U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 17 February 2004

Case No. 2002-BLA-414

In the Matter of:
DORIS E. TREASE, Widow of
DAVID TREASE,
Claimant,

v.

VALLEY CAMP OF UTAH, INC.,
Employer,
and
VALLEY CAMP COAL COMPANY,
c/o ACCORDIA EMPLOYERS SERVICE,
Carrier,

and
DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
Party-in-Interest.

APPEARANCES:[1]
Martin J. Linnet, Esq.
On behalf of Claimant

William S. Mattingly, Esq.
On behalf of Employer/Carrier

BEFORE: THOMAS F. PHALEN, JR.
Administrative Law Judge

DECISION AND ORDER – DENIAL OF BENEFITS

This is a decision and order arising out of a claim for benefits under Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended by the Black Lung Benefits Act of 1977, 30 U.S.C. §§ 901-962, ("the Act") and the regulations thereunder, located in Title 20 of

[1] The Director, Office of Workers' Compensation Programs notified the undersigned through a letter dated March 31, 2003 that it did not intend to appear at the hearing.

the Code of Federal Regulations. Regulation section numbers mentioned in this Decision and Order refer to sections of that Title.[2]

On July 30, 2002, this case was referred to the Office of Administrative Law Judges by the Director, Office of Workers' Compensation Programs, for a hearing. (DX 26).[3] A formal hearing on this matter was conducted on July 17, 2003, in Price, Utah by the undersigned Administrative Law Judge. All parties were afforded the opportunity to call and to examine and cross examine witnesses, and to present evidence, as provided in the Act and the above referenced regulations.

ISSUES

The sole issue in this case is:

1. Whether the Miner's death was due to pneumoconiosis;

(DX 26). At the hearing, Employer withdrew as contested issues the issues of whether the miner had pneumoconiosis. Prior to the hearing, Employer did not contest the timeliness of the claim, status as a miner, post-1969 employment, coal mine employment of at least 32 years, that pneumoconiosis arose out of coal mine employment, whether Claimant was an eligible survivor, and it did not contest its identity as the proper responsible operator. However, for appellate purposes, Employer contested additional issues as listed in its letter dated January 19, 2001.

Based upon a thorough analysis of the entire record in this case, with due consideration accorded to the arguments of the parties, applicable statutory provisions, regulations, and relevant case law, I hereby make the following:

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Background

David Trease ("Miner") was born on April 21, 1934 and he died on January 20, 2000. (DX 1, 5). He married Doris (Wright) Trease ("Claimant') on March 25, 1975, and they remained married until the time of his death. (DX 1, 4; Tr. 11). Claimant has not remarried since Miner's death. (Tr. 11). I find that Claimant is an eligible surviving spouse of Miner.

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[2] The Department of Labor amended the regulations implementing the Federal Coal Mine Health and Safety Act of 1969, as amended. These regulations became effective on January 19, 2001, and are found at 65 Fed. Reg. 80, 045-80,107 (2000)(to be codified at 20 C.F.R. Parts 718, 722, 725 and 726). On August 9, 2001, the United States District Court for the District of Columbia issued a Memorandum and Order upholding the validity of the new regulations. All citations to the regulations, unless otherwise noted, refer to the amended regulations.

[3] In this Decision, "DX" refers to the Director's Exhibits, "EX" refers to the Employer's Exhibits, "CX" refers to the Claimant's Exhibits, and "Tr" refers to the official transcript of this proceeding.

Procedural History

Miner filed an initial claim for benefits under the Act on October 19, 1989. (DX 1). Miner requested a formal hearing before the Office of the Administrative Law Judges after his claim was denied by the Director, Office of Workers' Compensation Programs ("OWCP"). Administrative Law Judge Daniel Stewart conducted a formal hearing and issued a decision and order denying benefits on October 26, 1992. Administrative Law Judge Stewart found that Claimant established 30 years of coal mine employment and found Employer to be the proper responsible operator. After he determined that Miner did not establish the presence of pneumoconiosis, Administrative Law Judge Stewart found that Claimant was not entitled to benefits. No further action was taken by Miner on his initial living miner claim.

Miner died on January 20, 2000. (DX 5). On September 27, 2000, Claimant filed a claim for survivor benefits under the Act. (DX 2). On April 23, 2001, OWCP determined that Claimant was entitled to benefits. (DX 16). Employer requested a formal hearing on May 4, 2001. (DX 17). On July 30, 2002, this matter was transferred to the Office of the Administrative Law Judges for a formal hearing. (DX 26). Administrative Law Judge Daniel Roketenetz was assigned to the matter and he issued a notice of hearing on March 11, 2003. On June 23, 2003, Administrative Law Judge Roketenetz reassigned the matter to the undersigned. A formal hearing was conducted by the undersigned on July 17, 2003.

Length of Coal Mine Employment

David Trease was a coal miner within the meaning of § 402(d) of the Act and § 725.202 of the regulations. I find that he engaged in at least 30 years of coal mine employment.

Responsible Operator

Liability under the Act is assessed against the most recent operator which meets the requirements of § 725.493(a)(1). The District Director identified Valley Camp Coal Company as the putative responsible operator. (DX 10). Valley Camp Coal Company does not contest their identification as the proper responsible operator. Valley Camp Coal Company is the employer with whom Miner spent his last cumulative one year period of coal mine employment and is properly designated as the responsible operator in this case. § 725.493(a)(1).

MEDICAL EVIDENCE

X-RAY REPORTS

Exhibit	Date of X-ray	Date of Reading	Physician/Qualifications	Interpretation
DX 21	11/30/99	10/17/01	Wheeler, BCR[4], B-reader[5]	No CWP

[4] A physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology, Inc., or the American Osteopathic Association. *See* 20 C.F.R. § 727.206(b)(2)(III). The qualifications of physicians are a matter of public record at the National Institute of Occupational Safety and Health reviewing facility at Morgantown, West Virginia.

DX 21	12/2/99	10/17/01	Wheeler, BCR, B-reader	No CWP
DX 21	12/9/99	10/17/01	Wheeler, BCR, B-reader	No CWP
DX 21	12/16/99	10/17/01	Wheeler, BCR, B-reader	No CWP
DX 21	12/27/99	10/17/01	Wheeler, BCR, B-reader	No CWP
DX 21	12/30/99	10/17/01	Wheeler, BCR, B-reader	No CWP
DX 21	1/2/00	10/17/01	Wheeler, BCR, B-reader	No CWP
DX 21	1/5/00	10/17/01	Wheeler, BCR, B-reader	No CWP
DX 21	1/15/00	10/17/01	Wheeler, BCR, B-reader	No CWP
DX 21	1/18/00	10/17/01	Wheeler, BCR, B-reader	No CWP

Hospital Records

Miner was admitted to Rose Medical Center in Denver, Colorado on November 6, 1995 by William Pluss, M.D. (EX 4). He submitted to a pulmonary function test ("PFT"), which Dr. Pluss interpreted as revealing severe airway obstruction with significant response to bronchodilation. He found that Miner's lung volumes reveal severe hyperinflation. Dr. Pluss also noted that Miner's diffusing capacity was severely reduced. Dr. Pluss commented that Miner was a sixty-one year-old male with a ten year history of emphysema who stopped smoking in 1989. He noted that Miner's symptoms were primarily shortness of breath, a chronic productive cough, occasional swelling in his ankles, and a general inability to do anything over the past five years. Dr. Pluss noted that Miner had been evaluated by physicians in Utah for potential lung transplantation, but Miner rejected such an approach. Miner was also evaluated by physicians in California but was turned down for surgical treatment of emphysema. Dr. Pluss noted that Miner had been using supplemental oxygen for the past six years. He noted that Miner was a truck driver and coal miner who smoked one pack per day for forty-three years, ending in 1989. Upon physical examination, Dr. Pluss detected markedly decreased breath sounds, but no wheeze or crackle. Dr. Pluss reviewed the aforementioned PFT, as well as an ABG. He noted that CAT scan of Miner's chest showed diffuse emphysema with Type III. He interpreted a chest x-ray as showing hyperexpanded lung fields and slightly enlarged pulmonary arteries. Dr. Pluss' impression was chronic obstructive pulmonary disease ("COPD"), but no acute process. He found that Miner's first problem was emphysema, noting that Miner was a reasonable candidate for STE surgery. He found that Miner also had problems related to hypothyroidism, penicillin allergy, and a history of reflux esophagitis. His plan was to admit

[5] A "B" reader is a physician who has demonstrated proficiency in assessing and classifying x-ray evidence of pneumoconiosis by successful completion of an examination conducted by or on behalf of the Department of Health and Human Services. This is a matter of public record at HHS National Institute for Occupational Safety and Health reviewing facility at Morgantown, West Virginia. (42 C.F.R. § 37.5l) Consequently, greater weight is given to a diagnosis by a "B" Reader. *See Blackburn v. Director, OWCP*, 2 B.L.R. 1-153 (1979).

Miner for STE surgery, followed with aggressive respiratory care postoperatively. He noted that Miner might need antibiotic care because of his chronic bronchitis.

On November 9, 1995, Dr. John Simon performed surgical laser treatment to Miner's right lung for Miner's emphysema. (EX 4). He noted that Miner tolerated the procedure well. Following the procedure, Miner developed tachycardia at a rapid rate and was transferred to the Intensive Care Unit. Dr. Cohen treated Miner's tachycardia. By November 13, 1995, Miner had developed increasing respiratory distress. A chest x-ray revealed an increasing pneumothorax, which was treated with chest-tube placement. Due to his respiratory distress, Miner was intubated and ventilated. He then underwent placement of four chest tubes for his pneumothoraces. Miner was discharged on November 18, 1995 in fair condition. Dr. Simon's diagnoses on discharge were emphysema, pneumothoraces, respiratory failure, atrial fibrillation, and urinary tract infection.

Dr. Pluss conducted a follow-up PFT on March 18, 1996. (EX 4). He interpreted the results as revealing severe airway obstruction with marked response to bronchodilators. Dr. Pluss found Miner's lung volumes to reveal moderate hyperinflation. He noted that Miner's diffusing capacity was severely reduced.

Doug Ross, M.D. evaluated Miner on July 1, 1998 at the Utah Valley Regional Medical Center. (EX 5). He noted that Miner was a sixty-six year-old man who had emphysema that was first diagnosed in 1985. Dr. Ross noted that Miner had lung volume reduction surgery performed three years ago in Denver, Colorado. He considered a fifty pack-year smoking history. He also noted that Miner was currently using supplemental oxygen and nebulizers. Dr. Ross noted a medical history of COPD, status post lung volume reduction surgery with laser surgery and lung resection, hyperthyroidism, gout, peptic ulcer disease, hernia repair, and deviated left septum. He did not conduct any laboratory testing. Dr. Ross' assessment was severe COPD that was status post lung volume reduction surgery, possible pulmonary fibrosis, history of atrial fibrillation, hypothyroidism, gout, and chronic hypoxemia. Dr. Ross did not plan any medication changes. He discussed Miner's advance lung disease and oxygen therapy.

Miner was admitted to the Utah Valley Regional Medical Center emergency room on July 7, 1998 by Dr. Michael Pearce due to an exacerbation of COPD. (EX 5). Dr. Pearce admitted Miner to the hospital due Miner's worsening symptomatology, started Miner on intravenous steroids, aggressive nebulizer treatment, and intravenous antibiotics. Miner was discharged on July 11, 1998 with diagnoses of COPD, hyperglycemia, atrial fibrillation, and histories of hypertension, peptic ulcer disease, gout, hernia repair, as well as being status post lung reduction surgery, and chronic hypoxemia.

Dr. Pearce conducted a follow-up visit with Miner on July 31, 1998. (EX 5). He noted that Miner was doing okay with his COPD, and he recommended a follow-up in three months.

On September 8, 1999, Miner returned to Dr. Ross, complaining of increasing dyspnea and some sweats. (EX 5). Dr. Ross' assessment was that Miner had advanced and severe COPD status post lung volume reduction surgery that was now in exacerbation. He also found that Miner was suffering from left lower lobe patchy commune acquired pneumonia, diabetes

mellitus, hypothyroidism, and hypertension. Dr. Ross admitted Miner to the hospital and administered intravenous antibiotics, respiratory therapy, and cortical steroids. He noted that Miner's lung problem was very advanced and considered any type of pneumonia a worrisome problem. Dr. Ross planned to follow Miner closely and treat him aggressively. During Miner's hospital course, he developed gastric outlet obstruction. A CT scan revealed a hematoma-like mass that was treated with tube feedings and parenteral nutrition, but it did not relieve spontaneously. Thus, an exploratory laporatomy with retroperitoneal biopsies, cholecsystomy with evacuation of the retroperitoneal hematoma was required. At the time of discharge on November 22, 1999, Dr. Ross noted that Miner had completed most of his care for respiratory disease, and Miner had cleared his pneumonia. Dr. Ross assessed Miner's respiratory condition as better. He noted that Miner needed to be stronger because he was quite de-conditioned. He discharged Miner to the transitional care unit. Dr. Ross' diagnoses on discharge were pneumonia, exacerbation of end-stage lung disease, gastric outlet obstruction, retroperitoneal hematoma, and deconditioning.

Steven Lewis, M.D. noted that Miner had been admitted to Dixie Regional Medical Center on November 30, 1999. (DX 7). He noted that Miner was an unfortunate sixty-five year-old man with a history of very severe obstructive airways disease. Dr. Lewis noted that Miner had a long history of heavy cigarette smoking who quit smoking ten years ago. He noted that Miner had a lung reduction surgery and an exploratory laparotomy in September 1999. Miner presented to the emergency room with marked hypercapnia and marked hypoxemia, which necessitated emergency intubation. Dr. Lewis was called to provide Miner's care in the intensive care unit. He listed Miner's problems as: (1) acute respiratory failure; (2) probably pneumonia; (3) hypotension; (4) status post cholecystectomy; (5) status post lung reduction surgery; (6) history of hypertension; (7) history of supraventricular tachy arrhythmias; and (8) history of diabetes mellitus. On physical examination, Dr. Lewis noted decreased breath sounds throughout all lungs zones, more so on the right than the left. He noted that a chest x-ray shows marked hyperexpansion of the lungs, with severe bullous disease on the right side. Dr. Lewis stated that he studied the film for a long while in order to rule-out a pneumothorax and determine that it is all bullous changes. Dr. Lewis commented that there is a question of lower lobe infiltrate. His assessment was acute respiratory failure with severe COPD and emphysema probably from aspiration pneumonia. He diagnosed hypotension and sought to rule-out sepsis. Dr. Lewis stated that Miner's prognosis was guarded due the severity of Miner's disease and his presenting condition. He planned to admit Miner to the intensive care unit, provide mechanical ventilation, antibiotics, aggressive small-volume nebulizer therapy, intravenous theophylline therapy, continued digoxin IV, and sedation with Diprivan.

James Brown, M.D. provided a consultative examination on December 8, 1999 due to Miner's feeding difficulties and high nasogastric output. (DX 7). Dr. Brown noted that Miner had not tolerated attempts to tube feed since he was admitted, and that Miner was malnourished. He noted that Miner had a recent hospitalization for gastric outlet obstruction, which led to a confirmation of a large retroperitoneal mass with encasement of duodenum, involvement of colonic mesentery, and extension into the lower abdominal area. Upon physical examination, Dr. Brown found Miner's lungs to be clear to auscultation anteriorly, with diminished breath sounds in the bases, and no wheezes. His impression was that Miner had a high nasogastric output, which led Dr. Brown to be concerned that Miner had a persistent bowel obstruction. He

found the retroperitoneal mass to be concerning, adding that it could represent dysmotility or other anatomic abnormality. He recommended an abdominal CT scan.

A CT scan of Miner's abdomen was interpreted by Dr. Steven Davis on December 9, 2000. (DX 7). His impression was that Miner's retroperitoneal mass did not result in bowel obstruction, and could represent an old walled off abscess or ruptured ulcer, inflammatory change related to a walled off duodenal diverticulum, unusual choledochal cyst, or a duplication cyst. Dr. Davis also found a small amount of insignificant ascites, advanced COPD, and several tiny low-density areas within Miner's liver.

Dr. Brown performed an endoscopy procedure on December 21, 1999 to insert a feeding tube. (DX 7). John Miller, M.D. performed a takedown of gastrojejunostomy, lysis of adhesions, and roux-en-Y gastrojejunostomy on December 28, 1999 for Miner's retroperitoneal mass and duodenal obstruction.

While Miner was at the Dixie Regional Medical Center, multiple portable chest x-rays were obtained and interpreted from November 30, 1999 through January 18, 2000. (DX 7). The chest x-rays did not conform to the standards for interpretation set forth at § 718.102. The undersigned was unable, through an exercise of discretion, to determine if the chest x-rays were negative for the presence of pneumoconiosis.

Dr. Lewis dictated a death summary on January 20, 2000. (DX 7). He listed nine final diagnoses: (1) respiratory failure; (2) severe COPD; (3) persistent ileus; (4) protein calorie malnutrition; (5) status post gastrojejunostomy; (6) takedown of gastrojejunostomy; (7) anemia; (8) pneumonia; and (9) recurrent supraventricular tachycardia. Dr. Lewis provided a brief history of a "gallant effort" by Miner who had recently been discharged by the Utah Valley Regional Medical Center for respiratory failure and creation of a gastrojejunostomy. Miner also had a large inflammatory mass in his abdomen of uncertain etiology. He noted that Miner was discharged to a nursing home, but he deteriorated from a pulmonary perspective and developed significant respiratory failure requiring intubation and mechanical ventilating in the intensive care unit. Dr. Lewis characterized Miner's hospital course primarily of respiratory failure. He noted that Miner was treated for severe COPD and a bronchopulmonary infection with intravenous antibiotics, theophyllines, corticosteroids, nebulizers, long-acting Beta-antagonists, and high-dose Atrovent. Miner showed slow improvement. He then developed a significant ileus, and gastrointestinal and surgical consultations were obtained, leading to a determination that Miner had a gastrojejunostomy created in the past that was probably necessitated by the history of the inflammatory mass surrounding his duodenum. Biopsies of the mass were non-malignant, and repeat CT scans showed that the mass had largely resolved. Since Miner's ileus persisted and his lung disease had improved, Miner's gastrojejunostomy was surgically taken down. After the surgery, Miner's ileus persisted and he could not tolerate tube feedings, so he required parenteral nutrition. Early in Miner's hospital course, physical therapy was instituted, but it was discontinued a few days prior to his death because Miner was unable to tolerate it. Miner also experienced supraventricular tachycardias, which required medication to control his heart rate. Based on Miner's condition, he was discharged from the intensive care unit to the intermediate care unit with orders not to reinstitute aggressive intensive care unit care with intubation and mechanical ventilating. Dr. Lewis stated that it became apparent that Miner's

gastrointestinal problems were resolving, other than the ileus, but Miner's pulmonary symptoms remained the key problem. Dr. Lewis characterized Miner's pulmonary symptoms as end-stage COPD with pulmonary artery hypertension, as was documented by a Swan-Ganz catheterization while in the intensive care unit. He stated that Miner slowly lost his ability to compensation his acid-base balance to achieve adequate oxygenation. Dr. Lewis noted that Miner deteriorated significantly and then expired.

Dr. Lewis signed Miner's Certificate of Death on January 27, 2000. (DX 5). Dr. Lewis listed the immediate cause of Miner's death as COPD with an underlying cause of pneumonia. He listed other significant conditions of gastric outlet and obstruction. Dr. Lewis noted that an autopsy had been performed, but he did not have those findings available to him prior to completing the cause of Miner's death.

Narrative Medical Evidence

Jerome Wiot, M.D., who is a board-certified radiologist and B-reader, interpreted a CT scan obtained on November 16, 1995. (EX 1). He found no evidence of coal workers' pneumoconiosis ("CWP"). Dr. Wiot did find that Miner had severe emphysema, stranding and loculation with pneumothorax and a thoracotomy tube in place.

Steven Lewis, M.D. issued a narrative report to Doug Ross, M.D. on May 4, 1998 after he examined Miner following a referral from physicians in Arizona. (DX 24). He found Miner to be a sixty-four year-old with profound chronic obstructive pulmonary disease ("COPD") and probably interstitial lung disease who had an episode of acute respiratory failure in Arizona that required intubation and mechanical ventilation. Miner's hospitalization was complicated by supraventricular arrhythmias. Dr. Lewis stated that Miner had been doing reasonably well since he was discharged, with no cough or sputum production. Dr. Lewis stated that Miner had a smoking history of at least a fifty pack-year history with a coal mining history of almost forty years. He noted that a chest x-ray was described to him as revealing fibrosis. Dr. Lewis noted that Miner's past medical history is remarkable for pulmonary disease. Upon physical examination, Dr. Lewis found almost no breath sounds anywhere, and when they were present, they were prolonged in the expiratory phase and there were diffuse crackles. Dr. Lewis stated that Miner obviously had severe obstructive airways disease. Additionally, he opined that Miner probably had an interstitial lung disease. He also found that Miner had cor pulmonale. He found that Miner was presently stable, although marginally. He referred Miner to Dr. Ross for further care.

On January 15, 2002, James Castle, M.D., who is board-certified in internal medicine and the subspecialty of pulmonary disease, issued a report. (DX 23). Dr. Castle reviewed and summarized Miner's medical records from Dixie Regional Medical Center, the certificate of death, the autopsy report, and the reports of Drs. Perper, Naeye, and Oesterling. Dr. Castle stated that he reviewed ten chest x-rays obtained between November 30, 1999 and January 18, 2000. He found that all ten were unacceptable for evaluation of pneumoconiosis because they were portable films and most of them were overexposed. He did identify evidence of bullous emphysema and varying degrees of pneumonia from the films. Dr. Castle opined, based on his review of Miner's medical histories, physical examinations, radiographic evaluations, hospital

records, pathology reports, and other data, that Miner did have minimal pathologic evidence of simple CWP. In addition to noting that Miner engaged in at least thirty years of coal mine employment, he also noted that Miner had a long history of heavy cigarette smoking. Dr. Castle found that Miner's coal mine employment history was of sufficient time to have developed CWP if he was a susceptible host. Similarly, he found that Miner had a sufficient smoking history to have caused COPD in the form of chronic bronchitis/emphysema and/or lung cancer in a susceptible host. Dr. Castle stated that Miner had a history of having such severe bullous emphysema that he had to undergo lung volume reduction surgery. Dr. Castle stated that there were no radiographic reports in the record to indicate the presence of CWP, but he found that the radiographic evidence did show significant bullous emphysema with varying degrees of pneumonia and atelectasis.

Dr. Castle stated that it was the opinion of the pathologists who reviewed Miner's autopsy material that there was evidence of simple CWP. He added that the prosector did not indicate that there was evidence in the final diagnosis CWP, but the prosector did indicate that the progression of findings was consistent with that process. Dr. Castle noted that Drs. Naeye and Oesterling both opined that Miner's CWP did not contribute to any impairment during Miner's life, nor did it contribute to or hasten Miner's death. He added that only Dr. Perper felt that Miner's CWP was a contributing factor to Miner's death and indicated that the centrilobular emphysema was due to Miner's coal mine dust exposure. Dr. Castle stated that Dr. Naeye explained in great detail why he disagreed with the conclusions of Dr. Perper. Dr. Castle then stated, "I would agree with his reasoning." He found it important to note that miner had severe bullous emphysema, which is not a condition associated with coal mine dust exposure. He stated that bullous emphysema involves very large dilation of air spaces and is seen with tobacco smoke induced pulmonary emphysema or from a congenital abnormality. He stated that it was also important that Miner's treating physicians did not diagnose a coal mine dust induced lung disease. In conclusion, Dr. Castle state that there was pathologic evidence of minimal simple CWP, but he opined that it was not sufficient enough to have caused him any disability during life. He found that Miner's death was due to an acute bronchopneumonia which resulted in respiratory failure complicated by surgical procedures on the abdomen. Dr. Castle added that Miner also had severe underlying bullous emphysema that was tobacco smoke induced. He opined that Miner's death was neither caused by, contributed to, or hastened in any way by the underlying simple CWP that was present pathologically.

Dr. Castle issued a second consultative report on November 13, 2002, after he reviewed additional medical evidence. (EX 8). Specifically, Dr. Castle reviewed and summarized his prior report, hospital records from 1995 through 1999 from Utah Valley Regional Medical Center and from Denver, Colorado, Dr. Wiot's x-ray report, the initial pathology report of Dr. Tomashefski and the supplemental pathology reports of Drs. Naeye and Oesterling. Dr. Castle opined, based on his review of all of the medical evidence submitted to him, that Miner did have minimal pathologic evidence of simple CWP. He opined that a review of the additional medical evidence only served to reinforce his previously stated opinions. Dr. Castle stated that the additional radiographic evidence established that there was no radiographic evidence of CWP, but he did find radiographic evidence of bullous emphysema. He found that Miner had numerous postoperative changes after undergoing volume reduction surgery complicated by multiple pneumothoraces. Dr. Castle stated that the additional physiologic studies showed that

Miner had severe airway obstruction with some degree of reversibility associated with hyperinflation and gas trapping. He noted that these were not findings typical of CWP, adding that CWP generally caused a mixed, irreversible obstructive and restrictive ventilatory defect that was not found in Miner. Dr. Castle stated that Miner's volume reduction therapy, or surgical treatment for emphysema, was a treatment for Miner's severe tobacco smoke induced bullous emphysema. He noted that bullous emphysema is not caused by CWP, it is caused by congenital abnormality or tobacco smoking. Dr. Castle characterized the newly available slides as continuing to show minimal, simple CWP. He commented that the pathologists characterized the simple CWP as so minimal "as to have not been sufficient enough to cause impairment during life or to contribute in any way to his work in the mines." Dr. Castle stated that he would concur with "these" opinions based upon his own review of the data. He then concluded that Miner's death was not caused by, contributed to, or hastened in any way by the underlying minimal simple CWP that was present pathologically. Dr. Castle noted that Miner had been admitted to the hospital for treatment of community acquired pneumonia and had developed a significant ileus. Subsequently, he noted that Miner required a choecystectomy for cholelithiasis and cholecystitis. He added that Miner then had a stormy course and was ultimately discharged to the transitional unit because of deconditioning. Dr. Castle then noted that Miner was re-admitted because of gastric outlet obstruction that required resection of his small bowel. He stated that Miner then developed respiratory failure and died as a result of complications of his surgery. Dr. Castle opined that Miner would have died as and when he did regardless of the presence of the minimal, simple CWP. He then added that Miner's bullous emphysema was caused by cigarette smoking.

Pathology Evidence

Jeffrey Goates, M.D. issued a tissue pathology report on November 9, 1995 after Miner underwent a wedge biopsy of the right upper and middle lung lobes at Rose Medical Center. (EX 4). Dr. Goates rendered a gross and microscopic description of the tissue he reviewed. In his gross description, he noted that the "moderately anthracotic pleura of each [lobe] is finely wrinkled and red to red-purple." Dr. Goates diagnosis was moderate-severe pulmonary emphysema. He also diagnosed pleural fibrosis, mild hypertensive vascular changes, anthracosis, respiratory bronchiolitis, focal organizing pneumonia, and focal dystrophic calcification.

Lalitha Gurunadham, M.D. performed an autopsy on January 20, 2000. (DX 6). The autopsy was limited to Miner's lungs. She noted a clinical history of COPD, and added she was going to investigate the lung tissue for the presence of "coal miner's disease." Dr. Gurunadham listed provisional anatomical diagnoses of severe obstructive pulmonary fibrosis with emphysema, pulmonary hypertension, and lung reduction surgery, right. Under the heading of "Block Listing and Microscopic Description," Dr. Gurunadham stated the following:

> Sections of the lungs show dilated respiratory bronchioles and small pulmonary arterioles. These are surrounded by many dust macules. Pigment laden macrophages are present within the alveoli. Patchy black parenchymal and subpleural nodules with collections of dust are also present. Emphysema is noted and appears centrilobular.

Dr. Gurunadham stated that the issue of whether the changes she detected in Miner's lungs are related to cigarette smoking is a subject of debate. She noted that the microscopic appearance of the coal dust nodule is usually characterized by accumulation of dust filed macrophages in the interstitium surrounding respiratory bronchioles and in the interlobular septa. Furthermore, the nodule may extend in a stellate fashion into surrounding alveolar septa. Additionally, fibrosis is absent or minimal and there is no evidence of inflammation. Dr. Gurunadham stated that the features she mentioned are noted in the lung tissue segments, which are findings in favor of the progression of pulmonary fibrosis secondary to CWP as compared to cigarette smoking. She added, that if further verification is indicated, the mineral content of Miner's tissue can be identified and quantitated by chemical tests or physical tests such as spectroscopy or x-ray diffraction. Dr. Gurunadham stated that light microscopy revealed fibrosis. She then stated that transmission and scanning electron microscopy can provide the location and specific identification of dust particles in pneumoconiosis. In providing her analysis, Dr. Gurunadham referenced "Surgical pathology of non-neoplastic lung disease."

On December 17, 2000, Joshua Perper, M.D., issued a consultative pathological report. (DX 8). He reviewed Miner's employment history, medical records, the January 20, 2000 autopsy report, four autopsy slides, the hospitalization records from November 30, 1999 through January 20, 2000, and Miner's certificate of death. He identified a coal mine employment history of an aggregate of thirty years. Dr. Perper cited to the November 30, 1999 admitting report of Dr. Lewis that identified a long history of heavy cigarette smoking that ended ten years prior. Dr. Perper set forth a detailed summary of Miner's hospital records, death certificate, and Dr. Gurunadham's autopsy report. Dr. Perper then rendered his findings from his microscopic observations of the four autopsy slides. In the pleura, he detected focal fibro-anthracosis with presence of birefringent silica crystals. He found severe centrilobular emphysema with focal interstitial fibrosis. Dr. Perper detected numerous anthracotic macules, small numbers of fibro-anthracotic mixed-type micro-nodules with surrounding scar emphysema and birefringent silica crystals that are present more around blood vessels and interstitial septa. He also found clusters of macrophages with anthracotic pigment and free anthracotic pigment in some of the alveoli. Dr. Perper noted dilated bronchi with focal chronic inflammatory infiltrates and dilated bronchioles. In places, he found small areas of surrounding alveoli filled with neutrophils. He detected slight to moderate thickening and sclerosis of small intra-pulmonary blood vessels. Dr. Perper found, in one place, a small foreign body granuloma with multiple multinucleated foreign body giant cells. Based on his microscopic review, Dr. Perper diagnosed simple coal workers' pneumoconiosis ("CWP"), slight to moderate, macular and micronodular. He diagnosed centrilobular emphysema, severe, with focal interstitial fibrosis. Dr. Perper opined that Miner had acute bronchopneumonia, slight to moderate, as well as sclerosis of small intra-pulmonary blood vessels consistent with the clinical laboratory finding of pulmonary hypertension, and a solitary foreign body.

Dr. Perper then answered "Medicolegal questions" posed to him by OWCP. He opined that Miner had evidence of slight to moderate simple CWP based on findings of: (1) over thirty years of occupational exposure; (2) clinical symptoms of shortness of breath that required treatment with bronchodilators, and a periodic cough with expectoration of mucus and occasional wheezing; (3) arterial blood gases indicative of severe hypoxemia and a need for

respiratory medication, supplemental oxygen, and eventual mechanical ventilation; (4) evidence of pulmonary hypertension by direct laboratory measurement and consistent microscopic pulmonary findings; (5) radiological findings were not interpreted as showing simple CWP, but no specific reading to evaluate for pneumoconiosis was performed and it is not uncommon for radiologists to miss diagnosis of simple CWP; and (6) Miner's pulmonary findings at autopsy were indicative of simple CWP, and the microscopic examination of the lung slides confirmed the presence of pneumoconiosis, with numerous macules and smaller numbers of micronodules, with associated interstitial and solid fibrosis, anthracotic pigmentation, silica crystals, scar emphysema and severe centri-lobular (centri-acinar) emphysema. Dr. Perper also noted that the autopsy prosector also recognized the microscopic evidence of simple CWP. Dr. Perper then stated that centrilobular emphysema is a known complication of heavy smoking and he noted that Miner had been a significant smoker in the past. However, Dr. Perper added that Miner had stopped smoking ten years before his demise, and noted that smoking related emphysema of lungs does not progress in severity after smoking cessation. He then stated that reliable scientific literature over the past decades has abundantly substantiated that centrilobular emphysema "is also a direct result of exposure to mixed coal mine containing silica and coal workers' pneumoconiosis." In a parenthetical following the foregoing quote, Dr. Perper stated that Dr. Naeye cited a contradicting reference but omitted the virtually tens of opposite references in the medical literature that found a causal correlation between exposure to coal mine dust and pneumoconiosis and centrilobular emphysema. He added that there is no logical reason to exclude coal mine dust as playing a significant role in causing Miner's centrilobular emphysema. Dr. Perper added that CWP and its emphysema complications have been shown in medical literature to progress after cessation of exposure to coal mine dust because of entrapped and retained intrapulmonary fibrogenic crystalline silica. Dr. Perper cited to his discussion later in his report and Appendix I to his report.

Dr. Perper also responded to the question of whether CWP was progressing in severity after cessation of occupational exposure to coal mine dust. He stated that the answer is positive. Dr. Perper argued that recent literature has substantiated that the pulmonary damage associated with exposure to coal mine dust and CW P may progress even after the cessation of occupational exposure to silica containing mixed coal mine dust. He added that such an argument is easily appreciated when considering that the inhaled silica remains trapped in the lungs and cannot be removed. Dr. Perper then cited to multiple articles and a medical-legal dictionary as sources that identified that pneumoconiosis is a progressive disease. The third question that Dr. Perper answered, was whether Miner's CWP was a result of his occupational exposure to coal mine dust. Dr. Perper stated that he answer was affirmative. He stated that Miner's occupational exposure of thirty years was more than sufficient to account for development of CWP. He added that the pathological findings of Miner's lungs clearly showed that Miner had pathological evidence of CWP with clear evidence of silica crystals in pneumoconiotic lesions of the lungs, indicative of exposure to mixed coal dust containing silica.

The fourth question that Dr. Perper answered was whether centrilobular emphysema can be caused by coal mine dust and CWP, and if so, was that the case in Miner's situation. Dr. Perper cited to several articles that establish that emphysema results from exposure to coal mine dust. Dr. Perper added that some of the studies he quoted controlled for smoking, and concluded that 'the relationship with dust exposures was only apparent among those with centriacinar

emphysema." In response to a fifth question, Dr. Perper answered that CWP was a substantial contributory cause of Miner's death both directly and indirectly through the associated centrilobular emphysema that caused hypoxemia that either triggered or aggravated fatal cardiac arrhythmia.

Dr. Perper then rendered four conclusions based upon his review of all of the medical records: (1) Miner had evidence of slight to moderate, simple CWP, with associated severe centrilobular emphysema; (2) Miner developed CWP as a result of occupational exposure to coal mine dust considering Miner's thirty years of occupational exposure; (3) Miner's cause of death was respiratory failure due to chronic and acute lung disease, with terminal bronchopneumonia; and (4) CWP with associated centrilobular emphysema was a substantial contributory cause of Miner's death both directly through hypoxemia and through hypoxemia precipitating a fatal arrhythmia. Attached to Dr. Perper's report were seven microphotographs of autopsy lung section. Dr. Perper identified the following in the seven microphotographs: (Fig. 1) fibro-anthracotic micronodule with surrounding scar emphysema; (Fig. 2) anthracotic macular pigmentation within thickened, fibrous septa. Clusters of macrophages with anthracotic pigment in alveoli; (Fig. 3) micronodule scar emphysema. Anthracotic pigmentation in adjacent, thick, fibrous septa; (Fig. 4) aggregate of fibro-anthracotic micronodules; (Fig. 5) fibro-anthracotic septum bordering large emphysematous space; (Fig. 6) foreign body granuloma with multiple multi-nucleated giant cells; and (Fig. 7) acute bronchopneumonia with alveoli filed with neutrophils (pus cells). Macular anthracotic pigmentation, perivascular and in the interstitial septa.

Dr. Perper also attached Appendix I, which was titled "Coal workers' pneumoconiosis and associated centri-lobular emphysema." In Appendix I, Dr. Perper argued that centri-lobular (centri-acinar) emphysema is known to be associated with both smoking and exposure to coal mine dust beyond any effect of smoking. He added that this is known based on numerous studies published in highly reputable medical journals. Dr. Perper alleged that some pathologists deny the role of CWP in the development of centri-lobular emphysema based on three-to-four publications, most of which he argued are obsolete and more than fifteen years-old. In support of his opinion, Dr. Perper cited to numerous articles, including an article published in 1996 by the World Health Organization and authored by the Director of the Division of Respiratory Disease of the National Institute of Occupational Safety and Health, as well as a textbook entitled *Spencer's Pathology of the Lung*, (6th Edition, 1996)(Dr. Perper quoted the following as found on p. 482 of the textbook: "postmortem studies have also shown that there is a significant excess of emphysema in coal workers with pneumoconiosis when compared to a non mining population. This is specific to centri-acinar emphysema and unrelated to pan-acinar emphysema. Its occurrence is associated with increasing amounts of dust retained in the lungs and palpable lesions.").

Dr. Perper is board-certified in anatomical and surgical pathology, as well as forensic pathology. (CX 1). Additionally, Dr. Perper obtained an LL.B. from the Law School of Hebrew University.

Richard Naeye, M.D. issued a report on May 25, 2001. (DX 19). He reviewed and summarized a copy of Miner's death certificate, an autopsy report and medical information from

Dixie Regional Medical Center, and the report of Dr. Perper. Dr. Naeye considered a coal mine employment history of thirty years and a heavy cigarettes smoking history for many years. Dr. Perper conducted a microscopic review of the four lung slides. He stated that the major abnormality present is widespread severe centrilobular emphysema in every piece of lung tissue available for review. He also noted that many foci of acute lobular pneumonia are present. Dr. Naeye detected "mucous with admixed neutrophils [sic] completely occludes airway lumina." He noted that air spaces often have large numbers of alveolar macrophages filed with a mixture of hemosiderin and anthracotic pigment. He found anthracotic pigment in small amounts beneath pleura and adjacent to small arteries and airways. Dr. Naeye recorded the presence of loosely arranged possible fibrous tissue admixed with the black pigment at some sites, which may not always be collagen. He stated that, at some sites, the fibrous tissue admixed with black pigment is likely comprised of fused alveolar walls that have broken in the genesis of the severe centrilobular emphysema. Dr. Naeye found further support for his previous statement by the absence of very tiny birefringent crystals of toxic free silica at the fused alveolar wall sites. He noted that the only birefringent crystals present within the macules are few in number and far too large in size to be toxic free silica. Dr. Naeye opined that the size of the crystals identifies them as silicates, which do not damage lung tissue.

Dr. Naeye stated that the minimal findings required to make the diagnosis of very mild, simple CWP may or may not be present. He identified the requisite criteria as the presence of a few small anthracotic macules or micronodules with evidence of associated tissue damage, with the damage being identified by the presence of fibrous tissue of focal emphysema. Dr. Naeye recalled that admixed tissue that may sometimes be collagen was found. He stated that any focal emphysema that may be present is obscured by the severe centrilobular emphysema that is present everywhere. Dr. Naeye opined that most of the findings that Dr. Perper interpreted as evidence of CWP are in fact not present or less prominent than he claims them to be. Dr. Naeye stated that anthracotic nodules that Dr. Perper identified are really macules. Dr. Naeye then wrote:

> Micronodules must be >1mm in diameter to qualify for this designation. All of the black deposits in these lungs are far smaller than 1 mm in diameter. All of the macules combined occupy no more space than 2% of all the lung tissues available for analysis.

Dr. Naeye then stated that there are no very tiny crystals of free silica in the tissue locations where foci of fibrosis may be present. He added, to identify such crystals, one must use the highest magnification possible with a light microscope and make observations in a darkened room. Using this technique, Dr. Naeye stated that there are no toxic free silica crystals in Miner's lungs. Considering all of his findings, the simple CWP in Miner's lungs is both too mild and limited in extent to have caused any measurable abnormalities in lung function, caused any disability, or hastened death. Dr. Naeye stated that Miner had a respiratory death, whose major causes were very severe centrilobular emphysema and mild to moderately severe chronic bronchitis. He opined that the terminal event was acute lobular pneumonia, which nearly reached the abscess stage at a few sites and occluded some airways.

Dr. Naeye then stated that his foregoing analysis left unanswered the question of whether Miner's occupational exposure could have had any role in the genesis of Miner's centrilobular emphysema. He opined that a critical review of the literature indicates that coal mine dust has only a clinically insignificant role in the genesis of centrilobular emphysema. Dr. Naeye identified FEV1 and FEV1/FVC values as the best way to measure the presence and severity of centrilobular emphysema. Citing to studies from 1986 and 1994, he stated that these values do not decrease with the number of years workers mine coal in longitudinal studies when age and cigarette smoking have been taken into consideration. Dr. Naeye stated that if mine dust produced centrilobular emphysema, those with the longest exposures would surely have the most emphysema and that is not the case. He contended that longitudinal studies that claim otherwise have not taken into consideration the fact that they represent a survivor population (i.e., a disproportionate number of those who left the industry before the studies concluded because of poor health or death) which greatly underestimates the effects of smoking on lung function and mortality in the coal miner populations under study.

Dr. Naeye is board-certified in clinical and anatomical pathology.

Everett Oesterling, M.D. issued a report on July 20, 2001. (DX 20). Dr. Oesterling reviewed medical records from Dixie Regional Medical Center, Miner's four autopsy slides, and the reports of Drs. Naeye and Perper. He also produced nineteen photomicrographs from the autopsy slides. Dr. Oesterling produced microphotographs one through five from slide number three. He detected a 2x3 mm area of tissue change with a 1 mm area of pigmentation, which was surrounded by enlarged air spaces constituting focal emphysema. Under polarized light, Dr. Oesterling detected infrequent birefringent silica crystals. Based on this nodule, Dr. Oesterling opined that the minimum criteria for a diagnosis of micronodular CWP is present. He added that this micronodule was the largest single aggregate of mine dust deposition noted in any four of the slides. Dr. Oesterling opined that the level of change he noted appears insufficient to have altered pulmonary function. Thus, he stated that mine dust would not have produced lifetime symptomatology, nor should it have contributed to or hastened Miner's death.

Dr. Oesterling found that microphotographs five through seven shows the presence of a small bronchus (airway) that was engaged in a protective response due to exposure to some toxic agent. He detected the presence of an inflammatory process and the destruction of lung tissue in microphotographs eight through sixteen. Dr. Oesterling added that these microphotographs also show early areas of organization or fibrosis. He characterized these areas of vascular congestion as showing a severe pneumonic process involving areas of Miner's lung that appeared functional. Dr. Oesterling found the loss of normal small structural component of the lung with the alveolus being replaced by much larger air sac in microphotograph seventeen. He detected giant cells in microphotographs eighteen and nineteen that were a response to foreign protein that indicates that miner experienced some component of aspiration of food particles, which he opined may have been the precipitating factor in initiating the extensive airway disease present at the time of Miner's demise. He stated that the process detected in microphotographs in eighteen and nineteen were unrelated to mine dust exposure. Dr. Oesterling stated that black pigment was absent in microphotographs seventeen through nineteen, which shows that Miner's severe destructive change is not related to mine dust exposure. Rather, he found that it was "a fairly significant centrilobular progressing to panlobular to bolus emphysema."

Dr. Oesterling found it notable that Miner underwent a lung reduction of the right lung. He opined that such a radical approach would indicate that Miner had severe bolus emphysema involving the right lung to the degree that the right lung was sacrificed with the hope that the left lung would compensate. However, Dr. Oesterling stated that Miner's left lung also had extensive emphysematous changes. He opined that the degree of change is not seen in miners, but typically is seen either in congenital abnormalities and/or cigarette smoking. Dr. Oesterling referenced the hospital record that document Miner's long history of heavy cigarette smoking. He then attributed Miner's severe pulmonary emphysema to Miner's history of smoking. He added that Miner's pulmonary disease was complicated by extensive purulent tracheobronchitis with bronchopneumonia apparently due to aspiration. He found that these processes were also unrelated to mine dust exposure. In the final paragraph of his report, Dr. Oesterling addressed the reports of Drs. Naeye and Perper in the following way:

> As for the report of Dr. Joshua Perper in which attempts to explain the extensive centrilobular, panlobular and bolus emphysema as related to mine dust exposure cannot be substantiated in literature. I believe this issue has been nicely addressed in the report of Richard Naeye, M.D. dated May 25, 2001, and I would merely echo his comments that the bulk of the medical literature and indeed my personal experience would not support Dr. Perper's conclusions that mine dust exposure was the causative factor in producing this gentleman's emphysema. Again, I would state that coalmine dust was not a factor in this gentleman's death.

Dr. Oesterling is board-certified in clinical and anatomical pathology, as well as in nuclear medicine.

Joseph Tomashefski, M.D. issued a report on December 13, 2001. (DX 22). He reviewed and summarized Miner's death certificate, autopsy report and slides, the Dixie Regional Medical Center records, and the reports of Drs. Perper, Naeye, and Oesterling. He considered a coal mine employment history of thirty years and a smoking history of heavy cigarette smoking. Dr. Tomashefski stated that his review of Miner's slides revealed severe, diffuse, airspace enlargement, with centriacinar accentuation, and "apparently detached" septa. He noted increased coarse black pigment being present within alveolar macrophages, as well as being deposited in alveolar septa and around bronchovascular structures. He identified approximately four peribronchiolar macules, each less than 1 mm in diameter, and altogether comprising less than one percent of the parenchymal tissue represented on the slides. Dr. Tomashefski stated that small bronchi were ecstatic with mucosal goblet cell hyperplasia and squamous metaplasia. Elastic pulmonary artery branches showed medial myxoid change. He stated that there are patchy neutrophilic exudates in alveoli and small airways throughout all sections.

According to his review of the records and the slides of miner's lung, Dr. Tomashefski opined that Miner had advanced, end-stage, mixed panacinar and centriacinar emphysema. Although he found that central airways were not included in the histological sample of Miner's lung, the presence of mucosal goblet cell hyperplasia and squamous metaplasis in peripheral

airways is consistent with chronic bronchitis. Dr. Tomashefski stated that the changes in the pulmonary vasculature further suggest the presence of pulmonary hypertension. He also found that Miner had acute bronchopneumonia. Dr. Tomashefski opined that the underlying cause of Miner's death was advanced emphysema and chronic bronchitis. He found that the immediate cause of Miner's death was bronchopneumonia, with small bowel obstruction serving as a contributory cause. Based on the presence of a few macules, Dr. Tomashefski found that Miner had simple CWP. However, he stated that the lesions of CWP are so minimal that they would not have caused Miner any respiratory symptoms, exercise limitation, or respiratory impairment. He also found that Miner's simple CWP was not a cause or contributory factor of Miner's death. He stated that the lesions of simple CWP are an incidental histological finding of no clinical relevance.

Dr. Tomashefski opined that neither Miner's minimal CWP, coal dust exposure, or coal mine employment were a cause of Miner's severe end-stage emphysema. He noted that emphysematous changes in Miner's lungs were not spatially associated with the few small coal macules which were present. He added that Miner's small bowel obstruction was unrelated to coal mine dust. Dr. Tomashefski opined that Miner's respiratory failure and death were unrelated to Miner's occupation as a coal miner. He also opined that Miner would have died at approximately the same time and in the same manner whether or not he ever worked in the coal mines or developed minimal CWP.

Dr. Tomashefski addressed Dr. Perper's findings of slight to moderate macular and micronodular CWP with findings of numerous anthracotic macules measuring up to 1-2 mm and small numbers of fibro-anthracotic mixed types of micro-nodules measuring up to 2 mm as an overestimation of the degree of simple CWP. He reiterated that he only found four coal macules, each less than 1 mm in diameter. He opined that there were no micronodules as defined in the Pathology Standards for CWP, citing to a 1979 publication. He strongly disagreed with Dr. Perper's conclusion that Miner had severe centrilobular emphysema significantly caused by coal mine dust and CWP. He reiterated that Miner's emphysema was advanced, severe and diffuse, of both centriacinar and panacinar patterns. He noted that the minimal degree of CWP involved less than 1% of Miner's lung tissue and cannot reasonably account for the extensive emphysema present. He stated that the emphysematous contribution of simple CWP is defined as 'focal emphysema" and is confined to the immediate vicinity of the coal macule. He opined that Miner's potential contribution of focal emphysema to Miner's severe diffuse emphysema is insignificant. Finally, Dr. Tomashefski stated that he disagreed with Dr. Perper's opinion that CWP with associated centrilobular emphysema was a substantial contributory cause of Miner's death. He referenced his previous statement that Miner's degree of simple CWP was too minimal to have been a cause or a contributory factor in Miner's death. Dr. Tomashefski added that cigarette smoke is an important cause of pulmonary emphysema and chronic bronchitis. He opined that Miner's advanced emphysema is the result of Miner's cigarette smoking habit.

On August 18, 2002, Dr. Naeye produced a second pathological consultative report based on his additional review of pathology reports from Rose Hill Medical Center, chest x-ray interpretations from Drs. Wheeler and Lewis, consultative reports from Drs. Wiot, Oesterling, Tomashefski, and Castle, as well as six previously unreviewed lung slides. (EX 6). Dr. Naeye stated that no new clinical information about Miner or his death were included in the medical

records from Rose Hill Medical Center. He summarized the other records he reviewed. Dr. Naeye stated that he reviewed "6 glass slides with 7 pieces of lung tissue that are different from the 5 pieces of lung tissue that were available for my 2001 review." He found that the new slides were of particular interest because microscopic examination shows there is less black pigment with associated fibrosis than was present in the five tissue samples he evaluated in his May 25, 2001 report. He found only "two<1.0mm anthracotic macules with associated fibrosis and rims of focal emphysema. He stated that the severe centrilobular emphysema that was present in the lung tissues supplied to him in 2001 was inconspicuous in the newly received tissues. Dr. Naeye stated that centrilobular emphysema is again severe. Dr. Naeye opined that the newly received information did not alter his original interpretations that lesions of very mild, simple CWP affected far too little lung tissue to have had any measurable effects on lung function, to have caused any disability or to have had any role in Miner's death. He commented that Drs. Oesterling and Tomashefski reached the same conclusions. Dr. Naeye commented that Dr. Goates, who was the first pathologist to review the lung tissues, did not conclude that CWP was present. Dr. Naeye did note that Dr. Goates found the presence of anthracosis without any associated tissue damage. He disagreed with Dr. Goates findings and conclusions. Dr. Naeye opined that very mild tissue damage is visible that is directly associated with the black pigment that includes fibrosis and focal emphysema, which meet the minimum criteria for the diagnosis of simple CWP. Dr. Naeye then stated that he "dealt with Dr. Perper's claims that anthracotic deposits > 1mm in diameter (micronodules) caused enough lung damage to lead to disability and contribute to death in my 2001 report." He added that even if fewer anthracotic lesions are present in the newly received lung tissue than were present in the lung tissues he reviewed in 2001, which he found further reduces the credibility of disability and death claims made by Dr. Perper.

Dr. Oesterling also issued another consultative pathological report, based on his review of six additional lung tissue slides, on September 12, 2002. (EX 7). He commented that the six additional slides provided some additional information. Dr. Oesterling utilized microphotographs to document his findings. He pointed to microphotograph one as containing the largest single aggregate of mine dust deposition, measuring approximately 2 mm, which is sufficient to qualify as a macule of CWP. Under polarized light, Dr. Oesterling detected weakly birefringent silica crystals, which he found to represent coal mine dust that is sufficient for a diagnosis of macular CWP. Dr. Oesterling concurred with the diagnosis rendered by Dr. Goates of respiratory bronchiolitis, which he characterized as the proper terminology for this condition that is known to be related to cigarette smoking. He stated that the impact of Miner's cigarette smoking history was clearly evident, referring to microphotograph eight, based on his findings of the remnant of pleural surface with a large area of free air, that demonstrate the presence of bullae indicating a severe bullous emphysema that has progressed from extensive panlobular emphysema that was described in his previous report. Dr. Oesterling detected ectopic bone formation or dystrophic ossification that may be the result of chronic passive congestion from left-sided heart disease. He also found evidence of pulmonary hypertension, which was illustrated in microphotograph fourteen. In this microphotograph, he found the muscularization that would be expected with the significant emphysema that Miner experienced. He also found smoker's macrophages in microphotograph fourteen. Dr. Oesterling concluded that the review of additional materials had been helpful because it clearly established the extent of Miner's emphysema, and because it clearly showed that the only additional mine dust that was present in

Miner's lung tissue consisted of rare macules that measured up to 2 mm in greatest dimension. Thus, he would re-classify Miner's coal workers' disease as a mild macular with focal micronodular CWP. He restated that the level of disease he detected was insufficient to have altered Miner's pulmonary function, thus, it was not a factor in Miner's lifetime nor was it a factor in his death.

Dr. Oesterling then concluded that Miner had a very mild form of CWP classified as minimal macular with focal micronodular disease. He stated that coal mine dust was in no way a factor in producing Miner's lifetime respiratory impairment; he attributed Miner's impairment to cigarette smoking. Dr. Oesterling opined that Miner died from a combination of respiratory failure complicated by severe ileus that had resulted in multiple operative procedures which failed to release Miner's intra-abdominal problem. Finally, Dr. Oesterling stated that death was not caused, contributed to, or hastened by any disease arising from coal mine employment.

On November 25, 2002, Dr. Tomashefski issued a supplemental pathology report after he reviewed and summarized additional medical records, which included Miner's hospitalization records from 1995 through 1999. (EX 9). Included among the records Dr. Tomashefski reviewed were six slides of lung tissue resected from Miner and he re-reviewed the four slides from Miner's autopsy. Dr. Tomashefski found that the additional evidence that he reviewed confirmed that Miner had disabling obstructive lung disease, as well as hypertension, hyperglycemia, cardiac tachyarrhythmias, gout, hypothyroidism, peptic ulcer disease, and a hernia repair. He characterized Miner's COPD as long-standing based on the PFTs he reviewed. He noted that Miner underwent a thoracoscopic lung reduction surgery of his right lung that removed 87 grams of lung tissue from Miner's right upper and middle lobe by YAG laser. He noted the surgical pathological description of the removed tissue. Dr. Tomashefski reviewed the six slides of resected lung tissue and found that it indicated the presence of moderately severe panacinar and centriacinar emphysema that was predominantly panacinar. He noted that a small bulla was in the middle lobe. He detected alveolar macrophages with coarse black pigment in all sections. Dr. Tomashefski found minimal interstitial fibrosis, mild pleural fibrosis, and mild chronic inflammation and fibrosis of membranous bronchioles. He noted that pulmonary arterioles show concentric intimal fibrosis. He also noted that a small amount of black pigment was deposited around blood vessels, but he did not find any coal macules.

Based on his initial and additional reviews, Dr. Tomashefski opined that Miner had severe, end-stage, mixed panacinar and centriacinar emphysema with features of chronic bronchitis. He stated that the clinical severity of the emphysema is emphasized in the additional records he reviewed, especially the CT scan and PFT data showing evidence of advanced emphysema. He still found that Miner's CWP was minimal. He added that the fact that no lesions of simple CWP were present in Miner's resected lung tissue further indicates the minimal extent of Miner's pneumoconiosis. He opined that his review of the additional medical evidence does not change his previous opinion that Miner's death was due to advanced emphysema caused by cigarette smoke exposure, complicated terminally by acute bronchopneumonia. It continued to be his opinion that CWP would not have caused Miner respiratory symptoms or respiratory impairment, and was not a cause of, or a contributory factor in Miner's death. He also continued to hold the opinion that neither coal dust exposure nor coal mine employment caused or contributed to Miner's death.

Deposition Testimony

Dr. Perper was deposed on November 5, 2002. (EX 3). Dr. Perper testified that he reviewed additional records after issuing his December 17, 2000 report, which included that reports of Drs. Naeye, Oesterling, Wheeler, Tomashefski, Castle, and Lewis. He stated that Miner had bullous and centrilobular emphysema. The articles that Dr. Perper reviewed did not link bullous emphysema with CWP. However, he testified that studies show that centrilobular emphysema may progress into bullous emphysema. Dr. Perper added that it is well recognized in the literature, including by NIOSH, that a causal relationship exists between pulmonary emphysema, exposure to coal dust, and CWP. Dr. Perper recognized that part of Miner's emphysema was caused by cigarette smoking, but he noted that the effects are additive. Dr. Perper stated that some of the studies he cited to were better controlled for smoking than others, but he added that the Department of Labor's implementation of the Federal Coal Mine Health and Safety amended as of December 20, 2000, quoted additional papers that he didn't quote in his report and he recognized that NIOSH felt that the criticism of those studies he relied upon and the ones mentioned in the Act were unwarranted. Dr. Perper testified that a long history of smoking could produce centrilobular and bullous emphysema like Miner had. When determining whether coal dust exposure or cigarette smoking was responsible for causing Miner's emphysema, Dr. Perper stated that Miner had two etiological agents, coal dust exposure and smoking. Although it is impossible to allocate the percentage of causation between them, Dr. Perper stated that it is possible to compare the relative significance of exposure. He stated that Miner's exposure to coal mine dust and cigarette smoke was significant. Based on history alone, Dr. Perper stated that he was able to identify that both etiological agents were responsible in some portion for Miner's emphysema, with the notation that Miner stopped smoking and the toxic action of smoking stops when the individual stops smoking while the toxic action of coal dust exposure and silica trapped in the lung continues beyond cessation of exposure. Dr. Perper testified that he found macular and micro-nodular disease. He allowed that the macular form of pneumoconiosis would usually not affect lung function. However, he stated that he detected macules, for whatever reason and even though it only had a small amount of fibrous tissue, that they were able to cause focal emphysema. He testified that he characterized Miner's CWP as slight to moderate because of the numerous amounts of large macules that contained silica in conjunction with small numbers of micro-nodules, all in the presence of clusters of macrophages with anthracotic pigment inside alveoli. Dr. Perper testified that the presence of a significant number of macrophages with anthracotic pigment in the alveoli might affect the gas exchange mechanism.

Dr. Perper stated that he did not agree with all of the conclusions of the autopsy prosector, noting that the autopsy prosector didn't recognize pneumoconiosis, even though the prosector described macules and nodules. Dr. Perper stated that centrilobular emphysema will stop progressing when a person stops smoking. He stated that some studies have shown that smokers who are also miners are sometimes worse with emphysema than miners who are not smokers. Dr. Perper stated that the relationship between smoking and pneumoconiosis to emphysema is an additive or synergistic relationship. He restated this as, "if you have one and you get the other, they make each other worse." Dr. Perper testified that some studies have shown that exposure to silica contained in coal dust interferes with an important enzyme in the

lungs that prevents scarring called alpha-1-antitrypsin inhibitor. He stated that exposure to silica destroys the inhibitor, which leaves people more prone to scarring.

Dr. Perper testified that he did not see bullous emphysema in the autopsy slides because bullous emphysema cannot be seen on tissue slides because they appear like a large balloon of air that might be several centimeters long. He added that panacinar (or bullous) emphysema has to start as centrilobular emphysema and then it progresses to panlobular emphysema. Dr. Perper testified that there is no disagreement between his autopsy slide reviews and the reviews of Drs. Naeye and Oesterling regarding the presence of CWP and severe centrilobular emphysema. He identified the difference between his opinion and those of Drs. Naeye and Oesterling as whether or not there is a relationship between coal dust exposure and the centrilobular emphysema that caused Miner's death. Dr. Perper stated that the other physicians dispute the fact that coal dust exposure and CWP cause centrilobular emphysema. Dr. Perper reiterated his opinion that the other physicians' view regarding the etiology of centrilobular emphysema is contrary to accepted medical literature, the position of NIOSH, and the position of the U.S. Department of Labor and its regulations. Dr. Perper criticized Dr. Tomashefski's report, arguing that Dr. Tomashefski missed the point regarding the etiology of Miner's centrilobular emphysema. Dr. Perper expanded on his criticism, stating that there are two types of emphysema present in Miner that were both caused by coal dust exposure and CWP: scar or focal emphysema that is localized around the coal macule or nodule, and diffuse centrilobular emphysema. Dr. Perper opined that the diffuse centrilobular emphysema associated with the generalized impact of the toxic effect of coal dust containing silica primarily contributed to Miner's death.

Dr. Oesterling was deposed on May 6, 2003. (EX 10). He reiterated the findings and conclusions contained in his prior reports. Dr. Oesterling testified that he saw two significant differences between the surgically removed lung tissue from 1995 and the tissue removed at autopsy. First, he was impressed by the very significant passive congestion that he found in the surgically removed specimen, as well as the presence of hemosiderin and macrophages and ectopic bone formation. He did not see evidence of passive congestion in the autopsy tissue specimens. He noted that the emphysematous changes were similar in both tissue sets. However, he did find some evidence of a micronodule of CWP in the autopsy tissue, while he only saw macules in the first tissue set. Dr. Oesterling noted that almost Miner's entire right lung was removed in 1995 to treat his emphysema because only five grams of right lung remained at Miner's autopsy. Dr. Oesterling commented that the emphysema he saw in Miner's right lung that was removed in 1995 had not significantly progressed based on the condition of the autopsy slides. Dr. Oesterling discussed the definitions of centrilobular and panlobular emphysema, as well as bullous emphysema. Dr. Oesterling attributed the emphysema that he detected in Miner to cigarette smoking because the changes due to Miner's dust exposure were relatively limited. He noted that there was no major compression of Miner's airways due to changes from dust deposition. He added that, to reach the point where panlobular emphysema can be considered to be related to coal workers' disease, there would have to be more evidence of compression of larger airways. Otherwise, based on "Dr. Spencer's textbook on lung disease, one of the very well known textbooks, makes a blanket statement that the emphysema related to mine dust exposure is limited to centrilobular emphysema, excludes panlobular emphysema." Based on his experience, he added that the foregoing citation was his feeling, too. Dr. Oesterling testified that a very minimal amount of black coal mine dust was present in Miner's lungs. He

noted that the crystals Miner breathed in during coal mine employment were not found in the areas of Miner's lungs where severe emphysema was present. Since the fine silica crystals were not present at the site of Miner's severe emphysema, it would indicate that the severe emphysema was due to the hyperinflation from the chronic bronchitis that is associated with cigarette smoking.

Regarding the cause of Miner's death, Dr. Oesterling testified that he would assume much of it had to be respiratory due to the fact that Miner had little lung functioning left, especially after 55-60% of Miner's primary lung tissue had been resected. The remaining lung tissue that Miner did have had emphysema with superimposed pneumonia that was destroying Miner's remaining lung function. He added that the emphysema was primarily affecting the upper portion of Miner's left lung and the pneumonia was affecting the lower portions. Dr. Oesterling opined that he did not think that there was enough of a structural change in Miner's lungs related to CWP to alter Miner's lung function enough to cause, contribute, or hasten Miner's death; he did not think that Miner's CWP produced a functional change. On cross-examination, Dr. Oesterling explained that Miner's emphysema, pneumonia, and previous heart failure contributed to Miner's death by producing function alterations in Miner's ability to oxygenate his blood and eliminate carbon dioxide. Dr. Oesterling testified that the four autopsy slides he reviewed represent only a very small percent of Miner's lungs; the tissue sections from the autopsy represent a fraction of a millionth of a portion of Miner's total lung, just as the one micronodule that Dr. Oesterling detected in his review of the autopsy slides impaired a fraction of a millionth of Miner's lung functioning. Dr. Oesterling stated that, had there been other areas that grossly looked like nodular change, the autopsy prosector would have sampled them because most people who work in the pathology field are aware of what must be done to sample lung to get an adequate representation because they usually end up being the subject of multiple reviews. Dr. Oesterling assumed that the one micronodule that was contained in the autopsy slides was the only one present because the autopsy prosector would have sampled other micronodules if they were present. He added that there was not much upper lobe left at the time of autopsy, which is the area pathologists typically look to diagnose the presence of CWP, because bullous emphysema had severe destruction of Miner's lungs.

Dr. Oesterling commented that he did diagnose the presence of bronchitis through the autopsy slides, and he added that the tracheobronchitis from the aspiration of Miner's lung was the primary cause of death in terms of Miner's final course of events because it destroyed the functioning lung that Miner had left. He noted that Miner probably did not have bronchitis caused by anything other than aspiration, but he could not be certain because of the destruction of the airways by Miner's disease process. Dr. Oesterling then stated that Miner probably had a great deal of bronchitis early on, adding that bronchitis is what leads to emphysema. Dr. Oesterling stated that Miner suffered from bronchitis due to cigarette smoking that developed into emphysema. He justified ruling out coal mine dust exposure as a cause of the early bronchitis because the form of panlobular and bullous emphysema that Miner had is not seen in coal miners who did not smoke. Thus, with the exception of airways that become occluded by extensive fibrosis related to coal dust exposure, he does not think that coal mine dust causes panlobular or bullous emphysema. Dr. Oesterling stated that CWP can lead to focal emphysema, which is a form of centrilobular emphysema. He added that the relationship between CWP and emphysema is due to the extent of dust that has been deposited and the extent of the change of

the lung. However, he found that Miner's centrilobular emphysema was not caused by CWP because the amount of dust present was not sufficient to produce physiologically the changes that would result in centrilobular emphysema.

Dr. Tomashefski was deposed on June 19, 2003. (EX 12). He reiterated the findings and conclusions contained in his prior reports. Dr. Tomashefski testified that he found minimal changes consistent with CWP based on his review of the autopsy slides that showed a few scattered coal macules that were tiny in size that constituted an insignificant portion of the total lung represented on the slides that he reviewed. He noted that he did not find any such lesions in Miner's surgically resected lung tissue, and he only found four lesions in Miner's tissue removed at autopsy. Dr. Tomashefski opined that Miner's centriacinar and panacinar emphysema was the primary cause of Miner's death. He found that both forms of Miner's emphysema were caused by Miner's cigarette smoking habit because the type of emphysema that Miner had is typical of that due to cigarette smoking and there was no other cause in the slides he reviewed. Dr. Tomashefski testified that coal dust exposure can cause focal emphysema, which is sometimes looks like centriacinar emphysema, but he did not detect any such changes in Miner's autopsy slides. Instead, he found that Miner's lesions of centriacinar emphysema were in the midst of diffused panacinar emphysema, with no coal macules or even significant coal dust deposits in this area of centriacinar emphysema.

Dr. Tomashefski specifically disagreed with Dr. Perper's conclusion that Miner's coal dust exposure led to Miner's centrilobular emphysema because he only detected a minimal amount of CWP. He also disagreed with Dr. Perper's statement that emphysema related to cigarette smoking does not increase in severity after the cessation of smoking because pigments, tars, and macrophages still persist in the lung and can continue to cause destruction. Regarding the autopsy prosector's report, Dr. Tomashefski commented that the prosector did not even diagnose CWP. He also found that there was a "disconnect" in the report of the prosector because the "prosector states fibrosis absent or minimal and then states that the fibers are in favor of the progression of pulmonary fibrosis." Dr. Tomashefski disagreed with many of the prosector's findings, including: (1) he stated that the prosector identified many dust macules, but he only found four; (2) the prosector detected patchy black parenchymal and subpleural nodules, but he did not see any parenchymal or subpleural nodules, he only saw a few small macules; (3) the prosector found centrilobular emphysema, and Dr. Tomashefski disagreed somewhat because he found the primary form of emphysema to be panlobular and not just centrilobular; and (4) he also disagreed with the prosector's diagnosis of severe chronic obstructive pulmonary fibrosis. Dr. Tomashefski added that the advantage of the autopsy prosector is that by seeing the gross tissue, the prosector can direct the tissue sampling and gent an impression of the dispersion and quantification by actually seeing the tissue as opposed to a reviewing pathologist who has to rely on the prosector's gross description of the tissue.

On July 28, 2003, Dr. Castle was deposed. (EX 13). He reiterated the findings and conclusions contained in his prior reports.

Smoking History

Claimant characterized Miner as a social smoker who smoke off-and-on from the time they were married in 1989 until he quit smoking completely in 1989. (Tr. 11, 12). Claimant added that she would "send two smokes in his lunch bucket, and sometimes he'd bring those home. One day he told me not to send them anymore." (Tr. 12). She noted that Miner began smoking again after they married in 1975, but she was unaware of the extent of his previous smoking habit. (Tr. 12-14).

At the hearing held in 1992 before Administrative Law Judge Stewart, Miner testified that he smoked about one-half pack of cigarettes per day from the age of fifteen to the age of twenty-five. (DX 1, Tr. 14-15). From the age of twenty-five, he smoked three to four cigarettes per day until he quit smoking completely in 1989. (DX 1, Tr. 15). Miner also testified that he had previously quit smoking from 1972 through 1976. (DX 1, Tr. 15). Miner also testified under cross-examination regarding the accounts of his smoking habit that he provided to physicians who examined him. (DX 1, Tr. 15). The medical evidence contained in the living miner's claim contains the following documentations of Miner's smoking history: (1) Dr. Barlow documented a one-half pack per day habit with a 40 pack-year history as of December 22, 1986; (2) on May 11, 1987 Dr. Barlow recorded a present habit of one-to-two cigarettes per day; (3) Dr. Barlow noted that Miner had stopped smoking by August 10, 1987; (4) Dr. Lincoln recorded a ten pack-year history as of November 17, 1989; (5) Dr. Farney considered a smoking history of one-half pack per day from the ages of fifteen to twenty-five; and (6) Dr. Morris documented one-half pack per day smoking history from the ages of fifteen to twenty-five. (DX 1). In 1995, Dr. Pluss noted that Miner had a forty-three pack-year smoking history that ended in 1989. In 1998, Dr. Ross noted that Miner had a fifty pack-year smoking history. Dr. Lewis found that Miner had a long history of heavy cigarette smoking in 1999.

Based on Miner's testimony and the documentation in the medical records, it is evident that Miner began smoking in 1949 and stopped smoking in 1989. Miner credibly testified that he smoked less than one pack per day for the entire forty years, and Claimant testified that Miner did not smoke one pack per day for the fifteen years she knew Miner before Miner stopped smoking. Since Miner smoked less than one pack per day for forty years, I find that Miner smoking history amounts to no more than a twenty pack-year history of cigarette smoking.

DISCUSSION AND APPLICABLE LAW

Mrs. Trease filed her survivor's claim on September 20, 2000. Entitlement to benefits must be established under the regulatory criteria at Part 718. *See Neeley v. Director, OWCP*, 11 B.L.R. 1-85 (1988). The Act provides that benefits are provided to eligible survivors of a miner whose death was due to pneumoconiosis. § 718.205(a). In order to receive benefits, the claimant must prove that:

1). The miner had pneumoconiosis;

2). The miner's pneumoconiosis arose out of coal mine employment; and

3). The miner's death was due to pneumoconiosis.

§§ 718.205(a). Failure to establish any of these elements by a preponderance of the evidence precludes entitlement. *See Anderson v. Valley Camp of Utah, Inc.*, 12 B.L.R. 1-111, 1-112 (1989); *Trent v. Director, OWCP*, 11 B.L.R. 1-26, 1-27 (1987).

Pneumoconiosis

In establishing entitlement to benefits, Claimant must initially prove the existence of pneumoconiosis under § 718.202. Claimant has the burden of proving the existence of pneumoconiosis, as well as every element of entitlement, by a preponderance of the evidence. *See Director, OWCP v. Greenwich Collieries*, 512 U.S. 267 (1994). Pneumoconiosis is defined by the regulations:

> For the purpose of the Act, "pneumoconiosis" means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical" pneumoconiosis and statutory, or "legal" pneumoconiosis.
>
> (1) *Clinical Pneumoconiosis*. "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconiosis, i.e., conditions characterized by permanent deposition of substantial amounts of particulate matter 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 or silicotuberculosis, arising out of coal mine employment.
>
> (2) *Legal Pneumoconiosis*. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.

Section 718.201(a).

Employer does not contest the issue of whether Miner suffered from pneumoconiosis arising out of coal mine employment. The medical reports of Drs. Perper, Tomashefski, Naeye, Oesterling, and Castle all find the presence of simple coal workers' pneumoconiosis based upon their review of Miner's medical evidence, which included surgically resected lung tissue and lung tissue removed at autopsy. I find that a preponderance of the autopsy evidence establishes that Miner suffered from pneumoconiosis arising out of coal mine employment. Therefore, I find that Claimant has established the presence of pneumoconiosis at the time of Miner's death by a preponderance of the evidence.

Arising out of Coal Mine Employment

In order to be eligible for benefits under the Act, Claimant must also prove that pneumoconiosis arose, at least in part, out of Miner's coal mine employment. § 718.203(a). For a miner who suffers from pneumoconiosis and was employed for ten or more years in one or more coal mines, it is presumed that his pneumoconiosis arose out of his coal mine employment. *Id.* As I have found that Miner engaged in at least thirty-years of coal mine employment, and since no rebuttal evidence was presented, I find that Miner's pneumoconiosis arose out of his coal mine employment in accordance with the rebuttable presumption set forth in § 718.203(b).

Death Due to Pneumoconiosis

Claimant has established that Miner suffered from pneumoconiosis arising out of coal mine employment. She must now prove that Miner's death was due to pneumoconiosis in order to be entitled to benefits. Subsection 718.205(c) applies to survivor's claims filed on or after January 1, 1982 and provides that an eligible survivor will be entitled to benefits if any of the following criteria are met:

1. Where competent medical evidence establishes that pneumoconiosis was the cause of the Miner's death, or

2. Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where death was caused by complications of pneumoconiosis, or

3. Where the presumption set forth in § 718.304 (evidence of complicated pneumoconiosis) is applicable.

20 C.F.R. § 718.205(c).

Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death. § 718.205(c)(5). The presumption set forth in § 718.304 is not applicable because Claimant has not established the presence of complicated pneumoconiosis. Therefore, in order for Claimant to be entitled to benefits, she must show that pneumoconiosis was the direct cause of Miner's death or that pneumoconiosis hastened Miner's death.

A death certificate, in and of itself, is an unreliable report of the miner's condition and it is error for an administrative law judge to accept conclusions contained in such a certificate where the record provides no indication that the individual signing the death certificate possessed any relevant qualifications or personal knowledge of the miner from which to assess the cause of death. *Smith v. Camco Mining, Inc.*. 13 B.L.R. 1-17 (1989); *Addison v. Director, OWCP*, 11 B.L.R. 1-68 (1988). Dr. Lewis signed Miner's death certificate, listing the cause of Miner's death as COPD with an underlying cause of pneumonia. Even though Dr. Lewis did not have the findings from Miner's autopsy available to him at the time he completed the death certificate, I find that Dr. Lewis possesses the relevant qualifications and knowledge from which to assess the cause of Miner's death. Dr. Lewis first examined Miner in 1998, and he followed Miner from

Miner's admission to Dixie Regional Medical Center on November 30, 1998 until Miner's death on January 20, 1999. He completed a death summary on January 20, 1999, in which he set forth an account of Miner's final hospital course. Accordingly, I find that the Dr. Lewis' opinion regarding the cause of Miner's death as indicated on the death certificate is entitled to probative weight.

Dr. Gurunadham performed an autopsy limited to Miner's lungs for the purpose of detecting the presence or absence of pneumoconiosis. She issued a written report. Dr. Gurunadham did not expressly diagnose the presence of pneumoconiosis, but she did render findings in favor of the progression of pulmonary fibrosis secondary to CWP as compared to cigarette smoking and she stated that the issue of whether the changes she detected in Miner's lungs are related to cigarette smoking is subject to debate. Dr. Gurunadham provided a gross and microscopic description of the lung tissue she reviewed. She set forth pathological findings and observations. Therefore, I attach probative weight to the findings noted by Dr. Gurunadham.

Four pathologists reviewed the tissue slides obtained during the autopsy of Miner and Miner's other medical records. All four reviewing pathologists, and a reviewing pulmonologist found that Miner suffered from simple CWP, severe emphysema, and pneumonia at the time of his death. All four reviewing pathologists and the reviewing pulmonologist found that Miner's death was due to his severe emphysema and superimposed pneumonia, as well as other complications that arose during Miner's final hospital course. The medical evidence is in conflict as to the severity of Miner's CWP and as to the etiology of Miner's emphysema.

Dr. Perper provided a consultative report in December of 2000. He considered an accurate account of Miner's smoking and coal mine employment history. He reviewed the hospital records from miner's final hospital course at Dixie Regional Medical Center and he reviewed the autopsy report and slides. In his report, he set forth microscopic pathological findings from his review of the autopsy slides. He opined that Miner had slight to moderate simple CWP based on five separate findings, which included Miner's history of exposure, symptoms, physiological tests, and his autopsy findings. Dr. Perper's autopsy findings of pneumoconiosis included findings of numerous macules and small numbers of micronodules, with associated interstitial and solid fibrosis, anthracotic pigmentation, silica crystals, scar emphysema and severe centrilobular emphysema. He cited to medical literature, which he characterized as reliable over the past decades, to support his finding that Miner's centrilobular emphysema was due to cigarette smoking, but also a direct result of exposure to mixed coal mine dust containing silica and CWP. Dr. Perper opined that Miner's CWP was a substantial contributory cause of Miner's death directly and indirectly through the associated centrilobular emphysema that caused hypoxemia that either triggered or aggravated fatal cardiac arrhythmia. Dr. Perper supported his findings by references to seven microphotographs taken from the autopsy slides, which identified the specific pathological findings he made. He also attached a document labeled "Appendix I", which contains a narrative analysis of the medical literature that supports his opinion that Miner's coal dust exposure and CWP contributed to the development of Miner's centrilobular emphysema.

Dr. Perper expanded on his opinion during his deposition in November of 2002. Prior to his deposition, he reviewed the pathological consultative opinions of Drs. Naeye, Oesterling, and

Tomashefski, as well as other medical evidence. He testified that Miner's coal dust exposure and CWP served as an additive factor in conjunction with Miner's smoking history to cause centrilobular emphysema. Dr. Perper testified that the relationship between coal dust exposure and centrilobular emphysema was supported by the medical literature, NIOSH, and the regulations implementing the Act as amended on December 20, 2000. He then testified, based on history alone, since Miner had two potential etiological agents, that Miner's centrilobular emphysema was caused by coal dust exposure and cigarette smoking. Dr. Perper conceded that the macular form of pneumoconiosis would not usually affect lung function. However, he detected macules that caused focal emphysema, even though they only had a small amount of fibrous tissue. Finally, Dr. Perper testified that some studies have shown that exposure to silica contained in coal dust interferes with the enzyme alpha-1-antitrypsin, leaving the lung more prone to scarring. He criticized the other reviewing pathologists for relying on medical literature contrary to accepted medical literature, the position of NIOSH, and the position of the implementing regulations of the Act to argue against his opinion that Miner's coal dust exposure resulted in centrilobular emphysema. He concluded by opining that the diffuse centrilobular emphysema associated with the general impact of the toxic effect of coal dust containing silica primarily contributed to Miner's death.

I find that Dr. Perper's opinion is entitled to probative weight enhanced by his credentials as a board-certified clinical and anatomical pathologist. Dr. Perper considered an accurate account of Miner's smoking and coal mine employment histories. He set forth clinical and pathological findings and observations, and his reasoning is supported by adequate data and medical literature. Dr. Perper's opinion is reasoned and documented.

Dr. Naeye issued a consultative pathological report in May 2001. The records he reviewed Miner's hospital records from Dixie Regional Medical Center, the autopsy report, and the autopsy slides. He stated that the minimal findings to diagnose very mild, simple CW P may or may not be present. Dr. Naeye opined that any focal emphysema that may be present is obscured by the severe centrilobular emphysema that is present everywhere. Dr. Naeye stated that all of the macules he detected only occupied 2% of the total lung tissue available from the autopsy. He noted that there are no very tine crystals of free silica in the tissue locations where foci of fibrosis may be present. Based on all of his findings, Dr. Naeye opined that Miner's simple CWP was too mild and limited in extent to have caused any measurable abnormalities in lung function, caused any disability, or hastened death. He found that Miner's death was respiratory in nature, with major causes of very severe emphysema and mild to moderately severe chronic bronchitis. Dr. Naeye then addressed the relationship between coal dust exposure and centrilobular emphysema. He asserted that the medical literature indicates that coal mine dust only has a clinically insignificant role in the genesis of centrilobular emphysema.

In a second consultative pathological report issued in August 2002 after reviewing additional medical records that included slides from the surgical resection of Miner's right lung in 1995 and the other pathologists reports, Dr. Naeye continued to find that Miner's very mild, simple CWP affected far too little lung tissue to have had any measurable effect on Miner's death. He noted that Drs. Oesterling and Tomashefski reached the same conclusion.

I find that Dr. Naeye's report is entitled to probative weight enhanced by his credentials as a board-certified clinical and anatomical pathologist. He set forth clinical and pathological findings, and his reasoning is supported by adequate data and medical literature. He considered an accurate account of Miner's smoking and coal mine employment histories. Dr. Naeye's opinion is reasoned and documented.

In July of 2001, Dr. Oesterling issued his first consultative pathological report based upon his review of Miner's medical records, which included the hospital records from Dixie Regional Medical Center, the autopsy report, and the autopsy slides. He prepared microphotographs from the autopsy slides. He detected a 2x3 mm area of tissue change with surrounding focal emphysema and infrequent birefringent silica crystals that satisfied the minimum criteria for diagnosis CWP. Dr. Oesterling identified a severe pneumonic process caused by aspiration, which may have been the precipitating factor in initiating the extensive airway disease present at the time of Miner's demise. He found that the degree of emphysematous change present in the tissues he reviewed is typically seen with congenital abnormalities or cigarette smoking. Dr. Oesterling testified that Miner's pulmonary disease was complicated by tracheobronchitis with bronchopneumonia apparently due to aspiration. He concurred with Dr. Naeye's criticism of Dr. Perper's "attempts to explain the extensive centrilobular, panlobular and bolus emphysema as related to mine dust exposure." He did not believe the relation could be substantiated in literature. Dr. Oesterling concluded that coal mine dust was not a factor in Miner's death.

Dr. Oesterling issued a second consultative pathological report in September 2002 after he reviewed slides containing tissue from Miner's surgically resected right lung from 1995, as well as other medical evidence. He again produced microphotographs. He detected the impact of Miner's cigarette smoking history based on his findings of a large area of free air that demonstrated the presence of bullae indicating that a severe bullous emphysema had progressed from extensive panlobular. Dr. Oesterling also found pulmonary hypertension and chronic passive congestion from left-sided heart disease. Dr. Oesterling again attributed Miner's impairment to cigarette smoking. He found that Miner died from a combination of respiratory failure complicated by severe ileus that had resulted in multiple operative procedures. Dr. Oesterling concluded that Miner's death did not cause, contribute to, or hasten Miner's death.

In his deposition testimony provided in May of 2003, Dr. Oesterling continued to find that Miner's emphysema to cigarette smoking because the changes due to Miner's dust exposure were relatively limited and he did not find any major compression of Miner's airways due to changes from dust deposition. He added that there was only a minimal amount of black coal mine dust in Miner's lungs and that the crystals related to coal mine employment were not found in the areas where severe emphysema was present. In the absence of fine silica crystals at the location of Miner's emphysema, Dr. Oesterling opined that Miner's emphysema must be due the hyperinflation from chronic bronchitis caused by cigarette smoking. He testified that the cause of Miner's death was respiratory in nature. He noted that the resection of Miner's right lung had removed 55-60% of Miner's primary lung tissue. When the remaining functional tissue developed emphysema with superimposed pneumonia, his remaining lung function was destroyed. More specifically, Dr. Oesterling stated that Miner's death was caused by emphysema, pneumonia, and previous heart failure that combined to produce functional

alterations in Miner's ability to oxygenate his blood and eliminate carbon dioxide. Dr. Oesterling also testified that Miner suffered from chronic bronchitis caused by cigarette smoking that developed into emphysema. He ruled out coal dust exposure as a cause of the bronchitis since the form of bullous and panlobular emphysema that was present was not seen in coal miners who did not smoke. He also found that Miner's centrilobular emphysema was not caused by CWP because the amount of dust present was not sufficient to produce physiologically the changes that would result in centrilobular emphysema.

I find that Dr. Oesterling's opinion is entitled to probative weight enhanced by his credentials as a board-certified clinical and anatomical pathologist. He considered an accurate account of Miner's smoking and coal mine employment histories. Dr. Oesterling set forth clinical and pathological findings and observations, and his reasoning is supported by adequate data. His opinion is reasoned and documented.

Dr. Tomashefski issued a consultative pathological report in December 2001 based on his review of Miner's hospital records, which included the records from Miner's final hospital course at the Dixie Regional Medical Center, the autopsy report, and the autopsy slides. He identified four macules, each less than 1 mm in diameter and comprising less than 1% of the parenchymal tissue represented on the slides. Based on the presence of these few macules, he opined that Miner suffered from simple CWP. He opined that Miner had advanced, end-stage, mixed panacinar and centriacinar emphysema, a well as chronic bronchitis and pulmonary hypertension. Dr. Tomashefski opined that the underlying cause of Miner's death was advanced emphysema and chronic bronchitis, with the immediate cause being bronchopneumonia with a small bowel obstruction serving as a contributory cause. He found that the lesions of Miner's simple CWP were so minimal that they would not have caused or contributed to Miner's death; he characterized his diagnosis of CWP as an incidental histological finding of no clinical relevance. Dr. Tomashefski stated that Miner's coal dust exposure and CWP did not cause Miner's severe end-stage emphysema since the emphysematous changes in Miner's lungs were not spatially associated with the few small coal macules. In addressing Dr. Perper's findings of slight to moderate macular and micronodular CWP with numerous anthracotic macules and small numbers of micronodules, Dr. Tomashefski pointed out that he found only four coal macules and no micronodules. Dr. Tomashefski stated that the emphysematous contribution of simple CWP appears as focal emphysema and is confined to the immediate vicinity of the coal macule. He found that the potential contribution of focal emphysema to Miner's severe diffuse emphysema is insignificant.

Dr. Tomashefski issued a second consultative pathological report in November 2002 after reviewing additional medical records, which included six slides of Miner's surgically resected right lung. From his review of the slides from 1995, Dr. Tomashefski observed moderately severe panacinar and centriacinar emphysema, as well as minimal interstitial fibrosis, mild pleural fibrosis, and mild chronic inflammation of the bronchioles. He did not detect any coal macules, but he found alveolar macrophages with coarse black pigment and black pigment around blood vessels. Based on his review of all of the medical evidence, Dr. Tomashefski opined that Miner suffered from end-stage mixed panacinar and centriacinar emphysema with features of chronic bronchitis. He also found that Miner suffered from minimal CWP. He continued to hold that Miner's death was due to advanced emphysema caused by cigarette smoke

exposure, and complicated terminally by acute bronchopneumonia. Dr. Tomashefski reasserted his opinion that Miner's CWP did not cause, contribute to, or hasten Miner's death. Dr. Tomashefski reiterated these opinions at his deposition in June 2003. He testified that he only detected a few scattered coal macules that were tiny in size and only occupied an insignificant portion of the total lung represented in the autopsy slides. Dr. Tomashefski again found that Miner's emphysema, in the form of centriacinar and panacinar, caused his death. He attributed both forms of Miner's emphysema to cigarette smoking because he detected no other cause from his pathological review. He noted that coal dust exposure can cause focal emphysema, but he stated that he did not detect any changes identified as focal emphysema.

I find that Dr. Tomashefski's opinion is entitled to probative weight. His curriculum vitae is not contained in the record, but the undersigned takes administrative notice that he is board-certified in clinical and anatomical pathology. Dr. Tomashefski considered an accurate account of Miner's coal mine employment and smoking histories. He set forth clinical and pathological observations and findings, and his reasoning is supported by adequate data. Dr. Tomashefski's opinion is reasoned and documented. I find that Dr. Tomashefski's opinion is entitled to probative weight enhanced by his credentials as a board-certified clinical and anatomical pathologist.

Dr. Castle, who is a board-certified pulmonologist, issued two consultative opinions and provided deposition testimony based on his review of the clinical and pathological evidence, which included the reports of all four pathologists. He found that Miner had the minimum pathological evidence to diagnose simple CWP. Dr. Castle opined that he agreed with the reasoning employed by Dr. Naeye to rebut the opinion of Dr. Perper that Miner's CWP was a contributing factor to Miner's emphysema and subsequently to his death. He stated that it was important to note that Miner suffered from bullous emphysema, which is a condition not associated with coal dust exposure. He found that Miner's simple CWP was not sufficient enough to have caused Miner any disability during his lifetime. He attributed Miner's death to acute bronchopneumonia resulting in respiratory failure complicated by surgical procedures on Miner's abdomen. Dr. Castle concluded that Miner's death was not caused, contributed to, or hastened by his underlying simple CWP. In his second consultative opinion, Dr. Castle opined that the additional medical evidence he reviewed only served to reinforce his prior opinions. He commented that the additional physiological studies that he reviewed showed findings, a severe airway obstruction with reversibility associated with hyperinflation and gas trapping, that were not consistent with CWP. Dr. Castle reiterated his concurrence with the findings of the pathologists that Miner's simple CWP was so minimal that it was not sufficient enough to cause impairment during Miner's lifetime. Dr. Castle reiterated his findings and opinions in his deposition testimony.

I find that Dr. Castle's opinion is entitled to probative weight enhanced by his credentials as a board-certified pulmonologist. He considered an accurate account of Miner's smoking and coal mine employment histories. He set forth clinical observations and findings, and his reasoning is supported by adequate data. Dr. Castle's opinion is reasoned and documented.

At the time of his death on January 20, 2000, Miner had been suffering from emphysema for approximately fifteen years. By 1995, the emphysematous changes in Miner's right lung had

progressed to such a degree of severity, that Miner underwent surgical resection of his right lung. Miner's right lung was almost entirely removed. The surgically removed right lung demonstrated the presence of minimal, simple CWP. Miner did not tolerate the surgical resection of his right lung well; he developed tachycardia and respiratory distress leading to placement of chest tubes for pneumothoraces, intubation, and mechanical ventilation. Miner was hospitalized on several occasions between 1995 and 1999 due to his severe COPD and worsening symptomatology. The final course of Miner's life was spent in the hospital, beginning with his admission by Dr. Ross on September 8, 1999 for complaints of increasing dyspnea and sweats. Dr. Ross noted that Miner was suffering from an advanced and severe COPD. Miner developed pneumonia, which Dr. Ross considered worrisome due to the severity of Miner's lung problem. Miner's condition was further complicated by the development of a gastric outlet obstruction due to a hematoma-like mass in his abdomen. Miner underwent a surgical procedure to repair the bowel obstruction and his treatment led to the resolution of his pneumonia. However, by the time Miner was discharged on November 22, 1999, Dr. Ross considered Miner to be quite de-conditioned.

Eight days later, Dr. Lewis admitted Miner to the intensive care unit due to his severe obstructive airways disease. Dr. Lewis' assessment was that Miner was experiencing acute respiratory failure with COPD and emphysema, probably due to aspiration pneumonia. He termed Miner's prognosis as guarded. Dr. Brown examined Miner and found that he was malnourished due to feeding difficulties resulting from his bowel obstruction. Dr. Lewis continued to attend to Miner until Miner's death. In the death summary that Dr. Lewis prepared, he commented that Miner had been suffering from end-stage COPD with pulmonary artery hypertension. He stated that Miner lost his ability to achieve adequate oxygenation of his blood, deteriorated significantly, and then expired. Based on his treatment of Miner over the period of his life, Dr. Lewis credibly opined that Miner's death was due to COPD with underlying pneumonia and complications of gastric outlet and obstruction.

Essentially, all of the reviewing pathologists and Dr. Castle found that Miner's death was due to respiratory failure brought about by severe emphysema and an acute pneumonia caused by aspiration of Miner's lung. The evidence also establishes that Miner's condition just prior to his death was diminished by malnutrition due to a retroperitoneal mass in Miner's abdomen that obstructed his bowel and required surgical intervention. Miner effectively only had one lung after 1995, and that lung was besieged by severe centrilobular, panlobular, and bullous emphysema, as well as CWP at the time of Miner's death. Since Miner only had a limited amount of lung function remaining, the physicians stated that the presence of pneumonia was worrisome because it would interfere with whatever lung functioning Miner had. The evidence clearly establishes that Miner's death was respiratory in nature, and the presence of CWP, dating back to 1995, has been clearly established. What remains for determination is what clinical role Miner's CWP assumed in relation to Miner's death.

Dr. Gurunadham performed an autopsy of the remaining portion of Miner's right lung and Miner's left lung. All four pathologists who reviewed the autopsy slides prepared by Dr. Gurunadham found the presence of simple CWP. While Dr. Perper classified the amount of CWP present as slight to moderate macular and micronodular, Drs. Naeye, Oesterling, and Tomashefski classified the CWP as minimal. While Dr. Perper found numerous anthracotic

macules and small numbers of micronodules, Drs. Naeye, Oesterling, and Tomashefski, collectively at most, only identified four macules and one micronodule. Drs. Oesterling and Tomashefski found that the amount of CWP present in all of the lung tissue contained on the slides obtained at autopsy only amounted to 1-2% of the viewable lung tissue. Drs. Oesterling and Tomashefski used microphotographs to identify the limited nature of the CWP that they detected. While Dr. Perper also obtained microphotographs from his review of the autopsy slides, he did not specifically identify the microphotographs that demonstrated the presence of numerous macules or the small number of micronodules. The opinions of Drs. Castle, Naeye, Oesterling, and Tomashefski, establish that the amount of pneumoconiosis present at the time of Miner's death in his left lung was minimal. Drs. Oesterling and Tomashefski both used the microphotographs to identify the only macules and micronodule that they were able to identify. Dr. Perper's opinion on the quantity of the presence of macules and micronodules was more generic, and he did not use the microphotographs he obtained to document his finding of numerous macules and small number of micronodules. The pathological evidence establishes that the amount of pneumoconiosis present in Miner's left lung at the time of his death was minimal and clinically insignificant. Drs. Castle, Naeye, Oesterling, and Tomashefski all opined that the nature of Miner's CWP was so minimal that it would not have caused Miner an impairment during his lifetime.

The minimal, insignificant nature of Miner's CWP, however, does not preclude a finding that Miner's death was due to CWP arising out of coal mine employment. Dr. Perper presented reasoned medical evidence supported by citations to medical literature that Miner's coal dust exposure and existing CWP contributed to the development of Miner's emphysema. Since the medical evidence establishes the significant role Miner's severe emphysema played in bringing about Miner's respiratory failure, Dr. Perper's opinion presents substantial evidence that Miner's CWP was a substantially contributing cause of Miner's death. However, Dr. Perper's opinion does not stand unopposed. Drs. Castle, Naeye, Oesterling, and Tomashefski all opine that Miner's minimal, simple CWP was so insignificant that it did not cause, contribute, or hasten Miner's death. They specifically disagree with Dr. Perper's contention that Miner's coal dust exposure and CWP contributed to Miner's development of emphysema. Instead, Drs. Castle, Naeye, Oesterling, and Tomashefski attribute the cause of Miner's emphysema to Miner's history of cigarette smoking. While all physicians who provided opinions in this matter considered a smoking history that exceeds the smoking history that the undersigned determined herein actually existed, the miss-reliance is of little consequence since their opinions on the etiology of Miner's emphysema do not rest solely on the length of Miner's smoking history. Rather, the determination of the etiology of Miner's emphysema is chiefly decided by the actual findings the reviewing pathologists rendered from Miner's autopsy and surgical resection slides.

Dr. Perper contends that coal dust exposure and CWP can lead to the development of centrilobular emphysema, and even potentially panlobular and bullous emphysema through its causation of centrilobular emphysema. He cites to a substantial body of medical literature to support his opinion. Dr. Perper also cites to the administrative findings of NIOSH, the Department of Labor, and the regulations that implement the Black Lung Act. While specific at times, Dr. Perper's opinion and argument, especially during his deposition, amounts to this opinion: a substantial body of medical and administrative evidence establishes that coal dust exposure and CWP can cause emphysema, Miner has CWP and emphysema, Miner had two

etiological risk factors for developing emphysema in the form of smoking and coal dust exposure, therefore, Miner's coal dust exposure and CWP led to Miner's emphysema, which led to Miner's death, so Miner's death was caused by CWP. Dr. Perper attempted to head-off criticism of his opinion by stating that physicians who do not recognize coal dust exposure and CWP as a causative factor in creating emphysema are relying out-dated medical literature.

Drs. Castle, Naeye, Oesterling, and Tomashefski criticized Dr. Perper's opinion. Drs. Naeye, Oesterling, and Tomashefski criticized the studies that Dr. Perper relied upon. The undersigned cannot resolve the differences in opinion between Dr. Perper and Drs. Naeye, Oesterling, and Tomashefski regarding the medical literature, but such a resolution is not appropriate or necessary. Even assuming that Dr. Perper's opinion on the interpretation of the medical literature is correct, which means that coal dust exposure and CWP can lead to the development of emphysema, the opinions of Drs. Naeye, Oesterling, and Tomashefski rule out coal dust exposure and CWP as the actual cause of Miner's emphysema. Dr. Perper detected a few coal macules that were surrounded by areas of focal emphysema. However, the collective opinions of Drs. Naeye, Oesterling, and Tomashefski establish that any areas of macules surrounded by focal emphysema, which would be due to coal dust and coal dust exposure, were insignificant and of no clinical significance because they occupied an insufficient amount of lung tissue to have any measurable effect on Miner's lung functioning and thus they did not have any effect on Miner's death. Further supporting the finding that Miner's coal dust exposure and CWP did not contribute to the development of his emphysema are the findings by Drs. Naeye, Oesterling, and Tomashefski that the areas of Miner's diffuse emphysema did not contain the crystals of birefringent silica that would have been deposited by coal dust exposure. Moreover, as demonstrated in the deposition testimony of Dr. Oesterling, for Miner's CWP to reach the point where it could have been considered to be related to Miner's panlobular, there would have to be evidence of compression of Miner's airways, and such evidence was not found.

Dr. Perper's opinion that Miner's coal dust exposure and CWP led to the development of Miner's emphysema was primarily based on his finding that Miner had coal dust exposure and CWP, and the medical literature establishes that coal dust exposure and CWP can cause emphysema. In contrast, while Drs. Naeye, Oesterling, and Tomashefski presented disagreement with Dr. Perper's characterization of the medical literature, their opinion on whether or not Miner's coal dust exposure and CWP caused any of Miner's forms of emphysema were based on their actual findings regarding the degree of changes in Miner's lung tissue that could be attributed to coal dust exposure or CWP. Drs. Naeye, Oesterling, and Tomashefski found that there was not even a significant amount of coal dust in Miner's lungs, and much less fibrotic change associated with coal dust particles. Because the opinions of Drs. Naeye, Oesterling, and Tomashefski were based on more specific and concentrated pathological findings regarding the absence of significant changes related to the presence of any particles associated with coal mining, I find their opinions to be better supported by the objective pathological evidence. Drs. Naeye, Oesterling, and Tomashefski based their opinions on the absence of significant, verifiable changes related to coal dust exposure or CWP. Since the minimal amount of the structural changes to Miner's lungs that were related to coal dust exposure or CWP only occupied an insignificant portion of Miner's lung and did not produce any measurable clinical impairment, Miner's CWP cannot be determined to have caused, contributed to, or hastened Miner's death.

Dr. Perper's opinion is insufficient to establish, by a preponderance of the evidence, that Miner's death was due to pneumoconiosis based on the contradictory evidence contained in the better-reasoned opinions of Drs. Naeye, Oesterling, and Tomashefski. I find that Miner's death was not directly caused by pneumoconiosis, nor was it contributed to by pneumoconiosis or any complication related to pneumoconiosis. Therefore, I find that the evidence relied upon by Claimant does not establish, by a preponderance of the evidence, that Miner's death was due to pneumoconiosis under § 718.205(c).

Entitlement

Claimant, Doris Trease, has failed to prove, by a preponderance of the evidence, that Miner's death was due to pneumoconiosis. Therefore, Mrs. Trease is not entitled to benefits under the Act.

Attorney's Fees

An award of attorney's fees is permitted only in cases in which the claimant is found to be entitled to benefits under the Act. Because benefits are not awarded in this case, the Act prohibits the charging of any fee to the Claimant for the representation and services rendered in pursuit of the claim.

ORDER

IT IS ORDERED that the claim of Doris Trease for benefits under the Act is hereby DENIED.

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THOMAS F. PHALEN, JR.
Administrative Law Judge

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 of this decision, by filing notice of appeal with the Benefits Review Board, P.O. Box 37601, Washington, D.C. 20013-7601. **A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, D.C. 20210.**