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Office of Administrative Law Judges
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Issue Date: 30 March 2006

In the Matter of:

ETHEL E. TOLLIVER, on behalf of and
Survivor of MICHAEL I. TOLLIVER,
Claimant

Case No.: 2000-BLA-882

v.

EASTERN ASSOCIATED COAL CORP.,
Employer

and

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

Appearances:

S. F. Raymond Smith, Esq.
Rundle and Rundle, LLC
Pineville, West Virginia
For the Claimant

Mark E. Solomons, Esq.
Laura Metcoff Klaus, Esq.
Greenberg Traurig, LLP
Washington, D.C.
For the Employer

Before: Alice M. Craft
Administrative Law Judge

DECISION AND ORDER AWARDING BENEFITS ON REMAND

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 et seq. The Act and implementing regulations, 20 CFR Parts 410, 718, 725 and 727, provide compensation and other benefits to living coal miners who are totally disabled due to pneumoconiosis and their dependents, and surviving dependents of coal miners whose death was due to pneumoconiosis. The Act and regulations define pneumoconiosis, commonly known as black lung disease, as a chronic dust disease of the lungs and its sequelae, including

respiratory and pulmonary impairments, arising out of coal mine employment. 30 U.S.C. § 902(b); 20 CFR § 718.201 (2005). In this case, the Claimant, Ethel E. Tolliver, alleges on behalf of her deceased husband, Michael I. Tolliver, that he was totally disabled by pneumoconiosis, and on her own behalf, that she is the surviving dependent of Mr. Tolliver, whose death was due to pneumoconiosis.

The case is before me on remand from the Benefits Review Board. I issued a Decision and Order Granting Benefits on September 29, 2003. The Board vacated my findings that the Claimant had shown a material change in conditions since denial of the miner's initial claim; that the miner had coal workers' pneumoconiosis; and that pneumoconiosis caused the miner's disability and death. The Board remanded the case to me with instructions to reweigh the medical evidence in accordance with the standards in the Fourth Circuit and the APA.

Due to the breadth of the Board's instructions, in this decision and order, I have entirely reevaluated the medical evidence.

PROCEDURAL HISTORY

Mr. Tolliver filed his initial claim on February 8, 1993. DX 32-1. The claim was denied by the District Director of the Office of Workers' Compensation Programs ("OWCP") on July 20, 1993, on the grounds that the evidence did not show that the Claimant had pneumoconiosis, or that it was caused by coal mine work, or that the Claimant was totally disabled by pneumoconiosis. DX 32-18. An attachment to the District Director's determination indicates that Mr. Tolliver's pulmonary function test met the standard for disability, but other evidence failed to show that his lung impairment was caused by coal mine work. DX 32-18 at p. 6. The Claimant did not appeal that determination. DX 32.

More than one year later, on May 16, 1995, the Claimant filed a duplicate claim. DX 1. The duplicate claim was granted by the District Director, and the Employer appealed. The case was heard by Administrative Law Judge ("ALJ") Stuart Levin on July 20, 1999, DX 62, but Mr. Tolliver died on September 25, 1999, before Judge Levin issued a decision. The case was remanded to the District Director to be consolidated with Mrs. Tolliver's survivor's claim. DX 69.

Mrs. Tolliver filed her survivor's claim on October 15, 1999. DX 70. The District Director issued an initial determination granting benefits on the survivor's claim, and finding that the new evidence did not affect previous findings on the miner's claim, on May 4, 2000. DX 87. The Employer appealed, DX 88, 89, and the claims were referred to the Office of Administrative Law Judges for hearing on June 26, 2000. DX 91.

I held a hearing on the consolidated claims on June 26, 2002, and issued a decision awarding benefits on both claims on September 29, 2003. The Employer appealed, and the Benefits review Board vacated the award on October 22, 2004. I received the file on remand from the Benefits Review Board on January 28, 2005, and the parties requested until April 20, 2005, to submit briefs. The Employer submitted a brief; the Claimant did not. The case is now ready for decision.

APPLICABLE STANDARDS

Both claims were filed before the effective date of the current regulations. Accordingly, I will apply only the sections of the newly revised version of Parts 718 and 725 that have not been found to be impermissibly retroactive. In this Decision and Order, the "old" rules applicable to this case will be cited to the 2000 edition of the Code of Federal Regulations; the "new" rules will be cited to the 2005 edition.

Pursuant to 20 CFR § 725.309 (2000), in order to establish that Mr. Tolliver was entitled to benefits in connection with his duplicate claim, the Claimant must demonstrate that there was a "material change in conditions" since the denial of his previous claim. In order to establish entitlement to benefits under Part 718, the Claimant must establish that Mr. Tolliver suffered from pneumoconiosis, that his pneumoconiosis arose out of his coal mine employment, and that his pneumoconiosis was totally disabling. 20 CFR §§ 718.1, 718.202, 718.203, 718.204, and 725.103 (2005). I must consider the new evidence and determine whether the Claimant has proved at least one of the elements of entitlement previously decided against the miner. If so, then I must consider whether all of the evidence establishes that he was entitled to benefits. *Lisa Lee Mines v. Director, OWCP*, 86 F.3d 1358 (4th Cir. 1996). In this case, the District Director implicitly found in the first claim that Mr. Tolliver was totally disabled by a pulmonary impairment. Thus the Claimant must establish that he had pneumoconiosis to show a material change in conditions. She must also show that pneumoconiosis contributed to his pulmonary disability to establish his right to benefits.

A surviving spouse is entitled to benefits if the miner died due to pneumoconiosis which arose out of coal mine employment. See 30 U.S.C. § 901; 20 CFR §§ 718.205 and 725.212(a)(3) (2005). The Claimant must first establish that the miner suffered from pneumoconiosis. *See Trumbo v. Reading Anthracite Co.,* 17 B.L.R. 1-85 (1993). In claims filed after January 1, 1982, death will be considered to be due to pneumoconiosis if (1) competent medical evidence establishes that the miner's death was due to pneumoconiosis; (2) pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or the death was caused by complications of pneumoconiosis; or (3) the presumption set forth at 20 CFR § 718.304 applies, i.e., an irrebuttable presumption that death was due to pneumoconiosis where there is medical evidence of complicated pneumoconiosis; but not if (4) the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death. 20 CFR § 718.205(c) (2005). This claim turns on whether pneumoconiosis hastened Mr. Tolliver's death from ventricular fibrillation during a bout of pancreatitis.

Medical Evidence

Chest X-rays

Chest x-rays may reveal opacities in the lungs caused by pneumoconiosis and other diseases. Larger and more numerous opacities result in greater lung impairment. The following table summarizes the x-ray findings available in connection with the current miner's claim and

the survivor's claim. X-rays taken before the initial claim was denied on July 20, 1993, have been marked with an asterisk (*). Readings of those x-rays have not been considered in determining whether there has been a change in conditions. *See Cline v. Westmoreland Coal Co.*, 21 B.L.R. 1-69, 1-74 (1997). X-ray readings introduced into evidence in the first claim of x-rays taken in November 1992 and January and March 1993 also appear on a separate chart below. The parties had some of those x-rays re-read for the current claim.

The existence of pneumoconiosis may be established by chest x-rays classified as category 1, 2, 3, A, B, or C according to ILO-U/C International Classification of Radiographs. Small opacities (1, 2, or 3) (in ascending order of profusion) may classified as round (p, q, r) or irregular (s, t, u), and may be evidence of "simple pneumoconiosis." Large opacities (greater than 1 cm) may be classified as A, B or C, in ascending order of size, and may be evidence of "complicated pneumoconiosis." A chest x-ray classified as category "0," including subcategories 0/-, 0/0, 0/1, does not constitute evidence of pneumoconiosis. 20 CFR § 718.102(b) (2000). Any such readings are therefore included in the "negative" column. X-ray interpretations which make no reference to pneumoconiosis, positive or negative, given in connection with medical treatment or review of an x-ray film solely to determine its quality, are listed in the "silent" column.

Physicians' qualifications appear after their names. Qualifications have been obtained where shown in the record by curriculum vitae or other representations. If no qualifications are noted for any of the following physicians, it means that either they have no special qualifications for reading x-rays, or I have been unable to ascertain their qualifications from the record. Qualifications of physicians are abbreviated as follows: A= NIOSH certified A reader; B= NIOSH certified B reader; BCR= board-certified in radiology. Readers who are board-certified radiologists and/or B readers are classified as the most qualified. See *Mullins Coal Co. v. Director, OWCP*, 484 U.S. 135, 145 n. 16 (1987); *Old Ben Coal Co. v. Battram*, 7 F.3d 1273, 1276 n.2 (7th Cir. 1993). B readers need not be radiologists.

Date of X-ray	Read as Positive for Pneumoconiosis	Read as Negative for Pneumoconiosis	Silent as to the Presence of Pneumoconiosis
09/16/92*			DX 72, DX 46 (EX 2) Ready Small areas of infiltrate and/or atelectasis
11/23/92*	EX 8 Renn B 1/2		DX 32-11 Smith Small nodular densities throughout both lungs

Date of X-ray	Read as Positive for Pneumoconiosis	Read as Negative for Pneumoconiosis	Silent as to the Presence of Pneumoconiosis
11/28-12/27/92*			DX 48 (EX 1), DX 32-11 Multiple chest studies taken during hospitalization were not read for pneumoconiosis but several showed interstitial and alveolar opacities DX 32-16 Sargent B/BCR One taken 12/23/92 unreadable
01/25/93*	DX 50, DX 48 (EX 4) Shipley BCR/B 1/0	DX 49, DX 48 (EX 3) Spitz BCR/B DX 42 Wiot BCR/B DX 32-15 Sargent BCR/B 0/1	DX 32-11 Van Dyck Chronic nodular interstitial pattern
03/04/93*	DX 50, DX 48 (EX 4) Shipley BCR/B 1/0 DX 32-17 Albin Not classified	DX 49, DX 48 (EX 3) Spitz BCR/B DX 42 Wiot BCR/B DX 32-14 Sargent BCR/B	
07/08/93*	DX 78, DX 48 (EX 2) Jenkins 1/1		
01/14/94	DX 78, DX 48 (EX 2) Jenkins (Not classified but said unchanged)		
03/18/94	EX 8 Renn B 2/1		
09/03/94	EX 8 Renn B 2/1		
09/16/94	EX 8 Renn B 2/1		
10/19/94			DX 72, DX 46 (EX 2) Wilson Minimal pleural scarring in right lung and probable bronchitis
06/28/95	DX 12 Sargent BCR/B 1/0 DX 14, DX 13 Patel BCR/A 2/2 DX 26 Wiot BCR/B 2/1	EX 3 Repsher B	
07/04/95	EX 8 Renn B 2/1		DX 48 (EX 5) Van Dyck Chronic interstitial changes with a reticular nodular pattern stable since 1992

Date of X-ray	Read as Positive for Pneumoconiosis	Read as Negative for Pneumoconiosis	Silent as to the Presence of Pneumoconiosis
01/15/96	DX 46 (EX 4) Renn B 2/2 DX 46 (EX 6) Wiot BCR/B 2/1 DX 42 Dahhan B 1/1	EX 3 Repsher B	
07/27/96			DX 48 (EX 5) Van Dyck Chronic interstitial changes
10/17/96			DX 48 (EX 5) (EX 1) Van Dyck No significant abnormality
03/27/97			DX 48 (EX 5) Schroeder Lung fields clear
03/10/99	DX 78 Jenkins 1/2	DX 85 Sargent BCR/B 0/1	
09/24/99	DX 72, 73 Saikewicz Profusion not classified (2 x-rays)	EX 3 Repsher B DX 75, DX 77 Sargent BCR/B 0/1	
09/24-09/25/99			DX 72, DX 73 Multiple additional chest studies taken during hospitalization for last illness reflect impressions of nodules, interstitial fibrosis, or other interstitial markings

X-ray interpretations from the prior claim appear on the following chart. As noted above, additional readings of these x-rays were introduced in the current claims, but none of the readings of x-rays in existence before denial of the initial claim have been considered in determining whether there has been a material change in conditions.

Date of X-ray	Read as Positive for Pneumoconiosis	Read as Negative for Pneumoconiosis	Silent as to the Presence of Pneumoconiosis
11/23/92			DX 32-11 Smith Small nodular densities throughout both lungs

Date of X-ray	Read as Positive for Pneumoconiosis	Read as Negative for Pneumoconiosis	Silent as to the Presence of Pneumoconiosis
11/28-12/27/92			DX 32-11 Multiple chest studies taken during hospitalization were not read for pneumoconiosis but several showed interstitial and alveolar opacities. DX 32-16 Sargent BCR/B One taken 12/23/92 unreadable
01/25/93		DX 32-15 Sargent BCR/B 0/1	DX 32-11 Van Dyck Chronic nodular interstitial pattern
03/04/93	DX 32-17 Albin not classified	DX 32-14 Sargent BCR/B	

Biopsies

Biopsies may be the basis for a finding of the existence of pneumoconiosis. A finding of anthracotic pigmentation is not sufficient, by itself, to establish pneumoconiosis. 20 CFR § 718.202(a)(2) (2005). Section 718.106(a) provides that a biopsy report shall include a detailed gross macroscopic and microscopic description of the lungs or visualized portion of a lung. If a surgical procedure was performed to obtain a portion of a lung, the evidence should include a copy of the surgical note and the pathology report. The Benefits Review Board has held, however, that the quality standards are not mandatory and failure to comply with the standards goes only to the reliability and weight of the evidence. *Dillon v. Peabody Coal Co*., 11 B.L.R. 1-113, 1-114 (1988); *see Dagnan v. Black Diamond Coal Mining Co*., 994 F.2d 1536, 1540-1541 (11th Cir. 1992). Section 718.106(c) provides that "[a] negative biopsy is not conclusive evidence that the miner does not have pneumoconiosis. However, where positive findings are obtained on biopsy, the results will constitute evidence of the presence of pneumoconiosis."

An open lung biopsy was performed on Mr. Tolliver's right lung on November 30, 1992, DX 46 (EX 3), DX 32-11, DX 32-6, after he was admitted to the hospital with an abnormal chest x-ray with reticulonodular infiltrate and a right pleural effusion, and a bronchoscopy failed to obtain enough tissue, *see* DX 32-11. At admission, his doctor thought his chest x-ray, in conjunction with weight loss and night sweats, suggested pulmonary tuberculosis. Both the surgical note and the pathology report from the biopsy are in the record. Dr. James R. Clay prepared the pathology report, including gross and microscopic description, the latter of which said in part:

> . . . The lung is densely fibrotic in most areas with dense fibrous tissue filling the air spaces. The one fragment which is not fibrotic has thin and delicate septae. Heavily pigmented macrophages are around some blood vessels and there [sic] areas having

> heavy lymphocytic infiltrates. No granulomata are present. There is no acute inflammation or tumor.
>
> Acid fast and GMS stains are negative for organisms.

Dr. Clay's diagnosis was "end stage pulmonary fibrosis, cause not apparent." DX 32-11, DX 32-6.

The Department of Labor sent the surgical pathology report and one glass slide with tissue to Dr. Richard L. Naeye, a pathologist, for his review. The Employer later submitted additional slides from the biopsy and other information to Dr. Naeye, and to another pathologist, Dr. Jerome Kleinerman. Dr. Naeye is board certified in anatomic and clinical pathology. He is a Professor and the Chairman of the Department of Pathology of The Pennsylvania State University College of Medicine. DX 45. Dr. Kleinerman is also board certified in anatomic and clinical pathology, and is the Director of the Department of Pathology of the MetroHealth Medical Center in Cleveland, Ohio, and a Professor Emeritus of the Case Western Reserve University School of Medicine. DX 44.

In his initial report prepared for the Department of Labor on March 23, 1993, based on the pathology report and a single slide, Dr. Naeye stated:

> 5 tiny pieces of tissue removed by surgery from the lungs are mainly comprised of fibrous tissue. A small amount of black pigment is present at 2 sites. There is fibrous tissue admixed with the black pigment but the fibrous tissue extends far beyond the black pigment so it appears that where the two are associated the association is fortuitous. There are no tiny birefringent crystals admixed with the black pigment.
>
> Not enough tissue is available for examination to be able to evaluate the extent or severity of centrilobular emphysema. No large bronchi are available for examination so the ratio of mucous to serous glands in the walls of bronchi cannot be determined.
>
> INTERPRETATION: Coal worker's pneumoconiosis (CWP) is absent in the tissue that is available for examination but the amount of tissue present is too small to be completely certain that CWP is absent. Since coal worker's pneumoconiosis is presumably absent it could not be causing impairments in lung function that would prevent this man from doing hard physical work in the coal mining industry. If he has died there is no evidence in the available tissue that CWP hastened his death. From the information that is available I do not know the cause of the fibrosis that is present in the small fragments of lung tissue.

DX 32-7.

In his second report prepared for the Employer on March 21, 1997, Dr. Naeye reported that he had reviewed 4 sets of slides, more than 20 other items including the results of multiple pulmonary function studies, chest x-rays and arterial blood gas analyses undertaken between 1992 and 1996, reports from 13 doctors, and medical records from Cobb Memorial Hospital and

Athens Regional Medical Center. Dr. Naeye noted about 20 years of coal mining history and greater than 25 pack years of smoking. He said x-rays did not disclose consistent findings conclusive for CWP, and a CT scan found a fine reticular-nodular pattern most compatible with interstitial fibrosis. Only one set of slides had black pigment located around blood vessels, while some subpleural fibrosis and interstitial fibrosis did not seem primarily associated with black pigment. He concluded that the tissue did not show the characteristic findings of CWP, but the tissue sample was not adequate to exclude the presence of CWP. Overall, the arterial blood gas values, pulmonary function studies and chest x-rays did not have characteristic findings of either simple or complicated CWP, although the x-rays suggested an interstitial disease. Based on the facts that Mr. Tolliver left the mines in 1987, and the first abnormality in arterial blood oxygen level appeared in 1993, and on the premise that simple CWP does not advance after a miner quits the industry, Dr. Naeye found no convincing evidence that CWP was present, and said that his clinical history indicated that Mr. Tolliver was disabled due to some type of immunologic rather than occupational disease. DX 45.

Dr. Kleinerman reviewed employment and medical records for Mr. Tolliver, and blood gas studies, pulmonary function tests and x-ray readings, as well as 21 histological slides from the biopsy. In his report he recounted Mr. Tolliver's employment and medical history at length. Dr. Kleinerman summarized his findings as follows:

> After review of the clinical records, the pulmonary function study results, the reports of chest x-rays, the surgical pathology report, and the pathology specimens, it is my opinion based on reasonable medical certainty that there is no evidence of simple coal workers' pneumoconiosis (CWP), simple nodular silicosis, nor complicated pneumoconiosis. Neither is there evidence of asbestosis or asbestos-related lung disease. It appears, based both on the histologic findings and the clinical histories that Mr. Tolliver's lung disease was caused by a combination of a lung-based collagen disease, probably rheumatoid arthritis, and the sequelae of Diffuse Alveolar Damage or ARDS. The pathologic changes in both of these entities is nonspecific. The presence of subpleural fibrosis with little or no coal dust pigment, of lesions characteristic of the interstitial fibrosis of honeycombing and the absence of macules of simple coal workers' pneumoconiosis rule out a diagnosis of CWP. Mr. Tolliver had interstitial fibrosis of a nonspecific type. The lung fibrosis was responsible in part for Mr. Tolliver's total disability. However, this disability was in <u>no</u> way the result of coal mine dust induced lung injury or any other occupationally-induced lung injury.

DX 44.

Because the biopsy was performed before Mr. Tolliver's initial claim was denied, it has not been considered in determining whether there has been a change in conditions since that claim was denied. *See Cline v. Westmoreland Coal Co.*, 21 B.L.R. 1-69, 1-74 (1997).

<u>CT Scans</u>

CT scans may be used to diagnose pneumoconiosis and other pulmonary diseases. The regulations provide no guidance for the evaluation of CT scans. They are not subject to the

specific requirements for evaluation of x-rays, and must be weighed with other acceptable medical evidence. *Melnick v. Consolidation Coal Co*., 16 B.L.R. 1-31, 1-33-1-34 (1991). The record for the current claims contains one report of a CT scan of Mr. Tolliver's chest, resulting in the following interpretation:

Exhibit #	Date of CT	Reading Physician	Interpretation or Impression
DX 46 (EX 4)	01/15/96	Williams for Renn	Very fine reticular nodular pattern throughout the lung fields could be compatible with coal worker's pneumoconiosis in the appropriate clinical setting

The record in Mr. Tolliver's initial claim contains reports of three CT scans. These earlier CT scans have not been considered in determining whether there has been a change in conditions, as they were taken before the initial claim was denied. *See Cline v. Westmoreland Coal Co.*, 21 B.L.R. 1-69, 1-74 (1997).

Exhibit #	Date of CT	Reading Physician	Interpretation or Impression
DX 32-11	11/23/92	Smith for Jenkins	Reticulonodular pattern throughout both lungs. Tuberculosis would be the initial consideration, but the differential includes other disease processes such as metastatic disease, sarcoidosis, and fungal infection
DX 32-11	11/29/92	Barton for Jenkins	Limited views of lung bases demonstrate persistent multiple small nodules with interval development of some bibasilar consolidation which may be secondary to atelectasis; persistent bibasilar pleural thickening
DX 32-11	12/10/92	Barton for Jenkins	Limited views of the lung bases demonstrate bilateral pleural effusions with interval increase on the left; bibasilar focal parenchymal consolidation involving both lungs, also appears increased on left

Pulmonary Function Studies

Pulmonary function studies are tests performed to measure obstruction in the airways of the lungs and the degree of impairment of pulmonary function. The greater the resistance to the flow of air, the more severe the lung impairment. The studies range from simple tests of ventilation to very sophisticated examinations requiring complicated equipment. The most frequently performed tests measure forced vital capacity (FVC), forced expiratory volume in one-second (FEV_1) and maximum voluntary ventilation (MVV).

The following chart summarizes the results of the pulmonary function studies available in connection with the current claims. "Pre" and "post" refer to administration of bronchodilators. If only one figure appears, bronchodilators were not administered. In a "qualifying" pulmonary study, the FEV_1 must be equal to or less than the applicable values set forth in the tables in Appendix B of Part 718, and either the FVC or MVV must be equal to or less than the applicable table value, or the FEV_1/FVC ratio must be 55% or less. 20 CFR § 718.204(b)(2)(i) (2005).

Ex. No. Date Physician	Age Height[1]	FEV_1 Pre-/ Post	FVC Pre-/ Post	FEV_1/ FVC Pre-/ Post	MVV Pre-/ Post	Qualify?	Physician Impression
DX 48 (EX 2) 07/08/93 Jenkins	43 70"	1.60	1.91	84%		Yes	Severe restriction
DX 48 (EX 2), DX 78 12/02/94 Jenkins	45 70"	1.59	1.92	83%	55	Yes	Moderately severe restriction improved since last measurement.
DX 7 06/28/95 Rasmussen	46 68.5"	1.92 1.97	2.31 2.31	83.1% 85.3%	110 110	Yes Yes	Moderate irreversible restrictive impairment. Acceptable per Dr. Ramavaya, DX 8. Invalid per Dr. Vest, DX 26, Dr. Repsher, DX 23. Valid, suggests moderate restrictive defect per Dr. Renn, DX 27, Dr. Goodman, DX 24.

[1] The fact-finder must resolve conflicting heights of the miner recorded on the ventilatory study reports in the claim. *Protopappas v. Director, OWCP*, 6 B.L.R. 1-221, 1-223 (1983); *Toler v. Eastern Assoc. Coal Co.*, 43 F.3d 109, 114, 116 (4th Cir. 1995). As there is a variance in the recorded height of the miner from 68" to 70", I have taken the mid-point (69") in determining whether the studies qualify to show disability under the regulations.

Ex. No. Date Physician	Age Height[1]	FEV_1 Pre-/ Post	FVC Pre-/ Post	FEV_1/ FVC Pre-/ Post	MVV Pre-/ Post	Qualify?	Physician Impression
DX 46 (EX 4) 01/15/96 Renn	46 69”	1.97 2.01	2.47 2.47	80% 81%	88 96	Yes Yes	Moderate restrictive defect.
DX 78 05/04/99 Jenkins	50 69”	2.05 2.12	2.39 2.45	86% 87%	90	Yes Yes	Moderately severe restriction

The following chart summarizes the results of the pulmonary function studies available in connection with the prior claim. Tests performed before the denial of the previous claim have not been considered in determining whether there has been a change in conditions. *See Cline v. Westmoreland Coal Co.*, 21 B.L.R. 1-69, 1-74 (1997).

Ex. No. Date Physician	Age Height	FEV_1 Pre-/ Post	FVC Pre-/ Post	FEV_1/ FVC Pre-/ Post	MVV Pre-/ Post	Qualify?	Physician Impression
DX 32-8 03/04/93 Albin	43 68”	1.60	1.98	80.8%	65.60	Yes	Moderate to severe restrictive impairment. No obstruction. Acceptable per Dr. Kraman DX 32-9

Arterial Blood Gas Studies

Blood gas studies are performed to measure the ability of the lungs to oxygenate blood. A defect will manifest itself primarily as a fall in arterial oxygen tension either at rest or during exercise. The blood sample is analyzed for the percentage of oxygen (PO_2) and the percentage of carbon dioxide (PCO_2) in the blood. A lower level of oxygen (O_2) compared to carbon dioxide (CO_2) in the blood indicates a deficiency in the transfer of gases through the alveoli which may leave the miner disabled.

The following chart summarizes the arterial blood gas studies available in connection with the current claims. A “qualifying” arterial gas study yields values which are equal to or less than the applicable values set forth in the tables in Appendix C of Part 718. If the results of a blood gas test at rest do not satisfy Appendix C, then an exercise blood gas test can be offered. Tests with only one figure represent studies at rest only. Exercise studies are not required if medically contraindicated. 20 CFR § 718.105(b) (2000).

Exhibit Number	Date	Physician	PCO_2 at rest/ exercise	PO_2 at rest/ exercise	Qualify?	Physician Impression
DX 72	09/16/92	Cobb Memorial Hospital	40.3	114	No	[In hospital with chest pain]
DX 72	10/20/94 00:20	Cobb Memorial Hospital	32.6	70	No	[In hospital with pneumonia]
DX 72	10/20/94 06:55	Cobb Memorial Hospital	38.8	83	No	[In hospital with pneumonia]
DX 10	06/28/95	Rasmussen	42 41	71 60	No Yes	Marked impairment during exercise. Acceptable per Dr. Gaziano, DX 11
DX 48 (EX 5)	07/04/95	Athens Regional Medical Center	37.8	57.7	Yes	[In hospital for fever and dyspnea]
DX 46 (EX 4)	01/15/96	Renn	45	68	No	Normal
DX 72	09/24/99 5 studies between 07:50 and 23:10	Cobb Memorial Hospital	See impression	See impression	Yes	4 out of 5 studies taken this date resulted in $pCO_2 \geq 50$ [In hospital with pancreatitis]

The following chart summarizes the arterial blood gas studies available in connection with the prior claims. Tests performed before the denial of the previous claim have not been considered in determining whether there has been a change in conditions. *See Cline v. Westmoreland Coal Co.*, 21 B.L.R. 1-69, 1-74 (1997).

Exhibit Number	Date	Physician	PCO_2 at rest/ exercise	PO_2 at rest/ exercise	Qualify?	Physician Impression
DX 32-11	21 studies taken from 12/14/92-01/07/93	Athens Regional Medical Center	See impression	See impression	Yes	19 of 21 studies during hospitalization resulted in $pCO_2 \geq 50$ [In hospital with sepsis with respiratory failure]
DX 32-13	03/08/93	Albin	41.8	91	No	

Treatment Records and Medical Opinions

Medical opinions are relevant to the issues of whether the miner had pneumoconiosis, whether the miner was totally disabled, and whether pneumoconiosis caused the miner's disability and death. A determination of the existence of pneumoconiosis may be made if a physician, exercising sound medical judgment, notwithstanding a negative x-ray, finds that the miner suffers from pneumoconiosis as defined in § 718.201. 20 CFR §§ 718.202(a)(4) (2005). Thus, even if the x-ray evidence is negative, medical opinions may establish the existence of pneumoconiosis. *Taylor v. Director, OWCP*, 9 B.L.R. 1-22 (1986). The medical opinions must be reasoned and supported by objective medical evidence such as blood gas studies, electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. 20 CFR § 718.202(a)(4) (2005). Where total disability cannot be established by pulmonary function tests, arterial blood gas studies, or cor pulmonale with right-sided heart failure, or where pulmonary function tests and/or blood gas studies are medically contraindicated, total disability may be nevertheless found, if a physician, exercising reasoned medical judgment, based on medically acceptable clinical and laboratory diagnostic techniques, concludes that a miner's respiratory or pulmonary condition prevents or prevented the miner from engaging in employment, i.e., performing his usual coal mine work or comparable and gainful work. 20 CFR § 718.204(b)(2)(iv) (2005). With certain specified exceptions not applicable here, the cause or causes of total disability must be established by means of a physician's documented and reasoned report. 20 CFR § 718.204(c)(2) (2005).

Treatment Records

Records from the Cobb Memorial Hospital show that Mr. Tolliver was admitted to the hospital from September 16 to 19, 1992, for acute chest pain, rule out myocardial infarction. He was kept in the ICU until his condition improved. His doctor of record at the hospital was Dr. Yu Chia Chao. He consulted with Dr. Pennington, who reported that Mr. Tolliver's pain could be due to CAD, but he was not experiencing an acute infarction. Dr. Pennington suggested that the pain could also be due to pericarditis. He recommended further tests, but noted that Mr. Tolliver was uninsured and lacked funds to pay. Mr. Tolliver returned to the emergency room a few days later on September 23, 1992, complaining of indigestion, but was released and advised to see his treating physician for follow-up. DX 72, DX 46 (EX 2).

Mr. Tolliver was admitted to the Athens Regional Medical Center from November 23, 1992, to January 9, 1993, after an abnormal chest x-ray with fever and weight loss. Initially it was suspected he might have tuberculosis, but other causes were also considered possible, including pneumoconiosis, rheumatoid arthritis, empyema and histoplasmosis. Eight different specialists were consulted during his hospitalization. Eventually he was diagnosed with sepsis syndrome with respiratory failure. The etiology was never pinpointed but likely sources included his kidneys, lungs and esophagus. He had diffuse pulmonary infiltrates of a reticular nodular pattern; open biopsy showed end-stage lung disease. Other diagnoses included rheumatoid arthritis; candida esophagitis, biopsy-proven with invasive fungal elements; gastroesophageal reflux with gastroesophageal ulcerations; renal stones with urinary tract infection, treated with lithotripsy; cardiomyopathy, probably sepsis induced, resolving prior to

discharge; severe hypertension, controlled with multiple medications; carious teeth, which were removed; renal insufficiency; anemia; and a medication-associated seizure disorder. His respiratory failure required mechanical ventilation for a prolonged period, from November 30 to the middle of December, and again for part of January. His condition was so bad he was expected to die in December. By the end of his hospitalization, he was so weak he was hardly able to stand by himself. DX 48 (EX 1), DX 32-11. He returned to the hospital for further lithotripsy for his renal stones in late January. DX 32-11.

Mr. Tolliver was treated by Dr. F. Hugh Jenkins of Athens Pulmonary and Allergy, P.C., during his hospitalization and after his discharge from the hospital, from February 1993, to July 1999. DX 78, DX 48 (EX 2), DX 32-11, DX 32-10, DX 22. Dr. Jenkins' qualifications are not in the file. It appears that Dr. Jenkins was Mr. Tolliver's primary medical care provider during this period; his office notes reflect about 25 visits. Dr. Jenkins read x-rays in 1993 and 1994 to be positive for diffuse small pulmonary nodules he believed to be consistent with Mr. Tolliver's previous work underground in the coal mine. In February 1993, Dr. Jenkins listed Mr. Tolliver's problems to be prolonged hospital stay for respiratory failure associated with sepsis; abnormal chest x-ray, multiple pulmonary nodules, open lung biopsy Dec. '92 equals end stage lung disease; nephrolithiasis, s/p lithotripsy times two; rheumatoid arthritis, chronic steroid use; and severe hypertension. In April 1993, Dr. Jenkins called Mr. Tolliver's continued improvement miraculous given the severe illness he went through. In July 1993, Dr. Jenkins said in the problem list that Mr. Tolliver's multiple pulmonary nodules were "probably pneumoconiosis." Dr. Jenkins' assessments generally reflect that Mr. Tolliver had severe multi system disease characterized as active but stable. Periodically Mr. Tolliver was taking steroids for his rheumatoid arthritis. At times Dr. Jenkins noted Mr. Tolliver to be experiencing shortness of breath; at other times he noted Mr. Tolliver's breathing to be improving. Progress notes from Mr. Tolliver's last documented visit on July 12, 1999, listed his problems as hypertensive cardiovascular disease, diabetes mellitus, ischemic cardiomyopathy, rheumatoid arthritis, abnormal chest x-ray, increased interstitial markings bilaterally, and neck pain. Dr. Jenkins ordered an MRI of his cervical spine and referred him to Dr. Troy Sullivan to treat his diabetes and cholesterol.

Records from the Cobb Memorial Hospital show that Mr. Tolliver was seen in the emergency room and released to home on March 16, 1994, after a dizzy spell which was attributed to hypertension. An abnormal echocardiogram taken in September 1994 was reported to show an echogenic mass near the base of the aortic valve, leading to a recommendation for a transesophageal echo, the results of which are not in the file. He was admitted to Cobb Memorial Hospital from October 19 to 24, 1994, for probable septicemia and acute pneumonia. His doctor of record at the hospital was Dr. Chao. DX 72, DX 46 (EX 2).

Mr. Tolliver was admitted to Athens Regional Medical Center from July 4-7, 1995, with fever and dyspnea which improved with reinstitution of low-dose Prednisone. The cause for his fever was thought to be associated with rheumatoid arthritis and Prednisone withdrawal; other causes were not found. DX 48 (EX 5).

Mr. Tolliver was admitted to Athens Regional Medical Center from July 27 to August 1, 1996, for chest pain. He was diagnosed with an inferior wall infarction and underwent cardiac

catheterization and angioplasty. DX 48 (EX 5) (EX 1). After his release from the hospital, he went for office visits to Dr. Harvey Ouzts at Athens Cardiology Group, P.C., who had been a consulting physician during his November 1992-January 1993 hospitalization. On August 23, 1996, Dr. Ouzts reported his examination as normal with stable angina after infarction and PTCA. He also assessed pulmonary fibrosis and rheumatoid arthritis. DX 48 (EX 1). Mr. Tolliver was readmitted to Athens Regional Medical Center from October 17 to 20, 1996, with chest pain. Catheterization showed re-occlusion. He was diagnosed with subendocardial infarction due to severe coronary disease. DX 48 (EX 5) (EX 1). On November 8, 1996, Dr. Ouzts assessed him as being stable but with pretty severe angina after mild effort. DX 48 (EX 1).

Dr. Ouzts next saw Mr. Tolliver on March 3, 1997, and observed that his angina was the same, occurring with minimal activity. His assessment was "stable angina and pulmonary fibrosis." DX 48 (EX 1). Records from the Athens Regional Medical Center show that Mr. Tolliver visited the emergency room on March 27, 1997, complaining of chest pain. He was admitted overnight, and discharged with a diagnosis of non-cardiac chest pain. EX 10, DX 48 (EX 5) (EX 1). On September 23, 1997, Dr. Ouzts said his angina was still stable, and planned to see him again in a year. DX 48 (EX 1).

Mr. Tolliver went to the emergency room again in July 1998, complaining of back pain diagnosed as an acute lumbar strain with spasm. EX 10, DX 48 (EX 5). The following month he was seen for muscle spasm in his back at Cobb Memorial. DX 72. After his September 1998 annual visit to his cardiologist, Dr. Ouzts again reported that he had stable coronary artery disease with a stable angina pattern. DX 48 (EX 1).

Dr. Troy Sullivan's office treatment records for Mr. Tolliver are found in EX 9 and DX 73. Dr. Sullivan first saw Mr. Tolliver on July 14, 1999, when Dr. Jenkins referred him for management of his diabetes, which was poorly controlled. Mr. Tolliver was 50 years old, and had no particular complaints. Dr. Sullivan took a history and performed an examination. His impressions were Type II diabetes, hypertension and hyperlipidemia, mostly hypertriglyceridemia. Dr. Sullivan changed some of his medications. When Dr. Sullivan saw him in follow-up two months later, on September 14, 1999, he added poor sleep, possibly due to depression, and chronic lung disease with evidence of obstructive lung disease to the diagnoses.

Ten days after his second office visit, on September 24, 1999, Mr. Tolliver was admitted to the hospital with acute pancreatitis under the care of Dr. Sullivan. *See* DX 73. The records of his final hospitalization at Cobb Memorial Hospital are found at DX 72. The dismissal diagnoses were severe acute pancreatitis, renal failure, hyperkalemia, metabolic acidosis, respiratory acidosis, COPD, CAD, type II diabetes mellitus, coal worker's pneumoconiosis and rheumatoid arthritis. Dr. Sullivan described Mr. Tolliver's course in the hospital as follows:

> Mr. Tolliver was admitted in the early morning of 9/24/99 with severe pancreatitis. Amylase of 2696. Throughout the day of 9/24 he was treated with fluid resuscitation and supportive treatment of electrolyte abnormalities including hyperglycemia, first hypokalemia followed by hyperkalemia. Patient became more uncomfortable and in order to more easily treat his acidosis and fluid imbalance he was intubated in the early

> morning of 9/25. He had a successful intubation and in the early morning hours of 9/25 approximately 0140 he went into cardiac arrest, ventricular fibrillation from which the patient could not be resuscitated. He was pronounced deceased at 0204.

Dr. Sullivan signed Mr. Tolliver's death certificate. He identified the immediate cause of death as ventricular fibrillation "due to, or as a consequence of" electrolyte imbalance and renal failure. Under "other significant conditions" he identified severe pancreatitis. EX 10; DX 71.

Medical Opinions Given in Connection with the Claims

The record contains the following medical opinions submitted in connection with the current claims.

1. Dr. Rasmussen

On June 25, 1995, Dr. D. L. Rasmussen examined Mr. Tolliver on behalf of the Department of Labor. DX 9. Dr. Rasmussen's qualifications are not found in the record. He took occupational, social, family and medical histories, and conducted a physical examination, chest x-ray, blood gas studies and pulmonary function testing, Based upon his examination, Dr. Rasmussen concluded that Mr. Tolliver was suffering from coal worker's pneumoconiosis, based on 20 years of coal mine employment and x-ray evidence of pneumoconiosis; a history of ARDS; and a history suggestive of hyperactive airways disease. He said that Mr. Tolliver had at least moderate loss of respiratory functional capacity and was totally disabled for resuming his last coal mine job. As to the extent which each of the diagnoses contributed to Mr. Tolliver's impairment, he said:

> There are 3 possible risk factors for his impairment in lung function. These include his coal mine dust exposure with its resultant pneumoconiosis, his previous ARDS, and possibly his rheumatoid arthritis. It is not possible to separate these effects. His coal mine dust exposure must be considered a significant or major contributing factor. His possible asthma is not reflected in the current study.

DX 9 at 4. His examination and conclusions were discussed in detail in a narrative attached to his report on the Department of Labor's form.

Dr. Rasmussen reviewed additional records submitted to him by counsel for Mr. Tolliver and prepared a report dated December 30, 1996. DX 40. The materials he reviewed included the first two reports from Dr. Renn, described below, which in turn referred to Dr. Naeye's report on the biopsy. Dr. Rasmussen stated he did not have Dr. Naeye's report, and that it would be of interest for him to see the reports of other pathologists concerning the biopsy material. He went on to state:

> All the x-rays in this case are quite consistent with coal workers' pneumoconiosis predominately; in fact, almost exclusively, showing rounded pea-sized nodular densities in all lung zones; a pattern which is not typical of the usual diffuse interstitial pulmonary fibrosis. It is also quite possible that biopsy of the right lower lobe might miss the

> presence of coal workers' pneumoconiosis since the predominant lesion in coal workers' pneumoconiosis is in the upper lung zones. Thus, in my opinion, a diagnosis of coal workers' pneumoconiosis has not been excluded.

DX 40 at 3. He went on to cite studies showing interstitial pulmonary fibrosis in coal miners, sometimes associated with coal pigment deposition, and sometimes not; and studies showing active alveolitis associated with impaired function. He then concluded:

> Thus, one must include Mr. Tolliver's coal mine dust exposure as a possible significant contributing factor and any diffuse interstitial pulmonary fibrosis which he may have.
>
> Mr. Tolliver also certainly has other medical conditions which could contribute to or cause the same abnormality; mainly, history of having had a diagnosis of rheumatoid arthritis established and also having suffered adult respiratory stress syndrome. However, it is impossible to exclude the patient's coal mine dust exposure as a significant contributing factor in view of the evidence for interstitial fibrosis occurring among coal miners.
>
> Based on all of the above, it remains my opinion that Mr. Tolliver does suffer from coal mine dust related lung disease which is a significant contributing cause of his disabling pulmonary insufficiency.

DX 40 at 4.

2. Dr. Sullivan

On October 21, 1999, Dr. Sullivan wrote the following letter addressed to the Division of Coal Miners Workman's Compensation:

> I have been asked to write you a letter regarding the death and subsequent eligibility for benefits of Mr. Michael Tolliver and his widow Ethel Tolliver. Mr. Tolliver carried the diagnosis of coal worker's pneumoconiosis, he also suffered from type II diabetes mellitus and coronary artery disease with two previous myocardial infarctions. In addition he suffered from rheumatoid arthritis. In late September of the year Mr. Tolliver was admitted to the hospital for an acute severe pancreatitis, he subsequently died from complications of that illness.
>
> It is my impression that his pre-existing medical problems played a major role in his death from this condition. He ultimately died from ventricular fibrillation which I believe was brought on by electrolyte imbalances including acidosis and hyperkalemia. These were due to renal failure and inadequate ventilation. His ventilatory failure was a result of this acute illness compounding his previous lung disease. Mr. Tolliver had to be intubated in the early hours of the morning. Within about 30 minutes of the intubation he died. I therefore, believe that Mr. Tolliver would not have died from pancreatitis had it not been for his pneumoconiosis and other health problems.

DX 74 at 2, EX 9 at 4. In a follow-up letter to Claimant's counsel dated September 12, 2001, Dr. Sullivan stated:

> After reviewing my letter of October 21, 1999 and the comments of the occupational pneumoconiosis board,[3] I maintain my belief that Mr. Tolliver's pneumoconiosis played a major role in his death. His renal failure and inadequate ventilation led to acidosis and hyperkalemia. The acidosis and electrolyte abnormality caused death by inducing a cardiac arrhythmia.

EX 9 at 5.

3. Dr. Tuteur

Dr. Peter Tuteur reviewed Mr. Tolliver's medical records on several occasions on behalf of the Employer, and provided reports dated December 17, 1996, DX 42, June 7, 1999, DX 48 (EX 6), and May 29, 2002, EX 1. He was deposed twice, on July 6, 1999, DX 48 (EX 10), and June 18, 2002. EX 11. Dr. Tuteur is board-certified in internal medicine and pulmonary disease, and is an Associate Professor at the School of Medicine at the Washington University in St. Louis. EX 4, DX 42. He treats patients as a pulmonary consultant, as well as teaching medical students and providing continuing medical education. He is the director of the Pulmonary Function Laboratory at the University.

For his initial report, DX 42, Dr. Tuteur reviewed medical records and reports from 1992 to 1996, as well as the results of arterial blood gas analysis dated January 7, 1993, pulmonary function studies dated June 28, 1995 and January 15, 1996, seven radiographic reports, and lung biopsy reports by Dr. Naeye and Dr. Clay. Dr. Tuteur identified Mr. Tolliver's medical problems as including renal functional insufficiency, adult onset diabetes mellitus, rheumatoid arthritis, and urosepsis associated with adult respiratory distress syndrome (ARDS) and subsequent development of interstitial pulmonary fibrosis. Based on the biopsy results, physical examination revealing crackles characteristic of an interstitial pulmonary process, pulmonary function studies demonstrating a restrictive ventilatory defect, and chest radiographs an CT scans confirming the interstitial process subsequently confirmed by biopsy, Dr. Tuteur concluded that Mr. Tolliver had diffuse interstitial pulmonary process due to diffuse interstitial pulmonary fibrosis. He said that both coal workers' pneumoconiosis and post-ARDS interstitial fibrosis were potential etiologies, and that the differential can be made from the pathologic specimen, as follows:

> If coal dust macules, focal emphysema, and pigment-associated fibrosis were prominent features, then coal workers' pneumoconiosis must be considered the primary interstitial process. In contrast, even if pigment is present, as expected in any coal miner, if fibrosis is predominantly unassociated with pigment deposition and coal dust macules, then post-

[3] This may be a reference to findings dated December 19, 2000, in which the board stated, "After careful review of all of the available medical evidence, it is the opinion of the Occupational Pneumoconiosis Board that occupational pneumoconiosis was not a material contributing factor in Mr. Tolliver's death." EX 7.

> ARDS fibrotic process is most likely. Based on the description of the pathologic specimens by both pathologists, and based on the learned conclusions of Dr. Naeye, it is with reasonable medical certainty that the diffuse interstitial pulmonary process pathologically proved and associated with clinically-consistent parameters is not due to the inhalation of coal mine dust, but a result of adult respiratory distress syndrome caused by urosepsis and treated with prolonged mechanical ventilation. Neither the ARDS nor its sequelae are in any was related to, aggravated by, or caused by the inhalation of coal mine dust or the development of coal workers' pneumoconiosis. It is due to urinary tract infection that led to bacteremia and septicemia.

DX 42.

In his June 1999 report, DX 48 (EX 6), Dr. Tuteur listed medical records he had reviewed for the period from 1992 to 1998. He said that the newly available data provided detail and breadth to the previous data, and provided further support for his conclusion that coal dust did not contribute to Mr. Tolliver's interstitial pulmonary process. After summarizing the available data, including pulmonary function and arterial blood gas test results and radiological data, Dr. Tuteur stated:

> Many reviewers of this data set conclude that with reasonable medical certainty Mr. Tolliver does not have coal workers' pneumoconiosis. These conclusions are based not only on the absence of fulfillment of pathologic criteria . . . but also the review of the totality of available medical records . . . Dr. Rasmussen is not as definitive in supporting the absence of coal workers' pneumoconiosis. . . I agree that in a scholarly and rigorous sense, absolute exclusion of the presence of pathologically significant coal workers' pneumoconiosis has not been accomplished. Neither has the entire right lung been observed, nor have multiple areas been biopsied. Yet, if coal workers' pneumoconiosis were to be so extensive as to produce substantial influence on Mr. Tolliver's clinical course, one would have expected fulfillment of pathologic criteria even in a relatively small sample. Thus, though the diagnosis of CWP "has not been excluded", with reasonable medical certainty it is not present. The clinical symptoms and physical examination findings as well as physiologic impairment seen in Mr. Tolliver's records are typical for coal workers' pneumoconiosis because it is typical for interstitial fibrotic process of which coal workers' pneumoconiosis is one of many. On this basis, these factors only continue to prove the presence of an interstitial pulmonary process, not its etiology. . . .There is no pathological evidence to support the presence of coal workers' pneumoconiosis. Pulmonary fibrosis exists in areas where no coal mine dust was present in the lungs. In fact, there is relatively little coal mine dust present in Mr. Tolliver's lungs. No coal dust macules were observed. No coal dust associated nodules were observed. And, of extreme import, is that the interstitial pulmonary process is fully explained as a consequence of the adult respiratory distress syndrome possibly in a patient with rheumatologic disorder.

Dr. Tuteur concluded that Mr. Tolliver was totally and permanently disabled, but that his disability was in no way related to the inhalation of coal mine dust, but rather to his other multiple medical problems.

At his first deposition, DX 40 (EX 10), Dr. Tuteur attributed Mr. Tolliver's interstitial fibrosis to his bout with ARDS in 1992. He suggested that x-ray reviewers who found changes consistent with pneumoconiosis did not have complete clinical information. He said that open lung biopsy such as Mr. Tolliver had is the gold standard for the diagnosis of the presence of coal workers' pneumoconiosis, and that the pathologic data indicate that there was an interstitial fibrosis unrelated to the presence of coal mine dust. He said he knew that to be true because there was a minimal amount of dust in the sub plural area, and more in macrophages around blood vessels, which is the location of dust and associated fibrosis not found with coal workers' pneumoconiosis; where there was fibrosis, there was no dust; and none of the other required anatomical criteria for the diagnosis of pathologically significant coal workers' pneumoconiosis were present, i.e., there were no micro nodules, no macro nodules, no coal dust macules and no focal emphysema. He observed that the reports of all three pathologists were consistent in the description of the lung pathology. It was his opinion that Mr. Tolliver's fibrosis was not caused by rheumatoid arthritis or Caplan's syndrome (rheumatoid lung disease in conjunction with deposition of coal mine dust in the lungs) because the hallmark of both, necrobiotic nodules, were not present. Dr. Tuteur again disagreed with Dr. Rasmussen's view that the diagnosis of coal workers' pneumoconiosis had not been excluded; he agreed it could not be excluded with 100% certainty, but went on to state:

> . . . But the questions asked of me were not is it remotely possible, but with reasonable medical certainty did Mr. Tolliver have coal workers' pneumoconiosis. And with reasonable medical certainty he did not. He did not, because open lung biopsy didn't show it. He did not because the fibrosis that was present, absent the associated coal dust deposition, absent the coal dust macule, absent the macro nodules, absent the micro nodules, absent the focal emphysema, was fully explained by the clinical course of Mr. Tolliver's illness. He had adult respiratory distress syndrome superimposed on arteriosclerotic heart disease, hypertensive heart disease, renal functional insufficiency and recurrent urinary tract stones, which at this point in 1992 prior to the development of ARDS was associated with a urinary tract infection which eventually became urinary tract sepsis.
>
> Once that adult respiratory distress syndrome was established, one would have expected dust unrelated interstitial pulmonary fibrosis at the time of that biopsy and so, yes, I agree with Dr. Rasmussen, you cannot rigorously and scholarly absolutely exclude the presence of coal workers' pneumoconiosis somewhere in Mr. Tolliver's lungs, but one can exclude with the utmost of reasonable medical certainty that coal workers' pneumoconiosis was present in his lungs and caused physiologic changes, radiographic changes and physical exam changes.

DX 48 (EX 10 at 19-20). Dr. Tuteur said that Mr. Tolliver would be unable to do the last job he had in the coal mine industry because he had symptoms of exertional breathlessness. Except for some worsening of Mr. Tolliver's coronary artery disease, his other health problems were stable or had improved between 1993 and 1999.

In his most recent report, EX 1, Dr. Tuteur stated that he had reviewed treatment records and reports by other reviewers from 1999, and reconsidered all of the pulmonary function and radiographic data and other data available to him. Dr. Tuteur opined that the newly available data about Mr. Tolliver's last illness confirmed his view that Mr. Tolliver did not have pneumoconiosis. He concluded:

> . . . Mr. Tolliver's database was reviewed in detail once again. The additional medical information listed above was integrated into the totality of available medical data. These data continue to support the conclusions previously reached, that with reasonable medical certainty, Mr. Tolliver did not have clinical, physiologic, radiographic or pathologic evidence supporting the diagnosis of coal workers' pneumoconiosis or any other coal mine dust-induced disease process. He did have a primary pulmonary problem. That process was an interstitial fibrotic process secondary to the healing associated with a 1992 adult respiratory distress syndrome.
>
> As a result of his multiple major medical problems (coronary artery disease, hypertension, elevated lipids, uncontrolled diabetes mellitus, chronic mild renal functional insufficiency), Mr. Tolliver was totally and permanently disabled. This disability was in no way related to, aggravated by, or caused by either the inhalation of coal mine dust or the development of coal workers' pneumoconiosis.
>
> . . .
>
> Simply, Mr. Tolliver had multiple important and advanced medical health problems. From a previous acute respiratory illness (ARDS), he developed an interstitial pulmonary process that pathologically and microscopically differed from and did not fulfill the criteria for the diagnosis of coal workers' pneumoconiosis. When he developed acute pancreatitis superimposed on these problems, compensatory mechanisms were suboptimal and he developed electrolyte imbalance, metabolic acidosis, respiratory failure, renal failure, and despite aggressive medical management, ARDS once again complicated by ventricular fibrillation and death. Neither the inhalation of coal mine dust nor the development of coal workers' pneumoconiosis played any role in this sequence of events.

EX 1 at 5.

During his second deposition, EX 11, Dr. Tuteur stated that he sees individuals with occupational lung diseases in his practice, and has treated and evaluated coal miners for lung disease. As part of his duties, he is the director of the pulmonary function laboratory. Dr. Tuteur reviewed Mr. Tolliver's medical history, including adult onset diabetes mellitus, not well-controlled, complicated by renal insufficiency; coronary artery disease; rheumatoid arthritis; a history of urosepsis complicated by adult respiratory distress syndrome leading to interstitial pulmonary fibrosis, confirmed by lung biopsy which demonstrated no pneumoconiosis or coal macules; and hypertension contributing to heart and vascular diseases. Dr. Tuteur said that acute severe pancreatitis has about a 50% mortality rate. He attributed Mr. Tolliver's pancreatitis to hyperlipidemia. He said pancreatitis in a person with interstitial fibrosis can lead to

underventilation, which he said was documented in Mr. Tolliver by an elevated PCO_2. He said that underventilation produces acidosis in the blood, as does pancreatitis, out of control diabetes and renal failure, so that Mr. Tolliver had four independent active processes which cause acidemia and acidosis. In his opinion, Mr. Tolliver had scarring on his lungs as a result of ARDS because it appeared about the time of his episode of urosepsis and ARDS, and was not associated with coal dust macules or necrobiotic nodules, which are a hallmark of rheumatoid lung. He described Mr. Tolliver's death as "cardiorespiratory," by which he meant that independent causes influenced both cardiac failure and respiratory failure. He said Mr. Tolliver's spirometric studies reflected a stable restrictive ventilatory abnormality caused by ARDS interstitial fibrosis.

In order to determine the etiology of interstitial fibrosis, Dr. Tuteur said he would look at several factors. First would be whether the individual was at risk by exposure to coal mine dust. Then he would look at whether there is a temporal relationship, the development of progressive breathlessness, of late inspiratory crackles, of decreased lung expansion, of physiologic impairment in this pattern while exposed to coal dust, and did it progress subsequently; were x-rays abnormal while exposed to dust and did they subsequently progress; was a biopsy performed while still working in the mines or shortly thereafter that shows coal dust macules; macules associated interstitial fibrosis; focal emphysema, etc. In Mr. Tolliver's case, total lung capacity measurements were small and confirmed a restrictive abnormality. Arterial blood gas studies were consistent with mild impairment of gas exchange at rest, which worsens with exercise. Those findings are true of any interstitial pulmonary process, including pneumoconiosis, rheumatoid lung disease, post ARDS fibrosis and idiopathic pulmonary fibrosis. Carboxyhemoglobin levels were normal, consistent with the history that Mr. Tolliver had quit smoking.

Dr. Tuteur disagreed with Dr. Sullivan's statement that pneumoconiosis played a "major" role in Mr. Tolliver's death. He said there was "no convincing or even credible medical evidence" that supports Dr. Sullivan's statement. Dr. Tuteur did agree that Mr. Tolliver's other health problems contributed to his rapid death from acute pancreatitis, which is difficult to treat because of the rapid development of acidosis. In Mr. Tolliver's case, in addition to acidosis from pancreatitis, there was also metabolic acidosis from renal insufficiency and keto acidosis from his diabetes. He said that the pancreatitis adversely affected the function of Mr. Tolliver's diaphragm, contributing to ventilatory failure, but that the high CO_2 reading was not due to lung dysfunction. Dr. Tuteur reiterated that he found no evidence that Mr. Tolliver had pneumoconiosis, or that pneumoconiosis caused his disability or contributed to his death.

5. Dr. Repsher

Dr. Lawrence Repsher reviewed Mr. Tolliver's medical records on several occasions and provided reports dated December 16, 1996, DX 42, June 15, 1999, DX 58, DX 48 (EX 8), and May 31, 2002, EX 3, and reviewed additional records in preparation for his deposition. Dr. Repsher was deposed on June 21, 2002. EX 12. Dr. Repsher is board-certified in internal medicine and pulmonary disease, with sub-specialties in occupational and environmental lung diseases, and is a B-reader. EX 6, DX 42.

For his initial report in 1996, DX 42, Dr. Repsher reviewed Mr. Tolliver's employment record, reports of examinations by Drs. Rasmussen and Renn, Dr. Naeye's report for the Department of Labor, medical records from Cobb Memorial Hospital from June 10, 1992 to October 24, 1994, and x-ray reports for x-rays taken June 28, 1995, and January 15, 1996. Dr. Repsher said that although the chest x-rays were consistent with pneumoconiosis, among many other things, there were radiographic nuances suggesting that the abnormalities are not due to pneumoconiosis, as follows:

> These include the fact that this is described as a reticular nodular infiltrate, whereas coal workers pneumoconiosis is primarily a nodular infiltrate. Further, there appears to be more involvement of the lower lobes than the upper lobes, whereas coal workers pneumoconiosis primarily involves the upper lobes. Finally, the ultimate arbiter, which is the open lung biopsy, showed no evidence of coal workers pneumoconiosis.

As to the pulmonary function tests, Dr. Repsher stated that they were consistent with restrictive lunge disease, compatible with the histologically documented interstitial lung disease. He attributed limitations of cardiopulmonary reserve in exercise testing to probable underlying heart disease. He disagreed with Dr. Rasmussen's opinion that Mr. Tolliver had CWP, ARDS, reactive airways disease, and possible rheumatoid arthritis, and that coal mine dust was a significant factor to Mr. Tolliver's lung disease, saying that the lung biopsy showed no evidence of coal workers pneumoconiosis and the chest x-ray was atypical for pneumoconiosis; that it was never documented that Mr. Tolliver had ARDS or reactive airways disease; and that rheumatoid arthritis was specifically ruled out by the serologic studies in September 1992. Dr. Repsher agreed with Dr. Renn that there was no evidence of pneumoconiosis, but disagreed with Dr. Renn's diagnosis of rheumatoid arthritis with idiopathic pulmonary fibrosis. In Dr. Repsher's opinion, Mr. Tolliver had clear cut serologic evidence of active systemic lupus erythematosus. In his view, therefore, the interstitial lung disease was due to lupus. Summing up, Dr. Repsher said Mr. Tolliver suffered from a number of very serious medical conditions, and was totally and permanently disabled, but found no evidence of medical or legal pneumoconiosis. DX 42.

In his June 1999 report, DX 58, DX 48 (EX 8), Dr. Repsher reviewed additional records and again concluded that there was no evidence of pneumoconiosis or any other respiratory disease caused or aggravated by his coal mine employment. In his opinion, Mr. Tolliver was totally and permanently disabled as a result of severe ischemic heart disease and ischemic cardiomyopathy with severe angina pectoris and chronic congestive heart failure, as well as probable diastolic dysfunction as a result of sever hypertension and resulting left ventricular hypertrophy. Although Mr. Tolliver had some restrictive impairment of lung function with some impairment of his diffusing capacity, Dr. Repsher characterized the impairment as "modest and probably not limiting." He said the pulmonary impairment was due solely to his underlying collagen vascular disease, as there was no evidence of any disease arising from coal mine employment on biopsy. DX 58.

In his most recent report, EX 3, Dr. Repsher again stated that Mr. Tolliver had no radiographic or biopsy evidence of legal or medical pneumoconiosis. Although Mr. Tolliver was totally and permanently disabled, his disability was due to medical problems unrelated to exposure to coal dust, and, since he did not have pneumoconiosis, pneumoconiosis did not cause,

contribute to or hasten his death. At his deposition, EX 12, Dr. Repsher said he has examined hundreds of coal miners, including examinations he conducted for the Department of Labor. His opinions about Mr. Tolliver did not change over time, although they evolved in light of additional diagnoses and the events surrounding Mr. Tolliver's death. Dr. Repsher opined that Mr. Tolliver had sufficient exposure to coal dust to cause disease in a sensitive miner. He concluded, however, that he did not develop any coal-dust-induced disease, nor any chronic obstructive disease due to smoking. His medical history included high blood pressure complicated by left ventricular hypertrophy with probable hypertensive cardiovascular disease, severe four-vessel coronary artery disease with two prior heart attacks, type II diabetes mellitus, severe lipid abnormalities, which may have accounted for his terminal episode of pancreatitis, and severe systemic lupus erythematosus manifested by pericarditis, pleuritis, lupus pneumonia, end stage lupus lung disease, dermatitis, arthritis, anemia, recurrent fevers, probable cerebritis and possible Libman-Sacks endocarditis. None of his diseases were caused by inhalation of coal mine dust. Dr. Repsher testified that abnormalities he found on x-rays were due to lupus or left ventricular congestive heart failure. He explained that plain x-rays do not distinguish the changes of lupus from the changes of congestive heart failure. He said the results of the CT, which are more sensitive, confirmed the x-ray findings. Pulmonary function tests were abnormal. He said that Mr. Tolliver's lung disease had a significant impact on his lung function, although not disabling, but that did not tell the cause. With regard to Mr. Tolliver's last illness, Dr. Repsher stated that both high triglycerides and lupus can cause pancreatitis. Because it was accompanied by shock, it carried a high risk of dying. The immediate cause of death was ventricular fibrillation. Causes of the fibrillation were probably multi-factorial, including severe heart disease, with the effect of pancreatitis with shock, severe metabolic acidosis and high potassium. Based on review of Dr. Sullivan's records and report, Dr. Repsher believed that Dr. Sullivan was concerned mostly with Mr. Tolliver's severe ischemic heart disease. There was no mention of coal-dust-induced lung disease on the death certificate. Dr. Repsher found no evidence of complicated coal workers pneumoconiosis, or any coal-dust-induced disease, including in the lung biopsy. He said there was no evidence of pneumoconiosis at all.

6. Dr. Dahhan

Dr. Abdul Dahhan reviewed Mr. Tolliver's medical records on several occasions and provided reports dated December 11, 1996, DX 42, June 2, 1999, DX 48 (EX 7), and May 27, 2002, EX 2. Dr. Dahhan was deposed on June 24, 2002. EX 13. Dr. Dahhan is board-certified in internal medicine and pulmonary disease, and is a B reader. EX 5, DX 42. He practices in a geographic area dependent on coal mining for industry, and the bulk of his patients are active or retired coal miners and their families. He has been treating miners since 1974.

In preparation for his first report, DX 42, Dr. Dahhan reviewed Mr. Tolliver's medical records from 1992 to 1996, and reports by other doctors. He concluded that Mr. Tolliver had radiological evidence consistent with simple coal workers' pneumoconiosis, but that there was no evidence of progressive massive fibrosis or complicated pneumoconiosis. He said Mr. Tolliver had a moderate restrictive ventilatory impairment demonstrated by the pulmonary function studies, and did not retain the capacity to return to his previous coal mine work. He believed that the cause was multi-factorial, and that the bulk of Mr. Tolliver's restrictive ventilatory abnormality and secondary pulmonary impairment was due to pulmonary fibrosis

secondary to rheumatoid lung disease and post ARDS pulmonary fibrosis, both of which are well known and reported in medical literature. He concluded that Mr. Tolliver's restrictive ventilatory impairment did not result from coal dust exposure or occupational pneumoconiosis because his simple pneumoconiosis was not severe enough to cause the development of a restrictive impairment of the nature seen in the pulmonary function studies, absent evidence of complicated pneumoconiosis in chest x-rays or pathological tissue.

Dr. Dahhan reviewed additional treatment records and other reports for his 1999 report. DX 48 (EX 7). He continued to find insufficient objective findings to justify the diagnosis of coal workers' pneumoconiosis. He confirmed his view that Mr. Tolliver had interstitial pulmonary fibrosis resulting from rheumatoid arthritis, stating that it was "highly plausible" that the 1992 bout with adult respiratory distress syndrome contributed to the interstitial fibrosis. He observed that all physicians had noted that Mr. Tolliver did not retain the respiratory capacity to continue his previous coal mining work because of his interstitial fibrosis. His other multiple medical problems were not caused by, contributed to or aggravated by coal dust exposure.

In his most recent report, EX 13, Dr. Dahhan said that Mr. Tolliver died as a result of ventricular arrhythmia secondary to electrolyte imbalance and pancreatitis. Based on the pathology reports by Dr. Kleinerman and Dr. Naeye, Dr. Dahhan found no evidence of coal dust induced lung disease. He attributed Mr. Tolliver's pulmonary fibrosis to rheumatoid lung disease and previous adult respiratory distress syndrome. At his deposition, he said he never examined Mr. Tolliver but believed that he had sufficient data to evaluate his pulmonary status from reviewing his records. Dr. Dahhan said that Mr. Tolliver had sufficient exposure to coal dust to cause dust-induced disease in a susceptible individual. Dr. Dahhan opined that Mr. Tolliver did not have pneumoconiosis. He said the gold standard for making that diagnosis is by lung biopsy, which in Mr. Tolliver's case did not show any pathological data indicative of pneumoconiosis. Although Dr. Dahhan had interpreted an x-ray taken January 15, 1996, as consistent with pneumoconiosis, it was overruled or superseded by the biopsy evidence. He said that pulmonary function tests indicated that Mr. Tolliver had a restrictive abnormality which could be caused by the pulmonary fibrosis reported by the pathologist. The biopsy ruled out coal dust as the cause of the fibrosis. Other possible causes, including adult respiratory distress syndrome induced fibrosis, rheumatoid arthritis, and idiopathic pulmonary fibrosis of the variety of Hamman Rich Syndrome, could not be ruled out. He also said that Mr. Tolliver did not have cor pulmonale, nor any evidence of complicated pneumoconiosis. Dr. Dahhan said that Mr. Tolliver's respiratory impairment and death were not related in any way to coal mine dust exposure. He testified that Mr. Tolliver's final hospital admission and death resulted from pancreatitis which caused electrolyte imbalance and ventricular tachycardia resulting in his death.

6. Dr. Renn

Dr. Joseph Renn examined Mr. Tolliver on January 15, 1996, DX 46 (EX 4), and provided supplemental record reviews on June 18, 1999, DX 48 (EX 9), and May 13, 2002, EX 8. He was deposed on July 15, 1999, DX 48 (EX 11), and testified on behalf of the Employer at the second hearing, Tr. 10-42. He is board certified in internal medicine, pulmonary disease, and forensic medicine. He is also a B reader. DX 46 (EX 5). He maintains a clinical practice and

consults in pulmonary diseases, and in intensive care of critically ill individuals, especially those who require assisted ventilation. He treats coal miners in his practice, some of whom have coal workers' pneumoconiosis, including complicated pneumoconiosis, and some of whom have idiopathic pulmonary fibrosis.

During Dr. Renn's January 1996 examination of Mr. Tolliver, he took an occupational history, a cardiopulmonary history, a past medical history, history of tobacco use, list of medications, personal history, and family history; performed a physical examination; and performed laboratory evaluation. The laboratory evaluation included blood tests, resting electrocardiograph, echocardiogram, chest x-ray, high-resolution CT scan, spirometry, lung volumes, diffusing capacity and resting arterial blood gases. Dr. Renn also reviewed numerous medical records, listed in his report. Dr. Renn diagnosed idiopathic pulmonary interstitial fibrosis, chronic bronchitis, rheumatoid arthritis, systemic hypertension, left ventricular hypertrophy, adult onset diabetes mellitus, exogenous obesity, hyperlipidemia, drug-induced hepatic dysfunction, and, by past medical history, adult respiratory distress syndrome secondary to sepsis, congestive heart failure secondary to cardiomyopathy and gastroesophageal reflux disease. DX 46 (EX 4).

In his June 1999 report, DX 48 (EX 9), after reviewing medical records for the period from 1992 to 1998, Dr. Renn observed that the only apparent serious new medical development since his 1996 examination were acute myocardial infarctions which occurred in July and October 1996. Cardiac catheterization had revealed severe arteriosclerotic coronary vascular disease for which Mr. Tolliver had angioplasty, after which he continued to have angina pectoris. Dr. Renn also reviewed pulmonary function, gas exchange and x-ray data he had not seen before. He was still of the opinion that Mr. Tolliver had idiopathic pulmonary interstitial fibrosis, likely a result of either rheumatoid arthritis or ARDS, but not pneumoconiosis.

At his deposition, DX 48 (EX 11), Dr. Renn explained his opinion that Mr. Tolliver did not have coal worker's pneumoconiosis. Dr. Renn said that Mr. Tolliver was hospitalized in 1992 because a urinary tract infection which became blood borne, known as sepsis, which can in turn cause diffuse alveolar damage to the lungs, from adult respiratory distress syndrome. ARDS causes such severe compromise of function that the majority of people who have it require mechanical ventilation, as did Mr. Tolliver. He was also diagnosed with rheumatoid arthritis at that time, which can also affect the lungs in several different ways, by causing necrobiotic rheumatoid nodules, Caplan's syndrome and diffuse pulmonary interstitial fibrosis. He agreed that the evidence from chest x-rays, pulmonary function studies and arterial blood gas studies would be similar in some respects, whether he had interstitial pulmonary fibrosis or coal workers' pneumoconiosis. Thus he read the January 16, 1996, x-ray as consistent with pneumoconiosis. Nonetheless, it was his opinion that Mr. Tolliver did not have pneumoconiosis, based on the CT scan, the pathology reports and other factors. While some of the evidence was consistent with pneumoconiosis, other evidence was not; but the total pattern of studies was consistent with the damage caused by ARDS. Dr. Renn believed that Mr. Tolliver was disabled by his pulmonary impairment, but was certain that exposure to coal dust did not cause or aggravate it. He said coal dust is not known to cause the diffuse alveolar damage that Mr. Tolliver had; rather, it affects the primary and secondary respiratory bronchioles, but not the alveolar area. He said that from a pulmonary function standpoint, Mr. Tolliver would have been

in the same position if he had never been a coal miner. He disagreed with Dr. Rasmussen's statement that there was no reason to distinguish between pneumoconiosis and other types or causes of pulmonary fibrosis, because different types of interstitial diseases have prescribed treatments, while pneumoconiosis does not. Thus failure to distinguish between the causes limits therapeutic options. On cross examination, he said he could not say what treatment would have been appropriate for Mr. Tolliver because he did not have enough information, such as whether his disease was active. He could not determine the exact nature of the pulmonary fibrosis, but could offer the most likely causes.

In his most recent report, EX 8, Dr. Renn challenged Dr. Sullivan's suggestion that pneumoconiosis contributed to Mr. Tolliver's death as follows:

> Whether or not Mr. Tolliver would have survived the acute pancreatitis will not be known owing to the method of his demise. From the time of his hospitalization he was in a persistent state of metabolic acidosis which was not vigorously and adequately addressed as he continued to worsen throughout his hospitalization. . . . Dr. Sullivan recognized that Mr. Tolliver ". . . ultimately died from ventricular fibrillation which I believed was brought on by electrolyte imbalances including acidosis and hyperkalemia." His statement that Mr. Tolliver would not have died from pancreatitis had it not been for his pneumoconiosis and other health problems is without foundation as the mortality rate for an initial episode of acute pancreatitis is in excess of 50%. Further, Mr. Tolliver's primary problem was not respiratory failure; rather, it was metabolic acidosis for all the reasons enumerated above and it was proven that he could be adequately oxygenated with relatively low flow supplemental oxygen that did not require endotracheal intubation and mechanical ventilation.
>
> Enclosed are charts titled "Pulmonary Function and Gas Exchange Data" containing studies performed on May 4, 1999, . . . The ventilatory function is comparable to that obtained in this laboratory on January 15, 1996. That his ventilatory function some five months prior to his demise was comparable to that obtained more than three years previously objectively reveals that his ventilatory function had not deteriorated over that period of time and a respiratory death would not be expected. . . .

EX 8 at 5-6.

At the second hearing, Dr. Renn said that when he evaluates an individual for black lung, he keeps in mind both the medical and the legal definitions of pneumoconiosis. Mr. Tolliver had sufficient exposure to coal dust to cause coal-dust-induced disease in a susceptible individual. Symptoms such as shortness of breath can be caused by many different body systems, not just the respiratory system. Mr. Tolliver had a complex cardiopulmonary history. His shortness of breath was related to his respiratory system, his cardiovascular system and his musculoskeletal system. He also had productive cough and wheezing. He was taking medications for multiple problems, including diabetes, heart failure, reflux disease, rheumatoid arthritis and interstitial lung disease. About a third of people with rheumatoid arthritis develop interstitial lung disease which can be confused with disease caused by coal dust. When he examined Mr. Tolliver, his chest examination was normal, except for auscultation, which revealed fine inspiratory crackles

in the bases that did not clear after a deep cough, suggesting interstitial disease. Dr. Renn said that Mr. Tolliver's problem began between the wall of the alveolus and the blood vessel, which is different than that caused by coal dust, at the respiratory bronchiole. There are over 200 interstitial diseases, including coal workers' and other pneumoconioses. The distinction between causes can be based on the pathology. In Mr. Tolliver's case, the pathology found alveolar areas involved with the interstitial fibrosis, without the coal macule that would be present with medical coal workers' pneumoconiosis. Laboratory data revealed high blood sugar from diabetes; liver function abnormalities, possibly from medication; and an abnormal electrocardiogram. Echocardiogram showed an enlarged left side of the heart, but normal right side, so he did not have cor pulmonale. Chest x-ray showed rounded opacities q in all lung zones, profusion 2/2. CT scan showed a mixture of both linear and nodular opacities. Mr. Tolliver had a restrictive ventilatory defect with no evidence of obstruction, and normal resting arterial blood gases, with a reduction of profusing capacity which corrected to normal when volume was considered. Exercise blood gases could not be done because of resting tachycardia, high blood pressure, electrocardiogram results and his history of seizures and cardiac arrest. Dr. Renn diagnosed idiopathic pulmonary interstitial fibrosis. He called it idiopathic because there were too many diseases Mr. Tolliver had which could cause interstitial disease, including rheumatoid arthritis, a history of adult respiratory distress syndrome, and side effects from medication he was taking. On cross examination, Dr. Renn confirmed that Mr. Tolliver had both linear and nodular opacities on the CT scan, and agreed it was possible that Mr. Tolliver had at least a couple of disease processes going on. In his view, the linear opacities would have been more related to the rheumatoid disease, while the nodular opacities, as well as some linear opacities, could be more related to the adult respiratory distress syndrome or the diffuse alveolar damage that resulted from that.

Dr. Renn testified that Mr. Tolliver's final admission to the hospital and death resulted from acute pancreatitis, probably due to high triglycerides. The mortality rate from a first episode of pancreatitis is 50%. Dr. Renn believed that Mr. Tolliver died due to metabolic acidosis related to his pancreatitis, and not a respiratory problem. He did not understand why Mr. Tolliver's doctor put him on a ventilator when he was well oxygenated with relatively small amounts of oxygen. Intubation can cause cardiac arrhythmia, which made Mr. Tolliver more prone to develop ventricular fibrillation. He also received Dopamine, which also causes cardiac irritability. Although there were shadows on the x-rays which could be consistent with coal workers' pneumoconiosis, based on the other information available, Dr. Renn concluded that the shadows were not pneumoconiosis. In Dr. Renn's opinion, Mr. Tolliver did not have any pulmonary or respiratory impairment caused by work in the mines. Although he believed Mr. Tolliver to be totally disabled, he had no coal-dust-induced disease which had a material adverse effect on his pulmonary or respiratory conditions, or worsened another pulmonary or respiratory disease. Dr. Renn opined that coal-dust-induced disease did not contribute to Mr. Tolliver's disability, and did not contribute to or hasten his death.

7. Dr. Albin

The following opinion was given in connection with Mr. Tolliver's initial claim. It has not been considered in determining whether there has been a material change in conditions since the first claim was denied. *See Cline v. Westmoreland Coal Co.*, 21 B.L.R. 1-69, 1-74 (1997).

Dr. Robert Albin examined Mr. Tolliver on behalf of the Department of Labor on March 4, 1993, in connection with Mr. Tolliver's first application for benefits. DX 32-12. Dr. Albin's qualifications are not in the file. He took occupational, social, family and medical histories, and conducted a physical examination, chest x-ray, blood gas studies and pulmonary function testing. Based upon his examination, Dr. Albin concluded that Mr. Tolliver had a moderate to severe restrictive defect shown by pulmonary function testing. As to the etiology, he reported that it was possibly related to either rheumatoid arthritis or inhalation. He observed that Mr. Tolliver did not have evidence of desaturation, but could not be exercised due to a recent heart attack. A hemoglobin level of only 9.2 raised the question of whether anemia might be playing a role in some of Mr. Tolliver's symptoms. Dr. Albin characterized the degree of Mr. Tolliver's impairment as "moderate."

Existence of Pneumoconiosis

The regulations define pneumoconiosis broadly:

> (a) 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 pneumoconioses, i.e., the 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 silico-tuberculosis, 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.
>
> (b) For purposes of this section, a disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.
>
> (c) For purposes of this definition, "pneumoconiosis" is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.

20 CFR § 718.201 (2005).

20 CFR § 718.202(a) (2005) provides that a finding of the existence of pneumoconiosis may be based on (1) chest x-ray, (2) biopsy or autopsy, (3) application of the presumptions described in Sections 718.304 (irrebuttable presumption of total disability or death was due to pneumoconiosis if there is a showing of complicated pneumoconiosis), 718.305 (not applicable to claims filed after January 1, 1982) or 718.306 (applicable only to deceased miners who died on or before March 1, 1978), or (4) a physician exercising sound medical judgment based on objective medical evidence and supported by a reasoned medical opinion.

Pneumoconiosis is a progressive and irreversible disease. *Labelle Processing Co. v. Swarrow*, 72 F.3d 308, 314-315 (3rd Cir. 1995); *Lane Hollow Coal Co. v. Director, OWCP*, 137 F.3d 799, 803 (4th Cir. 1998); *Woodward v. Director, OWCP*, 991 F.2d 314, 320 (6th Cir. 1993). As a general rule, therefore, more weight is given to the most recent evidence. See *Mullins Coal Co. of Virginia v. Director, OWCP*, 484 U.S. 135, 151-152 (1987); *Eastern Associated Coal Corp. v. Director, OWCP*, 220 F.3d 250, 258-259 (4th Cir. 2000); *Crace v. Kentland-Elkhorn Coal Corp*., 109 F.3d 1163, 1167 (6th Cir. 1997); *Rochester & Pittsburgh Coal Co. v. Krecota*, 868 F.2d 600, 602 (3rd Cir. 1989); *Stanford v. Director, OWCP*, 7 B.L.R. 1-541, 1-543 (1984); *Tokarcik v. Consolidated Coal Co*., 6 B.L.R. 1-666, 1-668 (1983); *Call v. Director, OWCP*, 2 B.L.R. 1-146, 1-148-1-149 (1979). This rule is not to be mechanically applied to require that later evidence be accepted over earlier evidence. *Woodward*, 991 F.2d at 319-320; *Adkins v. Director, OWCP*, 958 F.2d 49 (4th Cir. 1992); *Burns v. Director, OWCP*, 7 B.L.R. 1-597, 1-600 (1984).

The miner's claim is a duplicate claim. In a duplicate claim, the threshold issue is whether there has been a material change in conditions since the previous claim was denied. As noted above, in denying the first claim, the District Director found that the evidence established that Mr. Tolliver was disabled by a pulmonary impairment, but failed to show that his exposure to coal dust was a contributing cause. Thus the first determination must be whether the Claimant has established with new evidence that Mr. Tolliver suffered from pneumoconiosis or other pulmonary or respiratory impairment significantly related to or aggravated by dust exposure. Absent a finding that he suffered from such an impairment, there has been no material change in conditions, and the miner's claim must fail. Thus I must first consider the evidence in the duplicate claim to determine whether it shows Mr. Tolliver had pneumoconiosis. If I so find, then I must consider all of the available evidence to determine whether he was entitled to benefits.

Two of the four methods of establishing the existence of pneumoconiosis are foreclosed from consideration on the issue of change of conditions in the miner's claim. The biopsy was performed before the initial claim was denied,[2] and there was no autopsy. None of the presumptions apply, because the evidence does not establish the existence of complicated pneumoconiosis, the claims were filed after January 1, 1982, and Mr. Tolliver died after March 1, 1978. In order to determine whether the evidence establishes the existence of pneumoconiosis, therefore, I must consider the new chest x-rays and medical opinions, including the new CT scan. Absent contrary evidence, evidence relevant to any category may establish the

[2] Although I have not separately considered the biopsy report in reaching a conclusion regarding change in conditions, all of the doctors except Dr. Rasmussen, and possibly Dr. Sullivan, had access to it, and took it into account in forming their opinions. For this reason, I have addressed it in the context of their opinions.

existence of pneumoconiosis. In the face of conflicting evidence, however, I must weigh all of the evidence together in reaching my finding whether the Claimant has established that Mr. Tolliver had pneumoconiosis. *Island Creek Coal Co. v. Compton*, 211 F.3d 203, 211 (4th Cir. 2000).

Of the x-rays taken after July 20, 1993, some have been read as positive, some as negative, and some as both positive and negative. For cases with conflicting x-ray evidence, the regulations specifically provide,

> … where two or more X-ray reports are in conflict, in evaluating such X-ray reports consideration shall be given to the radiological qualifications of the physicians interpreting such X-rays.

20 CFR § 718.202(a)(1) (2005); *Dixon v. North Camp Coal Co*., 8 B.L.R. 1-344 (1985); *Melnick v. Consolidation Coal Co*., 16 B.L.R. 1-31, 1-37 (1991). Readers who are board-certified radiologists and/or B-readers are classified as the most qualified. The qualifications of a certified radiologist are at least comparable to if not superior to a physician certified as a B-reader. *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211, 1-213 n.5 (1985). Greater weight may be accorded to x-ray interpretations of dually qualified physicians. *Sheckler v. Clinchfield Coal Co*., 7 B.L.R. 1-128, 1-131 (1984). A judge may consider the number of interpretations on each side of the issue, but not to the exclusion of a qualitative evaluation of the x-rays and their readers. *Woodward*, 991 F.2d at 321; *see Adkins*, 958 F.2d at 52. Whether an x-ray interpretation which is **silent** as to pneumoconiosis should be interpreted as **negative** for pneumoconiosis, is an issue of fact for the ALJ to resolve. *Marra v. Consolidation Coal Co.*, 7 B.L.R. 1-216 (1984); *Sacolick v. Rushton Mining Co.*, 6 B.L.R. 1-930 (1984).

The x-ray taken on January 14, 1994, has been read only as positive, but has not been classified as required by the regulations. Thus it cannot be used to establish the existence of pneumoconiosis.

Three x-rays taken March 18, September 3 and September 16, 1994, have all been read as positive, by Dr. Renn, a B reader. There are no negative readings. These x-rays are positive.

The only reading of the x-ray taken October 19, 1994, in the course of treatment, makes no mention of pneumoconiosis, but does mention pleural scarring. I find this x-ray is neither positive nor negative.

The x-ray taken June 28, 1995, has been read as positive by three dually qualified readers, all radiologists, two of whom were B readers (Drs. Sargent and Wiot), and one of whom was an A reader (Dr. Patel), and as negative by Dr. Repsher, a B reader. As more, better qualified readers read it to be positive, I find it to be positive.

The x-ray taken on July 4, 1995, was read as positive by Dr. Renn, a B reader. There are no negative readings. The radiologist who interpreted it at the time it was taken observed interstitial changes with a reticular nodular pattern. I find this x-ray to be positive.

The x-ray taken January 15, 1996, has been read and classified as positive by three readers, two B readers, Drs. Renn and Dahhan, and one dually qualified, Dr. Wiot. One B reader, Dr. Repsher, said it was negative. Based on the greater number of positive readings by well-qualified readers, including a dually-qualified radiologist, I find this x-ray to by positive.

The x-ray taken July 27, 1996, during the course of treatment, is silent as to pneumoconiosis, but mentions chronic interstitial changes. This x-ray is neither positive nor negative.

The only interpretation of the next x-ray taken for purposes of treatment, on March 27, 1997, characterized the lung fields as clear. I find this x-ray to be negative. I note, however, that it is anomalous, as most other x-ray readers observed some form of interstitial disease.

An x-ray taken March 10, 1999, was read as positive by Dr. Jenkins, qualifications unknown, but as negative (0/1) by Dr. Sargent, who is dually qualified. Dr. Sargent's reading is entitled to greater weight based on his known qualifications.

Two x-rays taken during Mr. Tolliver's final illness were said to show pneumoconiosis by Dr. Saikewicz, qualifications unknown, but the profusion was not classified. The x-rays were read as negative by Dr. Repsher, a B reader, and by Dr. Sargent, who is dually qualified. Again, the negative readings outweigh the positive reading. Other x-rays taken during Mr. Tolliver's last hospitalization were silent as to pneumoconiosis, but the findings were such that they should not be considered negative.

Although I have found that there were more positive than negative x-rays, I have also found that the most recent x-rays, all taken during Mr. Tolliver's last year of life, were either negative, or unclassified. Thus I find the x-ray evidence is inconclusive, and insufficient to establish the existence of pneumoconiosis.

Next I must consider the medical opinions submitted in connection with the current claim. The Claimant can establish that Mr. Tolliver suffered from pneumoconiosis by well-reasoned, well-documented medical reports. A "documented" opinion is one that sets forth the clinical findings, observations, facts, and other data upon which the physician based the diagnosis. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19, 1-22 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient's work and social histories. *Hoffman v. B&G Construction Co.*, 8 B.L.R. 1-65, 1-66 (1985); *Hess v. Clinchfield Coal Co.*, 7 B.L.R. 1-295, 1-296 (1984); *Justus v. Director, OWCP*, 6 B.L.R. 1-1127, 1-1129 (1984). A "reasoned" opinion is one in which the judge finds the underlying documentation and data adequate to support the physician's conclusions. *Fields*, above. Whether a medical report is sufficiently documented and reasoned is for the judge to decide as the finder-of-fact; an unreasoned or undocumented opinion may be given little or no weight. *Clark v. Karst-Robbins Coal Co.*, 12 B.L.R. 1-149, 1-155 (1989) (en banc). An unsupported medical conclusion is not a reasoned diagnosis. *Fuller v. Gibraltar Corp.*, 6 B.L.R. 1-1291, 1-1294 (1984). A physician's report may be rejected where the basis for the physician's opinion cannot be determined. *Cosaltar v. Mathies Coal Co.*, 6 B.L.R. 1-1182, 1-1184 (1984). An opinion may be given little weight if it is equivocal or vague. *Griffith v. Director, OWCP*, 49

F.3d 184, 186-187 (6th Cir. 1995); *Justice v. Island Creek Coal Co*., 11 B.L.R. 1-91, 1-94 (1988); *Parsons v. Black Diamond Coal Co.*, 7 B.L.R. 1-236, 1-239 (1984).

The qualifications of the physicians are relevant in assessing the respective probative values to which their opinions are entitled. *Burns v. Director, OWCP*, 7 B.L.R. 1-597, 1-599 (1984). More weight may be accorded to the conclusions of a treating physician as he or she is more likely to be familiar with the miner's condition than a physician who examines him episodically. *Onderko v. Director, OWC*P, 14 B.L.R. 1-2, 1-6 (1989). However, a judge "is not required to accord greater weight to the opinion of a physician based solely on his status as the Claimant's treating physician. Rather, this is one factor which may be taken into consideration in … weighing … the medical evidence …" *Tedesco v. Director, OWCP*, 18 B.L.R. 1-103, 1-105 (1994). In assessing the probative value of conflicting opinions, I must account for "the qualifications of the respective physicians, the explanation of their medical opinions, the documentation underlying their medical judgments, and the sophistication and bases of their diagnoses." *Sterling Smokeless Coal Co. v. Akers*, 131 F.3d 438, 441 (4th Cir. 1997). *See also*, *Clark v. Karst-Robbins Corp.* 12 B.L.R. 1-149 (1989) (en banc); *Lucostic v. United States Steel Corp.*, 8 B.L.R. 1-46 (1985). *Accord*, *Underwood v. Elkay Mining, Inc*.,105 F.3d 946, 950-951 (4th Cir. 1997).

The Claimant has not offered any evidence which substantiates the background and qualifications of the doctors who offered opinions favorable to the claim. The credentials of the pulmonologists consulted by the Employer, on the other hand, are well documented in the record. Dr. Rasmussen and Dr. Sullivan suggested that coal dust exposure contributed to Mr. Tolliver's pulmonary impairment. As the physician who attended Mr. Tolliver during his last illness, Dr. Sullivan's opinion letter primarily focused on the cause of death. Moreover, Mr. Tolliver was referred to Dr. Sullivan for treatment of his diabetes, rather than his pulmonary problems, which suggests that pulmonary problems were not his specialty. In addition, Dr. Sullivan saw Mr. Tolliver only twice before he was hospitalized with pancreatitis. It is unclear from the record what, if any, records from Mr. Tolliver's past treatment Dr. Sullivan had available to him. Treatment records from Dr. Jenkins, who attended Mr. Tolliver in the hospital when he suffered from sepsis with respiratory failure, and then treated him from 1993 to 1999, suggest that he is a pulmonary specialist. His notes indicate that he believed Mr. Tolliver's interstitial fibrosis to have been caused by coal dust exposure. He did not submit an opinion in the claim, however, and the basis for his view is largely unexplained. Dr. Sullivan may have relied on Dr. Jenkins' diagnosis of pneumoconiosis in formulating his opinion, i.e., in his choice of wording when he specifically attributed pneumoconiosis as a contributing cause of death. Because neither Dr. Jenkins nor Dr. Sullivan gave much explanation of their reasoning for diagnosing pneumoconiosis, I must weigh Dr. Rasmussen's opinion that coal dust was a factor in the lung impairment, on the one hand, against those of the four doctors consulted by the Employer that coal dust was not a factor, on the other.

Dr. Rasmussen was consulted in the context of the living miner's claim. He had the opportunity to take histories, conduct objective testing, and examine Mr. Tolliver, as well as to review some, but not all, of Mr. Tolliver's medical records made available to the Employer's consultants. He stated that Mr. Tolliver "suffer[ed] coal mine dust related lung disease which [was] a significant contributing cause of his disabling pulmonary insufficiency." Dr. Rasmussen

acknowledged that Mr. Tolliver's history of rheumatoid arthritis and adult respiratory distress syndrome could also have contributed to his interstitial fibrosis. He said that it is impossible to exclude coal mine dust exposure as a contributing cause in view of the evidence of interstitial fibrosis occurring among coal miners, with or without associated coal pigment. I find that his opinion was well-documented, well-reasoned, and sufficiently explained to support a finding of pneumoconiosis, unless outweighed by the contrary opinions.

Arrayed against his opinion are those of Dr. Tuteur, Dr. Repsher, Dr. Dahhan, and Dr. Renn, all board certified specialists in pulmonary disease. Drs. Tuteur, Repsher and Dahhan performed extensive reviews of Mr. Tolliver's medical records, and Dr. Renn examined him, as well as reviewing his records. As Dr. Rasmussen's qualifications are not in the record, all four must be deemed to possess superior credentials, and they had as much or more information than Dr. Rasmussen on which to base an opinion. On the other hand, their opinions are not consistent with each other, which undermines the effect of their documented qualifications.

At the outset, Dr. Repsher's opinion is vastly different than any of the other consulting or treating doctors'. He alone said Mr. Tolliver's lung disease was due to lupus, and he alone said it was not disabling. The latter statement is contrary to the objective evidence in the record. Pulmonary function tests consistently showed restrictive lung disease of sufficient degree to meet the requirement for disability found in the regulations. The only exercise arterial blood study also resulted in a qualifying result. Dr. Repsher's contention that Mr. Tolliver's lung impairment was not limiting undermines confidence in his unique diagnosis as well. Nonetheless, given his qualifications, his opinion undermines the others offered by the Employer.

Dr. Tuteur concluded that Mr. Tolliver had diffuse interstitial pulmonary fibrosis caused by ARDS, which is not related to exposure to coal dust. He said that coal workers' pneumoconiosis was an alternative potential etiology which he rejected because the biopsy pathology, together with the medical history, established ARDS as the cause. He ruled out other causes of the fibrosis, including pneumoconiosis, because of the absence of coal macules, and rheumatoid arthritis, because of the absence of necrobiotic nodules. He agreed that Mr. Tolliver was unable to perform coal mine work. Though more consistent with other opinions as to diagnoses and disability, Dr. Tuteur's opinion also has troubling aspects. He relied heavily on Dr. Naeye's interpretation of the biopsy. The regulations provide that a negative biopsy does not preclude a finding of pneumoconiosis. Dr. Tuteur conceded that Mr. Tolliver's clinical symptoms, physical examination findings and physiologic impairment were typical for pneumoconiosis as well as interstitial fibrosis from other causes. His focus on the amount and location of dust, nodules, and macules, and the absence of focal emphysema, suggest that he focused on clinical, rather than legal pneumoconiosis. In addition, his opinion is inconsistent with those of Drs. Dahhan and Renn, because he ruled out rheumatoid arthritis as a cause.

Dr. Dahhan initially diagnosed simple pneumoconiosis based on the x-ray taken for Dr. Renn in January 1996, but said that Mr. Tolliver's restrictive impairment was due to pulmonary fibrosis caused by rheumatoid lung disease and ARDS. Dr. Dahhan said the simple pneumoconiosis observed on x-ray was not severe enough to cause the development of Mr. Tolliver's impairment absent evidence of complicated pneumoconiosis on x-ray or biopsy. He agreed that Mr. Tolliver was disabled from coal mine work by his interstitial fibrosis. In a later

report, after seeing the pathology reports, Dr. Dahhan withdrew his diagnosis of pneumoconiosis. This is inconsistent with the regulation regarding the import of a negative biopsy. He said the restrictive impairment would be explained by the fibrosis, which could be due to ADR, rheumatoid arthritis, or idiopathic pulmonary fibrosis, none of which could be ruled out. As he, too, conceded that the x-ray evidence was consistent with pneumoconiosis, his reasoning for excluding pneumoconiosis is not persuasive.

Dr. Renn agreed that the January 1996 x-ray was consistent with pneumoconiosis, and he agreed that Mr. Tolliver was disabled. Dr. Renn also said that the x-ray, pulmonary function studies and arterial blood gas results would be similar whether Mr. Tolliver had interstitial fibrosis or coal workers' pneumoconiosis. However, Dr. Renn testified that coal dust does not cause the diffuse alveolar damage that Mr. Tolliver had. He diagnosed idiopathic interstitial fibrosis because the fibrosis could have been caused by rheumatoid arthritis, ARDS, or side effects from medication. The radiologist who took the CT scan as part of Dr. Renn's examination of Mr. Tolliver said that it was consistent with coal workers' pneumoconiosis. Dr. Renn thought it possible that there was more than one active disease process based on the CT scan results (ARDS and rheumatoid arthritis), but expressed certainty that pneumoconiosis was not a factor. Both Dr. Dahhan and Dr. Renn agreed with each other on the diagnosis of rheumatoid arthritis as a cause, but that finding was explicitly rejected by Dr. Tuteur. I find Dr. Renn's reasoning for rejecting coal dust as one of multiple factors contributing to Mr. Tolliver's lung impairment no more persuasive than Dr. Dahhan's.

In the final analysis, the fact that four well-qualified pulmonologists all agreed that there were several possible causes, but could not agree on the etiology of Mr. Tolliver's fibrosis, undermines the credibility of all of their opinions. While not conclusive in themselves, the x-ray, CT scan, treatment records, and treating physicians' opinions, together with Dr. Rasmussen's opinion, support the conclusion that Mr. Tolliver had legal pneumoconiosis. In my previous decision I stated:

> All of the physicians who provided opinions that Mr. Tolliver did not have pneumoconiosis acknowledged that he had sufficient exposure to coal dust to cause pneumoconiosis in a susceptible individual. They also agreed that there was evidence from x-rays and other objective studies to support the diagnosis, but placed great emphasis on the fact that the biopsy did not positively establish its existence. More than one testified to the effect that biopsy is the "gold standard" for diagnosing pneumoconiosis. None, however, addressed the limitations of the biopsy in terms of its location or size, and their arguments appear to run contrary to the rule which provides that a negative biopsy does not exclude the diagnosis. Nor did they have any other basis than the biopsy for rejecting the evidence of the x-rays and CT scans. Drs. Tuteur, Dahhan and Renn all acknowledged that Mr. Tolliver had multiple conditions which could have contributed to his pulmonary problem, including rheumatoid arthritis and his history of ARDS, but did not offer convincing explanations why pneumoconiosis, of all potential causes or contributors, should be excluded. The same may be said of Dr. Repsher, although he identified a different potential contributing cause, lupus.

Having reevaluated the medical evidence, my conclusion remains the same. I find that the newly submitted evidence establishes that Mr. Tolliver suffered from legal pneumoconiosis. As this represents a material change in conditions since denial of his initial claim, I must next weigh all of the medical evidence together.

This conclusion does not change when all of the medical evidence is considered together. As to the x-ray evidence, the September 16, 1992, and November 28-December 27, 1992 x-rays were not classified, but were not negative, either. The November 23, 1992 x-ray was positive, as Dr. Renn classified it as 1/2, and there were no negative readings. The January 25 and March 4, 1993 x-rays were negative, as more dually qualified readers classified them as negative than as positive. The July 8, 1993, x-ray was positive, as Dr. Jenkins classified it as 1/1, and there were no negative readings. Taken as a whole, the x-ray evidence before and after July 1993 was inconclusive. Similarly, the 1992 CT scans showed small nodules of undetermined cause. After his 1993 examination in connection with Mr. Tolliver's first claim, Dr. Albin said that Mr. Tolliver's restrictive defect "possibly" was related to inhalation, although he did not diagnose pneumoconiosis. In addition, although the biopsy may be viewed as negative for pneumoconiosis, according to the regulations, it does not provide conclusive evidence that Mr. Tolliver did not have pneumoconiosis. As I noted in my previous decision, Dr. Naeye's position that simple pneumoconiosis does not advance after a miner quits the mining industry is contrary to the regulations and therefore may be viewed as "hostile to the act." Nonetheless, even Dr. Naeye acknowledged that the tissue sample was not adequate to exclude the presence of coal workers' pneumoconiosis. Also as I noted previously, at least two other doctors, Dr. Rasmussen and Dr. Tuteur, commented that biopsy from the lower lobe might miss the presence of pneumoconiosis, which predominantly affects the upper lobes. The other pathologist asked to look at the biopsy results and medical history, Dr. Kleinerman, gave a conclusory opinion that did not acknowledge the limited nature of the biopsy or the distinction between clinical and legal pneumoconiosis. Thus all of the earlier evidence must be viewed as inconclusive, and does not provide any compelling reason to discount the later, positive evidence.

For the same reasons, considering all of the medical evidence, I find that the Claimant has established that Mr. Tolliver had pneumoconiosis for the purpose of the survivor's claim.

Causal Relationship Between Pneumoconiosis and Coal Mine Employment

The Act and the regulations provide for a rebuttable presumption that pneumoconiosis arose out of coal mine employment if a miner with pneumoconiosis was employed in the mines for ten or more years. 30 U.S.C. § 921(c)(1); 20 CFR § 718.203(b) (2005). Mr. Tolliver was employed as a miner for over 27 years, and therefore is entitled to the presumption. The Employer did not offer any evidence to overcome the presumption. I conclude that Mr. Tolliver's pneumoconiosis was caused by his coal mine employment.

Total Pulmonary or Respiratory Disability

A miner is considered totally disabled if he has complicated pneumoconiosis, 30 U.S.C. § 921(c)(3), 20 CFR § 718.304 (2005), or if he has a pulmonary or respiratory impairment to which pneumoconiosis is a substantially contributing cause, and which prevents him from doing

his usual coal mine employment and comparable gainful employment, 30 U.S.C. § 902(f), 20 CFR § 718.204(b) and (c) (2005). The regulations provide five methods to show total disability other than by the presence of complicated pneumoconiosis: (1) pulmonary function studies; (2) blood gas studies; (3) evidence of cor pulmonale; (4) reasoned medical opinion; and (5) lay testimony. 20 CFR § 718.204(b) and (d) (2005). Lay testimony may only be used in establishing total disability in cases involving deceased miners, and in a living miner's claim, a finding of total disability due to pneumoconiosis cannot be made solely on the miner's statements or testimony. 20 CFR § 718.204(d) (2005); *Tedesco v. Director, OWCP*, 18 B.L.R. 1-103, 1-106 (1994). There is no evidence in the record that Mr. Tolliver suffer from complicated pneumoconiosis or cor pulmonale. Thus I will consider pulmonary function studies, blood gas studies and medical opinions. In the absence of contrary probative evidence, evidence from any of these categories may establish disability. If there is contrary evidence, however, I must weigh all the evidence in reaching a determination whether disability has been established. 20 CFR § 718.204(b)(2) (2000); *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19, 1-21 (1987); *Shedlock v. Bethlehem Mines Corp.*, 9 B.L.R. 1-195, 1-198 (1986).

All of the pulmonary function tests met the standard for disability found in the regulations. Although there was some disagreement whether the June 28, 1995 test produced valid results, there were no similar questions about any of the other tests. I find that the pulmonary function tests support a finding of pulmonary disability.

Many of the arterial blood gas studies did not meet the standard for disability, and several of those that did were taken when Mr. Tolliver was ill. Due to his heart problems, Mr. Tolliver generally was not administered exercise tests; however, I note that the one exercise study available in the record, taken in 1995, did meet the standard for disability. In any event, the results of non-qualifying arterial blood gas studies are not contrary to the qualifying pulmonary function tests, as the two types of studies measure different aspects of lung function.

Finally, all of the doctors who expressed an opinion on disability, with the exception of Dr. Repsher, whose opinion I give little weight in view of the objective test results, said that Mr. Tolliver was disabled from performing his former work in the mines.

I find that the overwhelming weight of the evidence supports the conclusion that Mr. Tolliver was totally disabled by the condition of his lungs.

Causation of Total Disability

In order to be entitled to benefits, the Claimant must establish that pneumoconiosis is a "substantially contributing cause" to the miner's disability. A "substantially contributing cause" is one which has a material adverse effect on the miner's respiratory or pulmonary condition, or one which materially worsens another respiratory or pulmonary impairment unrelated to coal mine employment. 20 CFR § 718.204(c) (2005); *Hobbs v. Clinchfield Coal Co.*, 917 F.2d 790, 792 (4th Cir. 1990); *Robinson v. Pickands Mather & Co.*, 914 F.2d 35, 38 (4th Cir. 1990). In *Toler v. Eastern Associated Coal Co.*, 43 F.3d 109 (4th Cir. 1995), the Court found it "difficult to understand" how an Administrative Law Judge (ALJ), who finds that the claimant has established the existence of pneumoconiosis, could also find that his disability is not due to

pneumoconiosis on the strength of the medical opinions of doctors who had concluded that the claimant did not have pneumoconiosis. The Court noted that there was no case law directly in point and stated that it need not decide whether such opinions are "wholly lacking in probative value." However the Court went on to hold:

> Clearly though, such opinions can carry little weight. At the very least, an ALJ who has found (or has assumed arguendo) that a claimant suffers from pneumoconiosis and has a total pulmonary disability may not credit a medical opinion that the former did not cause the latter unless the ALJ can and does identify specific and persuasive reasons for concluding that the doctor's judgment on the question of disability does not rest upon her disagreement with the ALJ's finding as to either or both of the predicates in the causal chain.

43 F.3d at 116. *See also Scott v. Mason Coal Company*, 289 F.3d 263, 269-270 (4th Cir. 2002).

In this case, none of the treating physicians gave an opinion whether Mr. Tolliver was disabled or the cause. Dr. Rasmussen believed that Mr. Tolliver had pneumoconiosis. He also acknowledged the risk factors posed by ARDS and rheumatoid arthritis. In his opinion, the effects of all these factors could not be separated. He concluded that exposure to coal dust "must be considered a significant or major contributing factor" in causing Mr. Tolliver's disability. I can find no specific or persuasive reason for crediting the opinions on the cause of disability of the doctors who did not believe Mr. Tolliver had pneumoconiosis.

I therefore find that pneumoconiosis contributed to Mr. Tolliver's disability.

Death Due to Pneumoconiosis

The Fourth Circuit, in which this claim arises, has held that any condition that hastens the miner's death is a substantially contributing cause of death. *Shuff v. Cedar Coal Co*., 967 F.2d 977 (4th Cir. 1992). This principle has now been codified in the regulations at 20 CFR § 718.205(c)(5) (2005). There is no dispute in this case that pancreatitis was the immediate cause of death. The survivor's claim therefore turns on the question of whether Mr. Tolliver's pulmonary impairment hastened his death.

Although I have given little weight to Dr. Sullivan's opinion on whether coal dust was a factor in causing Mr. Tolliver's lung impairment, as the attending physician during Mr. Tolliver's final hospitalization, his opinion on whether the pulmonary impairment hastened death is entitled to great weight. During his final illness, Mr. Tolliver was intubated due to inadequate ventilation, and died shortly thereafter. Dr. Sullivan said that inadequate ventilation resulted from acute illness compounding previous lung disease. Dr. Sullivan believed that Mr. Tolliver could have survived his pancreatitis "had it not been for his pneumoconiosis and other health problems." Except for Dr. Repsher, whose opinion I have discounted, the pulmonologists consulted by the Employer agreed that Mr. Tolliver had a lung impairment, but differed as to whether it contributed to Mr. Tolliver's death.

In his testimony regarding the cause of Mr. Tolliver's death, Dr. Tuteur was not entirely clear whether interstitial fibrosis was a contributory factor or not. In his report, he said acute pancreatitis superimposed on interstitial fibrosis meant compensatory mechanisms were "suboptimal." At his deposition, he said that pancreatitis in a person with interstitial fibrosis can lead to underventilation, documented by an elevated PCO_2; the arterial blood gas studies taken in the hospital confirm that this occurred in Mr. Tolliver's case. Dr. Tuteur also stated that four independent processes contributed to acidosis and death, including underventilation, pancreatitis, out of control diabetes and renal failure. All of these statements suggest that fibrosis contributed to Mr. Tolliver's death. However, Dr. Tuteur also said that the high CO_2 reading was not due to lung dysfunction. That suggests the opposite. All in all, I find that Dr. Tuteur's testimony better supports the position that Mr. Tolliver's restrictive lung disease hastened his death than the contrary.

Dr. Dahhan attributed Mr. Tolliver's death entirely to pancreatitis which caused electrolyte imbalance and ventricular tachycardia. His testimony did not address the issues of the high CO_2 readings and intubation, nor did it directly answer the issue of whether death occurred sooner than it otherwise would have because of Mr. Tolliver's lung impairment.

Dr. Renn specifically disagreed with Dr. Sullivan's position that inadequate ventilation contributed to Mr. Tolliver's death. He pointed out that pancreatitis has a mortality rate of 50% for the initial episode. He said that metabolic acidosis, not respiratory failure, was Mr. Tolliver's primary problem. He seemed to criticize Dr. Sullivan's choices in treatment, suggesting that use of a ventilator was unnecessary, as Mr. Tolliver was well oxygenated on small amounts of supplemental oxygen, and the ventilator may have caused cardiac arrhythmia. All of these arguments beg the question as to whether the lung impairment *hastened* Mr. Tolliver's death.

I conclude that the most compelling and reliable evidence on this issue is the opinion of Mr. Tolliver's treating physician that multiple pre-existing medical problems, including pneumoconiosis, hastened his death from the complications of pancreatitis.

Date of Entitlement

In the case of a miner who is totally disabled due to pneumoconiosis, benefits commence with the month of onset of total disability. Where the evidence does not establish the month of onset, benefits begin with the month that the claim was filed. 20 CFR § 725.503(b) (2005). The Claimant filed his claim for benefits in May 1995. The earliest qualifying pulmonary function test was taken in March 1993. The regulation regarding subsequent claims also provides, however, that "In any case in which a subsequent claim is awarded, no benefits may be paid for any period prior to the date upon which the order denying the prior claim became final." 20 CFR § 725.309(d)(5). The District Director issued his proposed decision and order on Mr. Tolliver's prior claim on July 20, 1993. As Mr. Smith took no further action on that claim, it became final one year later, on July 20, 1994. I therefore find that the Claimant is entitled to benefits commencing on July 20, 1994.

FINDINGS AND CONCLUSIONS REGARDING ENTITLEMENT TO BENEFITS

The Claimant has met her burden to establish that there was a material change in conditions since denial of Mr. Tolliver's initial claim, by showing that Mr. Tolliver was disabled by pneumoconiosis. She has also met her burden to establish that pneumoconiosis hastened his death. Mr. Tolliver was entitled to benefits during his lifetime, and Mrs. Tolliver is entitled to survivor's benefits under the Act.

ATTORNEY FEES

By order dated November 14, 2003, I granted the Claimant's request to hold filing of a fee petition in abeyance pending the outcome of the Employer's appeal. That order remains in effect.

ORDER

The claim for benefits filed by Michael I. Tolliver on May 16, 1995, and the claim for benefits filed by Ethel E. Tolliver on October 15, 1999, are hereby GRANTED.

A

ALICE M. CRAFT
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).