

asbestosis conditions upon reviewing a chest x-ray report obtained for Dr. Glen A. Baker, Jr., an attending Board-certified internist specializing in pulmonary diseases and a B-reader.

By letter dated October 5, 2006, the Office advised the employee that the evidence submitted was insufficient to establish his claim. It addressed the additional factual and medical evidence he needed to submit. The Office requested a rationalized medical report from an attending physician which described his symptoms, results of examination and tests, diagnosis, treatment provided, the effect of treatment and opinion with medical reasons on whether exposure or incidents in the employee's federal employment contributed to his condition. It also requested that the employing establishment respond to the employee's claim and provide exposure data, including air sample surveys or statements of the type of asbestos exposure, frequency, degree and duration for each job held. The Office also requested pertinent dispensary records, including relevant laboratory test results and any chest x-ray reports.

On August 18, 2005 Dr. Baker stated that pulmonary function studies revealed Ghon Complex in the left lung. In an August 22, 2005 report, he reviewed a history of the employee's employment exposure to chemicals, including asbestos, cigarette smoking and family background. On physical examination, Dr. Baker reported essentially normal findings. On x-ray examination of the chest, he reported possible pleural plaque overlying the left seventh rib. Dr. Baker stated that prebronchodilator testing demonstrated a forced vital capacity (FVC) of 68 percent of the predicted amount and forced expiratory volume (FEV₁) of 68 percent of the predicted amount. Post-bronchodilator testing revealed a FVC of 72 percent of the predicted amount and a FEV₁ of 73 percent of the predicted amount. Dr. Baker stated that the FVC and FEV₁ improved by five percent and seven percent respectively. He related that this was an insignificant degree of improvement in the employee's pulmonary function studies. Dr. Baker found that the baseline and post-bronchodilator studies demonstrated a mild restrictive defect. He stated that there was no significant improvement following administration of the bronchodilators.

Dr. Baker diagnosed pleural plaque on the left lung that was probably secondary to asbestos with a long history of asbestos exposure. He also diagnosed mild restrictive defect with no significant improvement following bronchodilators and mild bronchitis. Dr. Baker determined that the employee sustained a Class II impairment, noting that the FEV₁ and/or FVC was between 60 percent and 79 percent of the predicted amount based on Table 5-12 on page 107 of the American Medical Association, *Guides to the Evaluation of Permanent Impairment* (A.M.A., *Guides*) (5th ed. 2001). He stated that, if indeed the employee sustained pleural plaque and pleural asbestosis, then he should have no further exposure to any type of substance that may contain asbestos since his condition could worsen. Dr. Baker recommended that appellant stop smoking as it could increase his risk for lung cancer. He recommended a computerized tomography (CT) scan to rule out the presence of pleural plaque in the left lung or other plaques not seen on a regular chest x-ray. Dr. Baker stated that a chest x-ray demonstrated calcified hilar nodes with calcified areas peripheral to the nodes, which suggested an old Ghon Complex, which suggested the possibility of old tuberculosis.

The employing establishment submitted dispensary records dated August 30, 1954 to June 11, 1987. In a July 5, 2006 report, Dr. Gary W. Daniel, a Board-certified family practitioner, noted that the employee had a history of hearing loss, obesity, cigarette and cigar

smoking, pneumonia, bronchitis, chronic colds and sore throat, pneumothorax, allergies, bilateral knee replacements and prostate cancer.

By letter dated April 3, 2007, the Office referred the employee, together with a statement of accepted facts, the case record and a list of questions to be addressed, to Dr. Kenneth C. Anderson, Board-certified in pulmonary disease, for a second opinion medical examination.

In an April 24, 2007 report, Dr. Anderson noted the employee's symptoms which included daily cough with no mucus and wheezing, dyspnea with exertion when he walked and occasional chest tightness. He reviewed a history of smoking 1 to 1½ packs of cigarettes per day for 15 to 20 years. On physical examination, Dr. Anderson reported moist mucus membranes, a large tongue, midline trachea and narrowing of the lateral pharyngeal sidewalls. Regarding the lungs, he stated that the employee's respirations were not labored with crackles in the bases. Dr. Anderson stated that pulmonary function studies revealed a FVC of 2.98 or 64 percent of the predicted amount, FEV₁ of 2.32 or 68 percent of the predicted amount and FEV₁/FVC of 78. He related that the employee's cough and dyspnea on exertion with pulmonary function testing suggested a restricted ventilatory defect that dated back to 1985 and 1987 when pulmonary function tests were performed at the employing establishment and to Dr. Baker's August 18, 2005 pulmonary function test. Although pulmonary restriction was suggested, the employee's uncorrected diffusion capacity of carbon monoxide was within normal limits. Dr. Anderson noted that no arterial blood gases were performed. Also, no lung volumes were performed due to the employee's inability to bend his right knee to adequately close the door on the pulmonary function test machines to perform a plasmography. Dr. Anderson noted the employee's history of tobacco abuse. He stated that all the pulmonary function tests reviewed did not appear to show significant obstructive lung disease. Dr. Anderson reported an abnormal chest x-ray which demonstrated findings that suggested Category 1 pneumoconiosis in the middle and lower lung fields and pleural plaques bilaterally.

Dr. Anderson found that the employee sustained dyspnea. He opined that the chest x-ray suggested abnormalities that could be consistent with asbestosis, as well as asbestos-related pleural disease. Dr. Anderson advised that the pulmonary function tests had been abnormal since 1985, which suggested a restriction. However, the diffusion capacity of carbon monoxide was normal which did not indicate that the employee sustained any significant asbestosis. Dr. Anderson related that chest x-rays were unreliable in documenting the true extent of asbestos-related pleural disease and recommended a CT scan of the chest with high resolution. He concluded that, if the CT scan was not remarkable, then the employee's restrictive lung defect was not secondary to asbestosis. Dr. Anderson concluded that the true extent of possible asbestos-related pleural disease could be definitely outlined.

On May 23, 2007 Dr. John R. Bies, Board-certified in occupational medicine, performed a chest CT scan. He found a small noncalcified right lower lobe pulmonary nodule measuring 0.7 centimeters (cm) by 0.5 cm. Dr. Bies also found a focal atherosclerotic calcification proximal to the left ascending distal (LAD) coronary artery and mild splenomegaly. He suggested a follow-up CT scan in six weeks to three months to assure stability.

In a June 18, 2007 supplemental report, Dr. Anderson reviewed the May 23, 2007 CT scan. He stated that there were no findings consistent with asbestosis or asbestos-related pleural

disease. Therefore, the employee did not have asbestos or asbestos-related pleural disease. As the pulmonary function tests had been relatively stable since 1985 with normal carbon monoxide diffusion capacity, Dr. Anderson was not convinced that a significant pulmonary impairment was present. He noted that the CT scan identified two abnormalities: a five millimeter (mm) by seven mm noncalcified nodule in the right lower lobe and calcification of the proximal LAD coronary artery. However, these abnormalities were not related to the employee's federal employment. Although the employee had decreased FVC and FEV₁ values which appeared to be restrictive in nature, a total lung capacity evaluation could not be performed due to the inability to close the door on the plethysmograph. Dr. Anderson opined that, based on the normal CT scan of the chest and normal diffusion capacity of carbon monoxide, the employee did not have any significant asbestosis or asbestos-related pleural disease. He found that the employee did not sustain a pulmonary impairment causally related to his federal employment.

By decision dated July 6, 2007, the Office denied the employee's claim. It found that Dr. Anderson's April 24, 2007 report constituted the weight of the medical opinion evidence and established that the claimed lung condition was not caused by the employee's accepted work-related asbestos exposure.

By letter dated July 24, 2007, the employee, through counsel, requested an oral hearing before an Office hearing representative. On December 26, 2007 Dr. Matthew A. Vuskovich, Board-certified in occupational medicine, reviewed the May 23, 2007 CT scan. He found that upper lung appearances consistent with simple coal workers' pneumoconiosis. Dr. Vuskovich stated that in contrast to the ILO (International Classification of Radiographs of Pneumoconiosis) classification system for posterior-anterior x-ray images, a CT scan standardized classification system had not been developed. He found that it was not possible to classify the demonstrated lung appearances.

By decision dated May 13, 2008, an Office hearing representative affirmed the July 6, 2007 decision. The hearing representative found that the employee's lung condition was not causally related to his accepted exposure to asbestos based on Dr. Anderson's April 24, 2007 report.

LEGAL PRECEDENT

An employee seeking benefits under the Federal Employees' Compensation Act² has the burden of establishing the essential elements of his claim including the fact that the individual is an "employee of the United States" within the meaning of the Act, that the claim was timely filed within the applicable time limitation period of the Act, that an injury was sustained in the performance of duty as alleged and that any disability and/or specific condition for which compensation is claimed are causally related to the employment injury.³ These are the essential

² 5 U.S.C. §§ 8101-8193.

³ *Elaine Pendleton*, 40 ECAB 1143, 1145 (1989).

elements of each compensation claim regardless of whether the claim is predicated upon a traumatic injury or an occupational disease.⁴

To establish that an injury was sustained in the performance of duty in an occupational disease claim, a claimant must submit the following: (1) medical evidence establishing the presence or existence of the disease or condition for which compensation is claimed; (2) a factual statement identifying employment factors alleged to have caused or contributed to the presence or occurrence of the disease or condition; and (3) medical evidence establishing that the employment factors identified by the claimant were the proximate cause of the condition for which compensation is claimed or, stated differently, medical evidence establishing that the diagnosed condition is causally related to the employment factors identified by the claimant. The medical evidence required to establish a causal relationship is rationalized medical opinion evidence. Rationalized medical opinion evidence is medical evidence, which includes a physician's rationalized opinion on the issue of whether there is a causal relationship between the claimant's diagnosed condition and the implicated employment factors. The opinion of the physician must be based on a complete factual and medical background of the claimant, must be one of reasonable medical certainty and must be supported by medical rationale explaining the nature of the relationship between the diagnosed condition and the specific employment factors identified by the claimant.⁵ Neither the fact that appellant's condition became apparent during a period of employment nor her belief that the condition was caused by his employment is sufficient to establish a causal relationship.⁶

ANALYSIS

The Board finds that appellant has failed to establish a causal relationship between the employee's pulmonary condition and his accepted employment-related asbestos exposure.

The employing establishment's dispensary records covering the period August 30, 1954 to June 11, 1987 and Dr. Daniel's July 5, 2006 report note that the employee had a history of hearing loss, obesity, cigarette and cigar smoking, pneumonia, bronchitis, chronic colds and sore throat, pneumothorax, allergies, bilateral knee replacements and prostate cancer. Neither the dispensary records nor Dr. Daniel addressed whether the diagnosed conditions were caused by the employee's accepted asbestos exposure. The Board finds that this evidence is insufficient to establish appellant's burden of proof.

Dr. Baker's August 22, 2005 report stated that the employee had a Ghon Complex in the left lung "probably" secondary to a long history of work-related asbestos exposure and mild restrictive defect. He determined that the employee sustained a Class II impairment and bronchitis based on pulmonary function studies results (A.M.A., *Guides* 107, Table 5-12). The Board finds that Dr. Baker's opinion regarding the causal relationship between the employee's pulmonary condition and his asbestos exposure is speculative and equivocal in nature and of

⁴ See *Delores C. Ellyett*, 41 ECAB 992, 994 (1990); *Ruthie M. Evans*, 41 ECAB 416, 423-25 (1990).

⁵ *Victor J. Woodhams*, 41 ECAB 345, 351-52 (1989).

⁶ *Kathryn Haggerty*, 45 ECAB 383, 389 (1994).

diminish probative value.⁷ Dr. Baker noted that additional diagnostic testing was required to rule out the presence of a pleural plaque. He did not provide adequate medical rationale in support of his opinion on counsel reaction. Dr. Baker did not sufficiently explain how the employee's employment-related asbestos exposure caused or contributed to a definite pulmonary condition. The Board finds that the report of Dr. Baker is insufficient to establish that the employee sustained a pulmonary condition causally related to the accepted employment factor.

In an April 24, 2007 report, Dr. Anderson, an Office referral physician, reviewed the history of the employee's work-related asbestos exposure. He provided his findings on physical examination which included moist mucus membranes, a large tongue, midline trachea and narrowing of the lateral pharyngeal sidewalls. Regarding the lungs, Dr. Anderson noted that the employee's respirations were not labored with crackles in the bases. He stated that pulmonary function studies revealed demonstrated a FVC of 2.98 or 64 percent of the predicted amount, FEV₁ of 2.32 or 68 percent of the predicted amount and FEV₁/FVC of 78. Dr. Anderson related that the employee's cough and dyspnea on exertion on pulmonary function testing suggested a restricted ventilatory defect that dated back to the employing establishment's 1985 and 1987 pulmonary function tests and to Dr. Baker's August 18, 2005 pulmonary function test. However, the employee's uncorrected diffusion capacity of carbon monoxide was within normal limits. He noted that no arterial blood gases and lung volumes were performed due to the employee's inability to bend his right knee to adequately close the door on the pulmonary function test machines to perform a plasmography. Noting the employee's tobacco abuse, Dr. Anderson stated that all the reviewed pulmonary function tests did not appear to show significant obstructive lung disease. He diagnosed Category 1 pneumoconiosis in the middle and lower lung fields pleural plaques bilaterally based on a chest x-ray. Dr. Anderson did not state that the diagnosed condition was caused by the employee's work-related asbestos exposure.

Dr. Anderson opined that the employee sustained dyspnea. He stated that the chest x-ray suggested abnormalities that could be consistent with asbestosis, as well as, asbestos-related pleural disease. Dr. Anderson stated that the pulmonary function tests had been abnormal since 1985 which suggested a restriction. He recommended a chest CT scan as chest x-rays were unreliable in documenting the true extent of asbestos-related pleural disease.

In a June 18, 2007 supplemental report, Dr. Anderson opined that the employee did not sustain asbestosis or asbestos-related pleural disease based on the May 23, 2007 CT scan and normal diffusion capacity for carbon monoxide. He was not convinced that any significant pulmonary impairment was present due to the normal carbon monoxide diffusion capacity. Dr. Anderson opined that the five mm by seven mm noncalcified nodule in the right lower lobe and calcification of the proximal LAD coronary artery were abnormalities not causally related to the employee's employment. Although the employee had decreased FVC and FEV₁ values, which appeared to be restrictive in nature, a total lung capacity evaluation could not be performed due to the inability to close the door on the plethysmograph. Dr. Anderson concluded that the employee did not sustain a pulmonary impairment causally related to his federal employment.

⁷ *L.R. (E.R.)*, 58 ECAB ____ (Docket No. 06-1942, issued February 20, 2007); *D.D.*, 57 ECAB 734 (2006); *Cecelia M. Corley*, 56 ECAB 662 (2005).

Similarly, Dr. Vuskovich's December 26, 2007 report is insufficient to establish appellant's claim. He stated that the May 23, 2007 CT scan demonstrated upper lung appearances consistent with simple coal workers' pneumoconiosis, although it was not possible to classify the demonstrated lung appearances in the absence of a CT scan standardized classification system. Dr. Vuskovich did not address whether the employee's condition was caused by the accepted work-related asbestos exposure. The Board finds that his report is insufficient to meet appellant's burden of proof.

The Board finds that Dr. Anderson's opinion is sufficiently well rationalized and based upon a proper factual background such that it is the weight of the evidence on the issue of whether the employee sustained a lung condition causally related to his federal employment. The Board has noted that in assessing medical evidence the weight of such evidence is determined by its reliability, its probative value and its convincing quality and the factors which enter in such an evaluation include the opportunity for and thoroughness of examination, the accuracy and completeness of the physician's knowledge of the facts and medical history, the care of the analysis manifested and the medical rationale expressed in support of the physician's opinion.⁸ Dr. Anderson's opinion is of such reliability, probative value and convincing quality. He extensively detailed the employee's factual and medical history and reported the findings based on his review of the employee's medical records and diagnostic testing. Dr. Anderson provided a proper analysis of the factual and medical history and objective test of record. His conclusions regarding the employee's condition are based on a thorough review of the medical evidence.

The Board finds that appellant has not established that the employee's lung condition was causally related to his asbestos exposure. Appellant did not meet her burden of proof.

CONCLUSION

The Board finds that appellant has failed to establish that the employee sustained a pulmonary condition in the performance of duty.

⁸ See *Ann C. Leanza*, 48 ECAB 115 (1996).

ORDER

IT IS HEREBY ORDERED THAT the May 13, 2008 decision of the Office of Workers' Compensation Programs is affirmed.

Issued: July 15, 2009
Washington, DC

Alec J. Koromilas, Chief Judge
Employees' Compensation Appeals Board

Michael E. Groom, Alternate Judge
Employees' Compensation Appeals Board

James A. Haynes, Alternate Judge
Employees' Compensation Appeals Board