disease. Determining the most likely cause will usually depend on assessing the levels of exposure to known causative agents; this will be based on epidemiologic studies of occupationally exposed populations. The assessment of impairment will depend primarily on evaluation of disturbance in lung function. Ultimately, guidelines must be established to facilitate this decision-making process, and these must be the subject of continuing review in light of emerging scientific evidence. We strongly suggest that these guidelines not be written into the law because of the dynamic nature of scientific knowledge. The legal system must accept the uncertainties of diagnosis, which invariably depends upon assessment of medical probabilities.

The structure of a workable system should first include a national panel of experts to develop, and periodically modify, guidelines and criteria for the adjudication of claims for asbestos-associated diseases. Secondly,

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there should be regional panels, which would, of course, include health professionals knowledgeable in occupational lung diseases, and which would be responsible for the case-by-case determinations. Finally, a mechanism must be developed to make possible an appeal of the medical decisions rendered by the regional panel.

While the selection of both the national and regional panels, and the development of an appeals system, should be dealt with in the legislation, the details of these aspects are beyond the scope of our testimony, although a few brief comments seem appropriate. The national panel of experts should consist of highly experienced and respected members of the biomedical community. They should be selected from outside the government, and be maximally immune to short-term political influences. Similar considerations should determine the selection of the regional compensation panels.

Recognizing the importance of the guidelines, which are the responsibility of the national expert panel, we have appended to this statement an outline of what we consider to be a reasonable set of guidelines concerning the asbestos-associated diseases based on present knowledge.

All of the diseases which have resulted from asbestos exposure in the workplace have serious consequences to health, and may impair function and reduce longevity with their implicit personal tragedy. For the individuals at risk because of past exposure to asbestos dust, public responsibility is to insure that those who are injured receive the necessary support and assistance. This has not always been the case. The blame for this must be shared by elements of the legal and medical system, as well as

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segments of management and labor. The system outlined in this testimony attempts to maximize rationality and equity. We have every expectation that the Congress will respond appropriately to this difficult challenge.

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PROPOSED GUIDELINES FOR CASE EVALUATION

BASED ON ASSESSMENT OF EVIDENCE AVAILABLE MARCH, 1983

The case evaluation process involves establishing the presence of disease (diagnosis), its cause, and the extent of its functional effect on the individual (impairment).

I. Diagnosis and Determination of Cause

A. Asbestosis

Asbestosis is a pneumoconiosis defined as diffuse fibrosis of the lungs caused by asbestos exposure. Its features include rales (crackles), breathlessness, finger clubbing, lung function abnormalities (usually reduced volumes and impaired gas exchange), radiographic changes (irregular and linear opacities), and histopathologic demonstration of fibrosis with tissue fiber identification. The first four of these are non-specific; changes on the x-ray and pathologic tissue examination have increasing, but by no means absolute, specificity. Individually, these are not sufficient to make a diagnosis; in combination, the diagnosis depends upon weighing probabilities and assessing the evidence in toto, an important component of which is estimation of past exposure. In practice, the diagnosis is usually established on radiographic and exposure evidence. An expert committee of the College of American Pathologists and the National Institute for Occupational Safety and Health has recently developed guidelines for the pathologic diagnosis of asbestos-associated disease. When lung tissue is available for histopathologic examination, a diagnosis of asbestosis depends

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minimally upon the demonstration of discreet foci of fibrosis in the walls of respiratory bronchioles associated with accumulations of asbestos bodies (light microscopy). Additional findings are diffuse interstitial pneumonia and fibrosis. Neither fibrosis or asbestos bodies alone is sufficient for the histopathologic diagnosis of asbestosis.

B. Non-malignant Pleural Effects

These include pleural effusions, focal hyaline thickening (plaques), and diffuse pleural fibrosis. Plaques indicate exposure, not disease, because they do not cause symptoms or functional impairment. Attributability is assessed on the basis of an exposure history and the absence of other causal factors.

C. Lung Cancer

The diagnosis of lung cancer in asbestos-exposed individuals is no different than in other clinical settings. In population studies of various occupational groups, evidence of asbestosis has been found when excess lung cancer risk is demonstrated for comparable levels of exposure. In the individual case of lung cancer, the cause cannot be determined precisely. For this reason, the judgement must take into account length, intensity and character of exposure, evidence of other asbestos-associated diseases, and smoking history.

D. Malignant Mesothelioma

The primary issue is valid diagnosis. This diagnosis can generally not be established clinically, or radiographically, or with pleural fluid examination, or with limited biopsy tissue. Since the potential for misdiagnosis is considerable, adequate tissue specimens should be examined by

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pathologists experienced and expert in the diagnosis of this tumor (e.g., mesothelioma panels). When the diagnosis has been established, a history of exposure, even if short, is sufficient for a judgement of causation.

II. Assessment of Impairment

This follows the establishment of an asbestos-associated disease. In cases of extensive asbestosis or extensive pleural fibrosis, reference should be made to criteria for impairment in restrictive disorders outlined in the ATS statement on evaluation of impairment/disability secondary to respiratory disease (attached). Use will be made of pulmonary function tests and exercise performance testing when necessary. Regional panels will be responsible for periodic updating of impairment level.

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Scientific Assembly News

only if they have not been presented and are not under consideration for presentation at other national meetings. The issue of whether each member of ATS should be limited to sponsorship of one abstract at annual meetings has been raised and will be discussed by the Program Committee, but not for implementation in 1982. Please let us know if you have opinions about this rather significant proposed change.

Best wishes for an enjoyable and productive summer.



Section on Pulmonary Circulation

RICHARD W. HYDE, M.D., *Chairman*

At the annual business meeting, we discussed presentations by posters compared to 10-minute presentations. There was general agreement that the quality of poster sessions should be upgraded to include separate rooms, and there should be elimination of the competition with the lunch period. While some members recommended all sessions be posters, the majority opinion was that 10-minute presentations should be continued.

In order to coordinate activities with the Cardiopulmonary Council of the American Heart Association, the Chairman of the Program Committee of the Cardiopulmonary Council will in the future have a standing invitation to be a member of the Annual Meeting Program Committee of the Pulmonary Circulation Section.

For the Annual Meeting in May of 1982, a symposium is being organized on "Recent Advances in the Pathogenesis and Treatment of Pulmonary Hypertension." Dr. John T. Reeves of the University of Colorado will be the chairman. Dr. Reeves would appreciate suggestions about the symposium from the membership.

Dr. Donald Fredrickson announced his resignation as Director of the National Institutes of Health effective July 1, 1981. Dr. Fredrickson has been head of NIH since July of 1975. In his letter of resignation to President Reagan, he stated personal reasons for leaving.

Evaluation of Impairment/Disability Secondary to Respiratory Disease

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Appendix A-Conversion Factors

Bibliography

There are several classification schemes used for defining the degree of respiratory impairment (2-8). Some of these schemes are based on actual values of pulmonary function tests (4, 5), while others use predicted values (7-9). One scheme is based on severe dyspnea (6). The Component Committee for Disability Criteria of the American Lung Association has reviewed these programs, as well as the best information available, in their efforts to develop impairment/disability criteria in respiratory disease that would standardize one's assessment of respiratory impairment. These recommendations are intended to apply equally to occupational as well as nonoccupational respiratory disease.

A distinction has been made between the terms impairment and disability and the following definitions are presented:

IMPAIRMENT: This is purely a medical condition. It reflects a functional abnormality which persists after appropriate therapy and with no reasonable prospect of improvement. It may or may not be stable at the time the evaluation is made. If severe, it frequently precludes gainful employment. It is always a basic consideration in the evaluation of disability.

DISABILITY: A term which indicates the total effect of impairment upon a patient's life. It is affected by such diverse factors as age, gender, education, economic and social environment, and energy requirements of the occupation.

This paper was prepared by the Component Committee on Disability Criteria of the American Lung Association Pediatric and Adult Lung Disease Committee. The purpose of the document is to set forth guidelines for the assessment of impairment due to respiratory disease to be used by those organizations responsible for the determination of disability compensation (e.g., Social Security Administration, Veterans Administration and Workmen's Compensation Boards).

The paper is presented here for membership review. Please send your comments to the American Thoracic Society, 1740 Broadway, New York, NY 10019.

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Two people with an identical impairment may be differently affected in their life situations.

The Committee believes that rating of health impairment falls within the province of a physician's expertise to quantitate. Determination of disability is an administrative decision which requires consideration of many nonmedical as well as medical variables.

I. Identifying Pulmonary Impairment

History, physical examination, chest radiographs, and pulmonary function tests are needed to establish a diagnosis and/or provide a frame of reference.

1. Medical History

Although comprehensive, the history taking should particularly address the specific problems of pulmonary impairment. For those who have an interest in developing epidemiologic data, the questionnaire of the American Thoracic Society's Epidemiology Standardization Project may be suitable (10). For those who do not prefer to use such a questionnaire, the history should include the following:

(1) Demographic and Clinical Information

A. Identification

Basic information includes name, address, social security number, telephone number, date and places of birth and residence, age, and gender. The patient's race, marital status, highest grade completed in school, regularity and extent of exercise, may be helpful in selected situations.

B. Dyspnea

One of the manifestations of reduced lung function and impairment is shortness of breath. Dyspnea cannot be used as a sole criterion for evaluating impairment because the causes of dyspnea are multiple and complex. Furthermore, individual responses to a given degree of dyspnea vary and are influenced by factors unrelated to the extent of lung disease such as difficulty of verbal communication, preoccupation with health, and socioeconomic and educational backgrounds. Dyspnea should therefore be considered in combination with other clinical and physiologic factors (2, 3, 11-14).

C. Cough, Sputum Production

Although cough occurs frequently in patients with chronic obstructive pulmonary diseases (COPD), only some will meet the criteria of chronic bronchitis, i.e., cough productive of daily sputum for at least three months out of the year for two consecutive years. Thus, a detailed description of sputum as to volume, color, odor, consistency, and when expectorated, becomes important. Simple streaking of blood in the sputum may be observed in chronic bronchitis.

D. Wheezing

The hallmark of reversible airway obstruction is the wheeze. Wheezing is so common in asthma that it is frequently forgotten that "all that wheezes is not asthma." Foreign bodies or tumors in airways can cause wheezing. Such wheezing is usually confined to one area of the chest. Wheezing may occur in patients with congestive heart failure. Usually these findings are of brief duration and associated with obvious signs of cardiovascular decompensation. An ominous sign in status asthmaticus is the absence of wheezing. Wheezing is presumably absent because the flow rates are low or the airways are filled with mucus, thus reducing the ventilation to large areas of the lungs. No discussion on wheezing is complete without a description as to frequency; occurrence during day, year; identification of causative factors; whether it limits one's capacity to function, etc.

E. Environmental Exposure and Chronologic Occupational Data

Although traditional screening questions relating to mining, rock drilling, or sandblasting may be of little value since the public is usually acquainted with these hazards, unfortunately there are enough workers engaged in these industries who are not aware of the dangers to justify the questioning. Job titles such as "plasterer," "fireman," or "engineer" may be of little value because official titles tend to remain the same while job activities and hazardous exposures may change dramatically as a result of the development of new technology.

A detailed history of one's employment background should be presented in chronologic order. Vacation and part-time school employment, etc. should be included. In some cases, the spouse's job and exposure may be helpful. Finally the presence of pets, the use of forced air humidifiers, and the nature of hobbies should be determined. The employee should be questioned about specific hazardous dust or fume exposure. If such an exposure occurred, the following indices are helpful: (1) year first exposed to hazardous agent, (2) total years exposed, (3) estimation of the hazard level, and (4) years since last exposed.

F. Use of Tobacco

Subjects should be classified as nonsmokers (never smoked), ex-smokers (stopped for at least one year), and present smokers. The smoking history should list type(s) of tobacco smoked (cigarette, pipe, or cigar), with an emphasis on cigarette smoking. The history should include such information as to age when the subject first started to smoke, the brands used, if and when the use of cigarettes was discontinued, and an estimate of pack-years smoked. Maximal smoking levels should be determined.

2. Physical Examination

Physical examination *should* contain specific observations which will enable the reviewer to obtain an understanding of the claimant's respiratory problem:

(1) General Description

Notation should be made as to the patient's position (standing, sitting, or supine) at the time blood pressure, heart, and respiratory rates are recorded. Height and weight should be noted, preferably in the metric system. Do the following occur during inspiration: *en bloc* movement of the chest, use of sternocleidomastoid and accessory muscles, paradoxical movement of the intercostal spaces posterolaterally and an attempt to elevate the chest cage by fixing the shoulders and leaning forward. Record the degree of thoracic muscle wasting, position, and extent of diaphragmatic motion as well as paradoxical movement between rib cage and the abdomen.

(2) Description of the Patient's Breathing

Does the patient exhale through pursed lips? Depth of breathing, tachypnea, hyperpnea, labored breathing at rest, as well as the inability to complete sentences because of dyspnea should be recorded. By listening over the trachea with a stethoscope, the forced expiratory flow following a maximal inspiration can be timed. Normally this takes less than four seconds whereas it may take several times this in patients with airflow limitation.

(3) Cyanosis and Clubbing

In the presence of a normal hemoglobin concentration, cyanosis (central) involving the buccal mucosa and lips may not be seen until the arterial blood oxygen saturation is usually less than 75 %. Clubbing, almost invariably absent in COPD but which occurs when lung abscess, empyema, bronchiectasis or asbestosis coexist, should consist of four components: softness of the nail bed, curvature of the nail, increase in soft tissue of the fingertips, and increased hyponychial angle of the right index finger (15).

(4) Adventitious Lung Sounds

The character of lung sounds should be noted during quiet and deep breathing.

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Wheezing or rhonchi and crackles (rales) should be described as to intensity, location, and relationship within the respiratory cycle, i.e., inspiration, expiration, or both. Crackles may be present in two-thirds of patients with chronic interstitial disease such as asbestosis and desquamative interstitial pneumonia. These crackles usually occur during late inspiration and are described as fine end-inspiratory crackles (16). Early inspiratory crackles may be heard in diseases of airflow obstruction and particularly in bronchiolitis obliterans (17).

(5) Cor Pulmonale

The term cor pulmonale means pulmonary heart disease and is associated with chronic bronchitis, emphysema, chronic diffuse interstitial disease, etc. If right heart failure accompanies cor pulmonale, neck vein distension occurs above the upper level of the manubrium sterni when the patient is lying supine. These veins fill from below, and their volume does not decrease when the thorax is elevated to a 45 degree angle from the horizontal. Dependent edema, increased hepatic size in the right midclavicular line, or both may confirm the existence of heart disease not secondary to other causes. Not uncommonly along the left sternal border or over the epigastrium there is a right ventricular early diastolic gallop rhythm which tends to be increased on inspiration (18).

3. Chest Roentgenograms

(1) Minimal Examination

Minimal radiographic examination should consist of posterior-anterior (PA) and lateral views taken on deep inspiration. Chest radiographic findings often correlate poorly with physiologic findings in diseases of airflow limitation such as asthma and emphysema. No correlation between work status and roentgenographic findings has been noted. However, as part of the overall evaluation for impairment/disability, the chest radiograph should be described in terms of hyperinflation, loss of vascular markings, presence of bullae, flattened diaphragms, increase in retrosternal air space, sternophrenic angle, and PA diameter. Cardiac abnormalities or evidence of cor pulmonale should be noted. Finally, almost 10 % of patients with histologically-confirmed diffuse infiltrative disease had normal film findings before biopsy (19).

In diseases with rounded opacities, such as coal worker's pneumoconiosis, correlation between physiologic and roentgenographic abnormalities is poor. The only exception is when there is radiographic evidence of progressive massive fibrosis (PMF). As the PMF intensifies there is sometimes a significant reduction in the ventilatory capacity. If the disease is a mixed reticular-nodular pattern such as sarcoidosis, the correlation is frequently quite poor. However, the extent of diffuse linear opacities seen in interstitial pneumonias or asbestosis, correlate with physiologic abnormalities (20). Although previously suggested (21), the Committee believes that the recently revised international code (ILO 1980) (22) for pneumoconiosis classification should be used in describing all types of diffuse interstitial lung disease.

II. Definitions and Abbreviations Used

The American Physiological Society Committee under the chairmanship of Pappenheimer (1950) (23) provides the basis for many of the existing systems of physiology symbols. A number of revisions have been suggested over the years; the pulmonary terms and symbols used in this report are those included in a Report of the ACCP-ATS Joint Committee on Pulmonary Nomenclature (24).

1. Common Qualifying Symbols

STPD Standard conditions: Temperature 0° C, pressure 760 mmHg, and dry (0 water vapor);

BTPS Body conditions: Body temperature, ambient pressure, and saturated with water vapor at these conditions;

ATPS Ambient temperature and pressure, saturated with water vapor at these conditions.

2. Forced Spirometry Measurements (All values are BTPS unless otherwise specified.)

FVC — Forced vital capacity, performed with a maximally forced expiratory effort.

FEV_t — Forced expiratory volume (timed). The volume of air exhaled in the specified time during the performance of the FVC, i.e., FEV_t for the volume of air exhaled during the first second of the FVC.

$FEV_t/FVC\%$ — Forced expiratory volume (timed) to FVC, ratio expressed as a percentage.

$FEF_{25-75\%}$ — Mean forced expiratory flow during the middle half of the FVC. (Formerly called the maximum mid-expiratory flow rate.)

MVV_x — Maximal voluntary ventilation. The volume (L/min) of air exhaled in a specified period during repetitive maximal effort. The respiratory frequency is indicated by a numerical qualifier, i.e., MVV_{60} as MVV performed at 60 breaths per minute. If no qualifier is given, an unrestricted frequency is assumed.

3. Diffusing Capacity Tests

D_X Diffusing capacity of the lungs expressed as volume (STPD) of gas (x) uptake per unit alveolar-capillary pressure difference for the gas used. When carbon monoxide (CO) is assumed to be the gas, i.e., D_X is D_{CO}.

A modifier can be used to designate technique, i.e., Dsb represents single breath CO diffusing capacity.

D/V_A Diffusion per unit of alveolar volume with D expressed STPD and V_A expressed as liters BTPS. This method is preferred to the occasional practice of expressing both values STPD.

4. Blood Gas Measurements

Pa_{O_2} Arterial oxygen partial pressure.

Pa_{CO_2} Arterial carbon dioxide partial pressure.

Sa_{O_2} Arterial oxygen saturation.

5. Energy Expenditure and Oxygen Utilization

$\dot{V}_E$ — Expired volume per minute (BTPS).

$\dot{V}O_2$ — Oxygen consumption per minute (STPD).

$\dot{V}O_2$ max — Plateau in $\dot{V}O_2$ with increasing work rate, or $\dot{V}O_2$ at maximum tolerated work rate.

MET — The basal oxygen requirement of the body in an inactive state sitting quietly. It is considered by most authorities to be approximately 3.5 ml/(kg·min). Metabolic requirements for various jobs, leisure activities published in Tennessee Heart Association Manual (25), as well as presented by Karvonen (26) and Astrand (27), were computed for a 70-kg person.

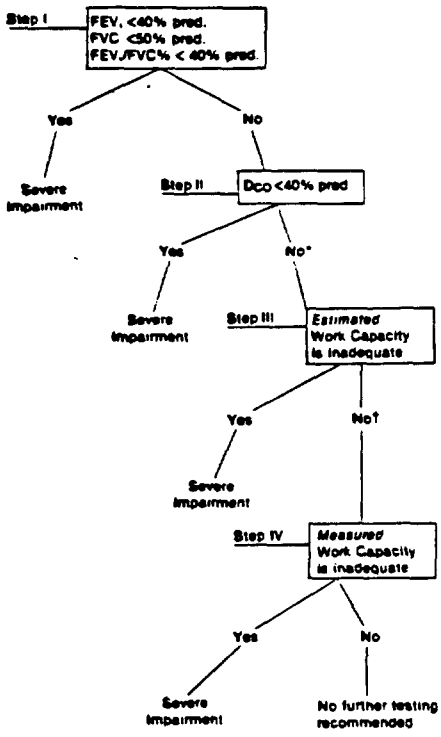
KPM/min — Kilopond meter/minute. The power needed to raise one kilogram the distance of one meter against the force of gravity in one minute.

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III. Rating of Respiratory Impairment

1. Test Schema

The committee recommends the following test schema for determining whether an individual is severely impaired by respiratory disease. (See text for further explanation.)



* If FEV,, FVC, FEV,/FVC% and D_{CO} are above the lower "cut-off" limits—stop.
† Not sure, see text when next step in Test Schema should be requested.

2. Selection of Tests

(1) Forced Spirometry Measurements (Step I)

Three measurements of pulmonary function are included in this step, i.e., FVC, FEV_1, and FEV_1/FVC%. If the results recorded in *any* of the three measurements are below the lower "cut-off" limits, the individual is declared severely impaired, and *no* further pulmonary function testing will be requested.

The subjects should be evaluated after they have received optimum therapy and are at their optimal health condition. If wheezing or other evidence of bronchospasm is present at the time of the examination, the ventilatory studies should be repeated after the administration of a bronchodilator. The bronchodilator most frequently used as an aerosol is isoproterenol, and one should wait ten minutes after administering two inhalations or a dose of 250 μg. If either metaproterenol or isoetharine is used, one should wait 15 to 20 minutes before repeating the spirometric studies.

The spirometric measurements should be performed as described in the Epidemiology Standardization Project (10). The equipment, methods of calibration, and technique used should meet the criteria outlined in the ATS Official Statement, "Standardization of Spirometry" (28).

Height should be measured with the subject standing in his/her stocking feet. If subjects cannot stand, or suffer from spinal deformities, height should be considered as equal to the arm span (maximal distance between the tips of the middle fingers when the arms are stretched out against the wall) (29).

The Committee acknowledges that studies have documented that the FVC and FEV_1 of black males are somewhat lower than those for white males of the same age and height (30, 31). However, a separate prediction equation is not recommended since such equations are not available for other ethnic groups.

When the Committee selected the FEV_1, FVC, and FEV_1/FVC% as the tests for classifying severe impairment, it had to decide between using actual or predicted values. This posed certain philosophical as well as scientific questions (32, 33). *Since the physician is concerned primarily with the definition of impairment and not the direct determination of disablement, the Committee recommends the use of percent predicted value where rating of impairment compares the individual's organ function with a comparable group of healthy individuals.*

A. Forced Expiratory Volume in First Second (FEV_1)

The measurement should be made on the best spirogram recorded either pre- or post-bronchodilator. *If a subject has a FEV_1 of 40 % of predicted or less, this individual is eligible for a severe impairment rating* (8). Normal is considered as a FEV, of greater than when the 95 % confidence interval is subtracted from the predicted value. The regression equations of Crapo et al (34) are acceptable for predicted values:

Test	Units	Equation	95% Confidence Interval
		Males	
FEV_1	L-BTPS	0.0414H-0.0244A-2.190	0.842
		Females	
FEV_1	L-BTPS	0.342H-0.0255A-1.578	0.561

(L-BTPS = liters at BTPS, H = height (cm), A = age (years). The 95% confidence interval when subtracted from the predicted value yields the lower limit of normal.)

B. Forced Vital Capacity (FVC)

The FVC is measured along with the FEV_1. *If a subject has a FVC of 50 % of predicted or less, this individual is eligible for a severe impairment rating* (8). Normal is considered a FVC of greater than when the 95 % confidence interval is subtracted from the predicted value. The regression equations of Crapo and associates (34) are acceptable for predicted values:

Test	Units	Equation	95% Confidence Interval
		Males	
FVC	L-BTPS	0.0600H-0.0214A-4.650	1.115
		Females	
FVC	L-BTPS	0.0491H-0.0216A-3.590	0.876

(L-BTPS = liters at BTPS, H = height (cm), A = age (years) The 95% confidence interval when subtracted from the predicted value yields the lower limit of normal.)

C. FEV_1/FVC Ratio Expressed as a Percentage

If a subject has a FEV_1/FVC% of 40 % of predicted or less, this individual is eligible for a severe impairment rating.[1] Normal is considered to be a ratio of greater than when the 95 % confidence interval is subtracted from the predicted value. The regression equations of Crapo et al. (34) are acceptable for predicted values:

Test	Units	Equation	95% Confidence Interval
		Males	
FEV_1/FVC	%	−0.1300H-0.152A + 110.49	8.28
		Females	
FEV_1/FVC	%	−0.2020H-0.252A + 126.58	9.06

(H = height (cm), A = age (years) The 95% confidence interval when subtracted from the predicted value yields the lower limit of normal.)

(2) Diffusing Capacity (Step II)

The D_{CO} should be performed when complaints of dyspnea continue unabated in spite of forced spirometric measurement results above the cut-off limits.

The methodology for performing the single breath diffusing capacity is basically that described in the 1978 American Thoracic Society Epidemiology Standardization Project (Part 2, pages 62-72) (10).

DeGraff et al (36) have shown that young persons residing at high altitude have higher apparent diffusing capacity for CO than their sea-level counterparts as a result of both increase in membrane diffusing capacity and lung capillary volume. In a study involving rapid changes of environmental pressure, Zamel (personal communication) found that with an increase in altitude, there was a significant increase in the D_{CO} and even more so in the D/V_A. Therefore, for altitudes above 4,000 feet the Com-

[1] These individuals have been found to have a statistically significant shortened life-span (35).

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mittee recommends the addition of supplemental oxygen to the inspired gas mixture in order to raise the exhaled PO_2 to a value which approaches 120 mmHg.

The Committee recommends those predicted values for D_{CO} developed by Crapo and Morris (37), which have been further normalized to a standard hemoglobin, using the accompanying Cotes' modification of the relationship described by Roughton and Forster in 1957 (38, 39):

Males

$$D_{CO}\ \text{corrected} = D_{CO}\ \text{measured} \frac{\text{Hgb} + 10.22}{1.7\ \text{Hgb}}$$

Females

$$D_{CO}\ \text{corrected} = D_{CO}\ \text{measured} \frac{\text{Hgb} + 10.22}{1.8\ \text{Hgb}}$$

The following are the hemoglobin corrected D_{CO} prediction equations and their 95 % confidence intervals as presented by Crapo and Morris (37):

	95% Confidence Interval
Males	
$D_{CO} = 0.391\ H - 0.196\ A - 25.04$	8.5
Females	
$D_{CO} = 0.267\ H - 0.148\ A - 10.34$	5.74

(H = height (cm), and A = age (years). The 95% confidence interval when subtracted from the predicted value yields the lower limit of normal.)

If a subject has a D_{CO} of 40 % of predicted or less, this individual is eligible for a severe impairment (8). Normal is considered a D_{CO} of greater than when the 95 % confidence interval is subtracted from the predicted value.

Since there is no prediction equation which will cover laboratories using different types of equipment, the Committee recommends that each laboratory check the D_{CO} equation it uses by testing 20 normal subjects and determining by the paired "*t*" test whether there is a significant difference between the observed and the predicted values. If there is, the laboratory could either develop its prediction equation or find another in the literature which would fit its needs.

(3) Estimated Work Capacity (Step III)

Work capacity studies are *not* recommended for individuals whose Steps I and II pulmonary function study results are either greater than the 95 % confidence interval or less than 40 % predicted. The Committee, however, recommends the use of work capacity studies under the following circumstances:

When the results of the Step I and II pulmonary function tests are above the severe impairment cut-off point or below the 95 % confidence interval and the claimant states he/she is physically unable to meet the demands of a specific job because of breathlessness.

When the claimant has not performed maximally or correctly in the Step I and II studies.

Exercise testing should not be performed on individuals who have the following: unstable angina pectoris, congestive heart failure, uncontrolled arrhythmias, uncontrolled severe hypertension, severe aortic stenosis, severe pulmonary hypertension, dissecting or ventricular aneurysms, acute illness, limiting neurological or musculoskeletal impairments, or for individuals on medications where performance of stress testing may constitute a significant risk (40, 41).

The criteria for stopping the test include: extreme fatigue, intolerable leg muscle pain, a decrease or lack of increase in systolic blood pressure with increasing workload, an increase in diastolic pressure of more than 20 mmHg above the resting value, severe systolic hypertension (greater than 260 mmHg), a burst of three or more successive premature ventricular contractures, recurring multifocal premature ventricular contractures, major left intraventricular conduction disturbance, symptomatic ST-T segment depression or elevation greater than 2.0 mm, tachyarrhythmia greater than 85 % of the age adjusted heart rate maximum, signs of insufficient peripheral circulation or cardiac output, i.e., pallor, cyanosis, clammy skin, nausea, dizziness, or muscle cramps (40, 41).

The Committee reviewed several articles (42-47) and decided to follow the Simple Clinical Exercise Testing described by Jones (48) in estimating the work capacity. This technique is used in his laboratory in more than 80 % of the subjects on whom exercise studies are performed.[2] The $\dot{V}O_2$ is not measured directly but rather can be obtained once the power output (KPM/min) is determined. Through the use of the Wick's graph (48), one can obtain comparable power output results with either the cycle ergometer or the treadmill (Appendix A). Equations for converting the KPM/min to an equivalent $\dot{V}O_2$ are presented in Appendix A. The equations for predicting one's maximal $\dot{V}O_2$ are also presented in Appendix A. Formulas for calculating the energy requirements for various activities such as treadmill walking, jogging/running, bicycle ergometry, etc., are presented in reference 50.

[2] This approach was presented in the report of a Task Force established by the Canadian Thoracic Society, etc. (49).

An incremental work test is most commonly used to obtain the maximum power output. On a cycle ergometer the subject starts exercising at 100 KPM/min and the power output is increased by 100 KPM each minute.

Small women and children start at 50 KPM and are increased by 50 KPM each minute (48). Ideally, the treadmill test should be performed at three mph (Wick's protocol [46]) but it may be necessary to start at a slower speed and increase it each minute, i.e., 1.5 mph 0 % grade for one minute, 2.5 mph 0 % grade for one minute, and 3.0 mph 0 % grade for one minute. Finally, leave speed at 3.0 mph and increase grade by 2.5 % each minute. The latter will increase work by increments of approximately 100 KPM/min (46).

Because of a linear relationship between $\dot{V}O_2$, heart rate, and ventilation, a concomitant rise in the latter two should be recorded during exercise (42, 49). If maximal heart rate values are of the order of 210- (0.65 × years), the participant is probably being adequately stressed (48). On average, when the subjects are working up to about 85 % of their maximal heart rate, they have reached 75 to 85 % of their $\dot{V}O_2$ max (25, 51). A reduction in the ventilatory capacity will also limit maximum exercise ventilation (MEV) and exercise will cease at a $\dot{V}O_2$ below the predicted maximum. In 1975, Spiro and associates (42, 52) presented this relationship for determining whether the $\dot{V}O_2$ max is acceptable in the COPD patients by comparing the observed $\dot{V}_E$ with the predicted ventilatory capacity which he calculated as follows:

$$\text{MEV} = (\text{FEV}_1 \times 18.9) + 19.7$$

For normal subjects or patients with nonobstructive pulmonary defects, Jones found the following to provide a more acceptable correlation (48):

$$\dot{V}_E\ \text{max} = \text{FEV}_1 \times 35$$

The equipment required for Step III includes a carefully calibrated treadmill or cycle ergometer, an electrocardiograph for recording the heart response to exercise, low resistance valve, dry gas meter for measuring inspired volume and fitted with a potentiometer and recorder for electrocardiograph and potentiometer signals (42, 48, 51). If hypoxemia is anticipated during exercise, ear oximetry is simple and often helpful. A five % drop in the Sa_{O_2} is considered meaningful. There should be ready access to emergency equipment and drugs (41, 51, 53).

The Committee has reviewed the findings of Roemmich and associates (54) where they concluded that for coal miners taller than 165.1 cm (5 ft. 5 in.), provided the FEV, was over 50 % of the predicted figure, the miner

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was capable of sustaining a level of work requiring a $\dot{V}O_2$ max of 1.75 L/min or 7.0+ METS.[3] This level of work is relatively strenuous and in excess of the usual energy demands made on coal miners. Michael et al (55) noted that their subjects could walk on a treadmill for eight hours, without undue fatigue, if the energy costs did not exceed 35% of the $\dot{V}O_2$ max. Three observations presented by Astrand were also evaluated (51):

Working at his/her own pace, work output can be sustained for an eight-hour period if one does not exceed 40 % of his/her attained $\dot{V}O_2$ max.

For shorter periods of time one can work effectively at about 50 % of achieved $\dot{V}O_2$ max.

If one functions under test conditions for at least five minutes at a rate of work demanding a $\dot{V}O_2$ of 2.5 L/min, he/she can most likely work under steady state conditions for hours with a $\dot{V}O_2$ of one L/min.

The Committee thus considers the claimant to be severely impaired if 30–40 % of his/her observed $\dot{V}O_2$ max does not allow the claimant to meet the $\dot{V}O_2$ costs of his/her occupational activities over an eight-hour period (25–27, 56). *If a $\dot{V}O_2$ max of 15 ml/(kg · min) cannot be reached, the participant should be considered impaired for practically all types of labor (49).*

(4) Measured Work Capacity (Step IV)

Measured work capacity studies should be requested whenever one is not sure that the work capacity was properly recorded in the Step III study. The following could serve as a basis for doubt:

Inappropriate changes observed in either the heart rate and/or $\dot{V}_E$.

Subject unable to complete the test because of nonrespiratory symptoms, i.e., leg muscle fatigue, etc.

Doubt about the predominant factor(s) responsible for the respiratory symptoms.

Step IV exercise protocol can be either that used by Jones or one the performing laboratory regularly uses (48). If the Jones's approach is used, the subject will be studied in the steady state[4] at one-third and two-thirds of the maximum power output attained during the Stage III incremental work test. In addition to recording the usual parameters including the KPM/min, $\dot{V}O_2$ max, Jones collects, as the occasion requires, an arterial or arterialized capillary blood specimen, enabling him to quantify such variables as cardiac output, alveolar ventilation, and physiologic dead space/tidal volume ratio. Other details concerning the performance of Step IV *per se* can be found in the narrative presented in Step III as well as the following references (57–59).

The definition of severe impairment is the same as presented for Step III.

(5) Other Tests — Not Recommended

A. Blood Gases

Abnormal arterial blood gas (ABG) values can be a partial reflection of chronically impaired lung function. However, ABG determinations are invasive and the results may be difficult to standardize because of the effects of hyperventilation, altitude, obesity, portal cirrhosis, etc. (60–63). For the obstructive lung diseases, the FEV_1 correlates better with an ability to work than the resting Pa_{O_2} level (63). The Committee does not recommend their use for initial screening. Arterial blood gas studies should be reserved for selected cases and then performed under rigidly controlled laboratory conditions.

The Committee recommends before a subject can be declared impaired because of hypoxemia, he/she should have evidence of one or more of the secondary conditions related to alveolar hypoxia including:

Pulmonary hypertension as determined by cardiac catheterization.

Cor pulmonale as exhibited by electrocardiographic and chest radiographic changes.

Increasing severity of hypoxemia as noted by blood samples from an indwelling arterial cannula or by the ear oximeter while exercising on a treadmill for no more than six minutes at two mph, zero % grade. Increase in hypoxemia can be reversed by O_2 via nasal cannula.

Erythrocytosis confirmed by complete blood count, and if necessary, the use of radioactive nuclides to measure red blood cell mass and plasma volume.

All of these conditions need not, of course, be present in each individual (64).

B. Maximum Voluntary Ventilation

The maximum voluntary ventilation (MVV) first presented in Germany more than 40 years ago was useful because the FEV_1 had not been described (65). It is not as useful as the FEV_1 for the following reasons:

The test has a much larger learning effect.

It is much more fatiguing.

It is influenced not only by the physical properties of the lungs, but also those of the chest wall.

Because of the more rapidly changing flow rates, the instrument requirements are more rigid.

In interstitial disease, the MVV may be normal even when there is a severe impairment of gas exchange.

The 1978 American Thoracic Society Epidemiology Standardization Project did not recommend the use of this test (10).

C. Miscellaneous Tests of Flow and Volume

The $FEF_{25-75\%}$, closing volume, closing capacity and volume of isoflow are tests designed to detect changes in the smaller airways of less than two millimeters in internal diameter and are not recommended for the assessment of impairment.

IV. Special Considerations when Interpreting and Reporting Findings

1. Applicant Severely Impaired by Asthma

An asthmatic is considered severely impaired if carefully documented severe attacks of bronchospasm requiring treatment in emergency room or hospital occur at least once every two months or on an average of at least six times per year despite therapy judged as optimum by a specialist in respiratory disease. The claimants should have prolonged expiration with wheezing or rhonchi between attacks (40).

2. Applicant Not Properly Diagnosed and/or Treated

The name or diagnosis given to the symptoms or syndrome that the medical profession accepts as evidence of ill health has importance for purposes of impairment/disability evaluation. The diagnosis provides a means to decide upon the type of evidence necessary to describe impairment and the form of examination and laboratory procedures necessary for diagnosis. Clearly, if an earlier diagnosis is incorrect, the examiner should submit the correct diagnosis.

3. Applicant Refuses or Has Not Received Commonly Accepted Regimen of Therapy

By definition, regardless of the reason(s) given, decisions concerning severe impairment should not be made until after appropriate therapy and only when there appears to be a reasonable prospect of no further improvement. If the participant is not being treated or has refused therapy generally considered likely to improve the claimant's condition, this should be recorded.

4. Applicant Has Co-Existing Diseases

When two disease processes co-exist, i.e., asthma and psychogenic overlay, it is important to present a separate evaluation for

[3] 70 Kg male.

[4] Steady State. A variation of less than ± 5 beats during a period of one minute of exercise. A variation in expired CO_2 and O_2 of less than ± 0.1% absolute concentration indicates a steady state of CO_2 output and O_2 intake (48).

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each diagnostic entity and indicate the major impairing condition. The examination should be sufficiently detailed in order that this distinction can be made. Although each disease by itself may not be disabling, the sum of the disorders may be.

5. Applicant's Refusal or Inability to Cooperate During Examination

If there is an unreasonable refusal or inability on the part of the claimant to cooperate in the performance of the requested studies, this should be recorded.

6. Applicant Deconditioned

A state of poor conditioning should be described when the level of impairment is primarily attributable to a deconditioned state resulting from illness, recent hospitalization, physical inactivity, obesity, malnutrition, etc.; this fact(s) should be recorded and retesting after appropriate remedial action should be recommended.

7. Applicant Beyond Retirement Age

Retirement age is irrelevant in determining cardiopulmonary impairment, but is important in a disability evaluation. Established procedures prevail for anyone referred for an evaluation.

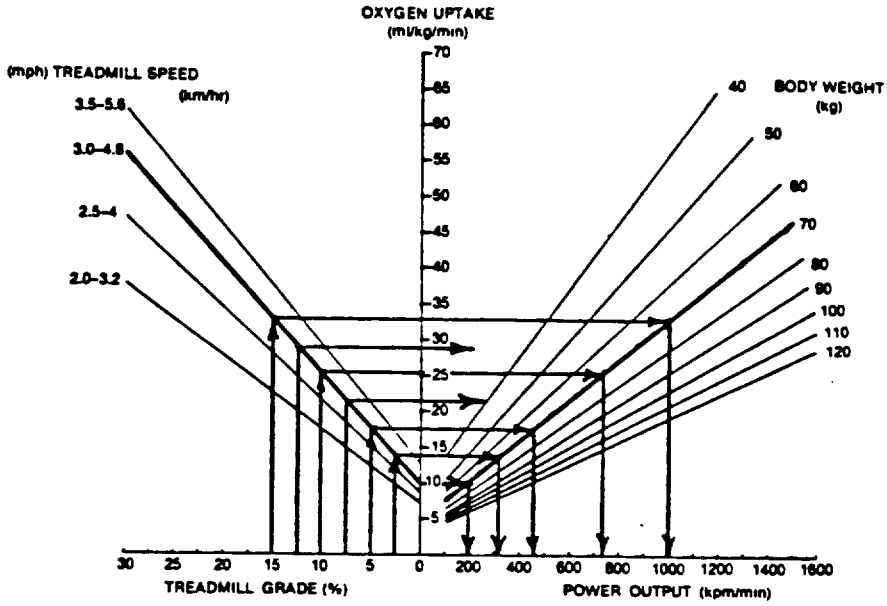
8. Death Cases

Whenever possible, obtain all medical evidence, including the death certificate, that may help in the determination of impairment and/or disability. The death certificate by itself is never sufficient.

9. Applicant Has Disability Without Impairment

Just as impairment can exist without disability, the reverse is true. Tuberculosis, bronchiectasis, sleep apnea (alveolar hypoventilation syndrome) are examples whereby disability can exist without measurable lung function impairment.

APPENDIX A
Conversion Factors



Equivalent Power Output Graph

Using the Wicks graph (48), the treadmill speed and elevation are converted into an equivalent power output obtained with a cycle ergometer as follows: A 70-kg subject is exercised at three mph, using progressive increments in elevation of 2.5 % each minute (46). A vertical line is drawn from the Treadmill Grade (%) to the speed isopleth. A horizontal line is next drawn to the isopleth for the subject's weight. Then a vertical line is dropped to the Power Output line. This is repeated for each increment of elevation reached.

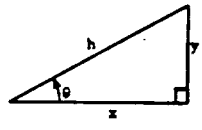
The graph may also be used to predict the $\dot{V}O_2$ from the data obtained during both the treadmill and cycle ergometer tests. Finally, other data such as cardiac frequency and ventilation may be compared with the treadmill and cycle ergometry results.

Direct Calculation of Power Output from Treadmill Testing

Power output (KPM/min) = Wt (kg) × Vertical rate of speed (m/min).

To calculate the power output of a subject walking (running) on a treadmill, the following are needed: (*1*) subject's weight (kilograms), (*2*) the rate of speed at which the subject is walking (meters/min), and (*3*) the angle θ between horizontal and the treadmill incline.

To find the vertical rate of speed, the angle θ must first be calculated.



Generally, treadmill incline is expressed as an elevation in terms of percent, where:

$$\text{Elevation (\%)} = \frac{y}{x} \times 100$$

By dividing this elevation by 100, the result is equal to the tan θ.

$$\tan \theta = \frac{y}{x}$$

To find θ take the arctan of the value $\frac{y}{x}$.

$$\theta = \arctan\left(\frac{y}{x}\right)$$

Now, sin θ will yield the vertical distance an object has been raised per unit of distance that same object has travelled along the hypotenuse. The same reasoning applies to the rates of speed on a treadmill so that:

$$\text{Vertical rate of speed (m/min)} = \text{Treadmill rate of speed (m/min)} \times \sin \theta$$

Note that when the elevation of the treadmill is equal to 0 percent, the equation will yield 0 KPM/min as a power output.

To convert treadmill speed from miles/hour to meters/minute:

$$\text{Speed (m/min)} = \frac{\text{Speed (mph)} \times 1609 \text{ meters/mile}}{60 \text{ min/hr}} = \text{Speed (mph)} \times 26.82$$

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Given the subject's weight in pounds, treadmill speed in miles/hour and treadmill elevation in percent, the complete equation is:

$$\text{Power output (KPM/min)} = \frac{\text{Wt (lb)}}{2.205} \times$$
$$[\text{Speed (mph)} \times 26.82] \times \sin(\arctan[\text{Elev (\%)}/100])$$

Example: Subject's weight: 180 lbs
Treadmill speed: 3.3 mph
Treadmill elevation: 16%

$$\text{Power output (KPM/min)} = \frac{180 \text{ lb}}{2.205} \times [3.3 \text{ mph} \times 26.82 \times \sin(\arctan[16\%/100])]$$
$$= 72.6 \text{ kg} \times [88 \text{ m/min} \times \sin(9.1°)]$$
$$= 72.6 \text{ kg} \times 13.9 \text{ m/min}$$
$$= 1015 \text{ KPM/min}$$

Conversion of Power Output to an Equivalent Oxygen Uptake

Unless directly measured during an exercise test, an approximation of the $\dot{V}O_2$ can be obtained by conversion of the power output (KPM/min) to an equivalent $\dot{V}O_2$ through use of the following formula:

$$\dot{V}O_2 \text{ ml/min} = 2 \times \text{power output (KPM/min)} + \text{basal } O_2 \text{ uptake}$$
where basal O_2 uptake = 3.5 × wt in kg

Example: Subject weighs 85.5 kg; and attained a maximum power output of 900 KPM/min. Thus his $\dot{V}O_2$ approximates 2.1 L/min.

$$\dot{V}O_2 \text{ max} = 900 \times 2 + 3.5 \times 85.5 = 2100 \text{ ml/min}.$$

Expressed per unit body weight = 2100 ÷ 85.5 = 25 ml O2(kg·min).

Prediction of Maximal O_2 Intake ($\dot{V}O_2$ MAX) (48)

Males
$\dot{V}O_2 \text{ max} = 4.2 - 0.032 \text{ age L/min (SD} \pm 0.4)$
$\dot{V}O_2 \text{ max} = 60 - 0.55 \text{ age ml/(kg·min) (SD} \pm 7.5)$

Females
$\dot{V}O_2 \text{ max} = 2.6 - 0.014 \text{ age L/min (SD} \pm 0.4)$
$\dot{V}O_2 \text{ max} = 48 - 0.37 \text{ age ml/(kg·min) (SD} \pm 7.0)$

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