



Workplace Safety and Insurance
Appeals Tribunal

Tribunal d'appel de la sécurité professionnelle
et de l'assurance contre les accidents du travail

Chronic Obstructive Pulmonary Disease (COPD)

Discussion paper prepared for

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Prepared by:

Dr. Dildar Ahmad

Emeritus Professor of Medicine
University of Western Ontario

Dr. Dildar Ahmad graduated from the Royal College of Surgeons in Ireland, Dublin in 1962. He did post-graduate training in Respiriology at the Institute of Chest Disease, London, England from 1964 to 1965. He was granted his fellowship in Respiriology in 1974. He joined the faculty at the University of Western Ontario in 1987 and currently holds the rank of Emeritus Professor of Medicine in the University of Western Ontario. His clinical and research interests were in pulmonary disease and heart lung transplants, and he has published widely in that area. He served as Director Lung Transplant program and as Staff Physician, University Campus London Health Sciences Centre from 1998 to 2003, and Consulting Physician, Respiriology at the London Cardiac Institute, London from 2003 to present. Dr. Ahmad has been an assessor for the Tribunal from 1995 to present.

This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

Chronic Obstructive Pulmonary Disease (COPD) is one of the most chronic of all known diseases. It is a major cause of mortality, morbidity and a major use of health care resources. In 2004, COPD was the fourth leading cause of death in both men and women in Canada (1). The burden of COPD has been increasing for the last two decades and is projected to increase in the next four (2). In 2000, COPD became fourth leading cause of death and is projected to be fourth leading cause for disability worldwide by 2009 (3). COPD is a leading cause of hospitalization and accounts for 19.9% of total hospitalizations for patients 63-75 years (4). The annual per patient costs for COPD parallel those of diseases, such as diabetes, arthritis and cardiovascular disease.

Definition

COPD is defined as a disease state characterized by airflow obstruction that is not fully reversible. The airflow obstruction is progressive and is associated with an abnormal inflammatory response in the lungs to noxious particles and gases thus causing airflow obstruction. Chronic airflow obstruction (CAO), Chronic Obstructive Lung Disease (COLD) and chronic airflow limitation (CAL) are synonymous terms used to describe the same disease.

Emphysema is defined as an abnormal permanent enlargement of airspace distal to terminal bronchioles accompanied by destruction of their walls and without fibrosis. Chronic bronchitis is described as a chronic cough, with mucous production for three months of the year for at least two successive years, where other causes have been excluded (5).

Asthma differs from COPD and is considered as a different entity. However some patients with asthma develop poorly reversible airflow limitation. These patients are indistinguishable from patients with patients with COPD and for practical purposes are treated as asthma. The high prevalence of COPD and asthma in general population results in the coexistence of both these diseases in many individuals.

Risk Factors:

Risk factors for developing COPD are genetic and environmental

Genetic factors:

In 1-3% of patients COPD is linked to genetic factors.. A low concentration of enzyme alpha-one antitrypsin in combination with smoking and other exposures increases the risk of pan-lobular emphysema that is seen in the lower lobes of the lungs. Several genes have been implicated in COPD, but the results have been inconsistent (7).

Tobacco Smoking:

Tobacco smoke exposure is the most important risk factor for COPD worldwide. Smoking is believed to be the cause of 80-90% of all COPD (8). However, only 10-20% of smokers develop clinically significant COPD. Recent evidence suggests this figure is as high as 50% (9). These observations suggest that genetic factors also have a role in causation.

Occupational dust vapors and fumes:

Some occupational environments are likely to involve a risk of COPD. The risk is less than that of smoking and interactions between smoking and occupational exposure to various agents are relevant. Occupational exposures to various dusts, chemicals, vapors and fumes act additively to increase a (13) person's risk of developing COPD. The fraction of COPD attributable to work was estimated as 19.2% overall and 31.1% in never smokers (10). Specific occupational exposures that contribute to development of COPD have accumulated over the past two decades (11, 12).

Air Pollution:

Air pollution is a risk factor for COPD, the risk of outdoor pollution is smaller than that of indoor pollution. According to WHO, in countries with low and middle income the risk of developing COPD is 35%. A recent report from China showed that persons who had never smoked and were exposed to biomass smoke had an increased risk of COPD.

Childhood Infections:

Studies have shown that infections during early childhood predispose an individual to COPD in adult life.

Socioeconomic Factors:

Poor populations have a higher risk of developing COPD. Many factors are responsible, poor nutrition, smoking, exposure to pollutants, crowding and early childhood infections.

Clinical Presentation

History:

The three most common symptoms in COPD are cough, sputum production and exertional shortness of breath. The morning cough and sputum production occurs in the fifth decade of life and can be present for months or years before a person seeks medical help. Shortness of breath on exertion usually does not occur till the 6th or 7th decade and occurs insidiously, as the airflow obstruction is a gradual process. Patients with COPD have periodic exacerbations, usually with an increased shortness of breath, increase in cough and sputum production with wheezing. The sputum changes in colour

from white (mucoid) to green or yellow with occasional blood streaks. These exacerbations usually occur in persons with compromised airflow obstruction and are known as acute exacerbation of chronic bronchitis (AECB). In advanced lung disease anorexia and weight loss is common and carries a worse prognosis. Psychiatric morbidity is high in COPD, reflecting social isolation due to chronicity and severity of the disease and the neurological effects of hypoxemia (low O_2 of arterial blood) and hypercapnea (high blood CO_2). Clinical examination is usually normal in early stage of the disease. As the disease progresses the resting respiratory rate increases to over 18/min. The thoracic cage is barrel shaped and on auscultation (listening to breath sounds) there is prolonged expiratory phase of breath sounds with or without wheezes. In patients presenting with <50 years of age, a strong family history or with minimal smoking history, serum levels of alpha-1-antitrypsin levels should be measured.

Investigations:

Chest x-rays are insensitive in establishing the diagnosis of COPD. In advanced disease patients develop hyperinflation with flattening of diaphragms. CT scans are most sensitive in diagnosing COPD. They can determine the type of emphysema that the person has.

Spirometry:

Spirometry is a "gold standard" for the diagnosis of COPD and it is an easy test to perform. Spirometry measures the maximum volume of air exhaled from a point of maximal inhalation. This volume is referred to as Forced Vital Capacity (FVC). The volume of air that is exhaled during the first second during the forced vital capacity is measured. This is referred to as Forced Expiratory Volume 1 Second (FEV1). With the spirometry machine on, the patient's age, height and ethnic group are entered to determine normal values for comparison. The patient completely fills the lungs, hears the command from technician, physician or a nurse to blow out for 6 seconds in one uninterrupted breath. The test is repeated a second time to check for repeatability, which must be within the 3%, to ensure that maximum effort has taken place. Extensive studies have shown that only three measurements are needed for a good Spirometry. Normally, 70% to 75% of FVC is blown out in the first second; thus, the FEV1/FVC ratio should be > 70%. (Table 1)

Severity of COPD:

There are a number of guidelines that classify severity based on the level of impairment of lung function and level of symptoms. The Canadian Thoracic Society published guidelines in 2007. They are listed in Tables 2 and 3.

Chronic Obstructive Pulmonary Disease (COPD)

Table 1

COPD Stage	Symptoms
MILD	Shortness of breath from COPD when hurrying on the level or walking up a slight hill. (MRC 2)
MODERATE	Shortness of breath from COPD causing the patient to stop after walking approximately 100 m (or after a few minutes) on the level. (MRC 3 to4)
SEVERE	Shortness of breath from COPD resulting in the patient being too breathless to leave the house, breathless when dressing or undressing (MRC 5), or the presence of chronic respiratory failure or clinical signs of right heart failure.

Table 2

COPD Stage	Spirometry
MILD	FEV1 > 80%, predicted. FEV1/FVC < 70
MODERATE	50% < FEV1 < 80% Predicted. FEV1/FVC < 70
SEVERE	30% < FEV1 < 50% Predicted. FEV1/FVC < 70
VERY SEVERE	FEV1 < 30% Predicted. FEV1/FVC < 70

The Medical Research Council Dyspnea Scale

Table 3

Grade	Description
1.	Not troubled by breathlessness except with strenuous exercise.
2.	Troubled by shortness of breath when hurrying on the level or walking up a slight hill.
3.	Walks slower than people of the same age on the level because of breathlessness or has to stop for breath when walking at own pace on the level.
4.	Stops for breath after walking about 100 yards or after a few minutes on the level.
5.	Too breathless to leave the house or breathless when dressing or undressing.

Chronic Obstructive Pulmonary Disease (COPD)

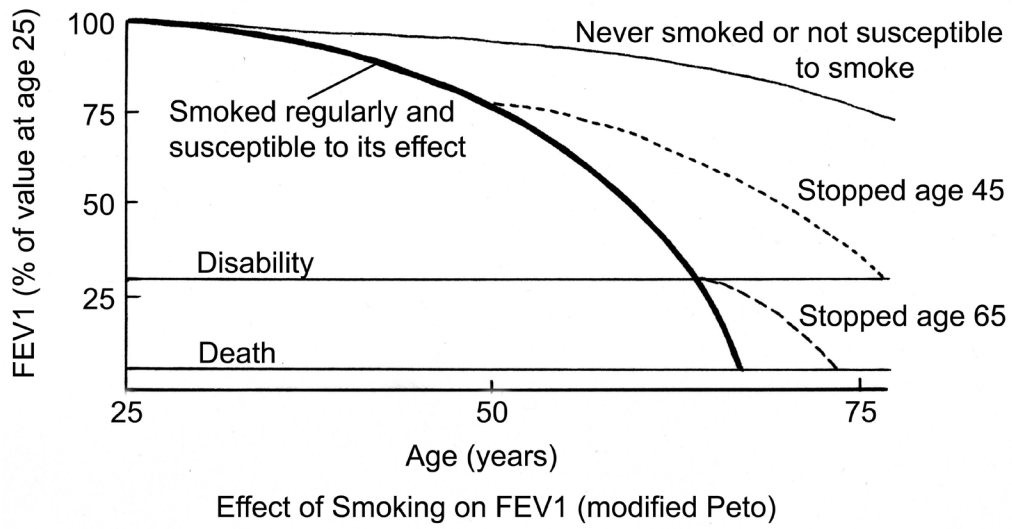


Figure 1 - Effect of Smoking on FEV1 (modified Peto)

Table 1

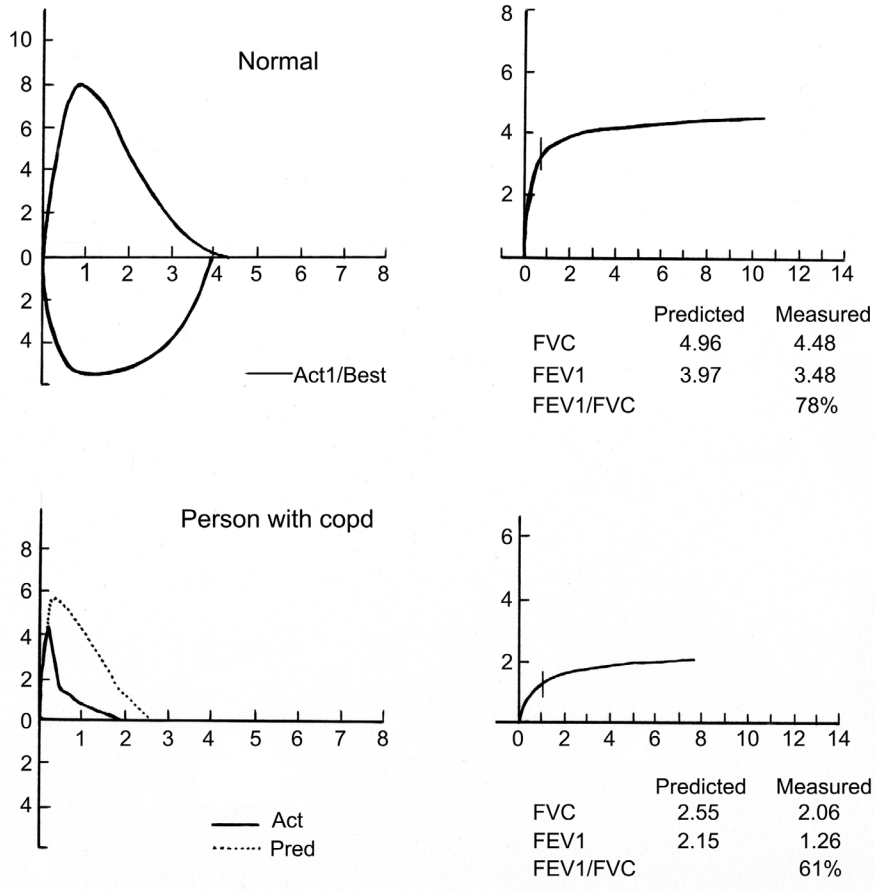


Figure 2 - Normal and person with copd

Natural History

The predominant feature of the natural history of COPD is development and relentless progression of airflow obstruction.

Occupation and COPD

Tobacco smoking is the primary cause; and increasing evidence indicates that occupational and environmental exposures influence the course of this disease. The importance of occupational exposures as a risk factor was observed by Fletcher and other epidemiologists in 1950. In 2002, the American Thoracic Society published a statement that attributes COPD to occupational exposure in 15-20% of cases. Thirty percent of this group were nonsmokers. Normally the rate of lung function declines with advancing age. After the age of 30 the FEV1 declines about 30ml a year in nonsmokers and about 40ml a year in smokers. The data suggests that risk of occupational exposures add an extra 7-8 ml/year (Fig. 3)

Occupational environments are risk factors for COPD. However, this risk is less than that of smoking. Selected occupational agents are listed below.

Gases:

Sulphur dioxide and ammonia

Minerals:

Coal, silica, silicates, asbestos, oil mists, Portland cement and manmade vitreous fibers.

Metals:

Welding fumes, cadmium and vanadium.

Organic dusts:

Cotton, grain dust, wood endotoxins

Smoke:

Internal combustion engine exhaust, environmental tobacco smoke and fine smoke.

Coal:

Coal miners have been more thoroughly investigated than other occupational groups. This is because of the enormous number of workers previously employed in this industry, and the potential influence of their investigations on early recognition of pneumoconiosis as a specific and early

identifiable illness. Most studies have identified a greater effect of coal dust in younger compared to older miners (13).

Other mineral dusts:

Several studies, cross-sectional and longitudinal have shown a relationship between hard rock miners exposed to silica dust and the development of COPD especially in gold miners (14).

Asbestosis:

In animal studies exposure to asbestos causes pulmonary fibrosis of small airways. A number of human studies have been reported that show an excess prevalence of airflow obstruction, but there are no large studies of ventilatory function abnormalities of asbestos-exposed populations, hence the clinical significance of asbestosis is uncertain (15).

Vapors fumes and dusts:

Welding fumes can cause COPD when combined with smoking the effect is additive. In some welders who are non smokers COPD can arise from exposure to welding fumes only (16).

Cadmium

Cadmium is used in production of alloys and other settings. Exposure to cadmium can cause COPD (17).

Organic dusts:

Cotton dust was the first agent to be recognized as a cause of COPD.

Grain and wood dust:

Grain and wood dust is known to cause COPD with an accelerated fall in FEV1 (18).

Tolulene diisocyanate:

Tolulene diisocyanate can cause COPD with an exposure of 2 ppb with a fall in FEV1 of approximately 10 ml/year.

Nuisance dusts:

Nuisance dusts exposure to a wide variety of nuisance dust can cause COPD (19).

Since the American Thoracic Society statement was published there have been a number of studies that support occupational exposures as important risk factors for COPD. In a Swedish longitudinal study of mortality data on 300,000 construction workers, those who were exposed to mineral dust had a higher mortality rate (20). In a third National Health and Nutritional

Examination Survey, a significant risk for COPD was found in workers of the following occupations: manufacturing involving plastic dust, leather, rubber and textiles; manufacturing; food products manufacturing; transportation and trucking; automotive repair; agriculture; construction; office cleaning services; health care and beauty care (). In an Australian study, exposure to biological dust was responsible for increased risk for COPD in women more than men (22). A recent study it was showed that COPD in men who were exposed to occupational fumes had an accelerated loss of lung function of approximately 10 ml/year (23).

Attributing COPD to occupation:

In a worker who has never smoked and who has developed COPD in the absence of other lung disease, workplace exposure to dust is a possible causative factor. The level of exposure determines the degree of risk. Given the variety of agents that have now been reported to cause COPD, it cannot be assumed that occupational exposure to an inhaled substance is free from risk.

References:

1. Deaths by selected grouped causes, sex and geography – Canada: Stats Canada 2007.
2. Lapez A.D; Shibuya K; Rao C; et al Chronic obstructive pulmonary disease : current burden and future projections. Eur Respir J:2006;(2) 397
3. Murray C.J;Lopez A. D; Alternative projection of mortality and disability by cause 1990-2020. Global burden of disease study. Lancet: 1997; 349:1498
4. Respiratory Diseases in Canada. <http://www.phac-aspc.gc.ca/cd-mc/crd-mrc/copd-mpoc-eng.php>
5. American Thoracic Society. Standards for the diagnosis and care of patients with chronic Obstructive Pulmonary Disease: ATS statement. Am.J.Respir Crit Care Med 1995;152: S77
6. Stoller J.K; Aboussouan L.S; et al Alpha-1- antitrypsin deficiency. Lancet 2005;23:2225
7. Sandford A.J; Weir T.D; Pare P. Genetic risk factors for Chronic Obstructive Pulmonary Disease. Eur Respir J 1997;10:1380

8. Davis R.M; Novotry T.E. Epidemiology of cigarette smoking and the impact on Chronic Obstructive Pulmonary Disease Am. Rev. Respir Dis 1989;140: S82
9. Rennard S.I; Vestbo J. COPD: The dangerous underestimation 15%. Lancet 2006; 367:1216
10. Hnizdo E; Sullivan P.A; Bang K.M; Wagner G. Association between COPD and Employment by industry and occupation in the U.S. population: a study of data From the Third National Health and Nutritional Examination Survey. Am J Epidemiol 2002; 156: 738
11. Hendricks D.J Occupation and COPD. Thorax 1996: 51:947
12. Becklake M.R Occupational Exposures : Evidence for a casual association with COPD Am. Rev Respir Dis 1989; 140: S85
13. American Thoracic Society committee on Occupational Contribution to Burden of Airways Disease Am Respir Crit Care Med 2003; 167:787
14. Anto J.M; Vermere P; Vestbo J et al Epidemiology of COPD Eur Respir J 2001; 17: 82
15. Wang J.M; Banks D.M; Airway obstruction and occupational dust exposure. In Banks DE Parker J. E eds Occupational Lung Disease an international prospective. London Chapman and Hall 1988
16. Beach J.R; Dennis J.H; Avery A.J; An epidemiologic investigation of asthma in welders Am. Respir. Crit. Care Med 1996; 154: 1394
17. Davidson A.G; Fuyers P.M; Newman Taylor A.J; et al Cadmium fume inhalation and emphysema. Lancet 1988; 1: 66
18. Glindmyer H.W; Lafante J.J; Jones R.J et al. Cotton dust and across shift cange in FEV1 as a predictor for annual change in FEV1. Am. J. Respir Crit Care Med 1994; 149:584
19. Bakke P. S; Balse V; Houa R; Giusvic A. Prevalence of Obstructive Lung Disease in general Population: relationship to title and exposure to ambient agents. Thorax 1993; 46: 863
20. Bergdahl I.A; Toren K; Ericksson K, et al. Increased mortality in COPD among Construction workers exposed to inorganic dust. Eur Respir J 2004;23:402
21. Trupin I; Earnest G; San Pedro M et al. The occupational burden of Chronic Obstructive Pulmonary Disease Eur Respir J 2003; 22:462

22. Matheson M.C; Benke G; Raven J et al Biological dust exposure to the workplace Is a risk factor for COPD *Thorax* 2005; 60:645
23. Torens K; Balmes J; Chronic Obstructive Pulmonary Disease Does Occupation Matter? *Am Respir Crit Crae Med* 2007; 176: 951