

U. S. DEPARTMENT OF LABOR

Employees' Compensation Appeals Board

In the Matter of MARLENE SINE, claiming as the widow of CHARLES SINE and
DEPARTMENT OF THE NAVY, BOSTON NAVAL SHIPYARD, Boston, Mass.

Docket No. 98-1776; Submitted on the Record;
Issued June 17, 1999

DECISION and ORDER

Before DAVID S. GERSON, WILLIE T.C. THOMAS,
BRADLEY T. KNOTT

The issue is whether appellant met her burden of proof to establish that the employee's death on November 28, 1993 was causally related to his accepted employment injury.

The Board has duly reviewed the case record in this appeal and finds that appellant has failed to meet her burden of proof to establish that the employee's death on November 28, 1993 was causally related to his accepted employment injury.

On April 15, 1992 the employee, then a laborer/sheet metal worker, filed an occupational disease claim (Form CA-2) alleging that he first became aware of his asbestos lung disease on July 3, 1991. The employee also alleged that he first realized that his condition was caused or aggravated by his employment on September 3, 1991.¹

The employee filed a claim for compensation on account of traumatic injury or occupational disease (Form CA-7) for the period beginning June 1, 1990.

By decision dated March 3, 1993, the Office of Workers' Compensation Programs found the evidence of record sufficient to establish that the employee sustained pleural plaques and that he was entitled to medical benefits for this accepted employment injury. The Office, however, found the evidence of record insufficient to establish that the employee was entitled to a schedule award inasmuch as the Federal Employees' Compensation Act did not provide compensation for permanent partial impairment of the lungs at the time of his last exposure to injurious employment factors. The Office further found the evidence of record insufficient to establish that the employee was entitled to compensation for the period beginning June 1, 1990 on the grounds that the evidence of record was insufficient to establish that the employee had any disability caused by his accepted employment injury. In a March 8, 1993 letter, the employee, through his counsel, requested an oral hearing before an Office representative.

¹ The employee worked for the employing establishment during the period 1942 to 1945.

By decision dated August 20, 1993, the hearing representative affirmed the Office's decision. In an October 8, 1993 letter, the employee, through his counsel, requested reconsideration of the hearing representative's decision.

In a January 21, 1994 decision, the Office denied the employee's request for modification based on a merit review of the claim. By letter dated January 28, 1994, appellant, through her counsel, requested reconsideration of the Office's decision and filed a claim for compensation by widow/widower and/or children (Form CA-5) based on the employee's death on November 28, 1993.

In an August 3, 1994 letter, the Office advised appellant to submit evidence supportive of her claim. By response letter dated August 8, 1994, appellant, through her counsel, submitted additional evidence.

By decision dated March 24, 1995, the Office denied appellant's request for modification based on a merit review of the claim.² In a March 14, 1996 letter, appellant, through her counsel, requested reconsideration of the Office's decision.

By decision dated July 16, 1996, the Office denied appellant's request for reconsideration without a review of the merits on the grounds that appellant neither raised substantive legal questions nor submitted new and relevant evidence.

In a July 19, 1996 letter, appellant's counsel requested an explanation regarding the delay in issuing a decision. In a September 9, 1996 response letter, the Office advised appellant's counsel that the evidence of record was insufficient to establish appellant's claim. The Office further advised appellant's counsel to submit additional medical evidence supportive of appellant's claim.

By letter dated February 28, 1997, the Office referred the employee's records along with a statement of accepted facts and a list of specific questions to Dr. Gerard Hayes, a Board-certified internist, for a second opinion medical report. Dr. Hayes submitted an April 22, 1997 medical report indicating that the employee's death was not causally related to asbestos exposure.

By decision dated July 16, 1997, the Office found the evidence of record sufficient to establish that the claimed incident occurred at the time, place and in the manner alleged, but insufficient to establish that the employee's death was caused by his accepted employment injury. In a December 16, 1997 letter, appellant, through her counsel, requested reconsideration of the Office's decision.

By decision dated March 6, 1998, the Office denied appellant's request for modification based on a merit review of the claim.

² Appellant appealed the Office's March 24, 1995 decision denying her request for modification to the Board. In an order dated January 29, 1996, the Board dismissed appellant's appeal based on her request to do so in light of her pending survivor's claim before the Office.

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 employment.³ This burden includes the necessity of furnishing rationalized medical opinion evidence, based on a complete factual and medical background, showing causal relationship.⁴ The mere showing that an employee was receiving compensation for total disability at the time of his death does not establish that his death was causally related to his employment.⁵ The medical evidence in this case fails to establish that the employee's death on November 28, 1993 was causally related to his accepted employment injury or to other factors of his employment.

In support of her claim, appellant submitted the medical reports of Dr. John Goodson, a Board-certified internist, Dr. Herbert H. Leventhal, a Board-certified radiologist, Dr. David L. Levy, a Board-certified internist and appellant's treating physician, Dr. Eva Statz, a Board-certified radiologist, Dr. Bimal Jain, a Board-certified internist, and Dr. Kenneth B. Bassion, a Board-certified radiologist. None of these medical reports, which predate the employee's death, addressed the cause of the employee's death on November 28, 1993, and thus, are not probative evidence to establish appellant's claim.

In further support of her claim, appellant submitted an undated pathology report of Dr. Gerald Feigin, a Board-certified pathologist, providing that the pleural and diaphragmatic plaques were strongly suggestive of asbestosis. Dr. Feigin's report is insufficient to establish appellant's burden because he failed to address a causal relationship between the employee's death and his accepted employment injury.

Appellant also submitted a November 30, 1993 death certificate revealing that the immediate cause of the employee's death was cardiopulmonary arrest and chronic obstructive lung disease. The death certificate further revealed that congestive heart failure and dementia contributed to the employee's death. The death certificate failed to indicate that the accepted employment condition of pleural plaques caused the employee's death on November 28, 1993.

In addition, appellant submitted a December 20, 1993 medical report of Dr. Ronald E. Gordon, a pathologist, indicating his findings on objective examination of the employee's lung tissue. Based on his findings, Dr. Gordon opined that the employee had an occupational asbestos exposure and that this exposure caused his lung disease which was the major cause of his death. Dr. Gordon failed to explain how or why the employee's death was caused by his lung disease.

An October 3, 1996 medical report of Dr. Lawrence Baker, a Board-certified internist, revealed a history of the employee's employment and medical treatment, and a review of medical records. Dr. Baker agreed with Dr. Gordon's opinion that the employee had occupational asbestos exposure that influenced his lung disease which was a major cause of his demise. He stated that the pathology was extraordinarily consistent with the disease asbestosis.

³ *Carolyn P. Spiewak (Paul Spiewak)*, 40 ECAB 552, 560 (1989); *Lorraine E. Lambert (Arthur R. Lambert)*, 33 ECAB 1111, 1120 (1982).

⁴ *Martha A. Whitson (Joe E. Whitson)*, 43 ECAB 1176, 1180 (1992).

⁵ *Isabell Craycraft (Harry Craycraft)*, 33 ECAB 1024 (1982); *Mary M. DeFalco*, 30 ECAB 514 (1979).

Dr. Baker further stated that the only exposures suffered by the employee were during the years that he worked for the employing establishment. He also stated that there was no question that the employee's asbestosis condition bore a direct relationship to those years he worked at the employing establishment given the history of no further exposures after leaving that employment. Dr. Baker noted the employee's other conditions and stated that notwithstanding those conditions, the asbestos-related lung disease brought about obstructive pulmonary disease symptomatology and honey-combing as evidenced on pathology at autopsy, probable pulmonary hypertension, and cor pulmonale with edema in the lower extremities around the penis and scrotum. He concluded that the employee's asbestosis which was pleural and parenchymal, caused alterations in ventilation and perfusion of the lung, brought about pulmonary hypertension, contributed to congestive phenomena and edema, and represented a major contributory factor in his demise on November 28, 1993. Dr. Baker failed to provide a detailed explanation of how the condition of asbestosis caused the employee's death.

A December 11, 1997 medical report of Dr. Richard Kradin, a Board-certified pathologist and internist, indicated a review of medical records and histological materials, and a history of the employee's employment. Dr. Kradin opined that based on his review of medical records, the employee's case met the clinical criteria for asbestosis. He stated that this diagnosis was based on known occupational exposure, appropriate latency, chest x-ray findings consistent with pneumoconiosis and rales on physical examination. Dr. Kradin further stated that he was unable to identify detailed pulmonary function test results in the record. He concluded that the employee's asbestosis contributed to his cardiovascular difficulties which led to both morbidity in life and his death. Dr. Kradin's report is not based on a complete review of the medical evidence inasmuch as he did not review the employee's pulmonary function test results.

The April 22, 1997 second opinion medical report of Dr. Hayes indicated a history of appellant's medical treatment and employment, and a review of medical records. Dr. Hayes diagnosed the following conditions: (1) coronary artery disease, status post myocardial infarction with cardiomyopathy, congestive heart failure and mitral regurgitation; (2) cerebrovascular disease, status post multiple strokes; (3) advanced Parkinson's disease; (4) chronic aspiration secondary to the first two diagnoses; (5) recurrent pneumonia, probably secondary to chronic aspiration; (6) status post gastrostomy tube placement consequent to advanced Parkinson's disease and chronic aspiration; (7) probable chronic obstructive lung disease noting that there was nothing to support restrictive or clinical apparent interstitial lung disease; (8) calcified benign pleural plaques consistent with asbestos exposure; and (9) microscopic evidence of pulmonary fibrosis possibly related to minimal clinically inconsequential asbestosis. Dr. Hayes opined that the employee's progressive dementia and death were related to medical problems separate from his asbestos exposure. He explained that there was no evidence to support a diagnosis of clinically significant asbestosis, but ample evidence to support a demise from obstructive, not

restrictive, lung disease, congestive heart failure and recurrent aspiration. Dr. Hayes further explained that:

“I believe [the employee] did have significant exposure over three years to asbestos from 1942 to 1945 and none thereafter. The calcified pleural plaques are in a typical location, have a typical radiographic appearance and are pathologically consistent with benign asbestos-associated pleural plaques. However, there is no evidence of pulmonary impairment due to restrictive physiology from these pleural findings, and thereafter I do not believe that the plaques in any way contributed to [the employee’s] medical and respiratory problems or his demise.

The autopsy findings of wet, congestive lungs and increased chest diameter support the clinical diagnoses of obstructive lung disease and congestive heart failure. Cor pulmonale, if it were ever present, could have likely been a consequence of obstructive lung disease. This patient also had dilated cardiomyopathy and mitral regurgitation, which leads to elevated pulmonary artery pressure (pulmonary hypertension) and right-sided heart failure which clinically is similar to cor pulmonale. Although emphysema was not grossly detected due to pulmonary congestion from heart failure, the increase A-P diameter of the chest very strongly suggests emphysema, there is little else that causes this finding. It is also important to note that no gross fibrosis was found at autopsy, suggesting that any microscopic fibrosis was very likely to be clinically unimportant.

Dr. Gordon’s findings of over 2 million asbestos fibers per gram of wet lung (about 1.5 million of which are amphiboles which are a cause of asbestosis) in association with the histologic findings described do suggest the pathologic diagnosis of asbestosis. However, the microscopic analysis does not clearly state the alveolar and peribronchial fibrosis were associated histopathologically on H&E section with asbestos bodies. It should be noted that this is a requirement for the pathologic diagnosis of asbestosis-associated pulmonary fibrosis (*i.e.* asbestosis) per the Pneumoconiosis Committee of the American College of Pathologist and the American Thoracic Society Ad Hoc Committee on environmental and Occupational Health. Other diseases which can result in lung fibrosis include recurrent healed pneumonia, recurrent aspiration, and idiopathic pulmonary fibrosis. The presence of asbestos bodies and asbestos fibers alone do not prove that lung fibrosis is due to this exposure, although high concentrations are more frequently associated with asbestosis. Nonetheless, the concentration of fibers in occupationally exposed individuals show a great deal of overlap between groups who develop clinical asbestosis and those who do not.

There is a distinction to be made between the histologic findings of fibrosis associated with asbestos fibers and the clinical entity asbestosis. The current American Thoracic Society guidelines for the diagnosis of clinical asbestosis (American Rev. Respir. Disease, 1986, 134:63) are accepted in all developed countries and require (1) a significant exposure history, (2) radiographic findings of pulmonary opacities suggestive of pulmonary fibrosis, (3) a characteristic restrictive pattern of impairment on pulmonary function tests and (4) presence of persistent middle to late inspiratory crackles on examination of the lung bases. The most important criteria are exposure history and chest film findings. As detailed above, the clinical

findings reported by the several physicians caring for [the employee] were consistent with obstructive, not restrictive, lung disease. There is no consistently reported association between obstructive lung disease and asbestos exposure. Where an association has been noted in asbestos-exposed populations, the strongest predictors of airflow obstruction relate to smoking exposure, not asbestos exposure. In any event, asbestosis is by definition a fibrosing alveolitis which results in restrictive interstitial lung disease, not obstructive lung disease. Dr. Baker's assertion that asbestos exposure resulted in [the employee's] obstructive lung disease and cor pulmonale is not consistent with the known effects of asbestos on lung function and is not consistent with a diagnosis of asbestosis.

The crackles heard on examination varied in the extent to which the lung fields were involved and seem to be more related to congestive heart failure than to pulmonary fibrosis. Further, there is no radiographic evidence of progressive pulmonary fibrosis as [the employee] deteriorated over the years. Progressive asbestosis causes a worsening of symptoms, and the radiographic progression of parenchymal opacities correlates to progression of symptoms and deterioration in lung function on pulmonary function tests. Dr. Levine correctly concludes that there is no evidence of asbestosis on clinical grounds, as [the employee] did not meet criteria of radiographic findings nor restrictive lung physiology. Dr. Goodson incorrectly asserts that pleural plaques are "pathognomonic" of asbestosis. The distinction between plaques (which are often present in the absence of asbestosis) and the disease asbestosis, is clearly drawn in the American Thoracic Society guidelines, and supported by several studies of more recent vintage.

A further refinement of terms should include the distinction between asbestosis-related findings and asbestosis-related disease. The asbestos-related diseases are limited to asbestosis as clinically defined above, mesothelioma and lung cancer. Benign pleural plaques as present in this case are not considered a disease. Therefore it is my conclusion that [the employee] did not meet the criteria for asbestos-related disease. Strictly speaking, it is not clear to me that he even meets accepted histologic criteria for asbestosis; Dr. Gordon's assertion notwithstanding, as there are other etiologies for pulmonary fibrosis which are quite plausible, given the clinical facts. I believe that Dr. Gordon's conclusion that [the employee's] "asbestosis exposure caused his lung disease" and that "his lung disease was the major cause of death" is misleading and does not incorporate the clinical findings, clinical course, and gross pathology findings with the cytocentrifugation, electron microscope and fiber burden analysis data. His lung disease appears to have been obstructive not restrictive in nature, and therefore incompatible with a clinical diagnosis of asbestosis. Even if one accepts that the histologic fibrosis is causally related to asbestos exposure, the rest of the clinical and radiographic data suggest that it was so mild as to have no impact on the progression of [the employee's] clinical deterioration and death. As described above, the gross pathologic findings do not support advanced asbestosis as the cause of death, or even a significant contributory factor."

The Board finds that Dr. Hayes provided a well-rationalized medical opinion based on an accurate factual and medical background. Inasmuch as appellant has failed to submit rationalized medical evidence establishing that the employee's death on November 28, 1993 was caused by his accepted employment injury of pleural plaques, she did not satisfy her burden of proof to establish her right to survivorship benefits.

The March 6, 1998 and July 16, 1997 decisions of the Office of Workers' Compensation Programs are hereby affirmed.

Dated, Washington, D.C.
June 17, 1999

David S. Gerson
Member

Willie T.C. Thomas
Alternate Member

Bradley T. Knott
Alternate Member