U. S. DEPARTMENT OF LABOR

Employees' Compensation Appeals Board

In the Matter of MARY JUNE FREME (claiming as widow of JOSEPH FREME) and DEPARTMENT OF LABOR, MINE SAFETY & HEALTH ADMINISTRATION, Morgantown, W.Va.

Docket No. 97-557; Submitted on the Record;
Issued November 19, 1998

DECISION and ORDER

Before DAVID S. GERSON, WILLIE T.C. THOMAS,
A. PETER KANJORSKI

The issue is whether appellant has met her burden of proof in establishing that the employee's death on September 25, 1992 was causally related to factors of his federal employment.

On March 15, 1995 appellant filed a claim for death benefits on account of the September 25, 1992 death of her husband, noting the nature of injury which caused the death as coal workers' pneumoconiosis. The employee had worked as a federal coal mine inspector from April 18, 1971 until he voluntarily retired on October 25, 1985. Prior to his federal employment, the employee had worked for 35 years as a coal miner with private coal companies from 1935 through 1970, except for three years during which he was in military service. The September 26, 1992 death certificate showed the immediate cause of death as pseudimonal pneumonia due to coal miners pneumoconiosis and emphysema.

In support of her claim, appellant submitted an Office of Workers' Compensation Programs' division of coal mine workers' compensation notice of initial finding, in which it determined that appellant was entitled to benefits due to the employee's death from pneumoconiosis.

Also in support of her claim appellant submitted a November 4, 1992 autopsy report from Dr. Jeffrey A Stead, a Board-certified pathologist. The immediate cause of death was noted as severe bilateral lobe pneumonia and the basic cause of death was noted as coal workers' pneumoconiosis. Dr. Stead explained that the employee's severe coal workers' pneumoconiosis was manifested by anthrasilicosis, emphysema and vascular changes and right ventricular hypertrophy indicating pulmonary hypertension. Dr. Stead noted that the employee's severe coal workers' pneumoconiosis rendered him simultaneously more likely to contract pneumonia and less likely to survive it than one not so afflicted.

A postmortem medical report dated July 19, 1993 from Dr. Richard L. Naeye, a Board-certified pathologist, based upon pathological specimens, also described the employee's cause of death as acute lobular pneumonia that was superimposed on severe simple coal workers' pneumoconiosis (CWP). Dr. Naeye stated: "The CWP is so severe it almost certainly hastened his death." He also noted that there was microscopic evidence of severe chronic bronchitis, but he did not include this in his interpretation of the cause of the employee's death.

In an attending physician's report dated February 12, 1995 Dr. Roger A. Abrahams, a Board-certified internist and pulmonary and occupational lung specialist, listed the direct cause of the employee's death as pseudomonas pneumonia/coal workers' pneumoconiosis and the contributory cause of death as coal workers' pneumoconiosis and emphysema. Dr. Abrahams noted as history of injury "coal worker" and in response to a question on the causal relationship of his history as a coal worker to the employee's death he noted "coal workers pneumoconiosis with progressive massive fibrosis and respiratory insufficiency predisposing risk for development of pneumonia."

Appellant submitted as history a July 3, 1989 report of Dr. Abrahams, which stated that when his August 8, 1985 report was prepared, he erroneously concluded that the employee did not suffer from pneumoconiosis and he based his opinion on a National Institute of Occupational Safety and Health certified B-reading by Dr. Joseph Renn. Dr. Abrahams stated that when he provided that opinion in 1985, he had little experience in interpreting chest x-rays for pneumoconiosis and relied completely on Dr. Renn's interpretation. Dr. Abrahams stated, however, that since he has now become a B-reader himself, his interpretation is quite different from Dr. Renn's and he believes that the employee suffered from progressive massive fibrosis as a direct result of his coal mining experience and also from bullous emphysema at least partially due to his years of cigarette smoking. Dr. Abrahams concluded that the employee was "severely impaired to the degree that he would not be able to perform his prior job as a federal coal mine inspector," and that this impairment was due to both (a) progressive massive fibrosis as a result of his exposure to coal dust and (b) emphysema with very severe obstructive airway disease as a result of his prior cigarette smoking and coal dust exposure.

Appellant also submitted a 1991 transcript of testimony in the Black Lung hearing before an administrative law judge. The employee testified that he developed breathing problems in 1981 or 1982 and he supposed his exposure to coal mine dust had an effect on his breathing.

Appellant further submitted several pages from a September 14, 1993 deposition from Dr. Naeye, which stated that he could not segregate the employee's exposures, nor tell which of the various exposures caused the various components of the coal workers' pneumoconiosis. Dr. Naeye opined that the coal workers' pneumoconiosis was a significant disease process in the employee's lungs and may very well have shortened his life. He also stated that he thought the employee would have lived somewhat longer if he had not had the coal workers' pneumoconiosis. Dr. Naeye noted that the employee's bronchitis did not have a clinically significant effect until after 1979.

Appellant additionally submitted one page from a deposition from Dr. Stead, in which he indicated: "Based on the organs that I had available to study and the clinical history and the findings of the presence of coal miners' pneumoconiosis … the basic cause of death [was] coal

miners' pneumoconiosis." Dr. Stead reiterated that pneumoconiosis was the substantially contributing cause of the employee's death.

In response to the Office's request for further information appellant's representative stated that the employee's exposure to coal dust continued for 14 years as a mine inspector, that he spent most of his working hours underground and that it was obvious that his pneumoconiosis was related to coal dust exposure underground in his federal employment.

Also in response the employing establishment responded that the employee worked as a coal mine inspector in their roof control section, that during those years he was required to inspect both underground and surface mines and that normally inspectors are exposed to respirable coal mine dust. The district manager noted that the employee may have been required to walk return airways that may have exceeded the respirable coal mine dust standards of two milligrams per cubic meter of air and indicated that it would be impossible to determine the amount of time the employee was exposed to respirable coal mine dust or any other toxic fumes or gases. He noted that appellant retired in 1985 and that coal mine inspectors were issued respirators in 1989.

By decision dated June 9, 1995 the Office denied appellant's claim for death benefits finding that the evidence of record failed to establish that the deceased contracted a disease, which led to his death as a result of exposure to federal employment factors. The Office found that all three reports, while identifying the underlying cause of death as being coal miners' pneumoconiosis, failed to explain how pneumoconiosis was causally related to factors of the employee's federal employment.

By letter dated June 15, 1995, appellant, through her representative, requested reconsideration. In support of her request appellant resubmitted pages 27 through 38 of testimony from the employee describing his employment and his illness. She also resubmitted previously submitted testimony and medical reports.

By decision dated September 11, 1995, the Office reviewed the case on its merits and denied modification of the prior decision. The Office found that the record lacked a reasoned medical report from a pulmonologist explaining how autopsy findings resulted from 14 years of federal exposure verses 35 years of private coal company exposure.

By letter dated June 7, 1996, appellant, through her representative, again requested reconsideration. In support of her request, appellant submitted a December 6, 1995 letter from Dr. Abrahams which stated: "As you know, [the employee] had total pulmonary disability due to his severe obstructive airway disease and coal workers' pneumoconiosis with progressive massive fibrosis. His coal workers' pneumoconiosis was due to the total and cumulative dust exposure experienced at both the 35 years as an underground coal miner and the 14 years as a federal mine inspector. Both exposures have caused his severe coal workers' pneumoconiosis and substantially accelerated his obstructive airway disease and pulmonary impairment."

Appellant further submitted the complete depositions of Drs. Naeye and Stead and of the employee. Dr. Naeye stated: "I feel that the pneumoconiosis (sic) was less important in his disability and death than the coal workers' pneumoconiosis, but there was very significant coal

workers' pneumoconiosis present also." He then stated that although it was not easy to quantify "the roles of all these various disease processes, except to say that the emphysema, the centilobular and panlobular emphysema were definitely more important and the bronchitis was more important than the coal workers' pneumoconiosis, also, in his final destruction of his lungs." Dr. Naeye stated that the employee's immediate cause of death was a breakdown of the defense mechanisms in his lungs that he developed pneumonia, from which he could not recover. In response to the question about whether the pneumoconiosis hastened the development of the pneumonia, Dr. Naeye stated that he doubted it and indicated that it was a sequence of things that happened during all those years and that the bronchitis which had been established much earlier in his life was "the single major factor that led to the progressive destruction of his lungs." Dr. Naeye stated that what he did not know was whether any significant part of the employee's bronchitis originated in occupational exposure, but then he opined: "The fact that this man worked in the industry for so many years and then became an inspector leads me to think that it was far more likely that his cigarette smoking set him up for this bronchitis and all of his long-term complications than did exposure to coal mine dust." Dr. Naeye stated that he could not, with any degree of certainty, split the employee's bronchitis up into what was caused by smoking and what might have been caused by industrial exposure to dust and he stated that he could not speak with medical certainty because the employee's medical information and history was not good enough to know what was happening to him in his early years and he admitted that his opinion was based upon anecdotal evidence from speaking to younger miners. Dr. Naeye stated that the employee's focal emphysema was definitely a component of coal workers' pneumoconiosis and he opined that the "centrilobular emphysema and the panlobular emphysema and the bronchitis were the single biggest contributors to his disability and death, but the coal workers' pneumoconiosis was also a significant disease process in his lungs and may very well have shortened his life."

In his December 1, 1994 deposition, Dr. Stead indicated that coal miners' pneumoconiosis was not one disease but had a component that might be related to chronic bronchitis, a pigment component, a silica component and a free radical component, which had been implicated in eliciting chronic bronchitis which led to chronic lung disease. He also indicated that progressive massive fibrosis and massive lesions in the lungs would be synonymous with complicated coal miners' pneumoconiosis. Dr. Stead noted that upon autopsy he found evidence of simple coal miners' pneumoconiosis, classified as the macronodular variety, complicated by silicosis and emphysema. He opined that the employee's pneumoconiosis made him more likely to get any type of pneumonia. Dr. Stead opined the pneumoconiosis was the substantially contributing cause of the employee's death in this case.

By decision dated August 15, 1996, the Office denied modification of the prior decision finding that the evidence submitted was insufficient to warrant modification. The Office found that all three physicians, Drs. Abrahams, Naeye and Stead were equivocal and speculative, that nobody addressed the cause of the pneumoconiosis in relation to appellant's employment and that no medical opinion addressed how the employee's death could be attributed to his federal employment, when he was "required to wear respiratory protection."

The Board finds that this case is not in posture for decision.

Proceedings under the Federal Employees' Compensation Act are not adversary in nature, nor is the Office a disinterested arbiter. While the claimant has the burden to establish entitlement to compensation, the Office shares responsibility in the development of the evidence to see that justice is done.[1] This holds true in death benefits claims as well as in initial traumatic injury and occupational illness claims. In the instant case, although none of the employee's treating physicians' reports or pathology reports following autopsy, contain rationale sufficient to completely discharge appellant's burden of proving by the weight of reliable, substantial and probative evidence that the employee died due to illness causally related to his federal employment, or that the employee's death was hastened by any condition resulting from his federal employment, they constitute substantial, uncontradicted evidence in support of appellant's claim and raise a inference of causal relationship between the employee's death and his original occupational exposure, that is sufficient to require further development of the case record by the Office.[2] Additionally, there is no substantial opposing medical evidence in the record.

The Board notes that in his reports and his testimony Dr. Naeye stated both that the employee's coal workers' pneumoconiosis was so severe it almost certainly hastened the employee's death, that the pneumoconiosis was a significant disease process, which may have very well shortened his life, but that he doubted that the coal workers' pneumoconiosis hastened the development of pneumonia. The Board finds that this contradiction diminishes the probative value of Dr. Naeye's reports and testimony, such that it does not constitute substantial opposing medical evidence. Further, Dr. Naeye stated that the bronchitis the employee developed earlier in life was the single major factor that led to the progressive destruction of his lungs, but that the employee's bronchitis did not have a clinically significant effect until after 1979 and that he could not state with any degree of certainty what part of the employee's bronchitis might have been caused by industrial exposure to coal dust and that he based his opinion on anecdotal evidence from speaking to younger miners. The Board finds that these conflicting statements and admitted speculation further reduce the probative value of Dr. Naeye's reports and testimony.

As the probative medical evidence of record, and the factual evidence submitted in support of the claim, reveal that the employee was exposed for 14 years to respirable coal dust while performing his duties as a mine inspector and that he died of terminal pneumonia due to underlying coal workers' pneumoconiosis, massive pulmonary fibrosis, panlobular emphysema and possibly coal dust-related bronchitis, the Office should refer the case to a pulmonary disease expert with appropriate questions addressing the vital issues, for a rationalized opinion as to whether the employee's 14 years of exposure to respirable coal dust caused or aggravated the development of his pneumoconiosis which caused of accelerated his ultimate demise.

[1] *William J. Cantrell*, 34 ECAB 1223 (1983).

[2] *John J. Carlone*, 41 ECAB 354 (1989); *Horace Langhorne*, 29 ECAB 820 (1978); *see also Cheryl A. Monnell*, 40 ECAB 545 (1989); *Bobby W. Hornbuckle*, 38 ECAB 626 (1987) (if medical evidence establishes that residuals of an employment-related impairment are such that they prevent an employee from continuing in the employment, he is entitled to compensation for any loss of wage-earning capacity resulting from such incapacity).

Consequently, the decision of the Office of Workers' Compensation Programs dated August 15, 1996 is hereby set aside and the case is remanded for further development in accordance with this decision and order of the Board.

Dated, Washington, D.C.
November 19, 1998

David S. Gerson
Member

Willie T.C. Thomas
Alternate Member

A. Peter Kanjorski
Alternate Member