

FACTUAL HISTORY

On January 28, 2013 appellant, then a 64-year-old mine safety and health inspector, filed an occupational disease claim alleging that he developed occupational pneumoconiosis, chronic obstructive pulmonary disease (COPD), and chronic bronchitis due to exposures during his federal employment. He stated that he first became aware of his condition on October 9, 2012 and first attributed his condition to his employment on that date.

Appellant stated that he worked for the employing establishment from June 16, 2000 until July 29, 2011. He inspected underground lead mines for approximately four years with exposures to gases, diesel fumes, dust, and chemicals. Appellant stated that he had dust exposure on a regular basis with accumulations on his skin and clothing. He also inspected underground limestone mines and surface limestone quarries with exposures to gases, diesel fumes, radon, and rock dust. Appellant experienced dust on his skin and clothing with coughing and nasal production. He did not wear a mask. Appellant reported his private sector employment and dust exposures. He stated that he had shortness of breath for several years and smoked cigarettes at the rate of one pack per day for 42 years. Appellant stopped smoking in September 2006. His left lung was removed in 2006 due to cancer and he developed pneumonia in 2010.

Dr. Glen Baker, a Board-certified pulmonologist and certified B-reader examined appellant on November 20, 2012 and submitted a report dated December 5, 2012. He noted appellant's federal and private-sector work exposures as well as his history of smoking. Dr. Baker stated that appellant underwent a left pneumonectomy in 2006 and that his medical history was positive for lung cancer. He reviewed a September 17, 2012 chest x-ray and found parenchymal abnormalities consistent with pneumoconiosis including small opacities category 1/2. He did not find any pleural abnormalities consistent with pneumoconiosis. Dr. Baker found that appellant's forced vital capacity (FVC) was 64 percent of predicted and that his forced expiratory volume (FEV₁) was 39 percent of predicted. He found severe obstructive defect with a mild degree of restriction. Post-bronchodilator values were FVC of 70 percent and FEV₁ of 43 percent of predicted. Dr. Baker found moderate obstructive ventilator defect with a mild degree of restriction. He diagnosed occupational pneumoconiosis, category 1/2, COPD with a severe obstructive defect on pre-bronchodilator values, chronic bronchitis, and obstructive sleep apnea. Dr. Baker stated:

“[Appellant] has a long history of dust exposure in the extraction of copper, lead, zinc[,] and limestone. He has x-ray changes of occupational pneumoconiosis, which is due to a combination of the rock dust and various mineral dusts he was exposed to during his period of employment. [Appellant] also has severe obstructive airway disease and a long history of cigarette smoking. The combination of cigarette smoke and rock dust can be either synergistic or additive in terms of the effects on the lungs according to the medical literature. Thus [appellant's] condition would be worse than if he had never smoked or never worked in the mining industry. His symptoms are due to his occupational exposure to various metals and his smoking history.”

Dr. Baker provided an impairment rating of 65 percent under the American Medical Association, *Guides to the Evaluation of Permanent Impairment*.²

The employing establishment submitted a statement dated February 7, 2013 and acknowledged that appellant worked from June 16, 2000 to July 28, 2011 as a mine inspector. It stated that he was exposed to dusts, gases, and fumes from rocks, mobile equipment, drills, channel burners, excavators, milling, and rock crushing facilities. The employing establishment stated that it encouraged the use of respirators and had provided respirators along with annual fit testing for respirators for inspectors. It stated that mine inspection work rarely exposed an inspector to high levels of dust or gases for any length of time with an average of six hours a day five days a week at a mine site.

In a letter dated April 2, 2013, OWCP requested additional factual and medical evidence in support of appellant's claim and provided him with a questionnaire. It referred appellant for a second opinion evaluation with Dr. Harold Dale Haller, Jr, a Board-certified pulmonologist, who completed a report on June 19, 2013 and reviewed the statement of accepted facts. Dr. Haller diagnosed severe COPD, history of lung cancer, and obstructive sleep apnea. He stated that he could not confirm or refute appellant's diagnosis of occupational pneumoconiosis. Dr. Haller attributed appellant's COPD to his 42-year smoking history. He further stated, "It is well known that any significant dust exposure can cause COPD alone, or in combination with cigarette smoking and can accelerate the rate of decline of lung function. So, it is entirely possible that his exposure at work did potentially accelerate the rate of decline of his COPD." In regard to pneumoconiosis, Dr. Haller found marked hyperinflation on his chest radiograph. He stated, "I do not see definite interstitial changes consistent with pneumoconiosis."

Dr. Kenneth Anderson, a colleague of Dr. Haller, a Board-certified pulmonologist and B-reader, examined appellant's June 19, 2013 studies on July 23, 2013 and found parenchymal abnormalities consistent with pneumoconiosis including small opacities with a profusion of 1/0. He found no pleural abnormalities consistent with pneumoconiosis. Dr. Haller reviewed this report on September 6, 2013 and stated that it did not change his previously stated opinion.

By decision dated November 22, 2013, OWCP denied appellant's occupational disease claim finding that he had not submitted the necessary medical opinion evidence to establish a causal relationship between his diagnosed condition and his employment exposures. It found that Dr. Haller's opinion that it was "possible that exposure at work accelerated the COPD" was speculative and that Dr. Anderson did not find any abnormalities consistent with pneumoconiosis. Counsel requested an oral hearing from an OWCP hearing representative on December 3, 2013.

Dr. Matthew A. Vuskovich, appellant's physician Board-certified in occupational medicine and a B-reader reviewed a September 17, 2012 x-ray and found parenchymal abnormalities consistent with pneumoconiosis specifically small opacities 2/2, but no pleural abnormalities consistent with pneumoconiosis.

² A.M.A., *Guides*, 6th ed. 2009.

Appellant testified at the oral hearing before an OWCP hearing representative on June 9, 2014. He described his employment duties of inspecting lead and copper mines. Appellant stated that he was exposed to diesel fumes, rock, and ore dust as well as gases. He noted that he also inspected limestone quarries and mines with exposures to rock dust. Appellant stated that he wore a respirator in areas that had already received citations for dust or gas, but did not normally wear a mask.

Dr. Baker submitted an additional report on June 23, 2014. He reviewed appellant's federal and nonfederal employment exposures. Dr. Baker stated, "The various dust, odors, fumes, and chemicals that he would have been exposed to have all played a part in his pulmonary condition. They have affected his lung function as well as the appearance of his chest x-ray. The sum total of his dust exposures have all contributed to his occupational pneumoconiosis, chronic obstructive airway disease, chronic bronchitis and possibly to the development of lung cancer as well with exposure to radon and lead." He opined, "I think the sum total of his dust exposure has caused his current condition in concert with his smoking history. The medical literature suggests that when both exposures are present, the effects on the lungs may be either synergistic or additive."

By decision dated August 25, 2014, the hearing representative found that there was no rationalized medical evidence in support of appellant's claim and that the weight of the medical evidence rested with Dr. Haller's reports.

LEGAL PRECEDENT

OWCP's regulations define an occupational disease as "a condition produced by the work environment over a period longer than a single workday or shift."³ 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 evidence required to establish causal relationship is rationalized medical opinion evidence, based upon a complete factual and medical background, showing a causal relationship between the claimed condition and identified factors. The belief of a claimant that a condition was caused or aggravated by the employment is not sufficient to establish causal relation.⁴

ANALYSIS

Appellant submitted factual and medical evidence in support of his claim for pulmonary disease causally related to his exposures to dust, gases, and fumes. Dr. Baker reviewed a September 17, 2012 chest x-ray and found parenchymal abnormalities consistent with

³ 20 C.F.R. § 10.5(q).

⁴ *Lourdes Harris*, 45 ECAB 545, 547 (1994).

pneumoconiosis including small opacities category 1/2. He did not find any pleural abnormalities consistent with pneumoconiosis. Dr. Baker diagnosed occupational pneumoconiosis, category 1/2, COPD with a severe obstructive defect on pre-bronchodilator values, chronic bronchitis, and obstructive sleep apnea. He described appellant's history of employment exposures and smoking cigarettes. Dr. Baker opined that appellant's pneumoconiosis was due to a combination of the rock dust and various mineral dusts he was exposed to during his period of employment. He stated that the combination of cigarette smoke and rock dust could be either synergistic or additive in terms of the effects on the lungs according to the medical literature. Dr. Baker concluded that appellant's symptoms are due to his occupational exposure to various metals and his smoking history.

OWCP referred appellant for a second opinion evaluation with Dr. Haller, who diagnosed severe COPD, history of lung cancer, and obstructive sleep apnea. Dr. Haller stated that he could not confirm or refute appellant's diagnosis of occupational pneumoconiosis. He attributed appellant's chronic obstructive pulmonary disease to his 42-year smoking history. Dr. Haller further stated, "It is well known that any significant dust exposure can cause COPD alone, or in combination with cigarette smoking and can accelerate the rate of decline of lung function. So, it is entirely possible that his exposure at work did potentially accelerate the rate of decline of his COPD."

The Board finds that this case is not in posture for a decision. Proceedings before OWCP are not adversarial in nature and OWCP is not a disinterested arbiter; in a case where OWCP "proceeds to develop the evidence and to procure medical evidence, it must do so in a fair and impartial manner."⁵ OWCP referred appellant to Dr. Haller for a second opinion evaluation. Dr. Haller made findings consistent with Dr. Baker noting that it was entirely possible that appellant's exposure at work did potentially accelerate the rate of decline of his COPD. Dr. Anderson reviewed appellant's June 19, 2013 x-ray studies on July 23, 2013 and, like Dr. Baker, found parenchymal abnormalities consistent with pneumoconiosis including small opacities. Like Dr. Baker he also found no pleural abnormalities consistent with pneumoconiosis. Dr. Haller reviewed this report on September 6, 2013 and stated that it did not change his previously stated opinion. The Board finds that Dr. Haller did not offer a clear opinion as to whether appellant's diagnosed COPD was aggravated or accelerated by his employment exposures. Once OWCP undertakes development of the record, it must do a complete job in procuring medical evidence that will resolve the relevant issues in the case.⁶ Dr. Haller also failed to explain why Dr. Anderson's findings of parenchymal abnormalities consistent with pneumoconiosis including small opacities did not support a diagnosis of pneumoconiosis. The Board finds that OWCP is obligated to request a supplemental report from him addressing these issues. After this and such other development as OWCP deems necessary, OWCP should issue a *de novo* decision.

⁵ *Walter A. Fundinger, Jr.*, 37 ECAB 200, 204 (1985).

⁶ *Richard F. Williams*, 55 ECAB 343, 346 (2004).

CONCLUSION

The Board finds that the case is not in posture for a decision as OWCP undertook development of the medical evidence and failed to complete the development by securing a detailed and well-reasoned report from the second opinion physician, Dr. Haller, as to whether appellant's pulmonary disease developed due to factors of his federal employment.

ORDER

IT IS HEREBY ORDERED THAT the August 25, 2014 decision of the Office of Workers' Compensation Programs is set aside and remanded for further development consistent with this decision of the Board.

Issued: January 21, 2015
Washington, DC

Christopher J. Godfrey, Chief Judge
Employees' Compensation Appeals Board

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

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