

condition and realized that it was causally related to his employment on July 11, 2008. Appellant retired on or about June 1, 2008.²

Appellant submitted an accident report dated November 30, 1979, noting that he was filling and charging batteries and inhaled battery acid fumes which caused him to experience chest pains. He submitted spirometry tests for January 28, 1986 to September 14, 1994 and an August 1, 1995 pulmonary function test which did not provide an interpretation. A March 2, 2009 pulmonary function report revealed mild restrictive ventilator defect and superimposed early obstructive pulmonary impairment. Also submitted was a summary of appellant's military service with the National Guard from 1966 to 2008.

On June 29, 2009 OWCP advised appellant of the type of evidence needed to establish his claim. It particularly requested that he submit a physician's reasoned opinion addressing the relationship of his claimed condition and specific work factors.

In a July 16, 2009 statement, appellant noted working in the Combined Support Maintenance Shop in 1969 where he sanded old paint, cleaned with lacquer and inhaled lacquer, enamel paint fumes and paint dust, five days a week for two years. He was provided with a mask. Thereafter, appellant worked as a mechanic for 12 years and replaced brake shoes and repaired asbestos brake systems on trucks, tanks and construction equipment and serviced lead acid batteries. He was exposed to dust from the brake shoes, diesel smoke and carbon monoxide and inhaled battery acid fumes and lead fumes. Appellant noted that the ventilation system was poor. In 1983, he worked as an automotive worker until his retirement in 2008 and was responsible for maintaining automobiles and was exposed to diesel fumes, brake dust, gasoline exhaust, paint dust and cleaning chemicals.

Appellant submitted pulmonary function tests dated May 26, 1998 to April 14, 2005 which revealed early-stage mild obstructive deficiency. On August 24, 2009 he was treated by Dr. Robert W. Boyd, II, an osteopath, for a hearing and pulmonary condition. Appellant reported being exposed to chemicals and equipment with aerosolized irritants and he also had a history of smoking. Dr. Boyd noted that a recent evaluation revealed underlying obstructive lung disease; however, he noted that it was difficult to determine how much smoking affected the underlying etiology for his lung disease compared to exposure to irritants. He noted that appellant continued to be exposed to chemicals and that it was feasible that it could have caused or aggravated his underlying lung difficulties. Dr. Boyd noted not having any information on the types of chemicals and irritants to determine the extent that appellant's condition could have been caused by the irritants. On October 20, 2009 he treated appellant for hearing and breathing issues. Dr. Boyd noted that appellant smoked and was exposed to aerosolized chemicals that can aggravate obstructive pulmonary disease. He noted that it was difficult to ascertain the true etiology of appellant's condition but opined that it was most likely multifactorial and it would be difficult to say how much the work exposure added to the previous smoking history.

² Appellant filed a traumatic injury claim on November 9, 1979, for chest pains which occurred after he inhaled battery acid fumes, claim number xxxxxx001. This claim is not presently before the Board.

On October 26, 2009 OWCP denied appellant's claim on the grounds that the medical evidence did not demonstrate that the claimed medical condition was related to the work-related events.

Appellant requested reconsideration and submitted an April 22, 2010 report from Dr. Nicolette Myers, a Board-certified pulmonologist. He reported smoking one pack of cigarettes per day from 1966 to 1991 and being exposed to fumes from lacquer paints, battery acid fumes, solvents and asbestos. Appellant noted being diagnosed with chronic obstructive pulmonary disease and indicated that when he stopped smoking his coughing ceased. On examination, he had regular heart rate and rhythm, no wheezes or crackles and normal chest excursions. Dr. Myers noted that a chest computerized tomography (CT) scan revealed no evidence of bronchiectasis, minimal septal thickening in the anterior lingual and right middle lobe and several tiny nodules. He noted that appellant had multiple exposures to inhaled paint fumes, battery acid fumes and asbestos at work from 1960 to 1980 and smoked cigarettes during this time. Dr. Myers opined that it was possible that fumes contributed to appellant's moderate airflow obstruction and his chronic dyspnea. She further opined that appellant's smoking could have led to a worsening of his underlying chronic obstructive pulmonary disease. Dr. Myers noted that appellant's obesity contributed to his underlying dyspnea. Appellant submitted CT scans of the chest dated December 9, 2004 to July 21, 2010, which revealed stable tiny nodules and nonmass-like opacities in both lungs and no acute abnormality.

On October 4, 2010 OWCP referred appellant to Dr. Thomas F. Mulrooney, a Board-certified pulmonologist. In a November 17, 2010 report, Dr. Mulrooney noted reviewing the records provided and examining appellant. He noted appellant's history of smoking one pack of cigarettes per day from 1967 to 1992 and exposure to asbestos on brakes, diesel fumes, carbon monoxide and fumes from batteries. Appellant had normal heart rhythm with no murmur or gallop. Diaphragms moved symmetrically, lungs were clear, the pulmonary function measurements of lung volumes and mechanics were normal and there was no change after bronchodilator. The forced expiratory volume and forced vital capacity (FVC) ratio was slightly decreased (67 percent of predicted) with a significant reduction (55 percent of predicted) in mid and late expiratory flow rates. Dr. Mulrooney noted that the values reported for lung volumes except for vital capacity were disproportionate and represented a technical error in the pulmonary function laboratory. The results suggested very mild, borderline air flow obstruction. Dr. Mulrooney diagnosed minimal chronic obstructive pulmonary disease, probable obstructive sleep apnea and cardiomyopathy. He advised that complaints of exertional shortness of breath with fatigue may be related to chronic airflow obstruction that resulted from long and rather heavy tobacco use. Dr. Mulrooney opined that the abnormality in the pulmonary function measurements, a reduction in late expiratory flow rates, was considered an inflammation in the small airways often found in cigarette smokers. He stated that appellant's fatigue while exercising was difficult to attribute to any measured abnormality in the lung function. Dr. Mulrooney noted that appellant was obese and middle aged, with a long history of cigarette smoking, and that the consideration of coronary artery disease was a possible cause of his symptoms. He stated that appellant's obesity and small oropharyngeal space indicated a high probability of sleep apnea. Dr. Mulrooney opined that the mild abnormality in appellant's lung function and his mild obstructive lung disease were fully explained by his history of smoking a pack a day for 25 years. He noted that the stability of the pulmonary function evaluation provided no convincing evidence of progression of lung disease. Dr. Mulrooney concluded that

appellant's minimal decrease in pulmonary function was not attributable to his federal employment.

In a decision dated December 8, 2010, OWCP denied modification of the decision dated December 8, 2010.

On June 23, 2011 appellant requested reconsideration. He submitted a June 23, 2011 report from Dr. Myers who noted that appellant had multiple chemical exposures while working for the Federal Government and that his shortness of breath may have been in part due to exposure to these solvents and fumes. Dr. Myers noted that the pulmonary function tests demonstrated a mild-to-moderate ventilator defect with an FVC of 76 percent of predicted. She indicated that appellant had exposures to inhaled paint fumes, battery acids and asbestos at work from 1960 to 1980 and also smoked during this time. Dr. Myers opined that it was difficult to demonstrate causality because appellant was smoking at the same time; however, he had significant airflow obstruction by 1999 which suggested that the solvents did cause harm to his lungs in addition to tobacco use.

In a decision dated October 20, 2011, OWCP denied modification of the prior decision.

LEGAL PRECEDENT

An employee seeking benefits under FECA has the burden of proof to establish the essential elements of his claim. When an employee claims that she sustained an injury in the performance of duty, she must submit sufficient evidence to establish that she experienced a specific event, incident or exposure occurring at the time, place and in the manner alleged. Appellant must also establish that such event, incident or exposure caused an injury.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) 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 causal relationship is generally 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

³ See *Walter D. Morehead*, 31 ECAB 188, 194 (1979) (occupational disease or illness); *Max Haber*, 19 ECAB 243, 247 (1967) (traumatic injury). See generally *John J. Carlone*, 41 ECAB 354 (1989); *Elaine Pendleton*, 40 ECAB 1143 (1989).

nature of the relationship between the diagnosed condition and the specific employment factors identified by the claimant.⁴

ANALYSIS

It is not disputed that appellant's duties as a surface maintenance mechanic included exposure to fumes, chemicals and solvents while performing his duties. It is also not disputed that he has been diagnosed with chronic obstructive pulmonary disease. However, appellant has not submitted sufficient medical evidence to establish that his pulmonary condition including chronic obstructive pulmonary disease was causally related to specific employment factors or conditions.

The August 24, 2009 report from Dr. Boyd noted obstructive lung disease but the etiology of appellant's lung disease was difficult to determine because he was also a heavy cigarette smoker. He noted that it was feasible that appellant's chemical exposure, if not the cause of his underlying lung difficulties, could have aggravated his condition. On October 20, 2009 Dr. Boyd again noted the difficulty to evaluate the underlying etiology of appellant's lung condition but opined that it was most likely multifactorial and opined that appellant's medical conditions could have been aggravated by the work environment. The Board notes that Dr. Boyd's report provides some support for causal relationship but is insufficient to establish the claimed pulmonary condition was causally related to his employment duties. His reports, at best, provide speculative support for causal relationship since he notes that it was difficult to determine the etiology of appellant's conditions and that it was "most likely" multifactorial and that appellant's medical condition "could be" aggravated by the work environment.⁵ Dr. Boyd provided no medical reasoning explaining how particular workplace substances caused or aggravated a diagnosed condition. The need for medical rationale is particularly important in view of appellant's long history of cigarette smoking which the physician also indicated could have been a cause of appellant's condition. Therefore, this report is insufficient to meet appellant's burden of proof.

Similarly, on April 22, 2010, Dr. Myers noted that it was possible that exposure to fumes and solvents contributed to appellant's moderate airflow obstruction and his chronic dyspnea. She further opined that appellant's smoking during this time could also have lead to a worsening of his underlying chronic obstructive pulmonary disease and that obesity could have contributed to his underlying dyspnea. On June 23, 2011 Dr. Myers again found that appellant's shortness of breath may be due in part to exposure to solvents and fumes while working. She opined that it was difficult to demonstrate causality because appellant smoked at the same time but that he had significant airflow obstruction by 1999 which suggested that the solvents did cause harm to his lungs on top of tobacco use. Although, Dr. Myers supported causal relationship, she did not provide medical rationale explaining the basis of his conclusion regarding the causal relationship

⁴ *Solomon Polen*, 51 ECAB 341 (2000).

⁵ Medical opinions that are speculative or equivocal in character are of diminished probative value. *D.D.*, 57 ECAB 734 (2006).

between appellant's pulmonary disease and work factors.⁶ She did not explain the process by which exposure to solvents and fumes would cause or aggravate the diagnosed chronic obstructive pulmonary disease and why such condition would not be due to any nonwork factors such as smoking and obesity. Therefore, these reports are insufficient to meet appellant's burden of proof.

OWCP referred appellant to Dr. Mulrooney who, in a November 17, 2010 report, noted appellant's tobacco history and his workplace exposures. Dr. Mulrooney diagnosed chronic obstructive pulmonary disease, minimal, probable obstructive sleep apnea and cardiomyopathy. Pulmonary function measurements of lung volumes and mechanics were normal while there was a very mild, borderline air flow obstruction. Dr. Mulrooney noted appellant's complaints of exertional shortness of breath with fatigue could have been related to chronic airflow obstruction which resulted from his long and rather heavy use of tobacco. He explained that appellant's abnormality in the pulmonary function measurements was considered an inflammation in the small airways often found in cigarette smokers. Dr. Mulrooney noted appellant's symptoms of fatigue during exercise was difficult to attribute to the measured abnormality in the lung function but, in view of his obesity and cigarette smoking history, coronary artery disease was a likely possibility. He opined that the mild abnormality in appellant's lung function and his diagnoses of mild obstructive lung disease was explained by his history of smoking a pack a day for 25 years. Dr. Mulrooney concluded that the minimal decrease in pulmonary function identified in appellant was not attributable to his federal employment. He found no basis on which to attribute any diagnosis to factors of appellant's employment.

The Board finds that the medical evidence does not establish that appellant has a pulmonary condition causally related to his employment. An award of compensation may not be based on surmise, conjecture or speculation. Neither the fact that appellant's condition became apparent during a period of employment, nor the belief that his condition was caused, precipitated or aggravated by his employment, is sufficient to establish causal relationship.⁷ Causal relationships must be established by rationalized medical opinion evidence. As noted the medical evidence is insufficient to establish appellant's claim. Consequently, OWCP properly found that appellant did not meet his burden of proof to establish his claim.

On appeal, appellant asserts that his pulmonary condition was caused by his workplace exposure to chemicals and insolvents. As noted above, the Board has found that allegation to be insufficiently rationalized. Reports from appellant's physician's failed to provide sufficient medical rationale explaining how appellant's pulmonary condition was causally related to particular employment exposures and why his condition was not related to nonwork-related conditions such as his history of tobacco use and obesity.

Appellant may submit new evidence or argument with a written request for reconsideration to OWCP within one year of this merit decision, pursuant to 5 U.S.C. § 8128(a) and 20 C.F.R. §§ 10.605 through 10.607.

⁶ *Franklin D. Haislah*, 52 ECAB 457 (2001) (medical reports not containing rationale on causal relationship are entitled to little probative value); *Jimmie H. Duckett*, 52 ECAB 332 (2001).

⁷ *See Dennis M. Mascarenas*, 49 ECAB 215 (1997).

CONCLUSION

The Board finds that appellant did not meet his burden of proof to establish that his claimed conditions were causally related to his employment.

ORDER

IT IS HEREBY ORDERED THAT the October 20, 2011 decision of the Office of Workers' Compensation Programs is affirmed.

Issued: July 26, 2012
Washington, DC

Colleen Duffy Kiko, Judge
Employees' Compensation Appeals Board

Patricia Howard Fitzgerald, Judge
Employees' Compensation Appeals Board

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