U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 24 March 2004

In the Matter of:

DELANA RAY, widow, and on behalf, of
CHARLES B. RAY
Claimant

Case No.: 2002 BLA 245

v.

EASTOVER MINING COMPANY
Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party in Interest

Appearances: Mr. Ron Carson, Personal Representative
For the Claimant

Mr. Stacy Huff, Attorney
For the Employer

Before: Richard T. Stansell-Gamm
Administrative Law Judge

DECISION AND ORDER –
DENIAL OF SURVIVOR BENEFITS
DENIAL OF BLACK LUNG DISABILITY BENEFITS

This matter involves claims filed by Mr. Charles B. Ray and his surviving spouse, Mrs. Delana Ray for disability and survivor benefits under the Black Lung Benefits Act, Title 30, United States Code, Sections 901 to 945 ("the Act"). Benefits are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis, or to survivors of persons who died due to pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as "black lung" disease.

Procedural Background

Mr. Charles Ray's Claims

First Claim

Mr. Ray filed his first application for Black Lung disability benefits on August 24, 1992 (DX 93).[1] His claim was denied on February 19, 1993 by a U.S. Department of Labor ("DOL") claims examiner. After DOL considered additional evidence and a formal hearing was held, Administrative Law Judge Paul H. Teitler issued a Decision and Order Denying Benefits on November 11, 1996 (DX 93-65). Specifically, based on a qualifying pulmonary function test and the consensus of medical opinion, Judge Teitler determined that Mr. Ray was totally disabled due to a pulmonary impairment. However, because the radiographic and medical opinion evidence failed to demonstrate that he had coal workers' pneumoconiosis, Judge Teitler denied the claim since Mr. Ray's respiratory impairment was not related to his past occupation as a coal miner.

Second Claim

On July 29, 1998, Mr. Ray filed his second, duplicate, claim (DX 1). After a review of the evidence, DOL denied the claim on November 25, 1998 (DX 36). Mr. Ray continued to submit additional medical evidence over the next three years. DOL treated the submissions as modification requests and twice denied the requests (DX 50 and DX 63). Another denial of a modification request occurred October 6, 2000 (DX 69). Since Mr. Ray had passed away the day before, his surviving spouse, Delana Ray, appealed the denial on his behalf on October 16, 2000 (DX 70). On February 12, 2001, DOL again denied Mr. Ray's claim (DX 72). After considering additional autopsy evidence, DOL issued its final denial of Mr. Ray's modification request on the denial of his duplicate claim on December 13, 2001 (DX 90).

Mrs. Delana Ray's Survivor Claim

After her husband's death on October 5, 2000, Mrs. Ray filed her claim for survivor benefits on October 24, 2000 (DX 2). DOL denied her claim on February 12, 2001 (DX 73). After consideration of autopsy evidence and new medical reports, DOL again denied her claim on December 13, 2001 (DX 91). Mrs. Ray's appeal of the denial of her survivor claim was consolidated with her appeal of the denial of Mr. Charles Ray's claim and both cases were forwarded to the Office of Administrative Law Judges on March 4, 2002 (DX 94). Pursuant to a Notice of Hearing, dated October 4, 2002 (ALJX I), I conducted a hearing in Knoxville, Tennessee on January 14, 2003 attended by Mrs. Ray, Mr. Carson and Mr. Huff. My decision in this case is based on the hearing testimony and the documents admitted into evidence (DX 1 to DX 93; CX 1; EX 1 and EX 2).

[1]The following notations appear in this decision to identify exhibits: DX -- Director exhibit; CX -- Claimant's exhibit; EX -- Employer's exhibit; ALJ -- Administrative Law Judge exhibit; and TR -- Transcript.

ISSUES

1. Whether Mr. Ray suffered from coal workers' pneumoconiosis.

2. If Mr. Ray suffered from coal workers' pneumoconiosis, whether his death was due to coal workers' pneumoconiosis.

3. If Mr. Ray suffered from coal workers' pneumoconiosis, thereby establishing a material change in conditions, whether he was totally disabled due to coal workers' pneumoconiosis.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Stipulations of Fact

At the hearing, the parties stipulated to the following facts: a) Mr. Ray was a coal miner with post-1969 coal mine employment; b) his length of coal mine employment was at least 20 years; c) Eastover Mining Company is the Responsible Operator; and d) Mrs. Delana Ray qualifies as the surviving spouse of the miner (TR, pages 10 to 12).

Preliminary Findings

Born on April 21, 1931, Mr. Ray married Delana Cress on February 23, 1953 (DX 11 and DX 93-8). Mr. Ray started coal mining in 1951 as an electrician in coal mine construction. He continued as an electrician and foreman throughout his mining career and was a Chief Electrician for Eastover Mining. He left coal mine employment in October of 1975 because "he could not pass a physical due to black lung" (DX 93). After that time, he worked as a state mine inspector (DX 93-52). His work as Chief Electrician involved maintenance of all electrical and mechanical equipment at the mine and included directing the work force, training personnel and assisting in the repair of equipment (DX 5 and DX 93-6). In this position, Mr. Ray would typically sit two hours per day, stand six hours per day, lift 20-75 pounds about 1-4 times each day, carry 25 pounds about 300 feet and carry 10 pounds about 600 feet two times each day. The record establishes that Mr. Ray continued working in and out of the mines as an inspector, instructor and electrical specialist for Virginia's Department of Mines, Minerals and Energy, from March of 1975 through the end of 1991.[2]

Mrs. Ray testified that her husband's health "went downhill" from the 1970's and that he could no longer go fishing or travel very far because of his shortness of breath (TR, page 19). He had difficulty sleeping and would require two pillows for elevation to breathe at night (TR, page 20). Eventually, he was placed on oxygen 24 hours a day (DX 13). Mrs. Ray testified that

[2]In his Decision and Order, Judge Teitler determined that Mr. Ray's employment with the Virginia Department of Mines qualified as coal mine employment under the regulations, but he did not disturb the District Director's determination that the Commonwealth of Virginia could not be considered a putative operator responsible for payment of benefits. Because of the parties' stipulation, the question of whether Eastover Mining is the Responsible Operator for this claim is no longer at issue.

Dr. Boggan was the miner's personal physician toward the end of his life, along with Dr. Foster and Dr. Ron Brock (TR, page 21). Mr. Ray died on October 5, 2000 and Mrs. Ray has not remarried (DX 12, DX 2, and TR, page 17). The miner and widow had no dependents at the time either of their claims was filed (DX 2, DX 93, and TR, page 17). Starting at age 17, Mr. Ray smoked "about all of his life," at an average rate of about 1 1/2 pack of cigarettes per day (DX 93-26 and TR, pages 21 and 22).

Survivor Claim

To receive survivor benefits under the Act, and the implementing regulations, 20 C.F.R. § 718.205 (a),[3] because a former coal miner's death was due to coal workers' pneumoconiosis, a surviving claimant must prove by a preponderance of the evidence several facts. First, the claimant must establish eligibility as a survivor. A surviving spouse may be considered eligible for benefits under the Act if she was married to, and living with, the coal miner at the time of his death, and has not remarried.[4]

Next, the claimant must prove the coal miner had pneumoconiosis.[5] "Pneumoconiosis" is defined as a chronic dust disease arising out of coal mine employment. The regulatory definitions include both clinical pneumoconiosis (the diseases recognized by the medical community as pneumoconiosis) and legal pneumoconiosis (defined by regulation as any chronic lung disease arising out of coal mine employment).[6] The regulation further indicates that a lung disease arising out of coal mine employment includes "any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment."[7] As courts have noted, under the Act, the legal definition of pneumoconiosis is much broader than medical pneumoconiosis. *Kline v. Director, OWCP*, 877 F.2d 1175 (3d Cir. 1989).

Once a determination has been made that a miner had pneumoconiosis, it must be determined whether the coal miner's pneumoconiosis arose, at least in part, out of coal mine employment.[8] If a miner who was suffering from pneumoconiosis was employed for ten years or

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[3]Since Mrs. Ray filed her claim, the U.S. Department of Labor has published new regulations concerning black lung disability benefits. Most of the provisions in Part 718 of those new regulations are applicable to her case and Mr. Ray's claim.

[4]20 C.F.R. § 718.4 indicates that the definitions in 20 C.F.R. § 725.101 are applicable. 20 C.F.R. § 725.101, in turn, refers to the term "survivor" as used in Subpart B of Part 725. 20 C.F.R. § 725.214 then sets out the spousal relationship requirements and 20 C.F.R. § 725.215 describes the dependency rules. According to § 725.214 (a) the spousal relationship exists if the relationship is a valid marriage under state law. Under § 725.215(a), a spouse is deemed dependent if she was residing with the miner at the time of his death.

[5]20 C.F.R. § 718.205 (a) (1) and see *Trumbo v. Reading Anthracite Co*., 17 B.L.R. 1-85 (1993).

[6]20 C.F.R. § 718.201 (a) (1) and (2).

[7]20 C.F.R. § 718.201 (b).

[8]20 C.F.R. §§ 718.203 (a) and 205 (a) (2).

more in one or more coal mines, there is a rebuttable presumption that pneumoconiosis arose out of such employment.[9] Otherwise, the claimant must provide competent evidence to establish the relationship between pneumoconiosis and coal mine employment.[10]

Finally, the surviving spouse has to demonstrate the coal miner's death was due to pneumoconiosis.[11]

In summary, a survivor claim filed after January 1, 1982 must meet four primary elements for entitlement. The claimant bears the burden of establishing these elements by a preponderance of the evidence. If the claimant fails to prove any one of the requisite elements, the survivor claim for benefits must be denied. *Gee v. W. G. Moore and Sons*, 9 B.L.R. 1-4 (1986) and *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985). The four elements are: (1) the claimant is an eligible survivor of the deceased miner; (2) the coal miner suffered from pneumoconiosis; (3) the coal miner's pneumoconiosis arose out of coal mine employment; and, (4) the coal miner's death was due to coal workers' pneumoconiosis.

Eligible Survivor

As noted above, the parties have stipulated that Mrs. Ray is an eligible survivor under the Act. As a result, she has established the first requisite element of entitlement.

Issue # 1 – Presence of Pneumoconiosis

According to 20 C.F.R. § 718.202, the existence of pneumoconiosis may be established by four methods: chest x-rays (20 C.F.R. § 718.202 (a) (1)), autopsy or biopsy report (20 C.F.R. § 718.202 (a) (2)), regulatory presumption (20 C.F.R. § 718.202 (a) (3)),[12] and medical opinion (20 C.F.R. § 718.202 (a) (4)). Since the record does not contain evidence that Mr. Ray had complicated pneumoconiosis and Mrs. Ray filed her claim after January 1, 1982, a regulatory presumption of pneumoconiosis is not applicable. As a result, Mrs. Ray must rely on chest x-rays, autopsy evidence, and medical opinions to establish the presence of pneumoconiosis in her husband's lungs.

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[9]20 C.F.R. § 718.203 (b).

[10]20 C.F.R. § 718.203 (c).

[11]20 C.F.R. § 718.205 (a) (3).

[12]If any of the following presumptions are applicable, then under 20 C.F.R. § 718.202 (a)(3) a miner is presumed to have suffered from pneumoconiosis: 20 C.F.R. § 718.304 (if complicated pneumoconiosis is present then there is an irrebuttable presumption the miner is totally disabled due to pneumoconiosis); 20 C.F.R. § 718.305 (for claims filed before January 1, 1982, if the miner has fifteen years or more coal mine employment, there is a rebuttable presumption that total disability is due to pneumoconiosis); and 20 C.F.R. § 718.306 (a presumption when a survivor files a claim prior to June 30, 1982).

Chest X-rays

In adjudicating Mr. Ray's first claim for benefits, Judge Teitler summarized the multiple interpretations of the 7 chest x-rays in the record (*see* DX 93-65). He concluded that the preponderance of the more probative interpretations by better qualified B readers and board certified radiologists established that the radiographic evidence did not support a finding of pneumoconiosis.

I have reviewed those chest x-ray interpretations and agree with his ultimate conclusion. Based on Dr. Navani's sole interpretations, the May 5, 1992 and May 24, 1993 chest x-rays are positive for pneumoconiosis. However, the interpretations of the more qualified readers of the remaining five films establishes that the October 26, 1992, March 4, 1993, March 17, 1993, May 20, 1993 and September 28, 1993 are negative for pneumoconiosis.

With two exceptions, the following table summarizes all chest x-ray interpretations admitted into evidence since Judge Teitler's denial of Mr. Ray's first claim.[13]

Date of x-ray	**Exhibit**	**Physician**	**Interpretation**
February 19, 1992	DX 21	Hernandez	Minimal interstitial markings, bilateral granulomata (negative for pneumoconiosis).
May 11, 1992	DX 33 & DX 93-40	Ramakrishnan	Severe chronic fibrosis. Emphysema (negative for pneumoconiosis).
February 23, 1997	DX 23	Cassedy	Prominent interstitial disease, pulmonary fibrosis (negative for pneumoconiosis).
February 26, 1997	DX 23	Phillips	Fibrocalcific changes (negative for pneumoconiosis)
March 3, 1997	DX 20	Hickman, BCR[14]	Scarring from pneumonia (negative for pneumoconiosis).
April 16, 1997	DX 21 & DX 23	McMurray, BCR	Emphysema, significant COPD (chronic obstructive pulmonary disease) (negative for pneumoconiosis).
April 24, 1997	DX 21 & DX 23	Cooper, BCR	Obstructive disease (negative for pneumoconiosis).
April 25, 1997	DX 21 & DX 23	Westerfield, BCR	Granulomatous calcification (negative for pneumoconiosis).
July 17, 1998	DX 42	Baker, B	Positive for pneumoconiosis, profusion category 1/0,[15] type s/t opacities.[16]

[13]I have included the February 19, 1992 and May 11, 1992 chest x-rays because they were not in the record considered by Judge Teitler.

[14]The following designations apply: B – B reader, and BCR – Board Certified Radiologist. These designations indicate qualifications a person may possess to interpret x-ray film. A "B Reader" has demonstrated proficiency in assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A "Board Certified Radiologist" has been certified, after four years of study and examination, as proficient in interpreting x-ray films of all kinds including images of the lungs. *See also* 20 C.F.R. § 718.202 (a) (1) (ii).

[15]The profusion (quantity) of the opacities (opaque spots) throughout the lungs is measured by four categories: 0 = small opacities are absent or so few they do not reach a category 1; 1 = small opacities definitely present but few in number; 2 = small opacities numerous but normal lung markings are still visible; and, 3 = small opacities very numerous and normal lung markings are usually partly or totally obscured. An interpretation of category 1, 2, or 3 means there are opacities in the lung which may be used as evidence of pneumoconiosis. If the interpretation is 0, then the assessment is not evidence of pneumoconiosis. A physician will usually list the interpretation with two

(same)	DX 44	Mathur, B, BCR	Positive for pneumoconiosis, profusion category 1/2, type q/t opacities.
(same)	DX 45	Marshall, B, BCR	Positive for pneumoconiosis, profusion category 2/1, type q/r opacities.
(same)	DX 46	Sargent, BCR, B	Negative for pneumoconiosis; questionable emphysema, calcified granulomas.
(same)	DX 47	Barrett, BCR,B	Negative for pneumoconiosis; emphysema, exposure to tuberculosis.
(same)	DX 48	Goldstein, B	Positive for pneumoconiosis, profusion category 1/1, type s/t opacities, COPD with emphysema.
August 17, 1998	DX 27	Baker, B	Positive for pneumoconiosis, profusion category 1/0, type t/s opacities, tuberculosis.
(same)	DX 28	Sargent, BCR, B	Negative for pneumoconiosis; calcified granulomas, questionable emphysema.
(same)	DX 30	Barrett, BCR, B	Negative for pneumoconiosis; emphysema, old granulomas.
Sept. 11, 1998	DX 62	Kiser	Probable old granulomatous disease.
Sept. 24, 1998	DX 34	Wiot, BCR, B	Negative for pneumoconiosis; emphysema.
(same)	DX 34	West, BCR, B	Positive for pneumoconiosis, profusion category 1/1, type s/t opacities, emphysema. In the absence of small rounded opacities, the present findings are more consistent with asbestosis or other chronic interstitial
(same)	DX 35	Spitz, BCR, B	Negative for pneumoconiosis; emphysema.
(same)	DX 35	Shipley, BCR, B	Negative for pneumoconiosis; emphysema.
October 9, 1998	DX 21	Fox	Bilateral interstitial prominence (negative for pneumoconiosis).
February 26, 1999	DX 23	Phillips	(Negative for pneumoconiosis).
June 29, 1999	DX 22	Foster	(Negative for pneumoconiosis).
(same)	DX 32	Sargent, BCR, B	Negative for pneumoconiosis; calcified granulomas, questionable emphysema and tuberculosis.
August 18, 1999	DX 22, DX 23, DX 54, &DX 61	Westerfield, BCR, B	Positive for pneumoconiosis, profusion category 1/2, type q/r opacities, significant COPD and emphysema, known carcinoma in right lung apex.
(same)	DX 55 & DX 61	Mathur, BCR, B	Positive for pneumoconiosis, profusion category 1/1, type q/t opacities.
(same)	DX 56 & DX 61	Dahhan, B	Negative for pneumoconiosis; emphysema,.

digits. The first digit is the final assessment; the second digit represents the category that the doctor also seriously considered. For example, a reading of 1/2 means the doctor's final determination is category 1 opacities but he considered placing the interpretation in category 2. Additionally, according to 20 C.F.R. § 718.102 (b), a profusion reading of 0/1 does not constitute evidence of pneumoconiosis.

[16]There are two general categories of small opacities defined by their shape: rounded and irregular. Within those categories the opacities are further defined by size. The round opacities are: type p (less than 1.5 millimeter (mm) in diameter), type q (1.5 to 3.0 mm), and type r (3.0 to 10.0 mm). The irregular opacities are: type s (less than 1.5 mm), type t (1.5 to 3.0 mm) and type u (3.0 to 10.0 mm). JOHN CRAFTON & ANDREW DOUGLAS, RESPIRATORY DISEASES 581 (3d ed. 1981).

(same)	DX 57 & DX 61	Wiot, BCR, B	Negative for pneumoconiosis; emphysema.
(same)	DX 58& DX 61	Spitz, BCR, B	Negative for pneumoconiosis; emphysema.
(same)	DX 58 & DX 61	Meyer, BCR, B	Negative for pneumoconiosis; emphysema, calcified granulomas.
(same)	DX 59, DX 60 & DX 61	Perme, BCR, B	Negative for pneumoconiosis; emphysema, possible cancer.
(same)	DX 66	Sargent, BCR, B	Negative for pneumoconiosis; questionable emphysema.
January 1, 2000	DX 19	Vongkasemsiri	Emphysema (negative for pneumoconiosis).
January 9, 2000	DX 19	Norris	Emphysema, calcified granulomata (negative for pneumoconiosis).
February 25, 2000	DX 19	Vongkasemsiri	Emphysema, chronic interstitial disease with scattered small nodules compatible with healed granuloma (negative for pneumoconiosis).
February 27, 2000	DX 19	Vongkasemsiri	Emphysema, healed granuloma (negative for pneumoconiosis).
March 3, 2000	DX 17	Norris	Scattered granulomata, increased density right lung apex (negative for pneumoconiosis).
March 5, 2000	DX 19	Vongkasemsiri	Emphysema, chronic interstitial lung disease, density right apex (negative for pneumoconiosis)
March 7, 2000	DX 17	Norris, BCR	Pulmonary emphysema (negative for pneumoconiosis).
March 13, 2000	DX 19	Vongkasemsiri	Emphysema, bilateral chronic interstitial disease (negative for pneumoconiosis).
March 14, 2000	DX 19	Vongkasemsiri	Emphysema, bilateral chronic interstitial disease (negative for pneumoconiosis).
March 16, 2000	DX 19 & DX 23	Norris	Emphysema (negative for pneumoconiosis).
(same)	DX 22	Phillips	Emphysema, granulomatous calcification (negative for pneumoconiosis).
May 5, 2000	DX 19	Vongkasemsiri	Emphysema, increased mass right upper lung (negative for pneumoconiosis).

Of the above 27 chest x-rays, the physicians who reviewed 24 of the films failed to find evidence of pneumoconiosis. As a result, I consider those films (February 19, 1992; May 11, 1992; February 23, 1997; February 26, 1997; March 3, 1997; April 16, 1997; April 24, 1997; April 25, 1997; September 11, 1998; October 9, 1998; February 26, 1999; June 29, 1999; January 1, 2000; January 9, 2000; February 25, 2000; February 27, 2000; March 3, 2000; March 5, 2000; March 7, 2000; March 13, 2000; March 14, 2000; March 16, 2000; and, May 5, 2000) negative for pneumoconiosis.

The medical experts disagreed on the results of the other four films. Dr. Baker, Dr. Mathur, Dr. Marshall, and Dr. Goldstein saw pneumoconiosis in the July 17, 1998 chest x-ray. Since their consensus outweighs the contrary opinions of Dr. Sargent and Dr. Barrett, I find the July 17, 1998 is positive for pneumoconiosis. In a similar manner, the negative findings by Dr. Sargent and Dr. Barrett, both dual qualified radiologists, for the August 17, 1998 chest x-ray prevail over Dr. Baker's sole positive finding as a B reader; and the negative readings of the September 24, 1998 film by Dr. Spitz and Dr. Shipley outweigh Dr. West's positive finding.

Consequently, the August 17, 1998 and September 24, 1998 chest x-rays are negative for pneumoconiosis. Finally, based on the negative readings by Dr. Dahhan, Dr. Wiot, Dr. Spitz, Dr. Meyer, Dr. Perme, and Dr. Sargent, in contrast to the two positive findings by Dr. Westerfield and Dr. Mathur, the August 18, 1999 chest x-ray is negative.

In summary, over the course of Mr. Ray's life, 34 chest x-rays were taken of his lungs. Three of those films, May 5, 1992, May 24, 1993, and July 18, 1999, were positive for pneumoconiosis. The remaining 31 chest x-rays, which clearly represent the preponderance of the radiographic evidence, were negative for pneumoconiosis. Consequently, Mrs. Ray is not able to establish the presence of pneumoconiosis in her husband's lungs by chest x-rays.[17]

Although Mrs. Ray is unable to demonstrate the presence of pneumoconiosis through the preponderance of the chest x-ray evidence, she may still establish this necessary element of entitlement through either autopsy/biopsy evidence or medical opinion.

Autopsy/Biopsy Evidence

(Note: the following summary of biopsy findings, and other remaining portions of this decision, contain detailed information obtained from the autopsy of Mr. Ray, submitted to support the black lung disability and survivor claims of Mr. and Mrs. Ray. While respecting the dignity and privacy of the deceased, some discussion of the detailed observations is necessary because I find the medical information relevant on determining whether Mr. Ray had pneumoconiosis.)

Prior to reviewing the autopsy report, a review of the regulatory provisions on the requisite standard for diagnosing pneumoconiosis based on a biopsy or autopsy is helpful. The regulation at 20 C.F.R. § 718.201 (a) (1) defines "clinical" pneumoconiosis as a condition:

> characterized by permanent deposition of substantial amounts of particulate matter, caused by coal dust exposure, in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis, and silicotuberculosis arising out of coal mine employment.

At the same time, because the regulatory definition of clinical pneumoconiosis requires both deposit of coal dust matter and lung tissue reaction to the deposit, an autopsy finding of anthracotic pigmentation, standing alone, is not sufficient to establish the presence of pneumoconiosis, 20 C.F.R. § 718.202 (a) (2).

[17] I note this conclusion is further supported by the preponderance of the more probative opinions on the autopsy findings and the absence of any pneumoconiosis findings in several chest CT scans.

Dr. Gary L. Adelson
(DX 21)

On October 9, 1998, Dr. Gary L. Adelson evaluated the tissue sample from a fine needle aspiration biopsy of the mass in Mr. Ray's right lung. He found malignant large cell carcinoma. The sample size was too small to determine any other pathology.

Dr. C. Blake
(DX 87)

On October 6, 2000, Dr. Blake, board-certified in anatomic, clinical and forensic Pathology, performed an autopsy. Prior to the procedure, Dr. Blake was briefly informed that Mr. Ray had been a coal miner who stopped working in the mines in 1975. Mr. Ray had smoked a pack to a pack and a half of cigarettes a day and was still smoking at the time of his death. He had been diagnosed with cancer in the right lung and received radiation therapy in lieu of surgery due to his impaired pulmonary capacity.

During gross examination of Mr. Ray's heart, Dr. Blake observed cardiac hypertrophy with severe coronary artery disease and several scars from myocardial infarctions. Upon examination of the lungs, Dr. Blake found "striking massive bullous emphysematous blebbing of the medial aspect of" the upper and middle lobes of the right lung. Additionally, the lungs were "very heavily anthracotic over the exposed anterior surface" with stippling. However, "no fibrotic nodules are present on the surface of the lung." Likewise, upon sectioning of the lungs, Dr. Blake reported, "the pulmonary parenchyma itself does not demonstrate nodular structures on the heavily anthracotic surface which would suggest or fulfill the gross criteria for black lung disease (coal miners' pneumoconiosis)." The left lung contains bullous emphysema. Dr. Blake found a 5 centimeter cancer tumor in the right lung which had penetrated into the thoracic wall. The bronchial passages were filled with material. Based on his gross examination, Dr. Blake concluded there was no "gross evidence of pathology consistent with black lung disease."

Under the microscope, cardiac and coronary artery tissue showed extensive scarring and evidence of old myocardial infarctions. The right upper lobe lung tissue slides contained lung cancer cells and some samples indicated "completely tumor-replaced lung tissue." The left lung tissue slides revealed massive mucus and purulent plugging of bronchial passages, very severe bullous emphysema, bronchopneumonia and patchy interstitial fibrosis with both bullous and pan-lobular emphysema.

According to Dr. Blake neither the gross nor microscopic examination of the lungs revealed the presence of coal miners' pneumoconiosis. His autopsy diagnosis included severe coronary artery disease, massive desmoplastic squamous carcinoma of right upper lobe of lung, and bilateral pulmonary emphysema with bronchopneumonia, pulmonary interstitial fibrosis and obstructive bronchiectasis.

Dr. Michael D. Boggan
(DX 87)

In March 2001, after reviewing the results of Dr. Blake's autopsy, and stating that the finding of bullous emphysema was consistent with clinical findings, Dr. Boggan, a board certified surgeon, opined that Mr. Ray also had black lung disease. Noting the observed presence of a heavy anthracotic background, Dr. Boggan stated that "there is little doubt that the heavy anthracosis was secondary to his exposure in coal mines (emphasis added)." Dr. Boggan explained that the "absence of nodules simply means that this exposure did not result in anthrasilicosis rather than primary anthracosis."

Dr. A. Dahhan
(DX 89)

In October 2001, Dr. Dahhan, board certified in internal and pulmonary medicine, reviewed Dr. Blake's autopsy report and Dr. Boggan's assessment. Based on Dr. Blake's detailed description, Dr. Dahhan concluded the autopsy failed to reveal the presence of coal workers' pneumoconiosis.

Dr. Ben V. Branscomb
(EX 1)

In July 2002, Dr. Branscomb, board certified in internal medicine, reviewed the autopsy findings. According to Dr. Branscomb, the observed finding of anthracotic covered lung surfaces does not support a diagnosis of coal workers' pneumoconiosis. He explained that the term "anthracosis" simply means the presence of carbon deposits which may be caused by urban living, cigarette smoking and coal mining. As a result, both Mr. Ray's coal mining and his cigarette smoking caused the observed anthracosis. In fact, based on Mr. Ray's smoking history, the discovery of anthracosis was "absolutely inevitable, even if he had not been a coal miner." The principle feature of anthracosis "is the absence of any sort of scarring, nodule formation, or other indication of any negative or adverse response by the body." On the other hand, coal workers' pneumoconiosis involves pathologic changes which may include "scarring lesions" or "nodules." In Dr. Branscomb's opinion, the "extensive pathologic examination conclusively established the absence of any occupational pneumoconiosis."

Discussion

Dr. Adelson's analysis and diagnosis of lung cancer is not particularly probative on the issue of pneumoconiosis because he did not link the lung cancer to Mr. Ray's coal mine employment[18] and was unable to express an opinion as to the presence of any other pulmonary pathology due to the insufficient amount of lung tissue.

[18]As summarized later, Dr. Boggan and Dr. Brock partially related Mr. Ray's coal mine employment to his development of lung cancer, which might support a finding of legal pneumoconiosis. However, that causation conclusion is outweighed by the opinions of Dr. Kiser, Dr. Fino, Dr. Burki, Dr. Dahhan and Dr. Branscomb that his lung cancer was caused by cigarette smoking and not exposure to coal dust. Further, neither Dr. Boggan nor Dr. Brock explained the basis for such a connection.. In contrast, both Dr. Branscomb and Dr. Dahhan emphasized that

The other four doctors to evaluate the autopsy findings reached conflicting conclusions. Dr. Boggan opined the gross examination of the lung tissue was sufficient to diagnose pneumoconiosis related to Mr. Ray's coal mine employment. Even though the autopsy failed to disclose the presence of nodules or fibrotic reaction associated with the pigment, Dr. Boggan concluded Mr. Ray's "anthracosis" was a sufficient basis for a black lung disease diagnosis. Dr. Blake, Dr. Branscomb, and Dr. Dahhan disagreed. Both Dr. Blake and Dr. Branscomb concluded the anthracotic covering of the lung surfaces alone did not satisfy the criteria for a pneumoconiosis diagnosis. While Dr. Dahhan was less specific, he also agreed the objective autopsy details did not show the presence of coal workers' pneumoconiosis. In light of this divergent medical opinion, I must assess the relative probative value of the respective assessments in light of documentation and reasoning.

As to the first factor, a physician's medical opinion is likely to be more comprehensive and probative if it is based on extensive objective medical documentation such as radiographic tests and physical examinations. *Hoffman v. B & G Construction Co.*, 8 B.L.R. 1-65 (1985). In other words, a doctor who considers an array of medical documentation that is both long (involving comprehensive testing) and deep (includes both the most recent medical information and past medical tests) is in a better position to present a more probative assessment than the physician who bases a diagnosis on a test or two and one encounter.

The second factor involves an evaluation of the connections a physician makes based on the documentation before him or her. A doctor's reasoning that is both supported by objective medical tests and consistent with all the documentation in the record, is entitled to greater probative weight. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). Additionally, to be considered well reasoned, the physician's conclusion must be stated without equivocation or vagueness. *Justice v. Island Creek Coal Co.*, 11 B.L.R. 1-91 (1988).

With these principles in mind, I first note that due to the detailed nature of Dr. Blake's findings, both upon gross examination and microscopically, each doctor based their pathology findings on essentially equal documentary footing.

However, in terms of reasoning, I am able to make a probative value distinction between the conflicting medical opinions because Dr. Boggan's diagnosis of pneumoconiosis has diminished probative value for two reasons. First, and significantly, Dr. Boggan's reasoning is inconsistent with the black lung regulations. Dr. Boggan used the term "anthracosis" to identify the autopsy finding of anthracotic coating. While under 20 C.F.R. § 718.201 (a) the term "anthracosis" is used to identify pneumoconiosis, the regulation's "anthracosis" means the presence of anthracotic pigmentation coupled <u>with</u> fibrotic reaction of lung tissue. As referenced in the beginning of this section, 20 C.F.R. § 718.202 (a) (2) specifically states the anthracotic pigmentation, standing alone, is insufficient to establish the presence of pneumoconiosis. Since Dr. Blake's autopsy report specifically indicates no tissue reaction was present in Mr. Ray's lungs, and Dr. Boggan acknowledged no lung tissue reaction was observed, his use of the term "anthracosis" does not support a finding of pneumoconiosis.

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no medical study had shown the incidence of lung cancer among coal miners was greater than the general population.

Second, and closely related, due to insufficient explanation, Dr. Boggan's comments do not support a finding that Mr. Ray had legal pneumoconiosis, which involves a pulmonary disease or impairment related to exposure to coal dust. Specifically, although anthracotic pigment was present, Dr. Boggan did not reasonably explain how the dust deposits affected Mr. Ray's lung disease and impairment represented by his lung cancer and bullous emphysema. Consequently, his post-mortem assessment does not establish that Mr. Ray's emphysema and lung cancer were in some way related to his coal mine employment in light of the anthracotic deposits. (*See also* footnote 18).

In contrast, the opinions of Dr. Blake and Dr. Branscomb that the autopsy results do not support a finding of pneumoconiosis are better reasoned, consistent with the regulatory definition of pneumoconiosis, and consequently more probative. Dr. Branscomb emphasizes that a finding of anthracotic covering only establishes that Mr. Ray was exposed to airborne elements containing carbon. In other words, both Mr. Ray's cigarette smoking and his coal mine employment, together, or separately, explain the anthracosis finding. As required by the regulation, Dr. Blake and Dr. Branscomb evaluated Mr. Ray's lung tissue in terms of a fibrotic reaction to the anthracotic coating. In Mr. Ray's case, under both gross and microscopic examination, no evidence of the requisite lung tissue reaction, such as associated fibrosis or nodules, was discovered. As a result, in the absence of tissue reaction to the coal dust deposits, the physicians reasonably determined that Mr. Ray's lungs did not contain coal workers' pneumoconiosis.

Additionally, although Dr. Dahhan did not provide much explanation, his conclusion was based on the absence of the objective medical criteria necessary for a pneumoconiosis diagnosis. His finding is consistent with conclusions of Dr. Blake and Dr. Branscomb and provides additional support.

Finally, even if the probative value of all four medical assessments were equal, the collective opinion of Dr. Blake, Dr. Branscomb, and Dr. Dahhan outweighs Dr. Boggan's conclusion. Their consensus prevails and demonstrates that the autopsy failed to establish the presence of pneumoconiosis.

Medical Opinion

Although the chest x-ray evidence and the autopsy/biopsy evidence do not establish the presence of pneumoconiosis, Mrs. Ray may still establish the presence of pneumoconiosis in her husband's lungs through documented and reasoned medical opinions. To help put these medical opinions into perspective, I will first set out the other medical evidence in Mr. Ray's present claim that was not previously summarized by Judge Teitler in his 1996 Decision and Order and then discuss the numerous opinions.

Pulmonary Function Tests

Exhibit	Date / Doctor	Age / Height[19]	FEV¹ pre[20] post[21]	FVC pre post	MVV pre post	% FEV¹ / FVC pre post	Qualified[22] pre Post	Comments
DX 14	8/17/98 Baker	67 72.75"	.86	2.81	39	31%	Yes[23]	Valid per Dr. Burki (DX 15).
DX 22 & DX 42	11/5/98 Foster	67 73"	.89 1.12	2.34 2.58	--- ---	38% 43%	Yes Yes	Invalid per Dr. Burki (DX 43).

Arterial Blood Gas Studies[24]

Exhibit	Date / Doctor	pCO_2 (rest) pCO_2 (exercise)	pO_2 (rest) pO_2 (exercise)	Qualified[25]	Comments
DX 26	8/17/98 Baker	44	70.7	No[26]	
DX 22	3/6/00 Holston Valley Medical Center	56	82	Yes[27]	

[19]In the two pulmonary examinations, separated by three months, presented in Mr. Ray's second claim, his height ranged from 72.75" to 73". I will take the average of these two heights, i.e. 72.785", for purposes of determining whether the resulting values are qualifying according to the tables in Appendix B to 20 C.F.R. § 718.

[20]Test result before administration of a bronchodilator.

[21]Test result following administration of a bronchodilator.

[22]Under 20 C.F.R. § 718.204 (b)(2)(i), to qualify for total disability based on pulmonary function tests, for a miner's age and height, the FEV1 must be equal to or less than the value in Appendix B, Table B1 of 20 C.F.R. § 718, **and either** the FVC has to be equal or less than the value in Table B3, or the MVV has to be equal **or** less than the value in Table B5, or the ratio FEV1/FVC has to be equal to or less than 55%.

[23]The qualifying FEV1 number is 2.16 for age 67 and 72.785″; the corresponding qualifying FVC and MVV values are 2.77 and 87, respectively.

[24]I have not included the arterial blood gas studies accomplished in February 1997 during Mr. Ray's hospitalization due to a wide range of tests results which occurred during a 48 hour period. Likewise, the March 2000 hospitalization blood gas studies are not included (*see* DX 23).

[25]To qualify for Federal Black Lung Disability benefits at a coal miner's given pCO^2 level, the value of the coal miner's pO^2 must be equal to or less than corresponding pO^2 value listed in the Blood Gas Tables in Appendix C for 20 C.F.R. § 718.

[26]For the pCO_2 of 40 to 49, the qualifying pO_2 is 60, or less.

[27] Any pO_2 value is qualifying if the pCO_2 value is above 50.

CT Scans

1. February 26, 1999 by Dr. James C. Phillips (DX 23), diagnosis: emphysematous changes and a few scattered minute densities.

2. August 30, 1999 by Dr. Sunnan Vongkasemsiri (DX 19), diagnosis: pulmonary emphysema, primary tumor right upper lung.

3. December 6, 1999 by Charles R. Hickam (DX 23), diagnosis: granuloma and emphysematous changes, metastastic lesion in right apex.

4. February 28, 2000 by Dr. David M. Norris (DX 17), diagnosis: pulmonary emphysema, evidence of prior granulomatous exposure, soft tissue mass right upper lung.

Bone Scan

August 31, 1999 by Dr. Vongkasemsiri (DX 19), diagnosis: possible metastasis to right ribs.

Lung Perfusion Test

March 16, 2000 by Dr. James C. Phillips (DX 22 and DX 23), diagnosis: emphysematous changes.

Physician Assessments[28]

In reviewing the radiographic and other objective medical evidence, I have referenced Judge Teitler's summaries of that type of evidence in Mr. Ray's first claim and then summarized in my decision the additional evidence associated with Mr. Ray's second claim and Mrs. Ray's survivor claim. However, I take a different approach concerning the medical opinion in this case. Because many of the physicians' recent assessments represent a continuation of their earlier evaluations of Mr. Ray's pulmonary condition in the first claim, I have included all of their reports in the summaries below.

Dr. Kenneth Kiser
(DX 33, DX 51, DX 61, DX 62, DX 93-30, and DX 93-40)

Dr. Kiser started treating Mr. Ray in 1986. He noted in a March 1993 statement that earlier medical records indicated Mr. Ray's moderate pulmonary dysfunction was first identified in 1997. Since 1986, Dr. Kiser had treated Mr. Ray several times for shortness of breath. Between January 1992 and March 1993, back pain and muscle soreness were Mr. Ray's major ailments. Dr. Kiser reported one episode of bronchospasms. He noted Mr. Ray was smoking two packs of cigarettes a day and diagnosed COPD. On one occasion, Dr. Kiser administered

[28]In general, I have not included medical notes related to Mr. Ray's abdominal ailments and other non-pulmonary problems, such as the successful treatment of colon cancer in the early 1970s.

bronchodilation therapy. He reported a March 1993 chest x-ray as positive for pneumoconiosis. A May 1993 chest x-ray only showed the presence of emphysema.

Through April 1999, Dr. Ken Kiser treated Mr. Ray for numerous ailments including chronic and progressive shortness of breath, chronic obstructive pulmonary disease, chronic bronchitis, severe bullous emphysema, granulomatous disease, and lung cancer. His initial notations include a diagnosis of coal worker's pneumoconiosis. At that time, Dr. Kiser recorded Mr. Ray's "progressive shortness of breath with decrease in patient's ability to function secondary to continued and progressive SOB [shortness of breath]. Patient unable to perform strenuous work." In other treatment notes describing many other ailments, Dr. Kiser consistently diagnosed chronic bronchitis and continued to prescribe inhalers. On occasion, he provided a nebulizer treatment. Dr. Kiser also noted Mr. Ray's unsuccessful attempt to stop smoking. In July 1996, Dr. Kiser reported that a pulmonary function test established the presence of an obstructive pulmonary defect which responded to bronchodilator. In December 1996, September 1998, and March 1999, Dr. Kiser added coal workers' pneumoconiosis as a diagnosis.

In a deposition taken July 29, 1999, Dr. Kiser indicated he first saw Mr. Ray in 1986 and diagnosed his first pulmonary problem of bronchitis in 1988. Over the course of years, Dr. Kiser treated Mr. Ray's pulmonary problems with inhalers and antibiotics. He is aware of both Mr. Ray's 20 years of coal mine employment and long term smoking history. The chest x-ray interpretations are mixed. The pulmonary function study indicates that Mr. Ray has an obstructive pulmonary defect, with slight reversibility, which is consistent with his clinical presentation. Due to his pulmonary impairment, Mr. Ray can not do any manual labor. Dr. Kiser's pulmonary diagnosis is chronic rhinitis, COPD with pneumoconiosis and lung cancer. Mr. Ray's cigarette smoking caused the lung cancer. The other pulmonary conditions were caused by a combination of allergies, tobacco abuse and pneumoconiosis; the later two causes were significant factors. Because both cigarette smoke and coal dust may cause an obstruction and diffusion problems, Dr. Kiser could not separate and quantify the degree of causation between cigarette smoke and coal dust. The slight response to bronchodilators simply indicated that Mr. Ray's pulmonary impairment has a broncho-spasm component. A September 1998 CT scan revealed the presence of emphysema and a density in the right upper lobe. A subsequent biopsy of the density found large cell carcinoma.

Dr. J. D. Pharaoh
(DX 93-13)

On October 26, 1992, Dr. Pharaoh conducted a pulmonary evaluation of Mr. Ray, who had 24 years of coal mine employment and had smoked cigarettes at up to two packs per day since he was a teenager. The chest x-ray was positive for pneumoconiosis. The blood gas studies were "good." According to the pulmonary function tests, Mr. Ray had a moderately severe and disabling pulmonary obstructive disease consisting primarily of bullous emphysema. The pulmonary obstruction was due to Mr. Ray's "tobacco abuse."

Dr. J.D. Sargent
(DX 33 and DX 93-52)

In September 1993, Dr. Sargent, board certified in pulmonary disease and internal medicine, evaluated Mr. Ray's pulmonary condition. Mr. Ray had 27 years of coal mine employment and 45 years of cigarette use at the daily rate of one to two packs, a "very significant" smoking history. He complained about shortness of breath and was being medicated with inhalers. The chest x-ray was negative for pneumoconiosis and positive for emphysema. On the film, a pocket of air, called a bulla, caused by centrilobular emphysema, which stems from cigarette use, was present. The pulmonary function test established the presence of COPD that was reversible. According to the arterial blood gas study, Dr. Sargent diagnosed hypoxemia at rest. Cigarettes caused the obstructive defect. Coal workers' pneumoconiosis does not cause only an obstruction impairment.

Dr. John H. Lobban and Dr. James J. Merrill
(DX 20 and DX 23)

Between February 23 and 28, 1997, after experiencing chest pain and suffering a myocardial infarction, Mr. Ray was hospitalized and treated by Dr. Lobban. Mr. Ray's medical history included coal workers' pneumoconiosis and an obstructive lung disease with emphysema. When a test established the presence of a total occlusion of the right coronary artery, a successful angioplasty was performed.

After a few days at home, Mr. Ray returned to the hospital on March 3, 1997 due to increasing shortness of breath. Dr. Merrill diagnosed COPD exacerbation possibly due to a drug reaction. Dr. Merrill also noted Mr. Ray's acknowledgement that he started smoking cigarettes when he returned home from the hospital. Upon examination, Dr. Merrill heard diffuse wheezing. Mr. Ray was discharged on March 6, 1997.

On March 25, 1997, Dr. Lobban examined Mr. Ray in a follow-up visit and found "no complaints in regards to angina or shortness of breath."

Dr. Glen Baker
(DX 24 and DX 25)

On August 17, 1998, Dr. Baker examined Mr. Ray. Mr. Ray had 30 years of coal mine employment. His smoking history covered 50 years at the rate of ½ to 3 packs of cigarettes per day. The physical examination disclosed "course rales." The chest x-ray was positive for pneumoconiosis. The pulmonary function test revealed a severe obstructive pulmonary disease. The blood gas test indicated the presence of mild hypoxemia at rest. Based on his examination and the test results, Dr. Baker diagnosed: a) coal workers' pneumoconiosis; b) COPD due to cigarette smoke and coal dust, and c) chronic bronchitis due to cigarette smoke and coal dust. In light of the pulmonary function test, Mr. Ray was totally disabled and unable to return to coal mining.

On October 12, 1998, recognizing that two other physicians failed to find pneumoconiosis by chest x ray, Dr. Baker reviewed his evaluation and noted that Mr. Ray had at least 21 years of coal mine employment and over 40 pack years[29] of cigarette smoking. Dr. Baker concluded that Mr. Ray's chronic obstructive airway disease was "probably most likely caused by his greater than 40 pack year history of smoking, but possibly contributed to by his 21 year history of dust exposure which is now listed as a cause of obstructive airway disease." Even though the chest x-ray was read as negative, Dr. Baker could not "exclude some contribution from his 21 year history or more of dust exposure." Concerning Mr. Ray's total disability, Dr. Baker believed the impairment was "caused mostly by his chronic obstructive airway disease with severe obstructive defect and only minimally by his pneumoconiosis."

Dr. Joseph Smiddy
(DX 22 and DX 23)

During the course of Mr. Ray's treatment with Dr. Foster, Dr. Smiddy performed a pulmonary evaluation of Mr. Ray in July of 1999. Based on the patient's medical history, examination, vital signs, past x-ray, pulmonary function study from November of 1998 and symptoms, Dr. Smiddy diagnosed "large cell lung carcinoma with underlying COPD and continued nicotine addiction." Dr. Smiddy stated that he would wait for the x-ray report that he had ordered to determine the "level of coal worker's pneumoconiosis."[30]

Dr. Larry J. Foster
(DX 22 and DX 23)

In November 1998, Dr. Foster examined Mr. Ray and attempted to improve his pulmonary capacity in order that Mr. Ray might undergo surgery for his lung cancer. Mr. Ray had 24 years of coal mine employment and had smoked cigarettes at the rate of a pack a day since he was 16 years old. Based on chest x-ray and a CT scan, Dr. Foster diagnosed "very severe obstructive lung disease," which he believed was "largely fixed" (permanent). Nevertheless, Dr. Foster attempted to improve Mr. Ray's breathing with medication. In a follow-up visit, Mr. Ray reported some improvement with his breathing; however, Dr. Foster observed he was still smoking cigarettes. Over the course of the next year, Dr. Foster followed Mr. Ray's radiation treatment and continued breathing difficulties as he experienced shortness of breath and struggled with severe COPD. Following several days of hospitalization of Mr. Ray for shortness of breath, Dr. Foster saw him in April 2000. Unfortunately, by the time of this last office visit, Mr. Ray's lung cancer had metastasized and the focus of the visit was his pain management.

Dr. Gregory Fino
(DX 33, DX 53, DX 61, and DX 93-52)

In April 1994, Dr. Fino, board certified in pulmonary disease and internal medicine, reviewed Mr. Ray's medical record up to that date. Mr. Ray had worked in the coal mining industry for 20 years. He also had smoked cigarettes at the rate of a pack and a half a day since

[29]A pack year equals the consumption of a pack of cigarettes a day for one year.

[30]The record contains no further information on Dr. Smiddy's opinion about the presence of pneumoconiosis.

he was a teenager. The preponderance of the chest x-ray evidence was negative. The arterial blood gas test results varied but nevertheless indicated Mr. Ray did not become hypoxic upon exercise. The pulmonary function tests showed a purely obstructive pulmonary defect that disabled Mr. Ray. Based on this evidence, Dr. Fino concluded Mr. Ray had a chronic pulmonary obstructive defect caused by his cigarette smoking. Dr. Fino was able to eliminate coal dust as an etiology because the pulmonary defect was purely obstructive, without a restrictive component which was more consistent with damage caused by cigarette smoke and asthma. Additionally, the blood gas test results did not show significant oxygen impairment that is usually associated with coal dust exposure and the varying nature of the test results was inconsistent with the permanent, irreversible damage caused by coal workers' pneumoconiosis.

In July 1999, after reviewing the medical evidence from Mr. Ray's second claim and noting his findings from a prior review of the evidence related to Mr. Ray's first black lung claim, Dr. Fino still found insufficient evidence of coal workers' pneumoconiosis. Notably, a CT scan of Mr. Ray's lungs revealed the presence of emphysema and failed to disclose the presence of coal workers' pneumoconiosis. Mr. Ray's lung cancer was not caused by his exposure to coal dust. Instead, Dr. Fino opined Mr. Ray's disabling impairment was due only to smoking and lung cancer.

Dr. Carroll E. Rose, Dr. Robert Bechtel, and Dr. Vivek Venkatesh
(DX 17)

On February 27, 2000, Mr. Ray was admitted to the hospital in the care of Dr. Rose due to shortness of breath and general weakness. He again presented at the emergency room on March 5, 2000 and March 14, 2000 for the same symptoms, coupled with nausea. Mr. Ray's medical history consisted of right sided lung cancer (post radiation and chemo therapies), COPD, and a prior myocardial infarction. Mr. Ray was using oxygen assistance at home. Upon examination, the physicians typically heard decreased breath sounds. After reviewing x-rays and CT scans of the chest and abdomen, the doctors diagnosed recurrent shortness of breath secondary to COPD and congestive heart failure.

Dr. N.K. Burki
(DX 16 and DX 61)

In April of 2000, Dr. Burki, board certified in internal medicine, reviewed Mr. Ray's medical evidence from 1998 and 1999, including the x-ray interpretations, all opinions and reviews, Dr. Kiser's notes and letter, and Mr. Ray's occupational history of 20 years mining coal and social history of smoking for 45 years, up to two packs a day. Based on this information, and noting lung cancer is not an occupational disease, Dr. Burki indicated Mr. Ray's lung cancer was "very likely due to the prolonged, extensive cigarette smoke exposure." Dr. Burki firmly believed that the patient's pulmonary disease was not caused by chronic dust disease of the lung or by past coal mine employment. Additionally, Dr. Burki observed that most of the B-readers interpreted the x-rays as negative for pneumoconiosis and a September 1998 CT scan did not show the presence of pneumoconiosis.

Dr. Jan Robbins, Dr. Rondal K. Brock, and Dr. Ronald J.C. Fejeran
(DX 19, DX 80, and CX 1)

Dr. Robbins, Dr. Brock and Dr. Fejeran were the primary care providers for Mr. Ray from July 1999 to October 2000 for numerous ailments. In their examinations, they consistently found decreased breath sounds. Mr. Ray's pulmonary diagnosis was COPD, bronchitis, and lung cancer. In December 1999, Mr. Ray's lung cancer had spread to his right ribs. In July 2000, Mr. Ray's condition was "end stage." Mr. Ray told them that he had worked in coal mines for 30 years. Chest x-rays and CT scans identified "masses," emphysema, hyperaeration, and scarring. Dr. Brock and Dr. Fejeran believed some of Mr. Ray's "problems were related to Black Lung Disease."

On December 19, 2002, Dr. Brock stated he had treated Mr. Ray several years for "chronic obstructive pulmonary disorder with bullous formation." Dr. Brock believed that the pulmonary function tests, x-rays and CT scans "all point to end stage chronic obstructive pulmonary disorder related to coal mine disease and smoking." In his opinion, "Mr. Ray's disease was a result of anthracotic emphysema related to exposure to coal mines which ultimately led to tumor formation as well."

Alison Bailey, R.N.
(DX 80)

Nurse Bailey attended to Mr. Ray during his final weeks. After his death, she wrote that the duration and extent of Mr. Ray's pulmonary impairment exceeded the symptoms she had observed in other lung cancer patients.

Dr. Robert L. Thomas
(DX 12)

On October 23, 2000, Dr. Thomas certified that Mr. Ray died on October 5, 2000. He listed carcinoma of the right lung as the immediate cause of death. Although an autopsy had been performed, Dr. Thomas indicated the autopsy findings were not available at the time he made his causation of death determination.

Dr. C. Blake
(DX 87)

Based on his gross and microscopic autopsy findings, Dr. Blake opined that the immediate cause of Mr. Ray's death was respiratory failure. This asphyxia was due to massive bilateral bullous emphysema and massive right upper lobe, infiltrative lung cancer. He explained that Mr. Ray's lung tissue had been replaced by both advanced emphysema and massive lung carcinoma. Additionally, the lung cancer had infiltrated into the chest wall and bronchial passage. Advanced focal bronchopneumonia was also present. Mr. Ray also had very severe coronary artery disease with almost no circulation remaining from the coronaries to the heart muscle. Neither the gross nor microscopic examination of the lungs revealed the presence of coal miners' pneumoconiosis.

Dr. Michael D. Boggan
(DX 18, DX 21, DX 23, DX 67, and DX 87)

In April 1997, Dr. Boggan repaired Mr. Ray's abdominal aortic aneurysm. Mr. Ray's medical history included a myocardial infarction a few weeks earlier with a subsequent angioplasty and chronic obstructive pulmonary disease. On physical examination, Dr. Boggan heard diffuse wheezing. Mr. Ray was still smoking a pack of cigarettes a day. Over the course of several months, Dr. Boggan monitored Mr. Ray's recovery. Then, in the fall of 1998, a mass developed in the upper lobe of Mr. Ray's right lung. A needle biopsy revealed the mass was cancerous. In considering treatment options, Dr. Boggan noted, "under most circumstances, surgical removal is definitely the therapy of choice." However, based on the Black Lung Clinic pulmonary function studies, Dr. Boggan concluded Mr. Ray's pulmonary capacity was "well below" the level considered safe for surgery. In an attempt to improve Mr. Ray's pulmonary capacity, Dr. Boggan recommended that he stop smoking cigarettes. Eventually, in January 1999, Mr. Ray received localized radiation therapy for his lung cancer. In an August 1999 follow-up, Dr. Boggan reported that Mr. Ray was doing well.

In a November 2000 letter, Dr. Boggan stated he treated Mr. Ray after he suffered a myocardial infarction. At that time, chest x-rays showed chronic obstructive pulmonary disease and an aortic aneurysm, which was subsequently repaired. In addition to Mr. Ray's cardiac condition, the discharge diagnosis included COPD with emphysema, coal workers' pneumoconiosis ("CWP"), and tobacco abuse. In 1998, a mass was discovered in Mr. Ray's right lung. A needle biopsy in October 1998 confirmed the presence of cancer in the mass. The biopsy sample was insufficient to diagnose any other pulmonary problems. Due to Mr. Ray's compromised pulmonary capacity, he was not a candidate for surgical removal of the lung. Instead, Mr. Ray received radiation therapy. Unfortunately, the mass continued to develop and Mr. Ray expired. Although Dr. Boggan had not seen the autopsy results, he believed coal workers' pneumoconiosis played a role in Mr. Ray's death. Dr. Boggan explained that while tobacco abuse can lead to lung cancer, that possibility "is multiplied by long exposure to mining over a long period of time." Mr. Ray had "a combination of COPD and severe CWP." As a result, Dr. Boggan believed the combination of these two conditions "measurably shortened his life," and coal workers' pneumoconiosis was "a significant contributing factor. . ."

Finally, in March 2001, as previously discussed, based in part on the autopsy findings, Dr. Boggan believed Mr. Ray had black lung disease.

Dr. A. Dahhan
(DX 33, DX 82, DX 89, DX 93-30, and DX 93-52)

Dr. Dahhan conducted a pulmonary evaluation of Mr. Ray on March 17, 1993. Mr. Ray claimed 27 years as a coal miner although he spent the last portion of his career as a state inspector. He had smoked cigarettes for at least 42 years at the rate of one and half to two packs a day. Upon physical examination, Dr. Dahhan heard bilateral wheezing. The arterial blood gas study showed hypoxemia at rest and normal levels upon exercise. According to the pulmonary function study, Mr. Ray had a severe airway obstruction with moderately severe air trapping. There was no restrictive component to his impairment. In the chest x-ray, Dr. Dahhan found

evidence of bullous emphysema. Based on his examination, Dr. Dahhan found insufficient evidence to diagnose coal workers' pneumoconiosis. The pulmonary function test, treatment with inhalers, and the chest x-ray indicated Mr. Ray had emphysema due to his 60 to 80 pack year cigarette smoking habit. Due to his respiratory impairment, Mr. Ray no longer had the capacity to return to coal mining. Mr. Ray did not have pneumoconiosis which is characterized by: a restrictive pattern defect demonstrated in the pulmonary function test, the worsening of blood gas test results upon exercise, and the presence of fibrosis in a chest x-ray. In Dr. Dahhan's opinion, Mr. Ray did not have any pulmonary condition, caused by, aggravated from, or contributed to by his coal mine employment.

In a deposition about a year later, Dr. Dahhan reiterated his conclusions and reasoning concerning Mr. Ray's pulmonary condition. Mr. Ray does not have coal workers' pneumoconiosis. Mr. Ray's "respiratory disease is emphysema and chronic bronchitis, a condition that has resulted from his 60 to 80 pack years of smoking." Mr. Ray is totally disabled by the pulmonary impairment. Dr. Dahhan was able to eliminate coal dust as a cause because coal workers' pneumoconiosis has a restrictive component, alters the blood gas exchange mechanisms upon exercise and appears as opacities and fibrosis in chest x-rays. None of these symptoms were present in Mr. Ray's case. Finally, Dr. Dahhan stated that coal workers' pneumoconiosis could be progressive even after the elimination of coal dust exposure only if the disease had become complicated. According to Dr. Dahhan simple coal workers' pneumoconiosis was not a progressive disease.

In June 2001, Dr. Dahhan reviewed additional medical evidence related to Mr. Ray's last year of struggle with lung cancer. His evaluation included a review of additional CT scans and chest x-rays. Based on this information, Dr. Dahhan opined that Mr. Ray died due to lung cancer and its complications. Since the lung cancer was unrelated to Mr. Ray's exposure to coal dust, Dr. Dahhan concluded that occupational dust played no role in his death.

In October 2001, Dr. Dahhan again addressed the cause of Mr. Ray's death after reviewing the autopsy report, Dr. Boggan's statement and Dr. Branscomb's September 2001 evaluation. Upon consideration of this information, Dr. Dahhan continued to believe Mr. Ray did not suffer from coal workers' pneumoconiosis. He explained that the "gold standard" for making this pulmonary diagnosis is actual tissue examination of the lung through a biopsy or autopsy. In this case, an autopsy was performed and the lung tissue failed to reveal the presence of coal workers' pneumoconiosis. Further, Dr. Dahhan unequivocally reported that Mr. Ray's cancer was not caused by, related to or contributed to by the inhalation of coal dust since it is a disease of the general public at large, "more so in the smoking population." He also opined that "Mr. Ray died as a result of cancer of the right upper lobe refractory to treatment with metastasis." Thus. Dahhan believed that Mr. Ray died only as a result of lung cancer.

Dr. Ben V. Branscomb
(DX 86, EX 1 and EX 2)

In September of 2001, Dr. Branscomb reviewed Mr. Ray's medical record from 1997 through 1999. While aware Mr. Ray had been an electrician and mechanic, Dr. Branscomb did not find any detailed occupational history in the record. He noted that most physicians

chronicled Mr. Ray's severe and "long standing" tobacco abuse. Most of the radiographic evidence, including CT scans was negative for pneumoconiosis. A pulmonary function test indicated Mr. Ray had a severe and totally disabling obstructive pulmonary disease. The arterial blood gases were either normal or inconclusive. After acknowledging his understanding of both the medical and legal definitions of pneumoconiosis, Dr. Branscomb stated that Mr. Ray did not have coal workers' pneumoconiosis. Mr. Ray was totally disabled by COPD. Cigarette smoking caused his lung cancer. Mr. Ray's coal mine employment played no role in his death.

In July of 2002, after considering the autopsy findings, as previously discussed, Dr. Branscomb explained the finding of anthracosis did not establish the presence of pneumoconiosis. Dr. Branscomb unequivocally found that the extensive pathologic examination conclusively established the absence of any occupational pneumoconiosis.

He also disagreed with at least two of Dr. Boggan's assertions. First, according to Dr. Branscomb, scientific studies did not show an increased incidence of lung cancer in coal miners. Second, Mr. Ray's emphysema was not due to his coal mine employment. Coal dust-related emphysema occurs in the presence of severe, or complicated pneumoconiosis, due to the associated extensive scarring. In Mr. Ray's case, the "pattern of emphysema, its autopsy characteristics, and its clinical findings . . . are not those of chronic lung disease aggravated or caused by coal dust exposure." Due to the severity of his lung cancer, the timing of Mr. Ray's death was "inevitable." According to Dr Branscomb, Mr. Ray "would not have lived longer had he had neither emphysema nor coronary disease."

In January 2003, after reviewing Dr. Brock's December 2002 assessment, Dr. Branscomb stated Dr. Brock's:

> reference to 'anthracotic emphysema related to exposure to coal mines which ultimately led to tumor formation as well' is medically meaningless. Anthracosis represents carbon deposits in the lung and is seen in most smokers, coal miners, and urban dwellers whether or not they have emphysema or whether or not they have CWP [coal workers' pneumoconiosis]. It is not a disease but the normal way carbon is handled. Neither anthracosis or emphysema cause or predispose to the formation of tumors. Coal mine dust exposure and CWP notably <u>do not</u> increase the likelihood or accelerate the rate of tumor formation.

To support his position, Dr. Branscomb emphasized that "cigarette smoking is the cause of lung cancer in over 95% of the cases." Regardless of whether Mr. Ray had legal or medial pneumoconiosis, he would have died in the same manner and at the same time due to the severe lung cancer. That lung cancer was caused by Mr. Ray's persistent heavy tobacco exposure.

Discussion

The following medical care specialists and providers either did not actually render a pulmonary diagnosis concerning the presence of pneumoconiosis or simply mentioned black lung disease as part of Mr. Ray's medical history: Dr. Merrill, Dr. Lobban, Dr. Rose, Dr. Bechtel, Dr. Venkatesh, Dr. Robbins, and Nurse Bailey. In a slightly different manner, even

though he administered a pulmonary evaluation, Dr. Smiddy did not state whether Mr. Ray had pneumoconiosis because he was waiting for a chest x-ray interpretation. If Dr. Smiddy ever responded to such an interpretation, it is not in the record. Similarly, Dr. Thomas' terse conclusion on Mr. Ray's death certificate that lung cancer killed Mr. Ray lacks the documentation associated with the autopsy results and does not address whether pneumoconiosis might also have been present in Mr. Ray's lungs.

The remaining medical opinions present conflicting views on whether Mr. Ray had black lung disease. Again, due to this disagreement among the medical experts, I must assess the relative probative value of their opinions.

Dr. Foster was Mr. Ray's pulmonary physician from 1998 to 2000 and aware that Mr. Ray was a retired coal miner. During the course of their relationship, Dr. Foster evaluated Mr. Ray's chest x-rays and CT scans, conducted pulmonary examinations, assisted in the treatment of his lung cancer, and hospitalized Mr. Ray for breathing problems. Consequently, he was in an excellent position to provide a well documented and reasoned medical opinion. His pulmonary diagnoses contained only two components: lung cancer and severe chronic obstructive pulmonary disease. His diagnostic silence in regards to pneumoconiosis might indicate that he believed Mr. Ray did not have black lung disease. However, Dr. Foster was focused principally on treatment of Mr. Ray's lung cancer and the attempt to restore some pulmonary capacity to permit surgery. As a result, since Dr. Foster was never asked to clarify his view on the presence of pneumoconiosis, I do not consider the absence of such a diagnosis in his medical reports to be of significant probative value.[31]

Although Dr. Pharaoh referenced a chest x-ray that was positive for pneumoconiosis, that conclusion has diminished probative value for two reasons. First, despite that chest x-ray interpretation, Dr. Pharaoh did not include pneumoconiosis in his pulmonary diagnosis, thus rendering his opinion somewhat equivocal. Second, due to the date of his evaluation, Dr. Pharaoh's opinion is not as well documented as the conclusion by doctors who were able to evaluate the autopsy findings.

Initially, after considering a positive chest x-ray, Dr. Baker diagnosed coal workers' pneumoconiosis, COPD, and chronic bronchitis. He opined the latter two pulmonary ailments were due to both cigarette smoke and coal dust. Subsequently, after he became aware that the chest x-ray interpretations were mixed on the presence of pneumoconiosis, Dr. Baker became less certain about the coal dust etiology. After citing cigarette smoke as the most likely cause of Mr. Ray's obstruction, Dr. Baker added that coal dust "possibly" was a contributing factor. Due to the equivocal nature of that final conclusion, Dr. Baker's opinion loses probative value.

Dr. Sargent's conclusion that Mr. Ray did not have pneumoconiosis has diminished probative value due to a reasoning deficiency. As previously mentioned, under the regulations, pneumoconiosis may be any pulmonary disease that is related to coal dust exposure (legal pneumoconiosis). Dr. Sargent's explanation that pneumoconiosis can not cause only an obstructive impairment, reflects an emphasis solely on medical pneumoconiosis. In other words,

[31]Interestingly, Dr. Smiddy's pulmonary evaluation, with its deferred assessment on pneumoconiosis, was part of Dr. Foster's treatment notes.

Dr. Sargent's explanation shows the absence of any consideration of whether Mr. Ray suffered from legal pneumoconiosis.

Dr. Fino's detailed assessment of Mr. Ray's medial record has the same reasoning defect. To explain how he was able to eliminate coal dust as an etiology, he explained that Mr. Ray had a "pure obstructive pulmonary defect," without a restrictive component. His emphasis on the absence of a restrictive component reflects a focus on medical pneumoconiosis and no consideration of whether Mr. Ray may have had legal pneumoconiosis.

Dr. Buki's conclusion that Mr. Ray did not have pneumoconiosis is somewhat diminished in probative value because his opinion is not as well documented as the assessments of other doctors who were able to consider the autopsy findings. Additionally, due to the terse nature of his assessment, I am not able to ascertain how he was able to eliminate coal dust as an aggravating factor in Mr. Ray's COPD.

Although Dr. Brock and Dr. Fejeran diagnosed pneumoconiosis in the form of coal-dust related emphysema, their opinions have diminished value for reasoning and documentation deficiencies. Neither doctor explained the basis for his respective conclusion that Mr. Ray's COPD was related to his exposure to coal dust and cigarettes.[32] Additionally, in light of the autopsy findings showing bullous emphysema without any visible connection to the coal dust deposits on Mr. Ray's lungs, their opinions are not as well documented as the other medical specialists who reviewed the pathology findings.

Dr. Kiser, as Mr. Ray's long term treating physician provided a reasoned, though not exceptionally detailed, conclusion that Mr. Ray's long term exposure to both cigarette smoke and coal dust exposure contributed to his pulmonary obstruction. Additionally, based on extensive contact with Mr. Ray, he was able to acquire significant documentation that added to the value of his diagnosis. Nevertheless, because Dr. Kiser apparently lost contact with Mr. Ray in April 2000, his opinion ultimately has diminished probative value because he was unaware of the nature and manner of Mr. Ray's death a few months later, and most significantly, the results of the autopsy report. Consequently, despite his status as treating physician, Dr. Kiser's opinion is not as probative as the assessments of the four doctors who considered the post-mortem evidence, which was very significant in determining whether a link existed between Mr. Ray's obstructive pulmonary impairment and his coal mine employment.

Not surprisingly, since for various reasons most of the assessments of the doctors who evaluated Mr. Ray's pulmonary condition during his life have diminished relative probative value, the determination of whether Mr. Ray had pneumoconiosis fundamentally rests on the opinions of the four doctors who based their conclusions, in part, on the very significant autopsy findings.

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[32]Dr. Brock also stated Mr. Ray's coal dust exposure contributed to the development of his lung cancer tumor. As previously noted, his causation opinion is outweigh by other medical opinion, *see* footnote 18. Additionally, Dr. Brock's statement is not well reasoned since he provided no explanation for that portion of his opinion. Finally, the autopsy findings disclosed no connection between the lung cancer tumor and the observed coal dust deposits.

Based on his familiarity with Mr. Ray's medial record as a surgeon who treated him and his review of the autopsy report, Dr. Boggan presented a well-documented conclusion that Mr. Ray had coal workers' pneumoconiosis. Prior to the autopsy, based on Mr. Ray's long history of coal mining and cigarette smoking, Dr. Boggan had diagnosed coal workers' pneumoconiosis. Following the autopsy, which revealed significant anthracotic deposits, Dr. Boggan believed his diagnosis was validated. Concluding the gross post-mortem examination findings showed the presence of anthracosis, Dr. Boggan presented a firm diagnosis of coal workers' pneumoconiosis. However, as I previously discussed, in terms of reasoning, Dr. Boggan's diagnosis is deficient for two reasons. First, although he used the term "anthracosis," Dr. Boggan's reliance simply on the presence of coal dust matter on the lungs does not satisfy the regulation's definition of medical pneumoconiosis because Mr. Ray's lung tissue associated with that deposit did not react to its presence. Second, to the extent that Dr. Boggan's conclusion represents a finding of legal pneumoconiosis, Dr. Boggan failed to explain how the anthracotic pigment on the outer surface of the lungs, and in the absence of tissue reaction, caused, contributed to, or aggravated his bullous emphysema and lung cancer tumor.

In contrast, based on his direct observations, Dr. Blake found the anthracotic pigmentation insufficient to meet the established criteria for a diagnosis of coal workers' pneumoconiosis. Dr. Branscomb and Dr. Dahhan,[33] using extensive documentation from Mr. Ray's medical records and the detailed autopsy findings separately reached the same conclusion. In particular, Dr. Branscomb's diagnosis of obstructive pulmonary disease and lung cancer due to cigarette smoke represents the best documented and reasoned medical opinion in the record and is most consistent with all the objective medical evidence associated with Mr. Ray's pulmonary condition.

The probative opinions by Dr. Blake, Dr. Branscomb and Dr. Dahhan, representing the most probative medical opinion on the issue of pneumoconiosis, definitively establish that Mr. Ray did not have black lung disease. As noted before, even if Dr. Boggan's conclusion that Mr. Ray had anthracosis was reasonably valid, his finding of pneumoconiosis would still be outweighed by the well documented and reasoned consensus of Dr. Blake, Dr. Branscomb, and Dr. Dahhan, that Mr. Ray's lungs did not contain pneumoconiosis. Accordingly, Mrs. Ray is not able to establish the presence of pneumoconiosis through medical opinion.

Disability Claim

Mr. Ray's claim for disability benefits under the Act comes to me as a denial of a modification request to the initial denial of his duplicate claim. As I informed the parties at the hearing, I will subsume the issue of whether Mr. Ray established either a change in condition or a mistake of fact by re-adjudicating Mr. Ray's second claim under the material change of condition provisions of the regulations.

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[33]Prior to Dr. Dahhan's review of the autopsy evidence, his opinion suffered a loss in probative value because he believed that coal workers' pneumoconiosis only in its complicated form was progressive, which is contrary to the regulatory determination at 20 C.F.R. § 781.201 (c) that pneumoconiosis, whether complicated or not, is a latent and progressive disease. That probative deficiency became less relevant to Dr. Dahhan's final diagnosis which he based on the observed autopsy findings rather than the purported non-progressive nature of simple pneumoconiosis.

Any time within one year of a denial or award of benefits, any party to the proceeding may request a reconsideration based on a change in condition or a mistake of fact made during the determination of the claim; *see* 20 C.F.R. § 725.310. However, after the expiration of one year, the submission of additional material or another claim is considered a duplicate claim which will be denied unless the claimant demonstrates a material change in conditions under the provisions of 20 C.F.R. § 725.309, as interpreted by the Benefits Review Board and federal Courts of Appeals. Under this regulatory provision, according to the Court of Appeals for the Sixth Circuit in *Sharondale Corp. v. Ross*, 42 F.3d 993, 997-998 (6th Cir. 1994):

> [T]o assess whether a material change is established, the ALJ must consider all of the new evidence, favorable and unfavorable, and determine whether the miner has proven at least one of the elements of entitlement previously adjudicated against him. If the miner establishes the existence of that element, he has demonstrated, as a matter of law, a material change. Then, the ALJ must consider whether all of the record evidence, including that submitted with the previous claims, supports a finding of entitlement to benefits.

I interpret the *Sharondale* approach to mean that the relevant inquiry in a material change case is whether evidence developed since the prior adjudication would now support a finding of an element of entitlement. The court in *Peabody Coal Company v. Spese*, 117 F.3d 1001, 1008 (7th Cir. 1997) put the concept in clearer terms:

> The key point is that the claimant cannot simply bring in new evidence that addresses his condition at the time of the earlier denial. His theory of recovery on the new claim must be consistent with the assumption that the original denial was correct. To prevail on the new claim, therefore, the miner must show that something capable of making a difference has changed since the record closed on the first application.

In determining whether there has been a material change in condition, I focus on the four basic conditions, or elements, a claimant must prove by a preponderance of the evidence to receive black lung disability benefits under the Act. First, the miner must establish the presence of pneumoconiosis. Second, if a determination has been made that a miner has pneumoconiosis, it must be determined whether the miner's pneumoconiosis arose, at least in part, out of coal mine employment.[34] Third, the miner has to demonstrate he is totally disabled.[35] And fourth, the miner must prove the total disability is due to pneumoconiosis.[36]

With those four principle conditions of entitlement in mind, the next adjudication step requires the identification of the conditions of entitlement a claimant failed to prove in the prior claim. In that regard, of the four principle conditions of entitlement, the only elements that are

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[34]20 C.F.R. § 718.203 (a).

[35]20 C.F.R. § 718.204 (b).

[36]*Id.*

capable of changing are whether a miner has pneumoconiosis or whether he is totally disabled. *Lovilia Coal Co. v. Harvey*, 109 F.3d 445 (8th Cir. 1997). That is, the second element of entitlement (pneumoconiosis arising out of coal mine employment) and the fourth element (total disability due to pneumoconiosis) require preliminary findings of the first element (presence of pneumoconiosis) and the third element (total disability).

In Mr. Ray's case, Judge Teitler determined that the evidence established Mr. Ray was totally disabled due to pulmonary impairment. At the same time, Judge Teitler still denied Mr. Ray's first claim in February 1993 because he failed to prove the presence of pneumoconiosis. Consequently, for purposes of adjudicating the present duplicate claim, I must evaluate the evidence developed since February 1993 to determine whether Mr. Ray can now prove the presence of pneumoconiosis, thereby establishing a material change in conditions.

I have already accomplished that evaluation. In the adjudication of Mrs. Ray's survivor claim, I determined that the preponderance of the more probative radiographic, autopsy, and medial opinion evidence, including the evidence developed since February 1993, failed to establish the presence of pneumoconiosis. That same finding applies to consideration of Mr. Ray's claim for black lung disability benefits. Specifically, the preponderance of the more probative evidence, in particular the autopsy assessments of Dr. Blake, Dr. Dahhan, and Dr. Branscomb, fail to show that Mr. Ray had black lung disease.

CONCLUSION

The preponderance of the chest x-rays, more probative autopsy evaluations, and more probative medical opinions in the record fail to establish the presence of coal workers' pneumoconiosis in Mr. Ray's lungs. Since an essential element of entitlement in Mrs. Ray's case is the presence of pneumoconiosis, her survivor claim must be denied. Likewise, because the preponderance of the evidence developed since the denial of Mr. Ray's prior claim does not show the presence of black lung disease, a material change in conditions has not been established and in accordance with 20 C.F.R. § 725.309 (d), Mr. Ray's present, duplicate claim for benefits under the Act must also be denied.

ORDER

Accordingly, the claim of MRS. DELANA RAY for survivor benefits, and the claim of MR. CHARLES B. RAY for disability benefits, under the Act are **DENIED**.

SO ORDERED:

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RICHARD T. STANSELL-GAMM
Administrative Law Judge

Date Signed: March 24, 2004
Washington, DC

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date this decision is filed with the District Director, Office of Worker's Compensation Programs, by filing a notice of appeal with the Benefits Review Board, ATTN.: Clerk of the Board, Post Office Box 37601, Washington, DC 20013-7601. See 20 C.F.R. § 725.478 and § 725.479. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, DC 20210.