

FACTUAL HISTORY

Appellant filed a claim for death benefits on October 18, 2001 due to the death of her husband, a mine-safety inspector. The employee died on May 16, 1998.¹ Appellant submitted a copy of the employee's death certificate in which Dr. Stephen C. Ulrich, a Board-certified family practitioner, stated that the cause of death was due to pulmonary failure as consequence of a stroke and multi-infarct dementia.

Appellant submitted several medical reports in support of her claim. In a November 6, 1998 report, Dr. Ulrich stated that the employee had pneumoconiosis. He noted that the employee had developed shortness of breath but did not like doctors and medical tests. Dr. Ulrich reported that the employee had strokes on November 21, 1995 and on April 23, 1998. The employee developed aspiration pneumonia in the hospital which resolved. The employee was sent home but developed recurrent pneumonia and died at home.

In an August 25, 1998 report, Dr. Katherine Tabatowski, a Board-certified pathologist, stated that an autopsy showed that the employee had black reticulation and macular pigment deposition on sectioning of the lungs. The pleural surfaces were stiff. Scattered tiny nodular areas were present with a five millimeter calcified black nodule in the right lower lobe. Dr. Tabatowski indicated that a microscopic view of sections of both lower lobes and the right middle lobe revealed areas of acute pneumonia in which alveolar spaces were filled by neutrophils, accompanied by congestion and hemorrhage. She found scattered deposits of anthracotic pigment throughout the lung sections and in the pleura. Dr. Tabatowski noted that some of the deposits were associated with a slight degree of fibrosis. She reported the sections of the right lower lobe showed fibrotic and calcified granulomas. Dr. Tabatowski commented that the hilar lymph nodes showed fibrotic and calcified granuloma and extensive sinusoidal anthracotic pigment deposits. Based on the autopsy, she diagnosed dust macules with associated centroacinar emphysema, consistent with coal workers' pneumoconiosis; acute bronchopneumonia in both lower lobes and the right middle lobe; calcified and fibrocaseous granulomas in the lungs and hilar lymph nodes with no identified microorganisms; microscopic pulmonary thromboemboli; mild coronary atherosclerosis; and status post cerebrovascular accident. She stated that the presence of dust macules and associated centroacinar emphysema were consistent with coal workers' pneumoconiosis, particularly in view of the employee's long-term underground coal employment and nonsmoking status.

In an undated report, Dr. Robert A.C. Cohen, a Board-certified pulmonologist, reviewed the employee's medical records and autopsy results. He concluded that the employee had coal workers' pneumoconiosis. Dr. Cohen noted that appellant had a history significant for coal dust exposure as a coal miner from 1950 to 1970, a period that predated the advent of federally mandated modern dust control. He indicated that the employee additionally worked for 22 years as a federal mine inspector with part of his work taking place in underground coal mines. The employee never smoked cigarettes and only occasionally smoked a pipe until 1970. Dr. Cohen stated that appellant had symptoms of chronic lung disease, including dyspnea from walking on

¹ The Office initially denied appellant's claim as untimely filed. The Board reversed the Office decision and found that the claim was timely filed in a decision dated September 22, 2003.

level ground and climbing steps as early as 1977. He indicated that the employee had signs of chronic lung disease on various physical examinations. Dr. Cohen commented that chest x-ray evidence was scant and showed no evidence of pneumoconiosis but the findings did not change the fact that the employee had clear, clinical, physiologic and pathologic evidence of pneumoconiosis and emphysema due to his coal dust exposure. He stated that there was clear pathological evidence of simple coal workers' pneumoconiosis on autopsy seen by all examiners, with evidence of silicosis, emphysema, chronic bronchitis, pneumonia and pulmonary emboli. Dr. Cohen commented that dust-induced lung damage had been shown to progress even after exposure ceases. He noted that the employee had extensive pathologic changes of interstitial lung disease due to coal workers' pneumoconiosis involving 20 to 50 percent of the employee's terminal respiratory units which would cause significant gas exchange abnormalities. Dr. Cohen stated that the employee's emphysema was due to his work as a coal miner and coal inspector. He stated that the combined impairments of emphysema and interstitial lung disease reduced the ability of the employee's lungs to withstand any additional respiratory compromise such as pneumonia and pulmonary emboli. Dr. Cohen indicated that patients who have significant underlying lung disease cannot withstand the further injury caused by the pneumonia and pulmonary emboli and would die sooner. He concluded that the employee's work resulted in the development of pneumoconiosis, emphysema and chronic bronchitis. These diseases resulted in substantial pulmonary impairment which caused the employee to be less able to withstand the effects of pneumonia and pulmonary emboli and, to a reasonable degree of medical evidence, caused him to die sooner than he would have had he not had the underlying lung disease.

In an August 7, 2000 report, Dr. Jeffery A. Kahn, a Board-certified pathologist, reviewed the medical record and microscopic slides of tissue taken during the autopsy. He stated that in one set of slides at least 50 percent of the terminal respiratory units were involved by coal macules and small coal nodules. In another set, only 20 percent of the units were involved. A third set showed more severe acute pneumonia than was present in the original slides. Dr. Kahn explained that the employee's focal emphysema was a distinct form of emphysema that involved expansion and distortion of the lumina of terminal respiratory units when a macule was formed. He commented that the coal dust contributed to the development of emphysema and might also have produced bronchitis. Dr. Kahn stated that, when broad areas of the lungs were involved by acute inflammation as in the employee's pneumonia, the pneumonia would be even more life threatening than in a person without the chronic lung disease because the chronic lung disease deprived the lung of its functional reserve and impaired its ability to overcome the insults. He stated that, in these situations, greater degrees of anoxia, acidosis and toxin formation would develop and result in complex interactive effects that produced systemic and multi-organ dysfunction. Dr. Kahn also pointed out that the coal macules and coal nodules formed in pneumoconiosis resulted in the distortion of the lung architecture, disturbances of air flow, and thereby predisposed the employee to the development of pneumonia. He concluded that the employee's emphysema, chronic bronchitis, pneumoconiosis and neurologic disease from the strokes each contributed to the employee's developing pneumonia. Dr. Khan indicated that each of the diseases produced pathophysiologic effects that aggravated the other diseases present so that the effects combined to accelerate the employee's death.

In a May 3, 2001 report, Dr. Francis H.Y. Green, a Board-certified pathologist, diagnosed severe simple coal workers' pneumoconiosis, macules and micronodules, moderately severe focal and centriacinar emphysema, chronic bronchitis, necrotizing bronchopneumonia,

pulmonary embolus with infarction and old healed infectious granulomata. The employee was hospitalized on April 23, 1988 and developed aspiration pneumonia during his stay in the hospital which was treated with antibiotics. Dr. Green reviewed Dr. Tabatowski's slides and concurred in her diagnoses of dust macules with associated centriacinar emphysema consistent with coal workers' pneumoconiosis, acute bronchopneumonia, old healed granulomata, thromboemboli and mild coronary atherosclerosis. He noted that the employee had over 40 years of exposure to coal dust, more than sufficient to develop occupational pneumoconiosis. Dr. Green indicated that the autopsy revealed evidence of simple coal workers' pneumoconiosis comprising numerous small macules with associated focal and centriacinar emphysema and micronodules. He pointed out that the severe simple pneumoconiosis present at the employee's autopsy would not be easily detected by x-ray because the size of the lesions in the lungs were below the limits of detection of standard chest x-rays and because the emphysema which involved the loss of lung tissue in contrast to pneumoconiosis which involved the deposition of scar tissue would cancel each other out on x-rays. Although the lesions of pneumoconiosis at the autopsy were small, they occupied approximately 50 percent of all available respiratory bronchioles. He concluded that this condition, combined with the emphysema and chronic bronchitis, would have produced a significant ventilatory defect of the predominately obstructive kind. Dr. Green declared that the employee's chronic bronchitis and emphysema, demonstrated at autopsy, were due entirely to exposure to coal mine dust in the underground mines. He stated that the immediate cause of the employee's death was respiratory failure due in part to the pneumonia and to the pneumoconiosis. Dr. Green commented that these two diseases would work together synergistically to produce the respiratory failure. He noted that pneumonia was a common problem for persons with strokes, but most patients would survive with appropriate treatment. In the presence of other significant lung disease, however, the prognosis would be graver, and in the employee's case led to his death. Dr. Green stated that the pneumoconiosis would have contributed to the employee's rapid respiratory failure in two ways. First, the chronic bronchitis induced by exposure to coal mine dust would predispose the employee to the development of infection in the respiratory tract due to the accumulation of mucous secretions and an inability to adequately clear the lungs of infection. Second, the pneumoconiosis would contribute to the hypoxemia caused by the pneumonia by reducing the transport of oxygen from the air into the blood. Dr. Green concluded that these two processes, in combination, caused the employee's premature death from respiratory failure. He stated that the employee died in respiratory failure due to the complications of his strokes and to pneumonia and an occupational pneumoconiosis. Dr. Green commented that the latter was a major contributing factor in the employee's death.

In an undated report, Dr. Perry Guariglia, a Board-certified cytopathologist, stated that his examination of the lung tissue slides led to his conclusion that the employee, at death, had acute necrotizing bronchopneumonia, moderate centrilobular emphysema, coal workers' pneumoconiosis with coal dust macules and silica, silicotic nodules and pulmonary thromboemboli. He stated that the employee's moderate centrilobular emphysema was probably causally related to his occupational coal dust exposure. Dr. Guariglia indicated that the employee's pneumoconiosis and emphysema caused and contributed to the acute necrotizing bronchopneumonia. He concluded that the cause of the employee's death was cardiorespiratory failure due to acute, necrotizing bronchopneumonia which was brought on in a substantial way, by emphysema which was related to occupational coal dust exposure as well as classical coal workers' pneumoconiosis.

In a November 18, 2003 decision, the Office denied appellant's claim on the grounds that the evidence of record was not sufficient to establish that the employee's death was caused by factors of his federal employment.

Appellant requested a hearing before an Office hearing representative. She submitted additional medical evidence contemporaneous with the death of the employee. In an April 23, 1998 admission note, Dr. Robert Andrus noted that the employee was hospitalized because of a possible stroke and had very little oral intake in the days prior to his admission. Appellant had tried to force feed soup to the employee that day but he could not swallow. Dr. Ulrich noted that a feeding tube was passed through the employee's nose to provide nutrition. In a May 6, 1998 discharge report, Dr. Ulrich diagnosed multi-infarct dementia with marked deterioration of white matter as shown on a computerized tomography scan. He indicated that the employee had a probable stroke which was not apparent at that time.

The hearing was conducted on July 21, 2004. In a September 28, 2004 decision, the Office hearing representative found that the Office medical adviser had not stated why the comprehensive reports of Dr. Cohen, Dr. Tabatowski, Dr. Ulrich, Dr. Green, Dr. Guariglia and Dr. Kahn were insufficient to establish causal relationship. She pointed out that the physicians generally supported the opinion that a work-related pulmonary condition complicated the employee's recovery from his stroke and ultimately resulted in his death. The Office hearing representative remanded the case for referral of the record back to the Office medical adviser for a review of the record and an opinion on whether there existed a causal relationship between the employee's pulmonary condition and his subsequent death and factors of his federal employment.

In a November 1, 2004 memorandum, the Office medical adviser noted that on April 23, 1998 appellant attempted to force-feed soup to the employee. He stated that the employee's stroke in April 1998 caused him to be unable to swallow. The Office medical adviser commented that the employee could not clear secretions that were normally swallowed and, as a result, the secretions were aspirated into the bronchial tree and subsequently the lungs. He stated that this occurred because of the stroke and had nothing to do with the employee's lung conditions that were claimed to be related to his employment. The Office medical adviser commented that the employee did not improve with a feeding tube but worsened. He stated that the placement of the feeding tube in the employee's nose compromised the ability of the gastroesophageal muscles to act as a deterrent for regurgitated contents from the stomach to be refluxed up the esophagus, giving a second basis for aspiration into the lungs. The Office medical adviser stated that the pulmonary failure which caused the employee's death was due to the aspiration pneumonia brought on by the employee's stroke and had nothing to do with his pneumoconiosis.

In a November 15, 2004 decision, the Office denied appellant's claim on the grounds that the weight of the medical evidence, as shown by the Office medical adviser, did not establish that the employee's death was caused or aggravated by exposure to factors of his federal employment.

LEGAL PRECEDENT

An appellant has the burden of proving by the weight of the reliable, probative and substantial evidence that the employee's death was causally related to his or her federal employment. This burden includes the necessity of furnishing medical opinion evidence of a cause and effect relationship based on a proper factual and medical background. The mere showing that an employee was receiving compensation for total disability at the time of death does not establish that the employee's death was causally related to his or her federal employment.²

ANALYSIS

The medical reports diagnosed pneumoconiosis, based on examination of lung tissue taken from the employee in an autopsy. Dr. Tabatowski stated that the presence of dust macules in the lungs with associated emphysema were consistent with pneumoconiosis. However, she did not address the issue of how the pneumoconiosis diagnosis caused or contributed to the employee's death. Dr. Cohen stated that appellant had evidence of emphysema and pneumoconiosis involving 20 to 50 percent of his terminal respiratory units which would have caused gas exchange abnormalities. He related these conditions to the employee's work as a coal miner and a coal miner inspector. Dr. Cohen explained that these diseases resulted in a substantial pulmonary impairment which caused the employee to be less able to withstand the effects of pneumonia and therefore die sooner than he would have without the underlying lung disease. Dr. Kahn indicated that appellant's pneumoconiosis, shown by the coal macules and coal nodules, caused changes in the lungs that led the employee to be predisposed for the development of pneumonia. He stated that the focal emphysema, caused by the coal dust, resulted in the expansion and distortion of the lumen of the terminal respiratory units when a macule was formed. Dr. Kahn commented that, when broad areas of the lungs were involved by acute inflammation due to pneumonia, the pneumonia would be even more life threatening because the pneumonia deprived the employee's lungs of its functional reserve and impaired its ability to overcome the insults. He concluded that the diseases of emphysema, chronic bronchitis, pneumoconiosis and neurologic disease due to stroke, produced pathophysiologic effects that aggravated the other diseases to the point that the effects of the combined diseases accelerated the employee's death. Dr. Guariglia concluded that the employee, at death, had acute necrotizing bronchopneumonia, moderate centrilobular emphysema, pneumoconiosis with coal dust macules and silica, silicotic nodules and pulmonary thromboemboli. He stated that the employee's moderate, centrilobular emphysema and pneumoconiosis, were probably caused by the employee's occupational coal dust exposure. Dr. Guariglia indicated that the employee's pneumoconiosis and emphysema caused and contributed to his necrotizing bronchopneumonia which caused the cardiorespiratory failure.

Dr. Green noted that the employee developed aspiration pneumonia while in the hospital and that the pneumoconiosis lesions occupied approximately 50 percent of all available respiratory bronchioles. He added that this condition, combined with the emphysema and chronic bronchitis, would have placed a significant ventilatory defect that was predominately obstructive. Dr. Green concluded that the employee's pneumoconiosis worked together with the

² Carolyn P. Spiewak (*Paul Spiewak*), 40 ECAB 552 (1989).

pneumonia to produce respiratory failure that was the immediate cause of the employee's death. He noted that pneumonia was a common problem with stroke victims but most patients would survive with appropriate treatment. Dr. Green indicated that in the presence of other significant lung diseases, the prognosis would be graver and, in the employee's case, led to his death. He commented that the pneumoconiosis would have contributed to the employee's rapid respiratory in two ways, first by predisposing him to infection of the respiratory tract, and second by contributing to the hypoxemia which, together with the pneumonia, reduced the transport of oxygen from the air into the blood. Dr. Green concluded that the employee died of respiratory failure due to complications of his stroke, the pneumonia and the occupational pneumoconiosis. He commented that the latter was a major contributing factor in the employee's death.

The Office medical adviser stated that the employee developed aspiration pneumonia because appellant tried to force feed him following the effects of the April 23, 1998 stroke. The Office medical adviser stated that the stroke was the cause of the employee's inability to swallow and that the attempt to force feed him led to the aspiration pneumonia. He indicated that the attempt to feed the employee through a tube in his nose led to reflux from the stomach into the esophagus, causing another source of aspiration. The Office medical adviser concluded that the employee died due to the aspiration pneumonia without any contribution by the employee's pneumoconiosis.

The reports of Dr. Cohen, Dr. Kahn, Dr. Young and Dr. Guariglia stated that the employee's pneumoconiosis and emphysema were causally related to his employment as a mine safety inspector and contributed to his death due to respiratory failure. The Office medical adviser concluded that the employee died due to aspiration pneumonia without any contribution from his employment-related pneumoconiosis and emphysema. The medical reports of record give rise to a conflict in the medical evidence. The case will therefore be remanded for referral of the employee's medical records, accompanied by a statement of accepted facts and the case record, to an appropriate impartial specialist, who should be requested to give a diagnosis of the employee's conditions, a conclusion on whether any of the diagnosed conditions were causally related, in whole or in part, to the employee's employment mine safety inspector. The impartial specialist should provide his opinion on whether the employee's employment-related conditions caused or contributed to the employee's death, with a reasoned explanation on how these conditions would or would not have caused the employee's death.

CONCLUSION

The case must be remanded to resolve a conflict in the medical evidence on whether the employee's employment-related conditions caused or contributed to his death.

ORDER

IT IS HEREBY ORDERED THAT the decision of the Office of Workers' Compensation Programs, dated November 15, 2004 be set aside and the case remanded for further action as set forth in this decision.

Issued: September 12, 2005
Washington, DC

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

David S. Gerson, Judge
Employees' Compensation Appeals Board

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