NIH Public Access
Author Manuscript
J Occup Environ Med. Author manuscript; available in PMC 2015 October 01.

Published in final edited form as:
J Occup Environ Med. 2014 October ; 56(0 10): S23–S29. doi:10.1097/JOM.0000000000000282.

Performing a Lung Disability Evaluation: How, When, and Why?

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Abstract

Objective: The objective of this review is to present a case-based clinical discussion on this topic.

Methods: The manuscript represents part of the proceedings of the Occupational and Environmental Lung Disease conference held by the American College of Chest Physicians (ACCP) at Toronto, Ontario, Canada in 2013, and is based upon a non-systematic review of the current literature by the author.

Results: While the American Medical Association Guides to the Evaluation of Permanent Impairment is the mainstay for evaluating respiratory impairment, many other impairment schemata are currently available in the United States. Impairment evaluation for a case with chronic respiratory disease and a separate case with asthma are discussed.

Conclusions: Pulmonary function tests are the cornerstone for evaluating respiratory impairment. Impairment values differ between various impairment schemata. Impairment evaluation for asthma may be particularly difficult.

Introduction

The management of a patient with lung disease does not end with its treatment. These patients often require additional assistance on issues related to respiratory impairment and disability. Yet, many physicians refrain from providing these services, often with disastrous consequences for the patient. Multiple reasons underlie the general physician reluctance in addressing impairment. These include a lack of inclusion as a curricular milestone in fellowship training (1), fear and poor understanding of the legal system, confusion about various compensation systems, a mistaken notion that those who seek impairment assistance are malingerers (2, 3), and a desire to avoid potentially uncompensated efforts in the context of an already burdensome clinical schedule.

Although the terms, impairment and disability, are often used interchangeably, they are not synonymous. In 1980, the World Health Organization issued a statement defining *impairment* as, "any loss or abnormality of psychological, physiological, or anatomical

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Conflicts of Interest: None

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structure or function," and *disability* as, "any restriction or lack, resulting from impairment, of ability to perform an activity within the range considered normal for a human being." (4) The goal of respiratory impairment evaluation is the objective measurement of the extent of loss of respiratory function, primarily through application of pulmonary function or exercise testing. The physician plays a key role in impairment evaluation. On the other hand, the impact of the respiratory impairment on a person's ability to perform day-to-day activities is called disability, which is typically determined through application of administrative and legal instruments by experts in these areas who may be physicians or non-physicians. These experts not only rely upon the impairment evaluation, but also take into consideration other social and legal issues, as well as the specific requirements of the job.

Patients seeking an impairment evaluation in the United States can be usually classified into three general types: (1) those with advanced lung disease who apply for disability benefits under the United States Social Security Impairment program; (2) those with work-related lung disease who usually apply under the Workers' Compensation System; and (3) those who develop lung disease as a consequence of active military service, such as the Veterans Administration. The most commonly used impairment guidelines in the United States are the Social Security Impairment program and the Workers' Compensation System. Issues relevant to impairment evaluation for lung disease are discussed using two examples in the text below.

Case 1

A 56 year old man presents with a five years history of progressive moderate dyspnea on exertion (corresponding to New York Heart Association class II dyspnea) and daily cough productive of white phlegm, meeting the clinical definition of chronic bronchitis. He gives a 42 pack-year history of smoking and an occupational history of working in a foundry for 28 years. He gives a history of sandblasting without adequate respiratory protection for the initial 12 years of this job. The foundry was shut down two years prior to his presentation when he was also laid off. His current medications include short acting beta agonist and long acting anticholinergic inhalers. His physical examination is significant for a height measurement of 170 cms. without shoes, respiratory rate of 18 per minute; heart rate of 76 per minute; and an oxygen saturation of 92% on room air. His chest wall is symmetric and auscultation of his lungs shows bilateral expiratory rhonchi. The rest of his examination is normal. A two- view chest x-ray reveals hyperexpansion of bilateral lung fields. His pulmonary function tests are summarized in Table 1.

Question 1: What is the percent impairment of the whole person, based on lung disease, assuming maximal medical improvement and using the sixth edition of the American Medical Association (AMA) Guides to the Evaluation of Permanent Impairment (Guides)?

American Medical Association (AMA) Guides to the Evaluation of Permanent Impairment (Guides)

There are six editions of the AMA Guides. (5) Since the various editions of the AMA Guides contain markedly different sets of recommendations on impairment evaluation, the physician must choose the ‘right’ edition, depending upon the requirement of the compensation system that the patient is applying for. Use of the ‘wrong’ edition may result in a different impairment rating. Generally speaking, the use of the sixth edition of the AMA Guides results in a lower impairment rating than the previous editions, which may translate to a lower financial compensation for the patient with pulmonary injury or illness. While the AMA Guides generally follow the American Thoracic Society (ATS) schema for evaluating respiratory impairment (6, 7), there exist substantial differences between the two guidelines. Further, while other major guidelines are available on the Internet without charge, use of the web-based sixth edition of the AMA Guides carries a user fee.

Unlike previous editions, the sixth edition of the AMA Guides uses a standardized grid that incorporates five classes of impairment severity, ranging from Class 0 to Class 4. (5) The grids incorporate an objective, test-based key factor for defining the impairment class, along with two non-key factors for fine-tuning the severity grade within a given class. There are five grades in each severity class except for class 0 where there are no grades. Among the various objective tests, the most severely affected test parameter is used to define the impairment class.

Using the post bronchodilator forced expiratory volume in one second (FEV_1) value of 45% predicted (*i.e.* the most severely affected test parameter); this patient meets the key factor criteria for class 3 impairment (using the grid summarized in Table 2). The initial default impairment rating is the central grade within the severity class, which corresponds to a severity grade of 3C or 32% impairment. For the first remaining non-key factor, one determines the most appropriate impairment class position and records the number difference to the key factor impairment class. This step is repeated for the remaining non-key factor. The history and physical examination status (non-key factors) satisfy impairment class 2 criteria. Since the impairment class for each of these non-key factors is one class level below the key factor class, two grades should be subtracted from the default grade to identify the appropriate impairment severity grade. Thus, the final impairment rating corresponds to grade 3A or 24%, using the sixth edition of the AMA Guides.

Resting Pulmonary Function Tests

Pulmonary function tests are the cornerstone for evaluating respiratory impairment and should be performed according to the most recent ATS standards. (8-11) Spirometry and diffusing capacity are the key pulmonary function tests for assessing respiratory impairment for chronic respiratory conditions. Post bronchodilator spirometry is used when airflow limitation is present. Although the presence of hypoxemia on arterial blood gas analysis was used to evaluate impairment under the fifth edition of the AMA Guides, the sixth edition does not include hypoxemia in the evaluation of impairment. (5) The sixth edition also endorses the use of specific NHANES III reference standards for spirometry for Caucasian Americans, Mexican Americans, and African Americans. (5, 12) For the remaining population subgroups, no clear guidelines are provided. For corrected single-breath carbon monoxide diffusing capacity (D_LCO), Crapo's reference standards are used. The cut points for impairment classification, as suggested by the AMA Guides, are set arbitrarily and may differ from those recommended for assessing degree of lung disease severity by other professional organizations, such as the 2005 ATS statement (9) or by the Global Initiative for Chronic Obstructive Lung Disease (GOLD). (13) Some investigators have suggested that lung function thresholds should be expressed as a z-score, which converts a raw measurement on a test to a standardized score expressed in units of standard deviations.(14, 15) This strategy, although scientifically valid, is not currently used for impairment evaluation.

Maximal Cardiopulmonary Exercise Tests

Maximal cardiopulmonary exercise tests are difficult to perform due to need for specialized equipment and trained personnel, are expensive and not readily available, and carry a risk to the patient. Test performance should strictly adhere to the 2003 ATS-ACCP guidelines.(16) Clear agreement on the role of exercise tests in the evaluation of respiratory impairment is lacking. Generally, in cases in which subjective dyspnea is disproportionate to the resting pulmonary function test results, or when pulmonary function tests are difficult to interpret because of submaximal performance, cardiopulmonary exercise tests may be considered. Exercise testing may also be useful in determining whether an individual can perform a specific job with a known energy requirement (5). Generally speaking, per the AMA Guides, cardiopulmonary exercise testing is infrequently needed in the practical investigation of pulmonary impairment (5).

Under the 1986 ATS guidelines for the evaluation of impairment/disability secondary to respiratory disorders, the estimation of impairment from oxygen consumption at peak exercise ($\dot{V}O_2$peak) is based on the widely held, but untested, assumption that a worker involved in manual labor can comfortably work at 40 percent of $\dot{V}O_2$ peak (corresponding to lower limit of generally accepted normal values for anerobic threshold) for prolonged periods. (7) This view is based on findings from several small exercise studies performed in controlled laboratory settings on motivated volunteers, usually athletic men, that show that exercise endurance can be predicted from anaerobic threshold. (17-20) This assumption is however inadequately tested in occupational settings. Many have argued that 40% is too low a threshold value since anaerobic threshold occurs at a much higher percentage of predicted VO2peak in fit individuals. (17, 19) Finally, none of the major guidelines currently advocate the use of anaerobic threshold over the $\dot{V}O_2$ peak value to determine respiratory impairment.

Some laboratories lacking cardiopulmonary exercise test equipment often estimate METs (or metabolic equivalents or multiples of basal oxygen consumption which is approximately 3.5 mL/min/kg) of activity based upon exercise speed and grade on a treadmill, duration of exercise, and heart rate or heart rate variability, instead of directly measuring oxygen consumption. (21-23) These methods are not considered sufficiently accurate. The AMA Guides and the ATS statements only use cardiopulmonary exercise testing for this purpose. (5, 7)

Under the 1986 ATS guidelines for the evaluation of impairment/disability secondary to respiratory disorders, it is further implied that $\dot{V}O_2$ requirements can be assigned to specific occupations and individuals whose $\dot{V}O_2$peak is ≤15 mL/kg/minute would be uncomfortable performing most jobs because they would find it difficult to travel back and forth to their place of employment.(7) Unfortunately, data on $\dot{V}O_2$ requirements of most jobs in modern workplaces are not currently available. Furthermore, jobs with the same title may vary considerably in their $\dot{V}O_2$ requirements from one work site to another.

Scientific rationale for choice of tests used for impairment evaluation

The premise for the use of pulmonary function and exercise tests for evaluating impairment is that $\dot{V}O_2$peak reasonably measures ability to work, and that resting pulmonary function tests, such as FEV_1 and D_LCO, reasonably predict $\dot{V}O_2$peak values and therefore predict ability to work.

Oxygen Consumption at Peak Exercise as the Gold Standard for Measuring Ability to Work

Limited available medical literature appears to support the view that $\dot{V}O2$peak value, expressed as ml/kg/min, may be the gold standard for assessing impairment.(24-26) With exercise on a cycle ergometer, $\dot{V}O2$ increases linearly with external work, (16) and $\dot{V}O2$peak represents the maximal work an individual can perform during a short burst of activity. Some have advocated use of percent predicted $\dot{V}O_2$peak values (i.e., loss of aerobic capacity), instead of $\dot{V}O_2$peak expressed in ml/kg/min (i.e., remaining aerobic ability) for evaluating impairment in patients with respiratory disease, since the latter approach overestimates impairment in older and obese subjects.(27, 28) In addition, some consider the value for $\dot{V}O_2$ at anaerobic threshold ($\dot{V}O_2$AT) as a better index for work ability than $\dot{V}O_2$peak. (28) Individuals are unable to sustain work rates above anerobic threshold values. However, no major guidelines currently suggest the use of percent predicted $\dot{V}O_2$peak values or $\dot{V}O_2$AT to rate impairment.

Comparison of Resting Pulmonary Function Tests with Oxygen Consumption at Peak Exercise

FEV_1 is linearly correlated with $\dot{V}O2$peak levels, (29) but the reported correlations vary widely between studies, resulting in variance (coefficient of determination or r^2) values ranging from 0.25 to 0.71.(29-34) Use of absolute *vs.* percent predicted values yield similar correlation measures.(33) Although some studies demonstrate that FEV_1 and forced vital capacity (FVC) have similar predictive value for $\dot{V}O_2$peak levels,(33) most report FEV_1 to be a stronger predictor than FVC. A 2005 ATS statement on interpretative strategies for pulmonary function testing indicated that percent predicted $FEV_{1,}$ rather than FVC, should be used to categorize severity of impairment for all respiratory diseases.(9) The predictive ability of FEV_1 for $\dot{V}O_2$peak increases if it is used in combination with another variable, such as D_LCO, minute ventilation ($\dot{V}_E$), or dead space ventilation measure during exercise (V_D/V_T).(33) D_LCO does not predict $\dot{V}O_2$peak among healthy controls, (30) but it does so among subjects with COPD and those with occupational lung diseases, where it may account for a variance (coefficient of determination or r^2) of 0.25 to 0.76 in various studies. (32, 33, 35)

Despite the previously noted correlations in population studies, resting pulmonary function tests cannot accurately predict $\dot{V}O_2$peak values among individuals, particularly those with occupational lung diseases. In a comparison study of impairment ratings obtained using simultaneous resting pulmonary function tests and exercise tests conducted in 216 ambulatory patients with COPD, the two methods resulted in similar impairment rating in only 30.1%.(25) Ratings were similar between the two methods in the extreme subgroups of normal or severely impaired individuals. 61.1% were found to be less impaired according to exercise testing than according to resting pulmonary function tests, and 8.8% were more impaired according to exercise testing than resting pulmonary function tests. These data suggest that use of resting pulmonary function tests and exercise testing for evaluating impairment often yields discrepant results.

Question 2: Given the limited information available, can the lung disease in Case 1 be *attributed* to his occupational inhalational exposures?

Although not a part of impairment evaluation *per se*, physicians may need to confirm the validity of the clinical diagnosis and may be asked to additionally render an opinion on causation and apportionment. *Causation* or *attribution* refers to whether an exposure has been a “substantial” contributing factor in either causing or exacerbating lung disease. The level of certainty required in determining causation for occupational lung disease is different from the usual standard of 95% certainty used in medical research. The commonly accepted standard of certainty for occupational cases is that the illness is substantially caused, or exacerbated by, an occupational exposure on a "more probable than not" basis, or a level of certainty greater than 50%.

Any attempt to define causation or attribution must start with a comprehensive review of the existing medical literature on the association between the exposure and disease. For instance, relevant to Case 1, COPD has been associated with inhalational silica exposure, independent of smoking (36-40). Studies from many different work environments suggest that exposure to working environments contaminated by silica at dust levels that appear not to cause roentgenographically visible simple silicosis can cause chronic airflow limitation and/or mucus hypersecretion and/or pathologic emphysema (41). A meta-analysis of 13 studies among coal and gold miners confirmed an excess of bronchitic symptoms and obstructive physiology, even among nonsmokers (40). Further, there may be an additive effect between tobacco smoke and occupational pollutants in producing chronic bronchitis and air flow obstruction (41). Given the strength of evidence that silica inhalational dust exposure is an independent risk factor for COPD as well as the reported duration and latency of exposure in this case, a level of certainty that is greater than 50% is reached. Therefore, this patient’s COPD can be attributed to his occupational inhalational exposures.

Apportionment describes the relative contribution of multiple factors to the total respiratory impairment. For instance, both chronic inhalational silica exposure and cigarette smoking are “substantial” contributory factors to COPD. From a scientific perspective, it is usually difficult, if not impossible, to "apportion" their relative roles in causation of COPD, a complex, multifactorial disease. Physicians are asked to state their opinion on apportionment in the context of the body of available knowledge in that area, which is usually very limited.

Further, retrospective exposures are usually poorly quantified. It is the opinion of this author that specific percentages of apportionment for dust *versus* cigarette smoke in causing occupational lung disease in this case are best avoided.

Workers' Compensation System

This patient may also be eligible to apply under the Worker's Compensation system. The Workers' Compensation system is a "no-fault" system of medical care and disability insurance in which private insurers or self-insured employers pay benefits to an employee sustaining an injury or illness due to workplace exposure. Under Workers' Compensation rules, workers cannot sue their employer for injury or illness. The rules for the Workers' Compensation system in the United States vary from one state to another, but they usually follow one of the six editions of the American Medical Association (AMA) Guides to the Evaluation of Permanent Impairment. Although not relevant to Case 1, dust exposed coal miners may apply for 'total disability' benefits under the 1972 Black Lung Benefits Act. (42)

Case 2

A 50 year old male chemist presents with adult onset severe persistent asthma that has involved eight unscheduled physician visits including four visits to the emergency room, all resulting in hospitalizations, in the previous 12 months. The patient complains of daily respiratory symptoms of cough, wheeze, and dyspnea even in between attacks along with frequent nocturnal awakenings. A laryngoscopy reveals no paradoxical vocal cord movement. The list of daily medications includes 10 mg oral Prednisone, combination of inhaled high dose corticosteroid and long-acting beta agonist, frequent short-acting inhaled beta agonist, Zafirlukast, and monthly Omalizumab infusions. Physical examination is significant for 158 cms. Height, a BMI of 35 kg/m^2, and persistent expiratory wheezes documented on lung auscultation at all clinic visits. Pre-and post-bronchodilator spirometry results are shown in Table 3. A methacholine bronchoprovocation test was not performed as the test was deemed unsafe.

A review of patient's occupational history revealed that this chemist had a new exposure to colophony fumes at his workplace prior to the onset of symptoms. On direct questioning, the patient stated that his symptoms were worse on the days he worked; and improved on weekends and vacations, with recurrence of symptoms on returning to work. His serial peak expiratory flow rate tracings were consistent with the diagnosis of work related asthma. The material safety data sheet (MSDS) for colophony mentioned rosin, which is a sensitizer. A specific inhalational challenge could not be performed due to the lack of clinical test availability in the United States. A diagnosis of occupational asthma was established and the patient was removed from workplace exposures.

Question 3: What is the percent impairment of the whole person, based on lung disease, assuming maximal medical improvement and using the sixth edition of the AMA Guides?

Unlike most chronic respiratory conditions, asthma is an episodic disease, and impairment evaluation for asthma is particularly difficult. Therefore, most impairment evaluation schemata incorporate guidelines for asthma that are separate from those for chronic lung disease. Impairment levels for asthma differ dramatically between various impairment evaluation schemata as well.

A methacholine bronchoprovocation test is useful for confirming the diagnosis of asthma as well as in evaluating impairment from asthma under the AMA Guides and ATS guidelines.(5, 6) The performance of methacholine bronchoprovocation tests should strictly adhere to the relevant ATS guidelines.(43) The methacholine PC_{20} (*i.e.* provocative concentration of methacholine, expressed as mg/mL that results in at least 20% drop in FEV_1 compared to the pre-test baseline) is a 'key factor' for evaluating asthma impairment under the sixth edition of the AMA Guides. (5) On the other hand, methacholine PC_{20} and extent of FEV_1 reversibility are given less weight than are either minimum medication need or post bronchodilator FEV_1 in the multi-component asthma impairment scoring scheme recommended by the 1993 ATS guidelines or by previous editions of the AMA Guides. (5, 6) Unlike Social Security impairment criteria, the AMA Guides and ATS guidelines do not incorporate frequency of acute exacerbations in the impairment evaluation for asthma. Given the efficacy of currently recommended asthma therapies, frequent emergency room visits or hospitalizations generally reflect inadequate treatment and failure to achieve the objectives of treatment. The AMA Guides and ATS guidelines instead use minimum medication need for asthma control as a better reflection of the severity of disease for the purpose of impairment assessment than frequency of asthma exacerbations. Hence, it's easy to see why impairment ratings for the same patient with asthma might vary widely among various compensation systems. Further, the use of the sixth edition of the AMA Guides may result in a lower impairment rating than previous editions of the AMA Guides, which in turn may translate to a lower financial compensation for the patient with asthma.

In the event that methacholine PC_{20} is unavailable or cannot be safely performed (as is the case for the patient above), the sixth edition of the AMA Guides accepts post bronchodilator **FEV_1 percent predicted** value as a key factor. Using that approach, this patient receives an impairment rating of the whole person due to asthma of 0%. Although this scenario is uncommon, this case reflects the difficulties associated with asthma impairment, particularly when there is a discrepancy between the minimum medication need and post bronchodilator spirometry values.

Determinants of Work Ability in Asthma

Determinants of work ability in asthma are inadequately studied. In one study of subjects with objectively confirmed asthma, factors associated with a lower self-reported work ability include lower PC_{20} value (i.e., higher degree of airway hyper-responsiveness), greater disease severity as defined by the minimum medication needed to control asthma, and the presence of respiratory symptoms at the workplace (44). Interestingly, work ability in subjects with asthma is not related to baseline FEV_1 or FVC in that study (even though FEV_1 enters the impairment evaluation schema for asthma under both the AMA Guides and the ATS guidelines). (44) This finding is consistent with another non work-related study that demonstrates that FEV_1 percent predicted is inferior to standardized asthma questionnaires in predicting clinical asthma outcomes. (45)

Comparison between Respiratory Symptoms and PC_{20}

For most subjects with asthma, greater breathlessness perceived during asthma attacks is not correlated with greater decline in peak expiratory flow rate (46) or with a lower PC_{20} value.(47)

Comparison between Minimum Medication Need and PC_{20}

Almost all medications used to treat asthma improve PC_{20} values, i.e., decrease bronchial hyper-responsiveness.(48-60) In one study, patients with asthma with the minimum medication needed to control symptoms were divided into four groups: (1) those who required no medication; (2) those who required short-acting β_2 agonist occasionally, but not daily; (3) those who required daily short-acting β_2 agonist; and (4) those who required additional inhaled corticosteroid dosing. The mean PC_{20} value was highest in group 1 and lowest in group 4; the differences between each group were significant.(61) Minimum medication need in asthma is, therefore, an important predictor for airway hyper-responsiveness as well as work ability.

Comparison between Percent Predicted FEV_1 and PC_{20}

In a small clinical population of smokers and non-smokers, as well as in a population of subjects with asthma with concomitant stable bronchiectasis, baseline FEV_1 has been shown to correlate with methacholine PC_{20} values.(62)

Impairment evaluation for occupational asthma is even more problematic than non-occupational asthma. In these cases, both temporary and long-term impairment evaluation should be performed.(6) Temporary impairment for patients with sensitizer-induced occupational asthma should be performed after removing the worker from exposure. Early cessation of exposure improves prognosis in sensitizer-induced occupational asthma. Sometimes, physiologic tests may be normal, and symptoms and need for treatment may subside after early cessation of exposure, resulting in 0% measureable impairment. However, such an individual should be considered as 100% disabled on a permanent basis from working in a job that exposes him or her to the specific sensitizing agent.(6) It is not necessary to wait for long-term impairment rating to initiate vocational rehabilitation in such a case.(6) The long-term impairment evaluation is performed using the impairment evaluation systems devised for nonoccupational asthma, usually at least two years after cessation of exposure, when improvement has been shown to plateau (5, 6, 63-67). This recommendation is based upon findings by Malo *et al*. that improvements may be found two years or more after stopping exposure (68, 69) and the systematic review by Rachiotis *et al.* which demonstrated a pooled estimate of 32% symptomatic recovery at a median duration of follow-up of 31 months after cessation of exposure, with a significant between-study heterogeneity.(70)

Conclusions

The following five steps often constitute the process of completing a respiratory impairment evaluation.

The first step involves the confirmation of the diagnosis of lung disease. Because of the medicolegal nature of the evaluation, the physician should have greater certainty of the medical diagnosis than is sometimes used in clinical practice. In other words, objective confirmation of the diagnosis is preferable.

The second step involves defining maximal medical improvement (MMI). MMI occurs at the point when, following maximal therapy, no further clinical or physiological improvement is expected to occur (although deterioration might). If therapy has not been maximized, the physician should either delay impairment evaluation or give a temporary evaluation. A permanent impairment evaluation should be performed only at, or after, MMI has been reached.

The third step is identifying the correct guideline for evaluating impairment. As discussed previously, several compensation systems exist, each with its' own unique guideline. Therefore, identification of the compensation system for which the patient is eligible is essential, and the evaluating physician must be familiar with the specific guideline to be used. Of course, some patients may be eligible for more than one compensation program and may apply for more than one program contemporaneously.

The fourth step is to supplement the history and physical examination findings with appropriate objective tests. Performance of these tests should strictly adhere to the ATS standards.(8-11)

The fifth and the most important step requires writing a comprehensive report of the patient's history, physical examination, and review of objective tests. The assessment should provide clear and accurate answers, in lay terms, to the questions asked. The evaluation should state the diagnosis and whether MMI has been reached, and it should make note of the presence and degree of respiratory impairment. The specific impairment scheme used, including the specific page and table of the guideline used, should be referenced. In work-related respiratory disorders, causation, apportionment, and work restrictions may also need to be addressed, as requested.

Acknowledgements

While the Occupational and Environmental Lung Disease Conference 2013 was sponsored by the American College of Chest Physicians (ACCP), the content of the presentation as well as that of this manuscript was not regulated or approved by the College or by other presenters at the conference. The author of this manuscript was the speaker on this topic at the above-mentioned conference but not the organizer. Several other topics from that multi-speaker conference are being published in the same special edition of the Journal of Occupational and Environmental Medicine.

Disclosure of Funding Received for This Work: National Institutes of Health (NIH) - K23 HL094531-01A1

Sources of Funding: National Institutes of Health (NIH) - K23 HL094531-01A1; 1UL1RR031977; and 5 UL1 TR000041-05; Health Resources and Services Administration (HRSA) - 1D33HP19042; Patient Centered Outcomes Research Institute (PCORI) Contract # 7738152

All Sources of Support That Require Acknowledgment: National Institutes of Health (NIH) - K23 HL094531-01A1

The author would like to thank the two reviewers for their careful review of the manuscript and their constructive criticism. Their comments have been effectively addressed in the revised manuscript. It is hoped that the revised manuscript will find favor with the reviewers and the editor.

References

1. Fessler HE, Addrizzo-Harris D, Beck JM, et al. Entrustable Professional Activities and Curricular Milestones for Fellowship Training in Pulmonary and Critical Care Medicine: Report of a multi-society working group. Chest. 2014
2. Robinson KM, Monsivais JJ. Malingering? No evidence in a predominantly Hispanic workers' compensation population with chronic pain. Pain management nursing : official journal of the American Society of Pain Management Nurses. 2011; 12:33–40. [PubMed: 21349447]
3. Demers RY, Fischetti LR, Neale AV. Incongruence between self-reported symptoms and objective evidence of respiratory disease among construction workers. Social science & medicine (1982). 1990; 30:805–810. [PubMed: 2315748]
4. Wood PH. Appreciating the consequences of disease: the international classification of impairments, disabilities, and handicaps. WHO chronicle. 1980; 34:376–380. [PubMed: 6449782]
5. American Medical Association. The Pulmonary System. In: Rondinelli, RD., editor. Guides to the Evaluation of Permanent Impairment. American Medical Association; 2008. p. 77-99.
6. American Thoracic Society. Medical Section of the American Lung Association. Guidelines for the evaluation of impairment/disability in patients with asthma. The American review of respiratory disease. 1993; 147:1056–1061. [PubMed: 8466106]
7. American Thoracic Society. Evaluation of impairment/disability secondary to respiratory disorders. The American review of respiratory disease. 1986; 133:1205–1209. [PubMed: 3509148]
8. Miller MR, Crapo R, Hankinson J, et al. General considerations for lung function testing. Eur Respir J. 2005; 26:153–161. [PubMed: 15994402]
9. Pellegrino R, Viegi G, Brusasco V, et al. Interpretative strategies for lung function tests. The European respiratory journal : official journal of the European Society for Clinical Respiratory Physiology. 2005; 26:948–968.
10. Macintyre N, Crapo RO, Viegi G, et al. Standardisation of the single-breath determination of carbon monoxide uptake in the lung. The European respiratory journal : official journal of the European Society for Clinical Respiratory Physiology. 2005; 26:720–735.
11. Miller MR, Hankinson J, Brusasco V, et al. Standardisation of spirometry. The European respiratory journal : official journal of the European Society for Clinical Respiratory Physiology. 2005; 26:319–338.
12. Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. American journal of respiratory and critical care medicine. 1999; 159:179–187. [PubMed: 9872837]
13. Rabe KF, Hurd S, Anzueto A, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med. 2007; 176:532–555. [PubMed: 17507545]
14. Miller MR, Pincock AC. Predicted values: how should we use them? Thorax. 1988; 43:265–267. [PubMed: 3406912]
15. Vaz Fragoso CA, Gill TM. Respiratory impairment and the aging lung: a novel paradigm for assessing pulmonary function. The journals of gerontology Series A, Biological sciences and medical sciences. 2012; 67:264–275.
16. ATS/ACCP Statement on cardiopulmonary exercise testing. American journal of respiratory and critical care medicine. 2003; 167:211–277. [PubMed: 12524257]
17. Sullivan CS, Casaburi R, Storer TW, Wasserman K. Non-invasive prediction of blood lactate response to constant power outputs from incremental exercise tests. European journal of applied physiology and occupational physiology. 1995; 71:349–354. [PubMed: 8549579]
18. Wasserman K. The anaerobic threshold measurement to evaluate exercise performance. The American review of respiratory disease. 1984; 129:S35–40. [PubMed: 6421216]
19. Yoshida T, Chida M, Ichioka M, Suda Y. Blood lactate parameters related to aerobic capacity and endurance performance. European journal of applied physiology and occupational physiology. 1987; 56:7–11. [PubMed: 3830147]

20. Tanaka K, Matsuura Y, Matsuzaka A, et al. A longitudinal assessment of anaerobic threshold and distance-running performance. Medicine and science in sports and exercise. 1984; 16:278–282. [PubMed: 6748926]
21. Smolander J, Ajoviita M, Juuti T, Nummela A, Rusko H. Estimating oxygen consumption from heart rate and heart rate variability without individual calibration. Clinical physiology and functional imaging. 2011; 31:266–271. [PubMed: 21672133]
22. Froelicher VF Jr. Thompson AJ Jr. Davis G, Stewart AJ, Triebwasser JH. Prediction of maximal oxygen consumption. Comparison of the Bruce and Balke treadmill protocols. Chest. 1975; 68:331–336. [PubMed: 1157538]
23. Haller JM, Fehling PC, Barr DA, Storer TW, Cooper CB, Smith DL. Use of the HR index to predict maximal oxygen uptake during different exercise protocols. Physiological reports. 2013; 1:e00124. [PubMed: 24303190]
24. Pichurko BM. Exercising your patient: which test(s) and when? Respiratory care. 2012; 57:100–110. discussion 110-103. [PubMed: 22222129]
25. Fink G, Moshe S, Goshen J, et al. Functional evaluation in patients with chronic obstructive pulmonary disease: pulmonary function test versus cardiopulmonary exercise test. Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine. 2002; 44:54–58. [PubMed: 11802466]
26. Sue DY. Exercise testing in the evaluation of impairment and disability. Clinics in chest medicine. 1994; 15:369–387. [PubMed: 8088099]
27. Rusanov V, Shitrit D, Fox B, Amital A, Peled N, Kramer MR. Use of the 15-steps climbing exercise oximetry test in patients with idiopathic pulmonary fibrosis. Respir Med. 2008; 102:1080–1088. [PubMed: 18457939]
28. Neder JA, Nery LE, Bagatin E, Lucas SR, Ancao MS, Sue DY. Differences between remaining ability and loss of capacity in maximum aerobic impairment. Braz J Med Biol Res. 1998; 31:639–646. [PubMed: 9698768]
29. Pineda H, Haas F, Axen K, Haas A. Accuracy of pulmonary function tests in predicting exercise tolerance in chronic obstructive pulmonary disease. Chest. 1984; 86:564–567. [PubMed: 6478895]
30. Bogaard HJ, Woltjer HH, van Keimpema AR, Postmus PE, de Vries PM. Prediction of peak oxygen uptake in men using pulmonary and hemodynamic variables during exercise. Med Sci Sports Exerc. 2000; 32:701–705. [PubMed: 10731016]
31. Ong KC, Ong YY. Cardiopulmonary exercise testing in patients with chronic obstructive pulmonary disease. Ann Acad Med Singapore. 2000; 29:648–652. [PubMed: 11126703]
32. Dillard TA, Piantadosi S, Rajagopal KR. Determinants of maximum exercise capacity in patients with chronic airflow obstruction. Chest. 1989; 96:267–271. [PubMed: 2752808]
33. Cotes JE, Zejda J, King B. Lung function impairment as a guide to exercise limitation in work-related lung disorders. Am Rev Respir Dis. 1988; 137:1089–1093. [PubMed: 3195806]
34. Dimopoulou I, Tsintzas OK, Daganou M, Cokkinos DV, Tzelepis GE. Contribution of lung function to exercise capacity in patients with chronic heart failure. Respiration. 1999; 66:144–149. [PubMed: 10202318]
35. Carlson DJ, Ries AL, Kaplan RM. Prediction of maximum exercise tolerance in patients with COPD. Chest. 1991; 100:307–311. [PubMed: 1907536]
36. Leung CC, Yu IT, Chen W. Silicosis. Lancet. 2012; 379:2008–2018. [PubMed: 22534002]
37. Ehrlich RI, Myers JE, te Water Naude JM, Thompson ML, Churchyard GJ. Lung function loss in relation to silica dust exposure in South African gold miners. Occupational and environmental medicine. 2011; 68:96–101. [PubMed: 20884796]
38. Rushton L. Chronic obstructive pulmonary disease and occupational exposure to silica. Reviews on environmental health. 2007; 22:255–272. [PubMed: 18351226]
39. Rushton L. Occupational causes of chronic obstructive pulmonary disease. Reviews on environmental health. 2007; 22:195–212. [PubMed: 18078004]
40. Oxman AD, Muir DC, Shannon HS, Stock SR, Hnizdo E, Lange HJ. Occupational dust exposure and chronic obstructive pulmonary disease. A systematic overview of the evidence. The American review of respiratory disease. 1993; 148:38–48. [PubMed: 8317812]

41. American Thoracic Society Committee of the Scientific Assembly on Environmental and Occupational Health. Adverse effects of crystalline silica exposure. American journal of respiratory and critical care medicine. 1997; 155:761–768. [PubMed: 9032226]
42. United States Government Printing Office. Part 718 - Standards for determining coal miners' total disability or death due to pneumoconiosis. 2000. available at http://www.ecfr.gov/cgi-bin/retrieveECFR?gp=1&SID=5c92730f5b89d3c7474009fc8605a504&ty=HTML&h=L&n=20y4.0.2.2.5&r=PART
43. Crapo RO, Casaburi R, Coates AL, et al. Guidelines for methacholine and exercise challenge testing-1999. This official statement of the American Thoracic Society was adopted by the ATS Board of Directors, July 1999. Am J Respir Crit Care Med. 2000; 161:309–329. [PubMed: 10619836]
44. Balder B, Lindholm NB, Lowhagen O, et al. Predictors of self-assessed work ability among subjects with recent-onset asthma. Respir Med. 1998; 92:729–734. [PubMed: 9713631]
45. Eisner MD, Yegin A, Trzaskoma B. Severity of asthma score predicts clinical outcomes in patients with moderate to severe persistent asthma. Chest. 2012; 141:58–65. [PubMed: 21885725]
46. Boudreau D, Styhler A, Gray-Donald K, Martin JG. A comparison of breathlessness during spontaneous asthma and histamine-induced bronchoconstriction. Clin Invest Med. 1995; 18:25–32. [PubMed: 7768063]
47. Boulet LP, Leblanc P, Turcotte H. Perception scoring of induced bronchoconstriction as an index of awareness of asthma symptoms. Chest. 1994; 105:1430–1433. [PubMed: 8181331]
48. Page CP, Cotter T, Kilfeather S, Sullivan P, Spina D, Costello JF. Effect of chronic theophylline treatment on the methacholine dose-response curve in allergic asthmatic subjects. Eur Respir J. 1998; 12:24–29. [PubMed: 9701409]
49. Perng DW, Huang HY, Lee YC, Perng RP. Leukotriene modifier vs inhaled corticosteroid in mild-to-moderate asthma: clinical and anti-inflammatory effects. Chest. 2004; 125:1693–1699. [PubMed: 15136378]
50. Svendsen UG, Frolund L, Madsen F, Nielsen NH. A comparison of the effects of nedocromil sodium and beclomethasone dipropionate on pulmonary function, symptoms, and bronchial responsiveness in patients with asthma. J Allergy Clin Immunol. 1989; 84:224–231. [PubMed: 2547858]
51. Kanniess F, Richter K, Bohme S, Jorres RA, Magnussen H. Montelukast versus fluticasone: effects on lung function, airway responsiveness and inflammation in moderate asthma. Eur Respir J. 2002; 20:853–858. [PubMed: 12412675]
52. Berkman N, Avital A, Bardach E, Springer C, Breuer R, Godfrey S. The effect of montelukast on bronchial provocation tests and exhaled nitric oxide levels in asthmatic patients. Isr Med Assoc J. 2003; 5:778–781. [PubMed: 14650101]
53. O'Connor BJ, Towse LJ, Barnes PJ. Prolonged effect of tiotropium bromide on methacholine-induced bronchoconstriction in asthma. Am J Respir Crit Care Med. 1996; 154:876–880. [PubMed: 8887578]
54. Overbeek SE, Rijnbeek PR, Vons C, Mulder PG, Hoogsteden HC, Bogaard JM. Effects of fluticasone propionate on methacholine dose-response curves in nonsmoking atopic asthmatics. Eur Respir J. 1996; 9:2256–2262. [PubMed: 8947069]
55. Silkoff PE, McClean PA, Slutsky AS, et al. Exhaled nitric oxide and bronchial reactivity during and after inhaled beclomethasone in mild asthma. J Asthma. 1998; 35:473–479. [PubMed: 9751064]
56. Derom EY, Pauwels RA, Van der Straeten ME. The effect of inhaled salmeterol on methacholine responsiveness in subjects with asthma up to 12 hours. J Allergy Clin Immunol. 1992; 89:811–815. [PubMed: 1560164]
57. Yates DH, Sussman HS, Shaw MJ, Barnes PJ, Chung KF. Regular formoterol treatment in mild asthma. Effect on bronchial responsiveness during and after treatment. Am J Respir Crit Care Med. 1995; 152:1170–1174. [PubMed: 7551366]
58. Zu Wallack RL, Kass J, Shiue ST, et al. Effect of inhaled triamcinolone on bronchial hyperreactivity and airways obstruction in asthma. Ann Allergy. 1990; 64:207–212. [PubMed: 2301782]

59. van Rensen EL, Straathof KC, Veselic-Charvat MA, Zwinderman AH, Bel EH, Sterk PJ. Effect of inhaled steroids on airway hyperresponsiveness, sputum eosinophils, and exhaled nitric oxide levels in patients with asthma. Thorax. 1999; 54:403–408. [PubMed: 10212103]

60. Bel EH, Zwinderman AH, Timmers MC, Dijkman JH, Sterk PJ. The protective effect of a beta 2 agonist against excessive airway narrowing in response to bronchoconstrictor stimuli in asthma and chronic obstructive lung disease. Thorax. 1991; 46:9–14. [PubMed: 1871705]

61. Juniper EF, Frith PA, Hargreave FE. Airway responsiveness to histamine and methacholine: relationship to minimum treatment to control symptoms of asthma. Thorax. 1981; 36:575–579. [PubMed: 7031972]

62. Ip M, Lam WK, So SY, Liong E, Chan CY, Tse KM. Analysis of factors associated with bronchial hyperreactivity to methacholine in bronchiectasis. Lung. 1991; 169:43–51. [PubMed: 2011043]

63. British guideline on the management of asthma. Thorax. 2003; 58(Suppl 1):i1–94. [PubMed: 12653493]

64. Tarlo SM, Malo JL. An official American Thoracic Society proceedings: work-related asthma and airway diseases. Presentations and discussion from the Fourth Jack Pepys Workshop on Asthma in the Workplace. Annals of the American Thoracic Society. 2013; 10:S17–24. [PubMed: 23952871]

65. Beach, J.; Rowe, BH.; Blitz, S., et al. Diagnosis and management of work-related asthma: evidence report/technology assessment. Agency for Healthcare Research and Quality, United States Department Of Health And Human Services; Rockville, MD: 2005.

66. National Asthma Council Australia. Australian Asthma Handbook. 2002. available online at www.nationalasthma.org.edu

67. Tarlo SM, Balmes J, Balkissoon R, et al. Diagnosis and management of work-related asthma: American College Of Chest Physicians Consensus Statement. Chest. 2008; 134:1S–41S. [PubMed: 18779187]

68. Malo JL, Cartier A, Ghezzo H, Lafrance M, McCants M, Lehrer SB. Patterns of improvement in spirometry, bronchial hyperresponsiveness, and specific IgE antibody levels after cessation of exposure in occupational asthma caused by snow-crab processing. Am Rev Respir Dis. 1988; 138:807–812. [PubMed: 3202454]

69. Malo JL, Ghezzo H. Recovery of methacholine responsiveness after end of exposure in occupational asthma. American journal of respiratory and critical care medicine. 2004; 169:1304–1307. [PubMed: 15070824]

70. Rachiotis G, Savani R, Brant A, MacNeill SJ, Newman Taylor A, Cullinan P. Outcome of occupational asthma after cessation of exposure: a systematic review. Thorax. 2007; 62:147–152. [PubMed: 17040933]

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Table 1

Summary of pulmonary function tests for Case 1.

Pulmonary Function Parameter	Observed value	Percent predicted value
Post bronchodilator Forced Vital Capacity (FVC)	3.48 L	80%
Post bronchodilator Forced Expiratory Volume in One Second (FEV_1)	1.60 L	45%
Post bronchodilator FEV_1/FVC Ratio	46%	
Carbon Monoxide Diffusing capacity (D_LCO)	16.0 ml/min.mm Hg	51%

Table 2

A summary of the grid used by the sixth edition of the AMA Guides to determine the class and grade of impairment for chronic lung disease. The bold font shows the appropriate level of the rating factor in each column for Case 1. Readers are advised to look up the complete tables in the AMA Guides.

Criteria	**Key Factor Pulmonary Function & Exercise Tests**	**Non-Key Factor History of Clinical Presentation**	**Non-key Factor Physical Examination findings**
Severity Class	Class 3: 24-40% impairment	Class 2: 11-23% impairment	Class 2: 11-23% impairment
Severity Grade (%)	A B C D E 24 28 32 36 40	A B C D E 11 14 17 20 23	A B C D E 24 28 32 36 40
	FVC 50-59% predicted	Constant mild dyspnea despite continuous treatment	**Constant mild physical findings despite continuous treatment**
	FEV_1 45-54% predicted	**Intermittent moderate dyspnea despite continuous treatment**	Intermittent moderate physical findings
	D_LCO 45-54% predicted		
	VO_2max 15-17 ml/kg/min		
	VO_2max in 4.3-5.0 metabolic equivalent units or METs		

Footnote 1: FEV_1 is the most severely affected test parameter in Table 1 and is used as the ‘key factor’ for evaluating impairment. Even though the patient’s D_LCO value falls in the same impairment class as FEV_1, the former is not used as a ‘key factor’ since it is not the most severely affected test parameter.

Table 3

A summary of pulmonary function tests for Case 2

Parameter	Pre bronchodilator Parameter	Post bronchodilator Parameter	Percent Change in Parameter
Forced Vital Capacity (FVC)	3.0 L (75%)	3.68 L (92%)	23%
Forced Expiratory Volume in One Second (FEV_1)	2.1 L (62%)	3.0 L (88%)	42%
FEV_1/FVC Ratio	71%	82%	

Note: A methacholine bronchoprovocation tests was not performed as the test was deemed unsafe.

Table 4

A summary of the grid used by the sixth edition of the AMA Guides to determine the class and grade of impairment for asthma. The bold font shows the appropriate level of the rating factor in each column for Case 2. Readers are advised to look up the complete tables in the AMA Guides.

Criteria	**Key Factor methacholine PC_{20}**	**Non-key Factor - History of Clinical Presentation**	**Non-key Factor - Minimum Medication Need**
Severity Class		Class 0: 0% impairment	Class 4
Severity Grade (%)		-	A B C D E 45 50 55 60 65
		>80% post bronchodilator FEV_1 percent predicted despite continuous treatment	**Asthma not controlled by treatment**

Note: If methacholine PC_{20} is unavailable, post bronchodilator FEV_1 percent predicted (otherwise a non-key factor) is used as a key factor.