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Office of Administrative Law Judges
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Issue Date: 27 February 2004

Case No.: 1998-BLA-0678

BRB No.: 02-0137 BLA

In the Matter of:

BILL HOLBROOK
Claimant

v.

GOLDEN OAK MINING COMPANY
Employer

UNDERWRITERS SAFETY AND CLAIMS
Carrier

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party in Interest

BEFORE: JOSEPH E. KANE
Administrative Law Judge

DECISION AND ORDER ON REMAND - AWARDING BENEFITS

This proceeding arises from a claim for benefits under Title IV of the Federal Coal Mine Health and Safety Act of 1969, 30 U.S.C. § 901 *et seq.* (the Act). Benefits are awarded to coal miners who are totally disabled due to pneumoconiosis. Pneumoconiosis, commonly known as black lung, is a chronic dust disease of the lungs arising from coal mine employment. 20 C.F.R. § 718.201 (1996).

On April 3, 1998, this case was referred to the Office of Administrative Law Judges for a formal hearing. Following proper notice to all parties, a hearing was held on November 4, 1998 in Buckhorn, Kentucky. The Director's exhibits were admitted into evidence pursuant to 20 C.F.R. § 725.456, and the parties had full opportunity to submit additional evidence and to present closing arguments or post-hearing briefs.

Procedural History

On June 29, 1999, I issued a Decision and Order - Awarding Benefits in the above-captioned case. The parties stipulated that the Claimant had been a coal miner for twenty-four years, and I found that he was totally disabled due to coal workers' pneumoconiosis. I awarded benefits from February 1997, the month in which the claim was filed.

The Employer appealed the award to the Benefits Review Board ("the Board"). On November 30, 2000, the Board affirmed in part, vacated in part, and remanded the claim for reconsideration consistent with its opinion. *Holbrook v. Golden Oak Mining Co.,* BRB No. 99-1263 (Nov. 30, 2000) (unpublished). The findings pursuant to 20 C.F.R. §§718.202(a)(3) and 718.204(c) were affirmed as unchallenged on appeal. The onset date was also unchallenged. The Board remanded for reconsideration of the issues at §§ 718.202(a)(1), 718.202(a)(2), 718.203(b), and 718.204(b), with certain findings affirmed and certain findings vacated.

On September 28, 2001, I issued a Decision and Award of Benefits on Remand, which the Employer again appealed to the Benefits Review Board. *Holbrook v. Golden Mining Co.,* 1998-BLA-0678. The Board remanded the case again on October 31, 2002, holding erroneous my findings and analysis of the x-ray evidence under Section 718.202(a)(1) and the medical opinion evidence under Section 718.202(a)(2), (4), 718.203 and 718204(c). BRB 2002 Remand at p. 3-4. Thus, I am to reconsider the qualifications of the x-ray interpreters, to explain the basis of my previous assertion that some of the x-rays were not pertinent, to reconsider the x-ray readings of five interpreters as negative, and accord them appropriate weight, and to sufficiently analyze the medical opinion evidence for quality of reasoning, documentary support, and credibility. BRB 2002 Remand at p. 4-5. Furthermore, I am to provide a basis for my determinations of credibility, quality of reasoning and documentary support of the medical opinion and to analyze the opinions under the authority of *Jericol Mining, Inc. v. Napier*, 301 F. 3d 703 (6th Cir. 2002). After, I must weigh all the relevant evidence of record pursuant to the "appropriate standards" and state the basis of my decision. BRB 2002 Remand at p. 6. Thereafter, I must reweigh the evidence to determine whether claimant has pneumoconiosis, which arose from his coal mine employment, and whether his total disability is due to pneumoconiosis.

The Findings of Fact and Conclusions of Law that follow are based upon my analysis of the entire record, arguments of the parties, and the applicable regulations, statutes, and case law. They also are based upon my observation of the demeanor of the witness who testified at the hearing. Although perhaps not specifically mentioned in this decision, each exhibit and argument of the parties has been carefully reviewed and thoughtfully considered. While the contents of certain medical evidence may appear inconsistent with the conclusions reached herein, the appraisal of such evidence has been conducted in conformance
with the quality standards of the regulations.

The Act's implementing regulations are located in Title 20 of the Code of Federal Regulations, and section numbers cited in this decision exclusively pertain to that title. References to DX and EX refer to the exhibits of the Director and employer, respectively. The transcript of the hearing is cited as "Tr." and by page number.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Factual Background

The claimant, Bill Holbrook, was 46 years old at the time of the hearing and has a high school education. He has two dependents, his wife and a child in college, for purposes of augmentation of benefits. (Tr. 14-15, 24; DX 1, 9, 10, 11, 12, 53). The parties stipulated that the claimant was a miner for 24 years. He last worked in January 1997, when he stopped on doctor's orders. (DX 62, 2 - 3, 6 - 8; Tr. 25). The claimant currently smokes approximately half a pack of cigarettes per day. He began smoking in his early twenties, and for one period, smoked 1½ to 2 packs per day. (Tr. 35-37).

Medical Evidence

A. Chest X-rays

Date Ex. No.	Film of X-ray	Qual.	Physician/ Qualifications[1]	Interpretation
DX 48	5/7/85	-	Combs	Discoid atelectas in the right middle lobe.
DX 23	10/3/95	-	Hashem	Pneumoconiosis, 1/2, p/t, mid and lower zones.
DX 42	10/3/95	2	Halbert/BCR, B	Scar vs. atelectas on the left. No CWP.
DX 42	10/3/95	1	West/BCR, B	1/1, s/t, mid and lower zones. Pleural thickening. Not compatible with CWP. Compatible with asbestos exposure.
DX 42	10/3/95	1	Poulos/BCR, B	Completely negative.
DX 46	10/3/95	2	Lockey/B	Linear scarring left mid lung field. No CWP. Co.

[1] The symbol "BCR" denotes a physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology, Inc. or the American Osteopathic Association. 20 C.F.R. § 727.206(b)(2). The symbol "B" denotes a physician who was an approved "B-reader" at the time of the x-ray reading. A B-reader is a physician who has demonstrated expertise in assessing and classifying x-ray evidence of pneumoconiosis. These physicians have been approved as proficient readers by the National Institute of Occupational Safety & Health, U.S. Public Health Service pursuant to 42 C.F.R. § 37.51 (1982).

Ex. No.	Date of X-ray	Film Qual.	Physician/ Qualifications	Interpretation
DX 50,	10/3/95	2	Westerfield/B	Diffuse pulmonary fibrosis, mostly in lower
DX 48	11/7/96	-	Dochterman	Lung fields. Much soft tissue. Cardiac silhouette some somewhat enlarged. Prominent hilar shadows. Some lymphadenopathy in either hilar area could not be excluded.
DX 48	11/8/96	-	Gale	Moderate pulmonary congestion. (Portable chest).
DX 48	11/8/96	-	Gale	Mild congestive heart failure. (Portable chest)
DX 48	11/9/96	-	Gale	Mild perihilar congestion. (Portable chest).
DX 48	11/10/96	-	Gale	Congestive heart failure, increased since prior exam. (Portable chest).
DX 48	11/10/96	-	Kostelic	Congestive heart failure. (Portable chest).
DX 48	11/11/96	-	Kostelic	Clearing congestive heart failure. (Portable chest).
DX 42	1/18/97	-	Hashem	Persistent bilateral in filtrates with apparent increase since 5-7-96 and similar to 3-96. Differential diagnosis would include chronic infection, fungal disease, lymphoma or autoimmune disease.
DX 42	1/22/97	-	Kabir	Almost complete interval resolution of previously demonstrated bilateral basil pneumonic infiltrate. Persistent residual enlargement of both hilum associated with multiple surgical clips. A CT scan would be helpful.
DX 42	2/10/97	2	Halbert/BCR, B	Prominent central lung markings. Linear scars on left. No CWP.

Date Ex. No.	Film of X-ray	Qual.	Physician/ Qualifications	Interpretation
DX 42	2/10/97	2	Poulos/BCR, B	Findings compatible with previous surgical intervention to the anterior aspect of the left upper chest. No CWP.
DX 42	2/10/97	2	West/BCR, B	1/1, s/t, mid and lower zones. Pleural thickening. Not compatible with CWP. Chronic interstitial changes are suggestive of chronic interstitial inflammation or scarring as might be seen with asbestos exposure.
0DX 46	2/10/97	1	Lockey/B	Linear scarring left midlung field. No CWP.
DX 50,	2/10/97	2	Westerfield/B	Pulmonary fibrosis most in mid and lower lung fields. Much soft tissue.
DX 23	2/10/97	1	Myers	1/1, p/s, mid and lower zones. Borderline heart size.
DX 22	3/10/97	2	Baker/B	1/2, t/t, mid and lower zones. Pleural thickening. ? Changes on lateral chest wall. ? Pleural in nature secondary to dust exposure. Co - increased cardiac size.
DX 21	3/10/97	1	Barrett/BCR, B	Co. Congestive heart failure chronic? Acute right sided infiltrate. Follow-up.
DX 20	3/10/97	2	Sargent/BCR, B	Elevated right diaphragm. Post surgery. Atelectasis at left base. ? Enlarged right hilum. Lung volume loss at bases. Unknown etiology. Not CWP.
DX 42	3/10/97	1	West/BCR, B	1/2, s/t, mid and lower zones. Pleural thickening. Not CWP.
DX 42	3/10/97	2	Halbert/BCR, B	Mild central infiltrate bilaterally. Linear scarring on left. No CWP.

Ex. No.	Date of X-ray	Film Qual.	Physician/ Qualifications	Interpretation
DX 42	3/10/97	2	Poulos/BCR, B	Findings compatible with previous surgical intervention to the anterior aspect of the left upper chest. No CWP.
DX 46	3/10/97	2	Lockey/B	Linear scarring left midlung field. No CWP. Co.
DX 24	3/10/97	3	Broudy/B	1/1, s/t, mid and lower zones. Not characteristic of CWP. If due to pneumoconiosis, more likely to be related to asbestosis. (Read on 5/9/97).
DX 49	3/10/97[2]	3	Broudy/B	Completely negative. (Read on 9/12/97).
DX 42	3/10/97	1	Dineen/B	1/2, t/t, mid and lower zones.
DX 25	4/7/97	1	Broudy/B	0/1, u/t, mid and lower zones.
DX 42	4/7/97	U/R	Poulos/BCR, B	Unreadable (overexposed copies).
DX 46	4/7/97	U/R	Lockey/B	Unreadable (copies).
DX 50	4/7/97	U/R	Westerfield/B	Unreadable (copies).
DX 26	4/22/97	1	Broudy/B	1/1, t/t, all zones. Borderline cardiomegaly. Prominent right hilum.
DX 46	4/22/97	U/R	Lockey/B	Unreadable (copies).
DX 42	4/22/97	U/R	Poulos/BCR, B	Unreadable (overexposed copies).

[2] Although the ILO form gives an x-ray date of "9/10/97," the attached report indicates that Dr. Broudy reviewed the 3/10/97 x-ray.

Date Ex. No.	Film of X-ray	Qual.	Physician/ Qualifications	Interpretation
DX 50,	4/22/97	3	Westerfield/B	Fibrosis greater in lower lung fields. Much soft tissue.
EX 3	4/22/97	1	Kleinerman	0/1, s/s, both lower lung fields, fine interstitial lesion. Bilateral upper lobe emphysema. Cardiac shadow is enlarged, suggesting left ventricular hypertrophy.
DX 27	4/23/97	1	Broudy/B	1/2, s/t, mid and lower zones. Suggests interstitial disease not typical of CWP. More suggestive of asbestosis.
DX 44	7/10/97	2	Westerfield/B	Diffuse pulmonary fibrosis.
DX 51	7/15/97	1	Liber/BCR, B	Infiltrate, anterior segment, right upper lobe. Old films would be helpful. Possibilities sarcoidosis, lymphoids.
DX 51	7/29/97	1	Dineen/B	1/1, s/t, mid and lower zones.

. Pulmonary Function Studies

Date Ex. No.	Age/Hgt.	FEV1	FVC	FEV1/FVC	MVV	Comp/Coop
5/21/96 DX 58	44/68" *	2.35	2.57	91.43%	- -	Good
12/6/96 DX 42	45/68" *	2.24 2.22	2.52 2.52	88.88% 88.09%	- -	- -
3/10/97 DX 43	45/66.5"	2.00	2.43	- -	149	Good

Dr. N.K. Burki, a Board-certified pulmonologist, reviewed the above study on behalf of the OWCP and concluded that it was invalid due to suboptimal effort. He also found that the plateau at TLC suggested limitation of inspiratory capacity by spirometer bell. (DX 43).

Date Ex. No.	Age/Hgt.	FEV1	FVC	FEV1/FVC	MVV	Comp/Coop
4/7/97	45/67"	2.15	2.39	90%	102	Suboptimal
DX 13	*	2.41	2.59	93%	104	
4/22/97	45/67"	2.17	2.32	91%	104	Good
DX 14	*	2.23	2.40	93%	111	
7/10/97	45/66"	1.94	2.21	88%	105	Good
DX 45	*	2.03	2.26	90%	116	

Dr. Burki reviewed the above study and concluded that it was invalid due to suboptimal effort and due to the time to plateau being less than two seconds. (DX 45).

7/29/97	45/67"	2.20	2.50	88%	101	Good to
DX 51	*	2.09	2.52	83%	105	Fair
8/19/97	45/68"	1.68	1.89	89.3%	103.9	Fair/
DX 47	*	1.83	2.00	91%	105	Good

Dr. Burki also reviewed the above study on behalf of the OWCP and concluded that it was invalid due to suboptimal effort. (DX 47).

* Results obtained post-bronchodilator.

C. Arterial Blood Gas Tests

Date	Physician	pCO2	pO2	Ex. No.
11/8/96	Hospital	77.4	67.8	DX 48
	+	52.6	88.8	
11/9/96	Hospital	34.0	104.5	DX 48
		36.7	72.2	
		37.2	70.4	
		37.7	70.0	
11/10/96	Hospital	43.4	61.8	DX 48
	+	42.7	57.0	
11/11/96	Hospital	47	69.6	DX 48
3/10/97	Baker	38.4	74.3	DX 19

Date	Physician	pCO2	pO2	Ex. No.
4/22/97	Dineen	40.3	56.6	DX 14
7/10/97	Westerfield	39	61	DX 44, 54
7/29/97	Dineen	36.5	63.5	DX 51

+ Results obtained with exercise.

D. Medical Opinions, CT Scans and Biopsy Evidence

The records of St. Joseph Hospital show that the claimant was treated for depression in September 1984 on referral from Dr. George Caudill. A pulmonary function study was normal. An x-ray showed discoid atelectasis in the right middle lobe, but nothing was audible on examination to confirm this. He continued to be seen by Drs. Robert P. Granacher and Janet S. Kozel in 1984-87. The claimant underwent a sleep evaluation in January 1987. (DX 48).

The office notes of Dr. Edward P. Todd indicate that he began seeing the claimant on May 13, 1996 in consultation for Dr. Caudill. He obtained pulmonary function studies, a CT scan, a bronchoscopy, and after a consultation with Dr. John White, an open lung biopsy. Dr. Todd reported that "[t]he lymph nodes showed only reactive lymph nodes with hemosiderin laden macrophages. Both lung biopsies showed numerous interalveolar hemosiderin laden macrophages consistent with Goodpasture's syndrome, idiopathic pulmonary hemosiderosis or other bleeding tendencies." He then commented that "[b]asically, no particular bleeding tendency has been identified and the patient has denied any real hemoptysis. I strongly advised that he see a pulmonologist for further evaluation and treatment and he has agreed." The claimant proceeded to consult with Dr. Botto, a pulmonologist, and follow-up with Drs. Caudill and Todd. (DX 48).

The first CT scan was obtained on May 21, 1996. Dr. Darryl L. Dochterman interpreted it as showing:

1. Some normal sized nodes present in the fatty tissue of the superior mediastinum.
2. Some pathologically enlarged nodes in both hilar areas, and also in the posterior mediastinum adjacent to the esophagus.
3. Suspect that the hilar nodes are compressing the bronchi to some degree in both hilar regions.
4. Infiltration in both lung fields which radiate out from the hilar regions and into the lower lobe regions primarily.

(DX 48).

A bronchoscope, obtained on May 22, 1996, showed a few atypical cells consistent with pneumonia. (DX 48).

Another CT scan was obtained on July 15, 1996. Dr. Christine N. Riley interpreted it as showing "a prominent right hilar lymph node and somewhat smaller nodes in the left hilum and subcarinal region. The degree of adenopathy is unchanged. The bilateral infiltrates have slightly improved." (DX 48).

Dr. White's examination on October 7, 1996 revealed scattered rales and a few rhonchi at the bases bilaterally. A pulmonary function study revealed a mild restriction and a mild diffusion impairment. Dr. White's impression was interstitial lung disease, bilateral hilar adenopathy, dyspnea, chronic bronchitis, cigarette abuse, hypertension, obesity, hiatal hernia with history of peptic ulcer disease, and depression. He commented that:

> [The claimant] has had increasing dyspnea noted primarily since March of this year. I do not think he has an acute infection at this time, but I think he does have an active interstitial process in his lungs leading to his restrictive pattern on PFT's and abnormalities seen on chest x-ray. I think this might well be sarcoidosis. Other considerations include chronic infection such as TB or fungus. His hilar adenopathy could be caused by low-grade lymphoma, but I think that would be somewhat unlikely currently. He further has a long history of cigarette abuse and I think probably does have a component of chronic bronchitis. He is currently on bronchodilator inhalers. We will have him continue those for now. I will check P.P.D., fungal serologies, CBC, sed rate and ACE levels today. I think he does need a tissue diagnosis. . . .

(DX 48).

The claimant underwent a left lung open lung biopsy on November 8, 1996. Dr. John L. Wilhelmus, a pathologist, reviewed the tissue. The lymph node specimen showed a large amount of pigment present, both black carbonaceous pigment, and retractile and yellow-green pigment resembling iron. Dr. Wilhelmus diagnosed:

> 1) Lymph node, mediastinal: Reactive hyperplasia with focal hemosiderin-laden macrophages.
>
> 2 & 3) Lung biopsies, left lung: Numerous inter- alveolar hemosiderin-laden macrophages, emphysema, mild and focal interstitial fibrosis, predominantly subpleural.
>
> COMMENT: The most striking abnormality present is the large number of hemosiderin-laden macrophages within the alveoli suggesting the possibility of either Good-pasture's syndrome, idiopathic pulmonary hemosiderosis, or other bleeding tendency.

(DX 54). The principal diagnoses on discharge from St. Joseph Hospital were reactive hyperplasia with focal Hemosiderin laden macrophages, emphysema, and mild interstitial fibrosis. The attending physician was Dr. Todd. (DX 48).

Dr. Thomas V. Colby at the Mayo Clinic reviewed the lung tissue on referral from Dr. Wilhelmus. On December 11, 1996, he reported to Dr. Wilhelmus that:

> There is a dramatic increase in hemosiderin-filled macrophages, and one of the first things to think about in such a situation is pulmonary hemorrhage. According to the history on the pathology report, this patient has interstitial lung disease and increasing shortness of breath, and that does not sound like pulmonary hemorrhage syndrome to me. In addition, there is a history of "cigarette abuse" and bilateral hilar adenopathy.
>
> I think the lymph nodes show simply reactive hyperplasia, and that the pathologic changes of significance are in the lung. In addition to the patchy increase in macrophages, they appear to show a predilection for being around respiratory bronchioles, and there is mild associated interstitial fibrosis. Some mucostasis within small airways is also present. Around some of the bronchovascular bundles and in the pleura, there is a slight increase in pigment consistent with history of coal mining, but I do not think the changes are sufficient to warrant a diagnosis of pneumoconiosis. Since coal miners are exposed to anthracrotic pigment which is also seen in cigarette smoking, it may be difficult to completely separate the effects of exposure to coal from the effects of exposure to cigarettes. However, I have not seen the type of pathologic changes present in the biopsy from coal mining alone and think that they are, for the most part (and perhaps entirely), related to cigarette smoking.
>
> I think the changes fit with an exaggerated reaction of respiratory bronchiolitis (so-called respiratory bronchiolitis associated interstitial lung disease), producing some regions resembling desquamative interstitial pneumonia. (Incidentally, a case like this would have been called DIP in the past, and as such, steroids might be something to consider in the management in addition to cessation from smoking.) This reaction is associated with mild interstitial fibrosis. Many of the macrophages are pigmented and contain debris, typical of cigarette smoking. Some of the material is Prussian blue positive which is also typical of cigarette smoking. I think the mucostasis that is present is compatible with smoking.

> Respiratory bronchiolitis associated interstitial lung disease appears to form part of a spectrum which at one end is an asymptomatic histologic finding and at other ends is full blown desquamative interstitial pneumonia. I think this is a lesion that is associated with cigarette smoking, and I have not seen it as a finding really associated with coal mining. The obvious main point of management is to get the patient to quite (sic) smoking. In those who do quite (sic) smoking, there is usually slow improvement or at least no progression of the process.
>
> I refer you to references in the Mayo Clinic Proceedings (64:1373, 1989) and in the American Review of Respiratory Disease (135:880, 1987).
>
> My diagnosis reads as follows: Open lung biopsy showing changes most consistent with respiratory bronchiolitis associated interstitial lung disease; slight increase background dust consistent with history of coal mining; patchy mild interstitial fibrosis.

(DX 48).

The claimant was hospitalized at Whitesburg ARH Hospital from January 18 to 25, 1997. The attending physician was Dr. William Collins. The discharge diagnoses were pneumonia with bronchitis, idiopathic pulmonary fibrosis, depression, and hypertension. (DX 42).

Dr. Glen Baker examined the claimant on March 10, 1997 on behalf of the OWCP. He reviewed the claimant's histories, symptoms, and medications. Examination revealed bilateral basilar rales and wheezes. An x-ray was interpreted as positive for coal workers' pneumoconiosis, 1/2. A pulmonary function study showed a moderate restrictive defect. An arterial blood gas test revealed mild resting arterial hypoxemia. Dr. Baker diagnosed coal workers' pneumoconiosis, 1/2, based on the x-ray and occupational history, due to coal dust exposure; chronic bronchitis based on the history of cough, sputum production and wheezing, due to coal dust exposure; moderate restrictive defect based on the pulmonary function study, due to coal dust exposure; questionable pulmonary fibrosis, due to coal dust exposure; and arteriosclerotic heart disease based on history. Dr. Baker was aware of the claimant's smoking history. He concluded that the claimant had a moderate pulmonary impairment with decreased VC, decreased pO2, chronic bronchitis, and advanced coal workers' pneumoconiosis; and that he was totally disabled from his former coal mine employment. (DX 18).

Dr. Bruce C. Broudy examined the claimant on April 7, 1997 on behalf of the employer. He reviewed the claimant's histories, symptoms, and medications. Examination revealed bibasilar squeaks and inspiratory crepitations, and possible early clubbing of the fingers. A pulmonary function study showed a moderately severe restrictive defect. There was a slight improvement after bronchodilation, but the claimant's effort was not considered maximal. An arterial blood gas test revealed moderately severe to severe resting arterial hypoxemia. The

carboxyhemoglobin level was elevated at 8.3%, which was noted to indicate continued exposure to smoke. An x-ray was interpreted as negative, 0/1, u/t. Dr. Broudy diagnosed history of pulmonary fibrosis, obesity, depression, and hypertension. He commented that:

> I do not believe that Mr. Holbrook has coal workers' pneumoconiosis. There is some evidence that he has some type of interstitial pulmonary fibrosis of undetermined cause. Review of the St. Joseph Hospital biopsy specimens would be helpful, of course, to further confirm the findings. The patient does have some restrictive defect which may be partly related to less than maximal effort. There is hypoxemia which may be related to the interstitial fibrosis and obesity. Because of the hypoxemia and diminished lung function, I believe that he does not retain the respiratory capacity to perform the work of an underground coal miner or to do similarly arduous manual labor. I do not believe that there has been any significant pulmonary disease or respiratory impairment which has arisen from this man's occupation as a coal worker.

(DX 15). Dr. Broudy was deposed on December 18, 1997. He reiterated his examination findings. He further testified that:

> [I]diopathic pulmonary fibrosis is a disease or condition of the general public. It is not -- coal workers are not protected from getting it nor are they apparently more likely to get it than others. So, I believe that he would have had this disease whether or not he had worked in the coal mine.

As to the claimant's smoking history, he testified that:

> [S]moking is a major cause of pulmonary impairment and pulmonary disease. It usually causes chronic bronchitis or pulmonary emphysema or some combination thereof. It's frequently associated with obstructive airways disease. In this particular case it appears that the patient may have interstitial fibrosis that is unrelated to cigarette smoking, per se.

On cross-examination, Dr. Broudy estimated that the lung biopsy consisted of one per cent or less of the lungs, but that if biopsies were not representative, "we would never do a lung biopsy because we couldn't therefore generalize the best of what was wrong with the rest of the lung." He explained that:

> If there is a diffuse disease present in the lung; that is, a disease that's present in both lungs, then we make an assumption that biopsying a representative sample will allow us to generalize as to what may be going on in the entirely of both lungs, although there

> certainly can be more than one thing going on and it may not be present in all areas. I agree that that's a possibility.

He stated that a diagnosis of idiopathic interstitial lung disease excludes a diagnosis of coal workers' pneumoconiosis, because idiopathic indicates that the cause is unknown. As to Dr. Colby's report, Dr. Broudy stated that "anthracrotic pigment is from coal dust inhalation. It doesn't have anything specifically to do with cigarette smoke," and that "for him to say it's hard to distinguish the difference is a little perplexing to me . . . ." (EX 1).

Dr. John E. Myers, Jr., issued a letter on May 23, 1997, stating that:

> I reviewed . . . the pathology specimen on this man's lung biopsy and the report of Dr. Baker, and the x-rays, one of which I reviewed myself and agreed with Dr. Baker's interpretation.
>
> Obviously this man has significant pulmonary impairment which would be at least Class III under the AMA Guides. The biopsy specimens do not rule out coal workers' pneumoconiosis nor do they definitely substantiate it, which is not unusual. This man has an iron deposition disease of some sort within his lungs. Whether he incurred this iron exposure from his work or whether it represented an intrinsic disease such as Goodpasture's syndrome I can't say. I think we would have to conclude that he has coal workers' pneumoconiosis, that he may have other occupational disease or other disease not even related to his occupation as well.

(DX 41).

Dr. Byron T. Westerfield, a Board-certified pulmonologist, examined the claimant on July 10, 1997. Examination revealed coarse rales bilaterally, a few rhonchi, and mild clubbing. An x-ray was positive for diffuse pulmonary fibrosis. A pulmonary function study demonstrated severe restrictive ventilatory dysfunction. There was no significant improvement in flow rates following administration of bronchodilators. An arterial blood gas test revealed a mild decrease in oxygen on room air at rest. The carboxyhemoglobin level of 9.9% was noted to indicate heavy cigarette smoking. Dr. Westerfield concluded that the claimant was totally disabled, but that he did not have coal workers' pneumoconiosis. (DX 44, 54).

Dr. John F. Dineen, who is Board-certified in internal, pulmonary, and critical care medicine, examined the claimant on July 29, 1997 on behalf of the employer. (He had examined him earlier on April 22, 1997 as well; DX 16 - 17). Auscultation of the lungs revealed persistent Velcro-like rales at both lung bases. The fingers were clubbed. An x-ray was interpreted as showing bilateral parenchymal abnormalities consistent with pneumoconiosis, 1/1, s/t, both mid and lower lung zones. A pulmonary function study showed moderate restriction of lung volumes without an obstruction component, and no significant improvement in flow rates following the use of a bronchodilator. The claimant had minimal hypoxemia on room air. Dr. Dineen also reviewed the two pathology reports. He concluded that:

> Mr. Holbrook has interstitial lung disease. He has moderately severe respiratory impairment of the whole person (25-50%). I suspect that Mr. Holbrook has idiopathic interstitial pulmonary fibrosis. Coal workers' pneumoconiosis is an unlikely cause of his interstitial pulmonary fibrosis based on the following observations:
> (1) The open lung biopsy did not demonstrate coal workers' pneumoconiosis. That is the gold standard for diagnosing interstitial lung diseases.
> (2) Finger clubbing is not usually a part of the clinical spectrum of coal workers' pneumoconiosis.
> (3) The lower lung zones are predominantly involved and the small interstitial opacities are irregular in shape. Typically coal workers' pneumoconiosis/silicosis involves the upper lung zones and the parenchymal changes are round in nature. I do not think Mr. Holbrook retains the pulmonary capacity to perform his former duties as a coal miner. He has moderately severe hypoxemia on room air and requires oxygen 18 hours a day to prevent the development of pulmonary hypertension. He does not retain the respiratory capacity to perform his former duties as a coal miner or similar arduous labor.

(DX 51).

Dr. George Caudill, the claimant's treating physician, submitted his treatment records, a report, and then was deposed on July 31, 1997. He testified that the claimant has been his patient since 1978, and that he has treated him for occasional bronchitis through the years (once every two to three years). Since January 1996, he has seen him more frequently for respiratory problems. He stated that:

> At that point [the claimant] came in with an acute respiratory infection, and another episode of bronchitis. And was from (sic) that moment on had had several episodes. He was treated with antibiotics then, and he got a little better. Then shortly there after he got pneumonia, and wound up in the hospital. And he has had recurring problems since that time.

In the hospital, the claimant was treated by Dr. Bill Collins.

Dr. Caudill testified that "[t]he initial treating diagnoses (sic) was just acute bronchitis, and then later it became pneumonia. And then interstitial fibrosis with enlarged lymph nodes. And then after that, of course, interstitial fibrosis proven by biopsy." He stated that the finding of interstitial fibrosis was very consistent with pneumoconiosis, and even more consistent considering his work history. He explained:

> [B]asically the lung biopsy showed that he had indeed . . . interstitial fibrosis. Some of the macrophages were noted to be containing carbonatious material consistent with pneumoconiosis. Some of them also contained hemosiderin macrophages, which made us entertain the diagnoses (sic) of possibly Goodpasture's syndrome. But nothing else has been brought out to confirm that. He's never had bleeding, or any other findings normally seen in Goodpasture's syndrome. So we are not absolutely certain what that significance is. The carbonatious material was certainly consistent with the diagnosis of pneumoconiosis.

Dr. Caudill further testified that the finding of a restrictive defect was consistent with interstitial lung disease and with pneumoconiosis. He related the claimant's breathing impairment to coal dust exposure. He found him to be totally disabled. Dr. Caudill's office notes were attached to the deposition. (DX 52).

Dr. Westerfield was deposed on November 12, 1997. He testified as to his examination findings. As to the x-rays, he stated that the one he obtained was of quality 2 due to incomplete inspiration, which is a result of the claimant's pulmonary condition. He explained that "[i]f the lungs are not fully expanded, the lung markings appear more concentrated, and in some cases, increasing the normal lung markings can be mistaken for the presence of pneumoconiosis or other pathology." He also explained that for a diagnosis of pneumoconiosis "[a] chest radiograph needs to show small opacities, which are usually rounded, but they can be irregular. They are present most commonly in the upper and mid lung zones and they must be at a profusion level of 1/0 or greater, based upon the ILO classification for pneumoconiosis." Dr. Westerfield further testified that the interstitial scarring is unrelated to the claimant's smoking, and most likely due to the previous pneumonia. The carbon particles in the lymph nodes, he related to smoking and emphysema. He stated that the lung biopsy would have shown pneumoconiosis if the claimant had pneumoconiosis.

On cross-examination, Dr. Westerfield conceded that coal workers' pneumoconiosis is a type of pulmonary fibrosis and that the claimant has pulmonary fibrosis; and that coal workers' pneumoconiosis more often than not causes a restrictive defect, which the claimant has. He explained that he did not check off the boxes at section 2 of the ILO form because he felt the condition was clearly not pneumoconiosis. He conceded that a lung biopsy was not performed on the right lung, and that coal workers' pneumoconiosis cannot be ruled out without an autopsy. He also stated that, while uncommon, coal workers' pneumoconiosis can occur only in the lower lung zones. (DX 54).

Dr. Broudy reviewed additional medical records on behalf of the employer and issued supplemental reports on November 18, 1997 and December 19, 1997. In the latter report, he stated that:

> To make a diagnosis of coal workers' pneumoconiosis one would need an adequate history of exposure and either characteristic findings on chest x-ray or typical findings of pneumoconiosis by lung biopsy. Although this gentleman had a history of exposure,

> he had neither the characteristic x-ray findings nor the typical findings of coal workers' pneumoconiosis by lung biopsy. The open lung biopsy was an adequate specimen from the upper lobes and did not show coal workers' pneumoconiosis as was stated by two pathologists in the record. As noted, the chest x-ray was not normal, but it did not show the characteristic small rounded opacities in the upper zones, but rather irregular opacities in the mid to lower zones.

He continued to find that the claimant was totally disabled. (DX 55, 56).

Dr. Broudy was deposed on December 18, 1997. He reiterated his examination findings. He further testified that:

> [I]diopathic pulmonary fibrosis is a disease or condition of the general public. It is not -- coal workers are not protected from getting it nor are they apparently more likely to get it than others. So, I believe that he would have had this disease whether or not he had worked in the coal mine.

As to the claimant's smoking history, he testified that:

> [S]moking is a major cause of pulmonary impairment and pulmonary disease. It usually causes chronic bronchitis or pulmonary emphysema or some combination thereof. It's frequently associated with obstructive airways disease. In this particular case it appears that the patient may have interstitial fibrosis that is unrelated to cigarette smoking, per se.

On cross-examination, Dr. Broudy estimated that the lung biopsy consisted of one per cent or less of the lungs, but that if biopsies were not representative, "we would never do a lung biopsy because we couldn't therefore generalize the best of what was wrong with the rest of the lung." He explained that:

> If there is a diffuse disease present in the lung; that is, a disease that's present in both lungs, then we make an assumption that biopsying a representative sample will allow us to generalize as to what may be going on in the entirely of both lungs, although there certainly can be more than one thing going on and it may not be present in all areas. I agree that that's a possibility.

He stated that a diagnosis of idiopathic interstitial lung disease excludes a diagnosis of coal workers' pneumoconiosis, because idiopathic indicates that the cause is unknown. As to Dr. Colby's report, Dr. Broudy stated that "anthracrotic pigment is from coal dust inhalation. It doesn't have anything specifically to do with cigarette smoke," and that "for him to say it's hard to distinguish the difference is a little perplexing to me . . . ." (EX 1).

Dr. Westerfield reviewed additional medical records on behalf of the employer and issued a report on January 7, 1998. While he concluded that the claimant was totally disabled from a respiratory standpoint, he concluded that disability was related to pulmonary fibrosis of uncertain cause, though probably related to pneumonia. He concluded that the evidence showed that the claimant did not have coal workers' pneumoconiosis. He explained that:

> [The claimant] has an adequate history of exposure to coal dust, but does not have radiographic or pathological evidence of pneumoconiosis. Mr. Holbrook's chest x-ray is not normal. There is definite pulmonary fibrosis present on the chest roentgenogram but the fibrosis is not consistent with pneumoconiosis. This is, also, the opinion of most of the B Readers interpreting Mr. Holbrook's many chest x-rays.
>
> Mr. Holbrook was fortunate enough to have undergone open lung biopsy 11/8/96 and adequate lung tissue was obtained to diagnose interstitial fibrosis. According to both Dr. John Wilhelmus of Lexington and Dr. Thomas V. Colby of the Mayo Clinic there is no Coal Workers' Pneumoconiosis present on the biopsy specimens. Actual pathological examination is the most accurate means of diagnosing pneumoconiosis and would take precedent over an x -ray diagnosis.

(DX 57).

Dr. Dineen also reviewed additional records and issued a supplemental report on January 14, 1998. His opinions remained the same. (DX 57).

Dr. James Lockey reviewed the medical records and depositions on behalf of the employer and issued a report on February 23, 1998. Dr. Lockey concluded that:

> [The claimant's] pulmonary complaints are not consistent with coal worker's pneumoconiosis or in any way related to his occupation as a coal miner. Mr. Holbrook's pulmonary condition apparently started after onset of a respiratory infection in early 1996. He subsequently developed bilateral hilar adenopathy and fleeting and changing pulmonary infiltrates involving the basilar segments of both lungs radiating out from the hilar area. These findings are not consistent with an occupational exposure to coal dust. This was confirmed when the patient underwent a subsequent open lung biopsy which did not demonstrate any changes consistent with coal worker's pneumoconiosis. In addition, coal worker's pneumoconiosis causes persistent abnormalities on the chest x-rays that do not vary over time, are not associated with clubbing, and commonly do not involve the lower lung fields.

> Respiratory bronchiolitis and desquamative interstitial pneumonitis are pulmonary diseases that are commonly associated with heavy cigarette smokers. I have included a copy of this description from a textbook entitled, OCCUPATIONAL AND ENVIRONMENTAL RESPIRATORY DISEASE, Edited by Harber, Schenkar, and Balmes, Mosby Publishing, 1996, Chapter 8, Page 117, which is supportive of this opinion.

Dr. Lockey further concluded that the claimant was totally disabled from a pulmonary standpoint. (DX 58). Dr. Lockey failed to address the treatment records of Dr. Caudill that reflect breathing problems prior to the 1996 pneumonia incident. Consequently, I find that this opinion is not consistent or accurate with Mr. Holbrook's medical history. I accord this opinion only some weight against a finding of pneumoconiosis.

Dr. Ben V. Branscomb, who is Board-certified in internal medicine, reviewed the medical records on behalf of the employer and issued a report on May 27, 1998. He noted that "[t]he lymph node contained black carbon, as is the usual finding in smokers, miners, or older urban dwellers." As to the pathology reports, Dr. Branscomb commented that:

> Because the smaller bronchioles were inflamed (respiratory bronchiolitis associated with interstitial lung disease) Dr. Colby thought tobacco smoke might be the cause of the problem. (The bronchioles are the primary site for tobacco injury. Dr. Colby was suggesting that the bleeding into the alveoli was secondary to this injury.) He found other changes that fit with cigarette smoking.

Dr. Colby noted that in some smokers the bronchiolar injury is minimal and asymptomatic. A more severe example would correspond with Mr. Holbrook. In the severest form there is a great deal of death, destruction, and sloughing off of cells. That very rare condition is called desquamative interstitial pneumonia or DIP. DIP is a process similar to the usual interstitial pulmonary fibrosis (UIP) except that the cells are predominantly macrophages and they are in the air spaces with some inflammatory infiltrate of the alveolar walls by scanty fibrosis. The exchange between Dr. Colby and Dr. Wilhelmus relates to a rather technical classification question concerning several very rare and closely interrelated pulmonary disorders. Was the primary defect leaking of blood into the air sacs or was the primary defect in the bronchiolar walls and hemorrhage into air spaces secondary? In any case, there were no pathologic findings of any of the pneumoconioses. There also was no pervasive generalized interstitial fibrosis.

Dr. Branscomb concluded that:

> 1. Mr. Holbrook does not have coal worker's pneumoconiosis nor any other pneumoconiosis. This is based on, among other conderations, the fact that the x-ray was not compatible with CWP but was typical of what the biopsy showed he had. Second, the biopsy consisted of ample tissue absolutely to rule out coal worker's pneumoconiosis as the disease. Third, the clinical events, pulmonary

> function changes, blood gases, and all other clinical aspects are not compatible with early simple pneumoconiosis and are typical of the combination of the effects of smoking plus the findings at biopsy.
>
> 2. He is totally disabled as a result of a rare disease falling into the category of the interstitial and alveolar diseases. These include UIP, DIP, and the pulmonary hemorrhagic diseases. Within this spectrum there has been some disagreement and speculation concerning how best to classify Mr. Holbrook's disorder. (We have this same discussion frequently at our pulmonary pathology conferences.) The important finding is that he does not have any evidence of any pneumoconiosis and that the process causing his symptoms and findings was established by biopsy. My own personal formulation is that he probably does have idiopathic pulmonary hemosiderosis since that disease commonly produces no hemoptysis, may be intermittent, and is associated with underlying fibrosis around the involved alveoli and under the pleura. I have certainly seen such cases in the past. It is also possible that the primary defect lies in the interstitial spaces and this has resulted in some hemorrhage into the alveoli. There is no basis whatever for ascribing to coal dust any contribution whatsoever to the pathologic process in the lung nor to his impairments and disability.

(EX 2).

Dr. Jerome Kleinerman, a Board-certified pathologist, reviewed the biopsy tissue and medical records on behalf of the employer, and issued a report on May 30, 1998. He concluded that:

> [The] pathologic findings are diagnostic of early desquamative interstitial pneumonitis. This is an exudative stage in the development of classical interstitial fibrosis. Mr. Holbrook's lung biopsy also shows a terminal and respiratory bronchiolitis associated with the desquamative interstitial pneumonitis. This combination of pathologic findings has been described in heavy and prolonged cigarette smokers. However certain pathologic features commonly present in this entity such as the presence of lymphoid nodules in the lung and tissue eosinophilia are <u>not present</u>.
>
> Nevertheless it is my opinion with reasonable medical certainty that Mr. Holbrook does not have simple or complicated CWP. Coal Workers' Pneumoconiosis with reasonable medical certainty is not the cause of any of Mr. Holbrook's respiratory disability.

> I believe that Mr. Holbrook, even if provided with proper medical treatment for his respiratory ailment would be unable to perform his former coal mine work. Proper medical treatment would include: 1) total cessation of cigarette smoking, 2) weight loss of 30-40 pounds, 3) treatment with oral corticosteroids for his desquamative interstitial pneumonitis, and 4) careful observation for any subsequent change in Mr. Holbrook's pulmonary status.

(EX 3).

DISCUSSION AND APPLICABLE LAW

Because the claimant filed his application for benefits after March 31, 1980, this claim shall be adjudicated under the regulations at 20 C.F.R. Part 718. Under this part of the regulations, claimant must establish by a preponderance of the evidence that he has pneumoconiosis, that his pneumoconiosis arose from coal mine employment, that he is totally disabled, and that his total disability is due to pneumoconiosis. Failure to establish any of these elements precludes entitlement to benefits. *See Anderson v. Valley Camp of Utah, Inc.*, 12 BLR 1-111, 1-112 (1989).

Pneumoconiosis

"Pneumoconiosis" is defined as:

> [A] chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, progressive massive fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment. For purposes of this definition, a disease "arising out of coal mine employment" includes any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.

1. X-ray Evidence

Turning to the x-ray evidence under § 718.202(a)(1), I note that there are forty-five x-ray readings of seventeen x-rays in the record. *Supra.* Two of these interpretations were by Dr. Broudy of the same x-ray. Six of the readings were classified as positive (Category 1), and of those, two readings were made by physicians with no special qualifications, Dr. Myers and Dr. Hashem, and four were made by B-readers, Drs. Baker, Dineen and Broudy. Five x-rays were unreadable. As for the negative readings, eighteen readings were negative for pneumoconiosis despite showing evidence of other lung conditions such as linear scarring or pleural thickening incompatible with pneumoconiosis. The remaining x-rays listed some observations but did not

make a finding of pneumoconiosis. X-ray interpretations cannot support a finding of pneumoconiosis where the administrative law judge infers pneumoconiosis from the diagnoses. To establish the existence of pneumoconiosis, a chest x-ray must be classified as Category 1, 2, 3, A, B, or C. 20 C.F.R. §718.102(b).

In *Shumaker v. Peabody Coal Co.,* the administrative law judge impermissibly relied upon his own interpretation of the medical data according to the Benefits Review Board. BRB No. 97-0896 BLA (March 23, 1998)(unpublished). In this case, the readings of two x-rays were not classified in the form required to constitute evidence of pneumoconiosis but instead noted only "right middle lobe infiltrate and atelectasis" and diagnosed "persistent right middle lobe syndrome." Nevertheless, the administrative law judge offered his own opinion that the description of atelectasis was consistent with the existence of pneumoconiosis. The interpretation of medical data, including x-rays, is for the medical experts and the administrative law judge may not substitute his own medical judgment for that of a physician. *See Marcum v. Director, OWCP*, 11 BLR 1-23 (1987).

The Benefits Review Board vacated the administrative law judge's findings because he relied on his own inferences regarding the medical significance of the x-ray findings to conclude that the x-ray evidence established the existence of pneumoconiosis. *See Marcum, supra*. Therefore, where the interpretations of the x-ray readers are unclassified and do not mention pneumoconiosis, clinical or legal, I will not interpret those x-rays as indicative of pneumoconiosis.

The first x-ray, taken on May 7, 1985 by Dr. Combs, lists "discoid atelectas" but does not diagnosis pneumoconiosis and, therefore, it does not support a finding of pneumoconiosis and will be treated accordingly. (DX 48). I find this x-ray is not supportive of a diagnosis of pneumoconiosis.

Six physicians interpreted the second x-ray, taken on October 3, 1995. (DX 23, 42, 46, 50). Dr. Hashem interpreted the x-ray as positive for pneumoconiosis and Dr. Westerfield listed "diffuse pulmonary fibrosis" but not pneumoconiosis. Drs. Halbert and Poulos, both dually-qualified B-readers and Board-certified radiologists, interpreted the x-ray as negative but equally-qualified Dr. West, diagnosed pneumoconiosis's of 1/1, s/t, showing signs inconsistent with Coal worker's pneumoconiosis. (DX 42). The last interpretation of this x-ray, by B-reader Dr. Lockey, was also negative or "No CWP". I find that the superior qualifications of Drs. Halbert and Poulos entitle their interpretations to great weight in support of a finding that this x-ray is negative for pneumoconiosis. However, Dr. West is also dually-qualified and his positive interpretation, even where he addressed a different etiology than coal dust exposure, must still be considered positive evidence of pneumoconiosis under *Cranon v. Peabody Coal Co.,* 22 BLR 1-1 (Oct. 29, 1999) (Decision and Order on Reconsideration). Comments related to the cause and not the existence of pneumoconiosis are not considered under S 718.203(a)(1) but may be used to rebut the presumption at § 718.203(b).

Additionally, Dr. Lockey's interpretation, as a B-reader, is entitled to greater weight than the positive interpretation of Dr. Hashem, who has no radiological qualifications. Lastly, because a positive interpretation cannot be inferred from Dr. Westerfield's comments, his interpretation and that of Dr. West's does not overcome the probative weight against a finding

that this x-ray is positive for pneumoconiosis. *See, Marcum* and *Shumaker, supra.* Consequently, I find that this x-ray is negative for pneumoconiosis.

Dr. Dochterman, whose qualifications do not appear in the record, interpreted the November 7, 1996 x-ray to indicate "Prominent hilar shadows. Some lymphadenopathy in either hilar area could not be excluded." Where pneumoconiosis may not be inferred, this interpretation cannot support a finding of pneumoconiosis and, therefore, I find this x-ray does not support a finding of pneumoconiosis. *Shumaker, supra.*

Two interpretations appear for the November 8, 1995, x-ray. (DX 48). Both interpretations, by Dr. Gale, do not support a finding of pneumoconiosis and, therefore, this x-ray is not dispositive under § 718.202(a)(1). The same is true for Dr. Gale's interpretation of the November 9, 1996, x-ray. (DX 48). "Mild perihilar congestion" does not constitute a positive reading for pneumoconiosis due to coal mine employment and therefore, will not be considered probative for establishing Claimant's pneumoconiosis.

This analysis also applies to both interpretations of the November 10, 1996 x-ray. Drs. Gale and Kostelic, whose radiological credentials are not of record, read the x-rays in conjunctt-ion with Claimant's congestive heart failure and consequently, I find these x-rays are not probative in this issue of pneumoconiosis. (DX 48). The same is true of the x-ray read the next day, November 11, 1996, by Dr. Kostelic. He noted "clearing congestive heart failure" but this is not a positive reading and is not probative for pneumoconiosis and, therefore, it cannot support a finding of pneumoconiosis.

On January 18, 1997, Dr. Hashem read the x-ray as and commented "persistent bilateral in filtrates [sic] with apparent increase since 5-7-96 and similar to 3-96. Differential diagnosis would include chronic infection, fungal disease, lymphoma or autoimmune disease." None of the listed diagnoses pertains to pneumoconiosis and therefore, this interpretation does not support a finding of pneumoconiosis.

Dr. Kabir, whose radiographic qualifications are not in the record, concluded an x-ray taken on January 22, 1997, indicated "Almost complete interval resolution of previously demonstrated bilateral basil pneumonic infiltrate. Persistent residual enlargement of both hilum associated with multiple surgical clips." (DX 42). Again, I am not at liberty to infer the presence of pneumoconiosis and therefore, this x-ray does not indicate probative evidence of pneumoconiosis.

Six interpreters read the next x-ray, taken on February 10, 1997. (DX 42, 23, 50). Dr. Myers and Dr. West found this x-ray positive for pneumoconiosis. (DX 23, 42). Dr. Myers has no radiographic qualifications in the record but Dr. West is dually-qualified. However, two dually-qualified Board-certified, B-readers, Drs. Poulos and Halbert, classified the x-ray as negative for the existence of pneumoconiosis as did two B-readers, Drs. Locke and Westerfield. I find that this x-ray is negative for the presence of pneumoconiosis based on the greater probative weight accorded to the numerous interpretations of physicians with superior qualifications.

Turning to the twelfth x-ray, taken on March 10, 1997, there are ten interpretations in the record with two interpretations by Dr. Broudy. (DX 20, 21, 22, 24, 42, 49). Four readings are positive for pneumoconiosis, those of Dr. Baker and Dr. Dineen, who are both B-readers as well as Dr. West, dually-certified, and Dr. Broudy, a B-reader read the x-ray stating "Not characteristic of CWP. If due to pneumoconiosis, more likely to be related to asbestosis" however, under *Cranon*,*supra*, this still qualifies as a positive interpretation of 1/1, s/t. (DX 22, 24, 42). Drs. Sargent, Halbert, and Poulos, all dually-qualified readers, interpreted the x-ray as negative. (DX 20, 42). Admittedly, Dr. Halbert noted mild infiltrate bilaterally; he also noted that this was not "CWP." Another B-reader, Lockey, also read the x-ray as negative for pneumoconiosis. Dr. Broudy subsequently re-read the x-ray as completely negative.

The last interpretation, by Dr. Barrett, a Board-certified radiologist and B-reader, questioned indications of congestive heart failure and acute right sided infiltrate and suggested a follow-up. (DX 21). He did not diagnose or interpret the presence of pneumoconiosis and because it cannot be inferred, I do not find that this interpretation is indicative of pneumoconiosis despite the reader's superior qualifications. I do find, however, that the superior qualifications of three dually-qualified readers entitle their negative readings to greater probative weight than the positive readings by B-readers or the "infiltrate" reading of Dr. Barrett. I also find that the negative readings of the B-readers do not support a finding of pneumoconiosis and therefore, I find that this x-ray is negative for pneumoconiosis.

In addition, I declare the April 7, 1997, x-ray negative for pneumoconiosis where three readers found the radiographic image unreadable and one B-reader, Dr. Broudy, classified the x-ray "0/1", which is considered negative under the regulations. (DX 25, 42, 46, 50).

Five readers interpreted the April 22, 1997 x-ray, however, two readers, Drs. Poulos (Board-certified, B-reader) and Lockey (B-reader); found the quality too poor to read. (DX 46, 42). Of the remaining interpretations, Dr. Broudy, a B-reader, determined that the film represented pneumoconiosis characterized as "1/1, t/t, all zones." (DX 26). Dr. Kleinerman, whose radiographic qualifications are absent from the record, diagnosed "0/1, s/s, both lower lung fields, fine interstititial lesion. Bilateral upper lobe emphysema." (EX 3). However, a "0/1" reading is not a diagnosis of pneumoconiosis under the regulations and, therefore, this interpretation weighs against a finding of pneumoconiosis. Dr. Westerfield, also a B-reader, diagnosed "fibrosis greater in lower lung fields. Much soft tissue." (DX 50).

In sum, I find that the superior qualifications of Dr. Broudy permits his x-ray interpretation to be accorded some probative weight, however, his reading is contradicted by the "unreadable" determinations of Dr. Poulos, who possesses superior dual qualifications, and Dr. Lockey, a B-reader. I also note that Dr. Westerfield did not diagnose pneumoconiosis and his qualifications are equivalent to Dr. Broudy. Consequently, I find this x-ray does not support a pneumoconiosis finding.

The next x-ray, dated April 23, 1997 and read by B-reader Dr. Broudy, is classified as "1/2, t/s, mid and lower zones." (DX 27). Dr. Broudy concluded that the x-ray "suggests interstitial disease not typical of CWP" and was more suggestive of asbestosis. (DX 27). I find this x-ray weighs in favor of a finding of pneumoconiosis due to coal mine employment under

Cranon v. Peabody Coal Co., 22 BLR 1-1 (Oct. 29, 1999)(Decision and Order on Reconsideration).

Dr. Westerfield, a B-reader, diagnosed a July 7, 1997, x-ray as indicating "diffuse pulmonary fibrosis". (DX 44). A diagnosis of massive pulmonary fibrosis meets the definition of legal pneumoconiosis under the regulations but not diffuse pulmonary fibrosis. Additionally, the definition of legal pneumoconiosis is a disease "arising out of coal mine employment" [and] includes any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment". Dr. Westerfield did not clarify or indicate whether this was pneumoconiosis or whether this was a coal exposure-induced lung disease.

In his deposition, Dr. Westerfield stated that he felt the observed condition was clearly not pneumoconiosis. Consequently, this interpretation does not provide probative evidence of the absence or presence of pneumoconiosis. Dr. Westerfield noted the presence of pulmonary fibrosis (scarring) on his readings and testified as follows:

Q15. Was there a reason that you didn't fill out the section, Section 2B, as to the shape and size in the zones of the profusion?

A. The fibrosis did not have the appearance of pneumoconiosis.

Q16. Can you reflect that in the shape and size of the opacities, Doctor? Can you --

A. Well, pneumoconiosis has either rounded opacities which are from p, q, and r, vary from up to 3 millimeters in size, or irregular which are linear or just not rough. They're also very small. The type of fibrosis that Mr. Holbrook has is -- it's much more extensive than that. If you were looking at the radiograph rather than seeing little bitty dots or little bitty lines, you see long lines, large amounts of it. The entire tissue is involved. So, it's a different radiographic appearance than what we see with pneumoconiosis.

Q17. In you opinion, can you not describe that appearance on -- using the ILO form?

A. Well, it's not a parenchymal abnormality that's consistent with pneumoconiosis, so there's no--**...** box for scoring that type. All we do is note that the fibrosis is present.

Q18. Doctor, some of the physicians who reviewed x-rays in this claim went ahead and filled out the ILO form, but then stated an opinion that it was or was not coal workers' pneumoconiosis, but you don't think that that's possible to do given this form and the descriptive

terms that you have available to you as far as the shape and size of the opacities.

A. I think that's up to the interpreting physician if he wants to describe it that way. My opinion is this is clearly not pneumoconiosis and therefore I would not check the 2 box.

Q19. So if I were to ask you to actually make a-- state an opinion on the ILO form in regard to the profusion, the shape and size, and the zones, understanding that you don't believe it's pneumoconiosis, you couldn't do that?

A. That's correct.

Q20. Some doctors, who have read the chest x-ray, Doctor, have described the opacities as being t/t in shape and size or p/t and p/s. Would that just be a difference in medical opinion as to what you see and how you describe it as to how you rate your chest x-ray?

A. I can't comment on other physicians' interpretation. All I can say is that the qualified B-readers will oftentimes give a different interpretation of the same chest radiograph.

Q21. And different physicians will also give different interpretations of the same radiograph, is that correct?

A. That's correct.

Q22. It's all--whether or not you can describe what you see on Mr. Holbrook's chest x-ray depends in large part on your training, experience and your opinion, each physician's opinion; is that correct?

A. That's correct. I think I have an advantage on Mr. Holbrook's x-ray in that I completely evaluated Mr. Holbrook and performed tests and history and physical; also lung biopsy and I got to read these additional chest x-rays. So, I feel very comfortable about that radiographic interpretation and my diagnosis because of that.

Q23. Doctor, were you provided any medical reports prior to your examination of this gentleman or in conjunction with your examination of Mr. Holbrook besides the pathology report?

A. I have some other medical reports from Mr. Holbrook, Appalachian Regional Hospital. There's additional medical information that I reviewed, yes, sir.

Q24. And that was provided to you by defense counsel?

A. By Mr. Sturgill, yes, sir.

Q25. And you had the opportunity to review those medical reports in conjunction with your examination and prior to producing your report on this gentleman?

A. That's correct.

(DX 54, depo. at pp. 22-26).

The definition of statutory pneumoconiosis is not as narrowly defined as medical/ classical coal workers' pneumoconiosis, and consequently, the Act provides the claimant with a rebuttable presumption that <u>any</u> pneumoconiosis arose from his coal mine employment. 20 C.F.R. §§ 718.201, 718.203(b). The failure to properly classify an x-ray can be an attempt to circumvent that presumption. For example, Dr. Westerfield's readings and invocation of § 718.203(b) would give claimant the presumption that the parenchymal and pleural changes were due to coal workers' pneumoconiosis, and employer the burden to prove that they were due to asbestosis unrelated to dust exposure in claimant's coal mine employment.

My impression from this exchange is that the medical evidence he reviewed prior to reading the x-rays influenced Dr. Westerfield, and therefore, while the x-rays had changes that could have been classified as pneumoconiosis, Dr. Westerfield did not classify them as such because he had reached a different diagnosis. Furthermore, Dr. Westerfield opined that Claimant's x-ray changes were not due to pneumoconiosis. Consequently, and in accordance with the Benefits Review Board, I find that this x-ray is negative for pneumoconiosis. (Decision and Order Remand p. 4).

Next, Dr. Liber, a dually-qualified physician, read the x-ray taken on July 15, 1997. (DX 51). He also noted infiltrate stating that old films would be helpful. He concluded that the "possibilities would include sarcoidosis, lymphoids." In reliance on the rule of *Shumaker*, *supra*, I will not substitute my own inferences in lieu of the doctor's conclusions and because he has not diagnosed a lung disease arising out of coal mine employment, I find this x-ray is not probative for the presence of pneumoconiosis.

Lastly, Dr. Dineen, a B-reader, interpreted that the July 29, 1997 x-ray was positive for pneumoconiosis (1/1, s/t, mid and lower zones). (DX 51). The credentials of B-readers entitle their opinions to probative weight, therefore, I find that this x-ray is probative as evidence of pneumoconiosis.

In accordance with the preceding findings regarding each x-ray, the sum of my findings indicates that there are two x-rays positive for pneumoconiosis, by B-readers. The remaining x-rays either do not support a finding of pneumoconiosis or are negative for pneumoconiosis. Therefore, I find that the Claimant fails to establish pneumoconiosis by x-ray evidence under § 718.202(A)(1).

2. Biopsy Evidence

Under § 718.202(a)(2), pneumoconiosis may be established by biopsy evidence. As to the biopsy evidence, I reaffirm my previous weighing of the reports of the three pathologists who reviewed the tissue biopsied from Mr. Holbrook's left upper lobe on November 8, 1996. The biopsy was ordered due to recurrent bouts of pneumonia. I found that Pathologist, Dr. Wilhelmus, diagnosed mild and focal interstitial lung disease, numerous interalveolar hemosiderin-laden macrophages and emphysema. Dr. Wilhelmus reported numerous interalveolar hemosiderin-laden macrophages, emphysema, mild and focal interstitial fibrosis, predominately sub pleural. However, he did not find pneumoconiosis or relate the biopsy findings to coal dust exposure. Instead, he opined that Claimant could be suffering from Goodpasture's syndrome, idiopathic pulmonary hemosiderosis or other bleeding tendency, and obtained a consultation from Dr. Colby. (DX 54). I find that Dr. Wilhelmus' report is credible.

Dr. Thomas Colby, pathologist, reviewed the lung tissue sample and in his opinion, cigarette smoking played a major role if not the only role in the pathological changes evident from the tissue samples. (DX 48). He was equivocal, however, as to the role of coal dust exposure stating that there exists a slight increase in pigment consistent with coal mine exposure but that the changes were not sufficient to warrant a diagnosis of pneumoconiosis. Further, he stated, "it may be difficult to completely separate the effects of exposure to coal from the effects of exposure to cigarettes." He wrote "I have not seen the type of pathological changes present in this biopsy from coal mining alone and I think that they are, for the most part (and perhaps entirely), related to cigarette smoking." He also stated:

> There is a dramatic increase in hemosiderin-filled macrophages, and one of the first things to think about in such a situation is pulmonary hemorrhage. According to the history on the pathology report, this patient has interstitial lung disease and increasing shortness of breath, and that does not sound like pulmonary hemorrhage syndrome to me. In addition, there is a history of "cigarette abuse" and bilateral hilar adenopathy.
>
> I think the lymph nodes show simply reactive hyperplasia, and that the pathologic changes of significance are in the lung. In addition to the patchy increase in macrophages, they appear to show a predilection for being around respiratory bronchioles, and there is mild associated interstitial fibrosis. Some mucostasis within small airways is also present. Around some of the bronchovascular bundles and in the pleura, there is a slight increase in pigment

> consistent with history of coal mining, but I do not think the changes are sufficient to warrant a diagnosis of pneumoconiosis. Since coal miners are exposed to anthracrotic pigment which is also seen in cigarette smoking, it may be difficult to completely separate the effects of exposure to coal from the effects of exposure to cigarettes. However, I have not seen the type of pathologic changes present in the biopsy from coal mining alone and think that they are, for the most part (and perhaps entirely), related to cigarette smoking.
>
> I think the changes fit with an exaggerated reaction of respiratory bronchiolitis (so-called respiratory bronchiolitis associated interstitial lung disease), producing some regions resembling desquamative interstitial pneumonia. (Incidentally, a case like this would have been called DIP in the past, and as such, steroids might be something to consider in the management in addition to cessation from smoking.) This reaction is associated with mild interstitial fibrosis. Many of the macrophages are pigmented and contain debris, typical of cigarette smoking. Some of the material is Prussian blue positive which is also typical of cigarette smoking. I think the mucostasis that is present is compatible with smoking.
>
> <u>Respiratory bronchiolitis associated interstitial lung disease</u> appears to form part of a spectrum which at one end is an asymptomatic histologic finding and at other ends is full blown desquamative interstitial pneumonia. I think this is a lesion that is associated with cigarette smoking, and I have not seen it as a finding really associated with coal mining. The obvious main point of management is to get the patient to quite (sic) smoking. In those who do quite (sic) smoking, there is usually slow improvement or at least no progression of the process.

In sum, his diagnosis reads “Open lung biopsy showing changes most consistent with respiratory bronchiolitis associate with interstitial lung disease; slight increase background dust consistent with history of coal mining; patchy mild interstititial fibrosis.”

I additionally note that Dr. Colby's opinion shows that he was considering more than just the pathological changes associated with medical pneumoconiosis. However, the Sixth Circuit has held that the administrative law judge must consider biopsy evidence which indicates the presence of anthracotic pigment and consequently, I may dismiss this evidence as Dr. Colby has. *Lykins v. Director, OWCP*, 819 F.2d 146 (6th Cir. 1987) In *Griffith v. Director, OWCP*, 49 F.3d 184 (6th Cir. 1995), the Sixth Circuit held that a finding of pigmentation described as "yellow-black consistent with coal pigment" was insufficient to support a finding of pneumoconiosis yet here the pigment was specifically defined as “anthracrotic” as in the *Lykins* decision. Therefore, contrary to Dr. Colby’s, and later, Dr. Broudy’s assertions, I must consider the presence of

anthracrotic pigment in the biopsy evidence as some indication of pneumoconiosis. However, Section 718.202(a)(2) also provides that a finding in an autopsy of anthracrotic pigmentation shall not be sufficient, by itself, to establish the existence of pneumoconiosis.

Where Dr. Colby reviewed the Claimant's symptomology, coal dust exposure, as well as the report of Dr. Wilhelmus, I find that his report is well documented and somewhat well reasoned. However, he equivocates as to the degree of causation for the pulmonary findings. Therefore, I declare that his opinion is not as credible where he has not reached definite conclusions and his opinion presents only some evidence of legal pneumoconiosis but not clinical.

The body of Dr. Kleinerman's report indicates that he looked for the classic pneumoconiosis pathological changes. He came to a diagnosis of early desquamative interstitial pneumonitis, with associated terminal and respiratory bronchiolitis. He noted, however, "Certain pathologic features commonly present in this entity such as the presence of lymphoid nodules in the lung and tissue eosinophilia are <u>not present</u>." I find that this opinion does not support a finding of pneumoconiosis and is particularly probative due to his qualifications, as a pathologist and professor of pathology, and due to the fact that he did an extensive analysis of the pathological slides.

Dr. Edward Todd, the biopsy surgeon, examined Mr. Holbrook on November 7, 1996 and took a medical and smoking history but no work history. The examination revealed clear lungs sounds bilaterally. He listed the following impressions: interstitial lung disease, bilateral hillar adenopathy, dyspnea, chronic bronchitis, cigarette abuse, hypertension, obesity, hernia, and depression. The postoperative diagnosis was interstititial pulmonary disease of undetermined etiology. He strongly advised Claimant to see a Pulmonologist for further treatment and evaluation.

Reviewing the biopsy findings Dr. Todd noted, "Both lung biopsies showed numerous interalveolar hemosiderin laden microphages consistent with Goodpasture's syndrome, idiopathic pulmonary hemosiderosis or other bleeding tendencies" and "[b]asically, no particular bleeding tendency has been identified and the patient has denied any real hemoptysis." (DX 48). While this opinion evinces some evidence of pulmonary disease, it is only some evidence of pneumoconiosis due to coal mine employment where the etiology is not established in the opinion.

Dr. Myers reviewed the pathology specimen and the report of Dr. Baker, and the x-rays, stated that "[t]he biopsy specimens do not rule out coal workers' pneumoconiosis nor do they definitely substantiate it, which is not unusual." (DX 41). His report concluded:

> I reviewed **. . .** the pathology specimen on this man's lung biopsy and the report of Dr. Baker, and the x-rays, one of which I reviewed myself and agreed with Dr. Baker's interpretation.
>
> Obviously this man has significant pulmonary impairment which would be at least Class III under the AMA Guides. The biopsy specimens do not rule out coal workers' pneumoconiosis nor do

> they definitely substantiate it, which is not unusual. This man has an iron deposition disease of some sort within his lungs. Whether he incurred this iron exposure from his work or whether it represented an intrinsic disease such as Goodpasture's syndrome I can't say. I think we would have to conclude that he has coal workers' pneumoconiosis, that he may have other occupational disease or other disease not even related to his occupation as well.

While noting the somewhat equivocal nature of the etiology portion of the opinion, I do find that it provides a credible basis to support a pneumoconiosis finding where the report is well documented and includes numerous testing and reports. Dr. Myers provided the reasoning behind his observations and appears to have considered all relevant possibilities.

Dr. Dineen, Board-certified in internal, pulmonary, and critical care medicine, examined the claimant on July 29, 1997 on behalf of the employer. (He had examined him earlier on April 22, 1997 as well; DX 16 - 17). Auscultation of the lungs revealed persistent Velcro-like rales at both lung bases. The fingers were clubbed. An x-ray was interpreted as showing bilateral parenchymal abnormalities consistent with pneumoconiosis, 1/1, s/t, both mid and lower lung zones. A pulmonary function study showed moderate restriction of lung volumes without an obstruction component, and no significant improvement in flow rates following the use of a bronchodilator. The claimant had minimal hypoxemia on room air. Dr. Dineen also reviewed the two pathology reports. He concluded that:

> Mr. Holbrook has interstitial lung disease. He has moderately severe respiratory impairment of the whole person (25-50%). I suspect that Mr. Holbrook has idiopathic interstitial pulmonary fibrosis. Coal workers' pneumoconiosis is an unlikely cause of his interstitial pulmonary fibrosis based on the following observations - (1) The open lung biopsy did not demonstrate coal workers' pneumoconiosis. That is the gold standard for diagnosing interstitial lung diseases. (2) Finger clubbing is not usually a part of the clinical spectrum of coal workers' pneumoconiosis. (3) The lower lung zones are predominantly involved and the small interstitial opacities are irregular in shape. Typically coal workers' pneumoconiosis/silicosis involves the upper lung zones and the parenchymal changes are round in nature. I do not think Mr. Holbrook retains the pulmonary capacity to perform his former duties as a coal miner. He has moderately severe hypoxemia on room air and requires oxygen 18 hours a day to prevent the development of pulmonary hypertension. He does not retain the respiratory capacity to perform his former duties as a coal miner or similar arduous labor.

He asserted that he "suspect[s] that Mr. Holbrook has idiopathic interstitial pulmonary fibrosis." (DX 51). I find that this report is credible where Dr. Dineen reviewed two biopsy reports, examined Claimant twice, conducted pulmonary function studies, and chest x-rays.

Consequently, I find that this report does not support a finding of pneumoconiosis by biopsy evidence.

Dr. Caudill stated that the finding of interstitial fibrosis was very consistent with pneumoconiosis, and even more consistent considering his work history. He explained:

> [B]asically the lung biopsy showed that he had indeed **...** interstitial fibrosis. Some of the macrophages were noted to be containing carbonatious material consistent with pneumoconiosis. Some of them also contained hemosiderin macrophages, which made us entertain the diagnoses (sic) of possible Goodpasture's syndrome. But nothing else has been brought out to confirm that. He's never had bleeding, or any other findings normally seen in Goodpasture's syndrome. So we are not absolutely certain what the significance is. The carbonatious material was certainly consistent with the diagnosis of pneumoconiosis.

Dr. Caudill also testified, "[t]he initial treating diagnoses (sic) was just acute bronchitis, and then later it became pneumonia. And then interstitial fibrosis with enlarged lymph nodes. And then after that, of course, interstitial fibrosis proven by biopsy." He stated that the finding of interstitial fibrosis was very consistent with pneumoconiosis, and even more consistent considering his work history. DX 52, Depo. p.8). I find Dr. Caudill's opinion probative as to the existence of pneumoconiosis where he had the benefit of years of examinations, chest x-rays, and other objective tests to augment his conclusions. Further, he stated his reasoning behind the rejecttion of other possible sources of the carbonatious material.

Dr. Westerfield related the carbon particles in the lymph nodes to smoking and emphysema. He stated that the lung biopsy would have shown pneumoconiosis if the claimant had pneumoconiosis. He also testified that:

> The path report suggests some hemosiderin-laden macrophages and that's the iron present in the little patrol cells, as I call them. That could be from bleeding. There's no really good explanation for that and he does not have an iron storage disease that would cause him to accumulate iron. So, that's probably from some previous episodes of bleeding in his lung, that the cells engulfed the blood cells which contain iron and the iron was simply the residue. I think it's more likely that than an actual iron disease.

(DX 54). He also asserted that the carbon particles in the lymph nodes were related to smoking and emphysema. He stated that the lung biopsy would have shown pneumoconiosis if the claimant had pneumoconiosis.

On cross-examination, Dr. Westerfield conceded that coal workers' pneumoconiosis is a type of pulmonary fibrosis and that the claimant has pulmonary fibrosis; and that coal workers' pneumoconiosis more often than not causes a restrictive defect, which the claimant has. He

conceded that a lung biopsy was not performed on the right lung, and that coal workers' pneumoconiosis cannot be ruled out without an autopsy. He also stated that, while uncommon, coal workers' pneumoconiosis could occur only in the lower lung zones. (DX 54). However, Dr. Westerfield had previously testified that pneumoconiosis may be present in the middle and lower lung fields and Mr. Holbrook's x-ray reports evidenced this. The biopsy tissue sample was taken from the upper lung lobe yet Dr. Westerfield did not address this in his biopsy report. Consequently, I find Dr. Westerfield's report equivocal and do not assign it much probative weight.

Dr. Broudy stated "[a]lthough this gentleman had a history of exposure, he had neither the characteristic x-ray findings nor the typical findings of coal workers' pneumoconiosis by lung biopsy. The open lung biopsy was an adequate specimen from the upper lobes and did not show coal workers' pneumoconiosis as was stated by two pathologists in the record." (DX 55, 56). Dr. Broudy was deposed on December 18, 1997. He reiterated his examination findings. He further testified on cross-examination and estimated that the lung biopsy consisted of one per cent or less of the lungs, but that if biopsies were not representative, "we would never do a lung biopsy because we couldn't therefore generalize the best of what was wrong with the rest of the lung." He explained:

> If there is a diffuse disease present in the lung; that is, a disease that's present in both lungs, then we make an assumption that biopsying a representative sample will allow us to generalize as to what may be going on in the entirely of both lungs, although there certainly can be more than one thing going on and it may not be present in all areas. I agree that that's a possibility.

He stated that a diagnosis of idiopathic interstitial lung disease excludes a diagnosis of coal workers' pneumoconiosis, because idiopathic indicates that the cause is unknown. As to Dr. Colby's report, Dr. Broudy stated that "anthracrotic pigment is from coal dust inhalation. It doesn't have anything specifically to do with cigarette smoke," and that "for him to say it's hard to distinguish the difference is a little perplexing to me . . . ." (EX 1).

I find Dr. Broudy's report probative for the absence of pneumoconiosis where his opinion is well reasoned and his assertions are substantiated by his findings.

Dr. James Lockey reviewed the medical records and depositions on behalf of the employer and issued a report on February 23, 1998. Dr. Lockey concluded that the open lung biopsy did not demonstrate any changes consistent with coal workers' pneumoconiosis. (DX 58). He continued:

> He subsequently developed bilateral hilar adenopathy and fleeting and changing pulmonary infiltrates involving the basilar segments of both lungs radiating out from the hilar area. These findings are not consistent with an occupational exposure to coal dust. This was confirmed when the patient underwent a subsequent open lung biopsy which did not demonstrate any changes consistent with coal worker's pneumoconiosis.

I find Dr. Lockey's opinion probative for the absence of pneumoconiosis as it is well reasoned and well documented. (DX 58).

Dr. Branscomb, Board-certified in and a professor of internal medicine, provided a consultative report analyzing the medical evidence and discussing the pathological reports of the other physicians. He stated, "The biopsy consisted of ample tissue absolutely to rule out coal worker's pneumoconiosis as the disease" and:

> The important finding is that he does not have any evidence of any pneumoconiosis and that the process causing his symptoms and findings was established by biopsy. My own personal formulation is that he probably does have idiopathic pulmonary hemosiderosis since that disease commonly produces no hemoptysis, may be intermittent, and is associated with underlying fibrosis around the involved alveoli and under the pleura. I have certainly seen such cases in the past. It is also possible that the primary defect lies in the interstitial spaces and this has resulted in some hemorrhage into the alveoli. There is no basis whatever for ascribing to coal dust any contribution whatsoever to the pathologic process in the lung nor to his impairments and disability.

(EX 2). As to Dr. Colby's report, Dr. Branscomb stated:

> Because the smaller bronchioles were inflamed (respiratory bronchiolitis associated with interstitial lung disease) Dr. Colby thought tobacco smoke might be the cause of the problem. (The bronchioles are the primary site for tobacco injury. Dr. Colby was suggesting that the bleeding into the alveoli was secondary to this injury.) He found other changes that fit with cigarette smoking.
>
> Dr. Colby noted that in some smokers the bronchiolar injury is minimal and asymptomatic. A more severe example would correspond with Mr. Holbrook. In the severest form there is a great deal of death, destruction, and sloughing off of cells. That very rare condition is called desquamative interstitial pneumonia or DIP. DIP is a process similar to the usual interstitial pulmonary fibrosis (UIP) except that the cells are predominantly macrophages and they are in the air spaces with some inflammatory infiltrate of the alveolar walls by scanty fibrosis. The exchange between Dr. Colby and Dr. Wilhelmus relates to a rather technical classification question concerning several very rare and closely interrelated pulmonary disorders. Was the primary defect leaking of blood into the air sacs or was the primary defect in the bronchiolar walls and

> hemorrhage into air spaces secondary? In any case, there were no pathologic findings of any of the pneumoconioses. There also was no pervasive generalized interstitial fibrosis.

(EX 2). He also pointed out that Dr. Colby's report noted the history on the pathological request and not the actual pathological findings of interstitial lung disease with shortness of breath plus cigarette abuse. I find that Dr. Brascomb's qualifications and analysis of the records probative for the absence of pneumoconiosis.

In sum, the biopsy evidence shows that the claimant has interstitial lung disease. However, it is inconclusive as to its specifics and cause. Drs. Todd, Caudill, Myers, Dineen, Broudy, Lockey, Westerfield, and Branscomb, having not seen the tissue, cannot confirm or deny the contents of the biopsy reports. Their reports simply reiterated and considered the findings of the pathologists, who reviewed tissue comprising less than 1% of claimant's lungs. Furthermore, Dr. Wilhelmus indicated several possibilities, did not rule in or out coal dust exposure, and obtained a consultation from Dr. Colby. Dr. Colby's opinion is certain in that cigarette smoking played a major role, but equivocal as to the role of coal dust exposure. Dr. Kleinerman stated that coal dust exposure had nothing to do with the interstitial lung disease, but his opinion is countered by Dr. Colby's uncertainty. Dr. Todd found some evidence of pulmonary disease but listed no etiology. Dr. Myers opinion was equivocal and thus I afforded it little weight while Dr. Dineen's opinion that Mr. Holbrook's interstitial lung disease is not related to coal worker's pneumoconiosis is probative where he has superior credentials and was credible.

Dr. Caudill's opinion, however, was also probative for the presence of pneumoconiosis where he reviewed extensive reports and offered the most documented and substantiated opinion. Dr. Westerfield, on the other hand, offered a contradictory opinion and there fore, I find his opinion worthy of little weight. Dr. Broudy's opinion offers probative evidence against a finding of pneumoconiosis as does Dr. Lindy's and Dr. Brascomb's. I therefore find that the sum of the biopsy evidence does establish interstitial lung disease but does not establish pneumoconiosis due to coal mine employment under § 718.202(a)(2).

3. CT Scan Evidence

The first CT scan was obtained on May 21, 1996. Dr. Darryl L. Dochterman interpreted it as showing:

> 1. Some normal sized nodes present in the fatty tissue of the superior mediastinum.
> 2. Some pathologically enlarged nodes in both hilar areas, and also in the posterior mediastinum adjacent to the esophagus.
> 3. Suspect that the hilar nodes are compressing the bronchi to some degree in both hilar regions.
> 4. Infiltration in both lung fields which radiate out from the hilar regions and into the lower lobe regions primarily.

(DX 48).

Another CT scan was obtained on July 15, 1996. Dr. Christine N. Riley interpreted it as showing "a prominent right hilar lymph node and somewhat smaller nodes in the left hilum and subcarinal region. The degree of adenopathy is unchanged. The bilateral infiltrates have slightly improved." (DX 48).

The records of Dr. Todd show that the May 21, 1996 and July 15, 1996 scans were ordered to further define whether there was a significant pulmonary nodule (office notes of May 13, 1996) and to see if the nodes showed signs of involution (office notes of June 3, 1996). On August 19, 1996, Dr. Todd reported that the CT scan showed that there were no dominant masses. However, the scan did not establish a diagnosis. The left parasternotomy, mediastinal lymph node biopsy and open lung biopsy were subsequently undertaken. (DX 48). Dr. Broudy indicated that the report of the July 15 scan did not suggest the presence of coal workers' pneumoconiosis. (DX 56).

I find that the CT scans do not provide pertinent information, as they do not address the presence or absence of pneumoconiosis. While the reading of the July 15, 1996 scan indicated, "[n]o pulmonary nodules or mass is present," other changes were present and any relation to coal dust exposure was not discussed.

4. Medical Opinion Evidence

Numerous medical opinions were submitted as to whether or not the claimant had coal workers' pneumoconiosis, and whether his interstitial lung disease was due to coal dust exposure. Of these opinions, I give greatest weight to the opinion of Dr. Caudill, the claimant's treating physician since 1978 where his opinion is documented and well reasoned. Dr. Caudill played an active role in the investigation and treatment of the claimant's pulmonary condition. He received feedback from several consulting physicians: Dr. White considered sarcoidosis, tuberculosis and fungus as possible causes; Dr. Collins labeled the pulmonary fibrosis as idiopathic. He reviewed the biopsy report.

Dr. Caudill testified that the diagnoses have been acute bronchitis, pneumonia, interstitial fibrosis with enlarged lymph nodes, and interstitial fibrosis proven by biopsy. He related the interstitial fibrosis to coal dust exposure. While this opinion is sufficient by itself to outweigh all the contrary opinions, I do note that the opinion is supported by the opinion of Dr. Baker who related the changes on the x-ray to coal workers' pneumoconiosis, the restrictive defect to coal dust exposure, and the then-questionable pulmonary fibrosis to coal dust exposure as well. Dr. Wilhelmus found "[n]umerous interalveolar hemosiderin-laden macrophages, emphysema, mild and focal interstitial fibrosis, predominantly subpleural." He thought that the claimant might have either Goodpasture's syndrome, idiopathic pulmonary hemosiderosis or other bleeding tendency.

Dr. Broudy diagnosed idiopathic interstitial fibrosis unrelated to coal dust exposure, but also unrelated to cigarette smoking. Dr. Westerfield diagnosed diffuse pulmonary fibrosis unrelated to coal dust exposure and cigarette smoking, and most likely due to pneumonia. Dr.

Dineen diagnosed interstitial lung disease, probably idiopathic, with coal workers' pneumoconiosis being unlikely due to the lack of a diagnosis of CWP on biopsy, the presence of finger clubbing, and the shape and location of the opacities on x-rays. Dr. Lockey, a reviewer, diagnosed respiratory bronchiolitis and desquamative interstitial pneumonitis, commonly associated with heavy cigarette smokers. One of Dr. Lockey's reasons for concluding that coal workers' pneumoconiosis was not present, was because CWP causes persistent abnormalities on the chest x-rays that do not vary over time.

However, the classified x-rays were almost entirely read as Category 1 pneumoconiosis. The evidence also indicates that the standard they applied for the location and shape of opacities in coal workers' pneumoconiosis is based on what is common. Dr. Westerfield conceded that opacities can be irregular in coal workers' pneumoconiosis, and that it can occur only in the lower lung zones. The biopsy evidence, as I found, is inconclusive as to whether the changes were due to coal dust exposure, though it was positive for anthracrotic pigmentation.

Dr. White's examination on October 7, 1996 revealed scattered rales and a few rhonchi at the bases bilaterally. A pulmonary function study revealed a mild restriction and mild diffusion impairment. Dr. White's impression was interstitial lung disease, bilateral hilar adenopathy, dyspnea, chronic bronchitis, cigarette abuse, hypertension, obesity, hiatal hernia with history of peptic ulcer disease, and depression. He commented that:

> [The claimant] has had increasing dyspnea noted primarily since March of this year. I do not think he has an acute infection at this time, but I think he does have an active interstitial process in his lungs leading to his restrictive pattern on PFT's and abnormalities seen on chest x-ray. I think this might well be sarcoidosis. Other considerations include chronic infection such as TB or fungus. His hilar adenopathy could be caused by low-grade lymphoma, but I think that would be somewhat unlikely currently. He further has a long history of cigarette abuse and I think probably does have a component of chronic bronchitis. He is currently on bronchodilator inhalers. We well have him continue those for now. I will check P.P.D., fungal serologies, CBC, sed rate and ACE levels today. I think he does need a tissue diagnosis. . . .

(DX 48).

Although considered due to the biopsy, Dr. Caudill explained that there was no evidence to confirm the presence of a bleeding disorder, a finding reached as well by another treating physician, Dr. Todd. His finding that the claimant does not have a bleeding disorder outweighs the opinion of Dr. Branscomb, a reviewing physician, that the claimant has idiopathic pulmonary hemosiderosis. I note further that the other examining physicians also did not diagnose hemosiderosis.

The claimant was hospitalized at Whitesburg ARH Hospital from January 18 to 25, 1997. The attending physician was Dr. William Collins. The discharge diagnoses were pneumonia with bronchitis, idiopathic pulmonary fibrosis, depression, and hypertension. (DX 42).

Dr. Glen Baker examined the claimant on March 10, 1997 on behalf of the OWCP. He reviewed the claimant's histories, symptoms, and medications. Examination revealed bilateral basilar rales and wheezes. An x-ray was interpreted as positive for coal workers' pneumoconiosis, 1/2. A pulmonary function study showed a moderate restrictive defect. An arterial blood gas test revealed mild resting arterial hypoxemia. Dr. Baker diagnosed coal workers' pneumoconiosis, 1/2, based on the x-ray and occupational history, due to coal dust exposure; chronic bronchitis based on the history of cough, sputum production and wheezing, due to coal dust exposure; moderate restrictive defect based on the pulmonary function study, due to coal dust exposure; questionable pulmonary fibrosis, due to coal dust exposure; and arteriosclerotic heart disease based on history. Dr. Baker was aware of the claimant's smoking history. He concluded that the claimant had a moderate pulmonary impairment with decreased VC, decreased pO2, chronic bronchitis, and advanced coal workers' pneumoconiosis; and that he was totally disabled from his former coal mine employment. (DX 18). I find Dr. Baker's opinion and diagnoses probative and credible where he relied on objective tests, work history, examination and symptoms. I also find his diagnoses well reasoned where he relied on the complete pulmonary evaluation, testing, and histories to reach his diagnoses.

Dr. Bruce C. Broudy examined the claimant on April 7, 1997 on behalf of the employer. He reviewed the claimant's histories, symptoms, and medications. Examination revealed bibasilar squeaks and inspiratory crepitations, and possible early clubbing of the fingers. A pulmonary function study showed a moderately severe restrictive defect. There was a slight improvement after bronchodilation, but the claimant's effort was not considered maximal. An arterial blood gas test revealed moderately severe to severe resting arterial hypoxemia. The carboxyhemoglobin level was elevated at 8.3%, which was noted to indicate continued exposure to smoke. An x-ray was interpreted as negative, 0/1, u/t. Dr. Broudy diagnosed history of pulmonary fibrosis, obesity, depression, and hypertension. He commented that:

> I do not believe that Mr. Holbrook has coal workers' pneumoconiosis. There is some evidence that he has some type of interstitial pulmonary fibrosis of undetermined cause. Review of the St. Joseph Hospital biopsy specimens would be helpful, of course, to further confirm the findings. The patient does have some restrictive defect which may be partly related to less than maximal effort. There is hypoxemia which may be related to the interstitial fibrosis and obesity. Because of the hypoxemia and diminished lung function, I believe that he does not retain the respiratory capacity to perform the work of an underground coal miner or to do similarly arduous manual labor. I do not believe that there has been any significant pulmonary disease or respiratory impairment which has arisen from this man's occupation as a coal worker.

(DX 15).

Dr. Broudy's report appears to be well documented based on his complete pulmonary evaluation; however, I find his opinion to be less than well reasoned where he equivocates

regarding the etiology of Mr. Holbrook's diagnoses. He does not explain the reasoning behind his assertion that Mr. Holbrook does have pulmonary impairments and diminished lung function but it is not due to coal mine employment. He offered no other etiology to explain his diagnoses or his opinion that Mr. Holbrook was disabled. He also asserted that "patient has some restrictive defect which may be partly related to less than maximum effort," yet less than maximum effort would not indicate a restrictive defect. Instead, it would make the test results invalid. Consequently, I find that this opinion of Dr. Broudy's is not supported by the objective tests and is not well reasoned. Therefore, I accord it little weight for lack of credibility.

Dr. Broudy reviewed additional medical records on behalf of the employer and issued supplemental reports on November 18, 1997 and December 19, 1997. In the latter report, he stated that:

> To make a diagnosis of coal workers' pneumoconiosis one would need an adequate history of exposure and either characteristic findings on chest x-ray or typical findings of pneumoconiosis by lung biopsy. Although this gentleman had a history of exposure, he had neither the characteristic x-ray findings nor the typical findings of coal workers' pneumoconiosis by lung biopsy. The open lung biopsy was an adequate specimen from the upper lobes and did not show coal workers' pneumoconiosis as was stated by two pathologists in the record. As noted, the chest x-ray was not normal, but it did not show the characteristic small rounded opacities in the upper zones, but rather irregular opacities in the mid to lower zones.

He continued to find that the claimant was totally disabled. (DX 55, 56).

Dr. Broudy was deposed on December 18, 1997. He reiterated his examination findings. He further testified that:

> [I]diopathic pulmonary fibrosis is a disease or condition of the general public. It is not -- coal workers are not protected from getting it nor are they apparently more likely to get it than others. So, I believe that he would have had this disease whether or not he had worked in the coal mine.

As to the claimant's smoking history, he testified that:

> [S]moking is a major cause of pulmonary impairment and pulmonary disease. It usually causes chronic bronchitis or pulmonary emphysema or some combination thereof. It's frequently associated with obstructive airways disease. In this particular case it appears that the patient may have interstitial fibrosis that is unrelated to cigarette smoking, per se.

On cross-examination, Dr. Broudy estimated that the lung biopsy consisted of one per cent or less of the lungs, but that if biopsies were not representative, "we would never do a lung biopsy because we couldn't therefore generalize the best of what was wrong with the rest of the lung." He explained that:

> If there is a diffuse disease present in the lung; that is, a disease that's present in both lungs, then we make an assumption that biopsying a representative sample will allow us to generalize as to what may be going on in the entirely of both lungs, although there certainly can be more than one thing going on and it may not be present in all areas. I agree that that's a possibility.

He stated that a diagnosis of idiopathic interstitial lung disease excludes a diagnosis of coal workers' pneumoconiosis, because idiopathic indicates that the cause is unknown. As to Dr. Colby's report, Dr. Broudy stated that "anthracotic pigment is from coal dust inhalation. It doesn't have anything specifically to do with cigarette smoke," and that "for him to say it's hard to distinguish the difference is a little perplexing to me . . . ." (EX 1).

On direct examination during his deposition, he testified that he did not "know the origin of the pulmonary impairment and fibrosis noted on lung biopsy." (EX 1, p.10). In addition, his original report stated that he did observe small rounded opacities in the mid to lower lung zones but noted only a few and consequently, rate the film as negative. In a supplemental report, Dr. Broudy included his opinions regarding the biopsy report of Dr. Wilhelmus. (DX 55). He noted a "large amount of pigment, some of which consisted of carbonaceous pigment seen with coal dust exposure." He also stated, "the finding did not suggest coal workers' pneumoconiosis based on the report of Dr. Wilhelmus."

Dr. Broudy opined that the additional evidence tended to confirm his previous report, which "did not find definite evidence of coal workers' pneumoconiosis" but did note a slight increase in opacities in the lower and mid lung zones, which "could be characteristic of any type of nonspecific fibrosis such as was noted in this case pathologically." This statement tends to contradict his initial report where he classified the x-ray as negative due to the few number of small rounded opacities yet, subsequently, he noted an increase in the number. Admittedly, he may have found the increase too insignificant to change his opinion but the fact that he found it significant enough to mention indicates an inconsistency where the lack of small rounded opacities formed some of the basis for his rejection of pneumoconiosis as the cause of his interstitial disease.

I also note that during his deposition, when asked if the biopsy slides would indicate the presence or absence of pneumoconiosis, Dr. Broudy responded, "They may." He offered testimony regarding the biopsy report of St. Joseph's Hospital stating:

> Well the main question of interest here is whether or not the biopsy showed evidence of coal worker's pneumoconiosis. Just the mere presence of the carbonaceous material in the lymph nodes or in the lung would not be diagnostic of that disease. One would need to

> see the characteristic focal dust macule and the typical pattern of fibrosis to make a diagnosis of coal workers' pneumoconiosis. The pathologist described the findings in some detail but did not give a description that would be characteristic of coal workers' pneumoconiosis. He didn't raise – he specifically didn't raise that as a diagnosis…**he felt that the most striking finding was the presence of the so-called hemosiderin-laden macrophages in the alveoli suggesting the possibility of some bleeding into the lung from diseases that had not been suspected or diagnosed yet.** So, he really was unable to make a specific diagnosis. He did find interstitial fibrosis and listed some possible causes based on the findings of the hemosiderin-laden macrophages. (Emphasis added).

On cross-examination, Dr. Broudy conceded that the pathologists were not looking for pneumoconiosis. (EX 1, Depo. p. 19). Dr. Broudy stated that he would not be competent to view the slides and make a determination on his own. I find Dr. Broudy's opinion somewhat equivocal where he rules out smoking as the source of the impairments, because smoking causes obstructive, not restrictive, airway disease, yet he definitively rules out coal worker's pneumoconiosis even though he maintains that the etiology for the interstitial lung disease is unknown. In addition, Dr. Broudy did not adequately explain why the miner's diffusion impairment could not be related to his inhalation of coal mine dust.

Nor did he provide the basis for his opinion that the miner's fibrosis was not due to his coal mine dust exposure. Where a doctor would expect to see a restrictive defect if the miner's pulmonary fibrosis was due to his coal mine dust inhalation and all physicians of record have opined that Mr. Holbrook suffers from a restrictive rather than an obstructive impairment, this would provide some indication of coal dust exposure rather than cigarette smoking, for example.

Nevertheless, Dr. Broudy relied on the biopsy report, which he admitted was not done to diagnose pneumoconiosis and would not necessarily be definitive for the absence of pneumoconiosis, and the x-ray to make his determination that Mr. Holbrook does not have pneumoconiosis. The x-ray relied on was interpreted by three readers who found the radiographic image unreadable yet Dr. Broudy rated is a quality of "1" and used his interpretation to form the basis of his opinion. (DX 25, 42, 46, 50, Broudy Depo. p. 19-20).

Consequently, I find that although Dr. Broudy's report appears thorough, it is not well documented where the data he relied on are questionable. His conclusions are not well supported and are contradictory as to his basis of ruling out pneumoconiosis.
I accord this opinion little weight.

Dr. John E. Myers, Jr., issued a letter on May 23, 1997, stating:

> I reviewed **. . .** the pathology specimen on this man's lung biopsy and the report of Dr. Baker, and the x-rays, one of which I reviewed myself and agreed with Dr. Baker's interpretation.

> Obviously this man has significant pulmonary impairment which would be at least Class III under the AMA Guides. The biopsy specimens do not rule out coal workers' pneumoconiosis nor do they definitely substantiate it, which is not unusual. This man has an iron deposition disease of some sort within his lungs. Whether he incurred this iron exposure from his work or whether it represented an intrinsic disease such as Goodpasture's syndrome I can't say. I think we would have to conclude that he has coal workers' pneumoconiosis, that he may have other occupational disease or other disease not even related to his occupation as well.

(DX 41).

To the extent that this report supports Dr. Baker's x-ray interpretation and diagnoses, I find it probative but only somewhat credible, where he does not elaborate on the reasons behind his assertion "we would have to conclude that he has coal worker's pneumoconiosis." I find that this report presents some evidence supporting a finding of pneumoconiosis.

Dr. Byron T. Westerfield, a Board-certified pulmonologist, examined the claimant on July 10, 1997. Examination revealed coarse rales bilaterally, a few rhonchi, and mild clubbing. An x-ray was positive for diffuse pulmonary fibrosis. A pulmonary function study demonstrated severe restrictive ventilatory dysfunction. There was no significant improvement in flow rates following administration of bronchodilators. An arterial blood gas test revealed a mild decrease in oxygen on room air at rest. The carboxyhemoglobin level of 9.9% indicated heavy cigarette smoking.

After review of the biopsies, Dr. Westerfield concluded that the claimant was totally disabled, but that he did not have coal workers' pneumoconiosis. (DX 44, 54). Dr. Westerfield conceded during his deposition that opacities can be irregular in coal workers' pneumoconiosis, and that it can occur, only in the lower lung zones, yet he based his findings, in part, on a contrary conclusion. Additionally, Dr. Westerfield used the terms, coal workers' pneumoconiosis and pneumoconiosis, interchangeably and thus did not explicitly rule out other types of pneumoconioses. Dr. Westerfield had previously testified that pneumoconiosis may be present in the middle and lower lung fields and Mr. Holbrook's x-ray reports evidenced this.

Dr. Westerfield, deposed on November 12, 1997, testified about his examination findings. As to the x-rays, he stated that the one he obtained was of quality "2" due to income-plete inspiration, which is a result of the claimant's pulmonary condition. He explained that "[i]f the lungs are not fully expanded, the lung markings appear more concentrated, and in some cases, increasing the normal lung markings can be mistaken for the presence of pneumoconiosis or other pathology." He also explained that for a diagnosis of pneumoconiosis "[a] chest radiograph needs to show small opacities, which are usually rounded, but they can be irregular. They are present most commonly in the upper and mid lung zones and they must be at a profusion level of 1/0 or greater, based upon the ILO classification for pneumoconiosis."

Dr. Westerfield further testified that the interstitial scarring is unrelated to the claimant's smoking, and most likely due to the previous pneumonia. The carbon particles in the lymph nodes, he related to smoking and emphysema. He stated that the lung biopsy would have shown pneumoconiosis if the claimant had pneumoconiosis.

Dr. Westerfield reviewed additional medical records on behalf of the employer and issued a report on January 7, 1998. While he concluded that the claimant was totally disabled from a respiratory standpoint, he concluded that disability was related to pulmonary fibrosis of uncertain cause, though probably related to pneumonia. He concluded that the evidence showed that the claimant did not have coal workers' pneumoconiosis. He explained that:

> [The claimant] has an adequate history of exposure to coal dust, but does not have radiographic or pathological evidence of pneumoconiosis. Mr. Holbrook's chest x-ray is not normal. There is definite pulmonary fibrosis present on the chest roentgenogram but the fibrosis is not consistent with pneumoconiosis. This is, also, the opinion of most of the B Readers interpreting Mr. Holbrook's many chest x-rays.
>
> Mr. Holbrook was fortunate enough to have undergone open lung biopsy 11/8/96 and adequate lung tissue was obtained to diagnose interstitial fibrosis. According to both Dr. John Wilhelmus of Lexington and Dr. Thomas V. Colby of the Mayo Clinic there is no Coal Workers' Pneumoconiosis present on the biopsy specimens. Actual pathological examination is the most accurate means of diagnosing pneumoconiosis and would take precedent over an x-ray diagnosis.

(DX 57).

On cross-examination, Dr. Westerfield conceded that coal workers' pneumoconiosis is a type of pulmonary fibrosis and that the claimant has pulmonary fibrosis; and that coal workers' pneumoconiosis more often than not causes a restrictive defect, which the claimant has. He explained that he did not check off the boxes at section 2 of the ILO form because he felt the condition was clearly not pneumoconiosis. He conceded that a lung biopsy was not performed on the right lung, and that coal workers' pneumoconiosis cannot be ruled out without an autopsy. He also stated that, while uncommon, coal workers' pneumoconiosis could occur only in the lower lung zones. (DX 54). Where Dr. Westerfield's opinion and conclusions were contradicted by his deposition testimony on cross-examination, I find his opinion is not well reasoned and therefore, not credible where he equivocated regarding what can and cannot indicate pneumoconiosis.

Dr. Westerfield did not explain his reasons or the basis for excluding coal worker's pneumoconiosis. He offered no persuasive reasons for his conclusion that if the miner had interstitial pulmonary fibrosis it could not be related to his coal mine employment. He merely

opined that the miner did not have a coal mine dust-related disease. Where he reached differing conclusions during his testimony, I find his opinion less than well reasoned and therefore, not credible.

Dr. John F. Dineen, who is Board-certified in internal, pulmonary, and critical care medicine, examined the claimant on July 29, 1997 on behalf of the employer. (He had examined him earlier on April 22, 1997; DX 16 - 17). Auscultation of the lungs revealed persistent Velcro-like rales at both lung bases. The fingers were clubbed. An x-ray was interpreted as showing bilateral parenchymal abnormalities consistent with pneumoconiosis, 1/1, s/t, both mid and lower lung zones. A pulmonary function study showed moderate restriction of lung volumes without an obstruction component, and no significant improvement in flow rates following the use of a bronchodilator. The claimant had minimal hypoxemia on room air. Dr. Dineen also reviewed the two pathology reports. He concluded that:

> Mr. Holbrook has interstitial lung disease. He has moderately severe respiratory impairment of the whole person (25-50%). I suspect that Mr. Holbrook has idiopathic interstitial pulmonary fibrosis. Coal workers' pneumoconiosis is an unlikely cause of his interstitial pulmonary fibrosis based on the following observations - (1) The open lung biopsy did not demonstrate coal workers' pneumoconiosis. That is the gold standard for diagnosing interstitial lung diseases. (2) Finger clubbing is not usually a part of the clinical spectrum of coal workers' pneumoconiosis. (3) The lower lung zones are predominantly involved and the small interstitial opacities are irregular in shape. Typically coal workers' pneumoconiosis/silicosis involves the upper lung zones and the parenchymal changes are round in nature. I do not think Mr. Holbrook retains the pulmonary capacity to perform his former duties as a coal miner. He has moderately severe hypoxemia on room air and requires oxygen 18 hours a day to prevent the development of pulmonary hypertension. He does not retain the respiratory capacity to perform his former duties as a coal miner or similar arduous labor.

Where Dr. Dineen's report is based on all the objective testing and an examination, I find that his opinion is well documented. Furthermore, his x-ray interpretation of pneumoconiosis signifies the presence of opacities, but where his report rules out coal worker's pneumoconiosis, I find his opinion well reasoned and not contradictory. Findings of opacities do not necessarily signify the presence of coal worker's pneumoconiosis. Additionally, Dr. Dineen relied on the biopsy results to augment his objective finding and examination. Based on his superior qualifycations, I find his opinion is credible and worthy of probative weight against a finding of pneumoconiosis.

Dr. George Caudill, the claimant's treating physician, was deposed on July 31, 1997. He testified that the claimant has been his patient since 1978 and that he has treated him for occasional bronchitis through the years (once every two to three years) but has treated him much more

frequently for respiratory problems. (DX 52, Depo. p. 6). The record reflects over 100 office visits and or contacts through the years. Since January 1996, he has seen him more frequently for respiratory problems. He stated that:

> At that point, [the claimant] came in with an acute respiratory infection, and another episode of bronchitis. And was from (sic) that moment on had had several episodes. He was treated with antibiotics then, and he got a little better. Then shortly there after he got pneumonia, and wound up in the hospital. And he has had recurring problems since that time.

Dr. Caudill testified, "[t]he initial treating diagnoses (sic) was just acute bronchitis, and then later it became pneumonia. And then interstitial fibrosis with enlarged lymph nodes. And then after that, of course, interstital fibrosis proven by biopsy." He stated that the finding of interstitial fibrosis was very consistent with pneumoconiosis, and even more consistent considering his work history. He explained that:

> [B]asically the lung biopsy showed that he had indeed **. . .** interstitial fibrosis. Some of the macrophages were noted to be containing carbonatious material consistent with pneumoconiosis. Some of them also contained hemosiderin macrophages, which made us entertain the diagnoses (sic) of possibly Goodpasture's syndrome. But nothing else has been brought out to confirm that. He's never had bleeding, or any other findings normally seen in Goodpasture's syndrome… The carbonatious material was certainly consistent with the diagnosis of pneumoconiosis.

Dr. Caudill further testified that the finding of a restrictive defect was consistent with interstitial lung disease and that interstitial lung disease was consistent with pneumoconiosis. (DX 52, Depo. p. 8). He related the claimant's breathing impairment to coal dust exposure and found him to be totally disabled. Dr. Caudill's office notes were attached to the deposition and included numerous objective tests results obtained over the course of treating years. (DX 52). Dr. Caudill treated Mr. Holbrook with oxygen, Flovin and Antivan inhalers, several different types of Prevental inhalers and breathing treatments. (DX 52, Depo. p. 12). His diagnoses are pneumoconiosis, interstitial fibrosis, angina, hypertension, anxiety, recurrent bronchitis and depression. On cross-examination, Dr. Caudill stated that he relied on the objective test results of other physicians, such as the hospital doctors, to make his diagnoses. He also stated that interstitial fibrosis is a “very very rare” condition in individuals who have not been exposed to coal dust.

I find Dr. Caudill’s opinion deserving of the greatest probative weight, based on his status as treating physician, his rendering of an opinion after review of extensive medical evidence and the relatively recent examination of the Claimant. Furthermore, the opinion is based on a long history of objective testing, examination and frequent visits.

The Sixth Circuit, in *Jericol Mining v. Old Republic Insurance Co.*, outlined relevant factors to consider when assigning weight to the opinion of a treating physician. 301 F.3d 703 (6th Cir. 2002). These factors have since been codified into the regulations at revised 20 C.F.R. § 718.104(d) and include the nature and duration of the relationship and the frequency and extent of the treatment.

The record reveals a treatment history spanning twenty years and including over one hundred contacts or visits. Dr. Caudill has treated Mr. Holbrook for a variety of diseases and ailments requiring hospitalizations and various tests. He has been privy to the decline in Mr. Holbrook's respiratory condition and has observed first-hand his symptomologies. Consequently, I find that Dr. Caudill's opinion is worthy of the greatest probative weight based on his treating physician status, his extensive knowledge of the Claimant and his conditions, his review of all available data, and his well-reasoned and credible findings.

Dr. Dineen also reviewed additional records and issued a supplemental report on January 14, 1998. His opinions remained the same. (DX 57).

The Employer provided three consultative reports. Dr. James Lockey reviewed the medical records and depositions on behalf of the employer and issued a consultative report on February 23, 1998. (DX 58). Dr. Lockey, without examination, concluded that:

> [The claimant's] pulmonary complaints are not consistent with coal worker's pneumoconiosis or in any way related to his occupation as a coal miner. Mr. Holbrook's pulmonary condition apparently started after onset of a respiratory infection in early 1996. He subsequently developed bilateral hilar adenopathy and fleeting and changing pulmonary infiltrates involving the basilar segments of both lungs radiating out from the hilar area. These findings are not consistent with an occupational exposure to coal dust. This was confirmed when the patient underwent a subsequent open lung biopsy which did not demonstrate any changes consistent with coal worker's pneumoconiosis. In addition, coal worker's pneumoconiosis causes persistent abnormalities on the chest x-rays that do not vary over time, are not associated with clubbing, and commonly do not involve the lower lung fields.
>
> Respiratory bronchiolitis and desquamative interstitial pneumonitis are pulmonary diseases that are commonly associated with heavy cigarette smokers. I have included a copy of this description from a textbook entitled, OCCUPATIONAL AND ENVIRONMENTAL RESPIRATORY DISEASE, Edited by Harber, Schenkar, and Balmes, Mosby Publishing, 1996, Chapter 8, Page 117, which is supportive of this opinion.

Dr. Lockey further concluded that the claimant was totally disabled from a pulmonary standpoint. (DX 58).

Dr. Ben V. Branscomb, who is Board-certified in internal medicine, reviewed the medical records on behalf of the employer and issued a consultative report on May 27, 1998. He noted that "[t]he lymph node contained black carbon, as is the usual finding in smokers, miners, or older urban dwellers." As to the pathology reports, Dr. Branscomb commented that:

> Because the smaller bronchioles were inflamed (respiratory bronchiolitis associated with interstitial lung disease) Dr. Colby thought tobacco smoke might be the cause of the problem. (The bronchioles are the primary site for tobacco injury. Dr. Colby was suggesting that the bleeding into the alveoli was secondary to this injury.) He found other changes that fit with cigarette smoking. Dr. Colby noted that in some smokers the bronchiolar injury is minimal and asymptomatic. A more severe example would correspond with Mr. Holbrook.
>
> In the severest form, there is a great deal of death, destruction, and sloughing off of cells. That very rare condition is called desquamative interstitial pneumonia or DIP. DIP is a process similar to the usual interstitial pulmonary fibrosis (UIP) except that the cells are predominantly macrophages and they are in the air spaces with some inflammatory infiltrate of the alveolar walls by scanty fibrosis. The exchange between Dr. Colby and Dr. Wilhelmus relates to a rather technical classification question concerning several very rare and closely interrelated pulmonary disorders. Was the primary defect leaking of blood into the air sacs or was the primary defect in the bronchiolar walls and hemorrhage into air spaces secondary? In any case, there were no pathologic findings of any of the pneumoconioses. There also was no pervasive generalized interstitial fibrosis.

Dr. Branscomb concluded that:

> 1. Mr. Holbrook does not have coal worker's pneumoconiosis nor any other pneumoconiosis. This is based on, among other considerations, the fact that the x-ray was not compatible with CWP but was typical of what the biopsy showed he had. Second, the biopsy consisted of ample tissue absolutely to rule out coal worker's pneumoconiosis as the disease. Third, the clinical events, pulmonary function changes, blood gases, and all other clinical aspects are not compatible with early simple pneumoconiosis and are typical of the combination of the effects of smoking plus the findings at biopsy.
>
> 2. He is totally disabled as a result of a rare disease falling into the category of the interstitial and alveolar diseases. These include

> UIP, DIP, and the pulmonary hemorrhagic diseases. Within this spectrum there has been some disagreement and speculation concerning how best to classify Mr. Holbrook's disorder. (We have this same discussion frequently at our pulmonary pathology conferences.) The important finding is that he does not have any evidence of any pneumoconiosis and that the process causing his symptoms and findings was established by biopsy. My own personal formulation is that he probably does have idiopathic pulmonary hemosiderosis since that disease commonly produces no hemoptysis, may be intermittent, and is associated with underlying fibrosis around the involved alveoli and under the pleura. I have certainly seen such cases in the past. It is also possible that the primary defect lies in the interstitial spaces and this has resulted in some hemorrhage into the alveoli. There is no basis whatever for ascribing to coal dust any contribution whatsoever to the pathologic process in the lung nor to his impairments and disability.

(EX 2).

Initially, I find that Dr. Branscomb's report is reasonably well documented and well reasoned and accord it probative weight against a finding of pneumoconiosis where he discussed all the possible causes of the x-ray and biopsy test results and based his conclusions accordingly. He failed however, to take into consideration Mr. Holbrook's coal dust exposure history, his symptoms, or his arterial blood gas studies and pulmonary function tests and he did not examine Mr. Holbrook.

I also note that Dr. Branscomb is the only physician of record who diagnosed the rare pulmonary diseases listed in his report. The treating physicians and hospital staff have not reached the same diagnosis nor have the other examining or consulting physicians. Consequently, I find that Dr. Branscomb's report is unsubstantiated by any other reports or findings. He fails to explain the basis for determining that coal dust exposure does not affect or aggravate Mr. Holbrook's condition in any fashion. I find this report provides some probative weight against a finding of pneumoconiosis, but do not accord it significant weight where it is not well documented or well reasoned.

Dr. Jerome Kleinerman, a Board-certified pathologist, reviewed the biopsy tissue and medical records on behalf of the employer, and issued a report on May 30, 1998. He concluded that:

> [The] pathologic findings are diagnostic of early desquamative interstitial pneumonitis. This is an exudative stage in the development of classical interstitial fibrosis. Mr. Holbrook's lung biopsy also shows a terminal and respiratory bronchiolitis associated with the desquamative interstitial pneumonitis. This combination of pathologic findings has been described in heavy

> and prolonged cigarette smokers. However certain pathologic features commonly present in this entity such as the presence of lymphoid nodules in the lung and tissue eosinophilia are not present.
>
> Nevertheless it is my opinion with reasonable medical certainty that Mr. Holbrook does not have simple or complicated CWP. Coal Workers' Pneumoconiosis with reasonable medical certainty is not the cause of any of Mr. Holbrook's respiratory disability.
>
> I believe that Mr. Holbrook, even if provided with proper medical treatment for his respiratory ailment would be unable to perform his former coal mine work. Proper medical treatment would include: 1) total cessation of cigarette smoking, 2) weight loss of 30-40 pounds, 3) treatment with oral corticosteroids for his desquamative interstitial pneumonitis, and 4) careful observation for any subsequent change in Mr. Holbrook's pulmonary status.

I find that Dr. Kleinerman's well reasoned and documented report is probative on the limited issue of clinical pneumoconiosis. Consequently, it does not support a finding of pneumoconiosis in Claimant's favor. However, the definition of statutory pneumoconiosis is not as narrowly defined as clinical coal workers' pneumoconiosis.[3] Where Dr. Kleinerman's opinion does not address legal pneumoconiosis, it will not weigh against a finding of legal pneumoconiosis.

I find that pneumoconiosis is established by Dr. Caudill's probative, credible opinion, which is entitled to substantial weight under *Wolf Creek Collieries v. Director, OWCP [Stephens]*, 298 F.3d 511 (6th Cir. 2002). Pneumoconiosis is also established by Dr. Baker's credible and probative opinion, as substantiated by the opinion of Dr. Myers. Dr. Caudill's opinion, as Mr. Holbrook's treating physician, offers controlling and heavily weighted evidence due to its credibility in light of its reasoning and documentation, other relevant evidence and the probative record as a whole. *See, Jericol Mining, Inc. v. Director, OWCP [Napier]*, 301 F.3d 703 (6th Cir. 2002).

In *Gray v. Peabody Coal Co.*, Case No. 01-3083 (6th Cir. Apr. 19, 2002) (unpublished), the Sixth Circuit held that the administrative law judge erred in according greater weight to the consultative opinions of two physicians over the opinion of a treating physician on grounds that the consultative physicians had superior credentials. Citing to *Tussey v. Island Creek Coal Co.*, 9982 F.2d 1036 (6th Cir. 1993), the court held that "[w]here the ALJ determines that the treating physician's opinion is well reasoned and well documented, the ALJ must give more weight to that opinion than to those of other physicians, even where those other physicians have superior qualifications."

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3 "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.

Weighing all of the evidence, both like and unlike, on the issue of pneumoconiosis, I find that the claimant has established pneumoconiosis. The x-rays showed some changes consistent with pneumoconiosis and the biopsy evidence is inconclusive as to whether the interstitial changes were due to coal dust exposure, but did show a background of anthracrotic pigmentation according to the Mayo Clinic's findings. The medical opinions of the claimant's treating physiccian, Dr. Caudill, the examining report of Dr. Myers and the District Director's report by Dr. Baker, all provide credible evidence supporting a finding that the claimant has pneumoconiosis and interstitial lung disease due to coal dust exposure. However, the examining reports of Drs. Broudy and Westerfield and the consultative reports of Drs. Dineen, Lockey, Branscomb and Kleinerman opine that Mr. Holbrook does not have pneumoconiosis due to coal mine employment and are not as credible as the contrary opinions for the above-discussed reasons. Where I found the reports of Drs. Broudy and Westerfield not credible, those opinions do not overcome the greater probative value of those physicians who found pneumoconiosis.

As the claimant has established that, he has pneumoconiosis (interstitial lung disease/ pulmonary fibrosis) he is entitled to benefits under the Act if he shows that the pneumoconiosis is caused by his coal mine employment and he is totally disabled due to pneumoconiosis.

Causation of Pneumoconiosis

Once it is determined that the miner suffers from pneumoconiosis, it must be determined whether the miner's pneumoconiosis arose, at least in part, out of coal mine employment. 20 C.F.R. § 718.203(a). If a miner who is suffering from pneumoconiosis was employed for ten years or more in one or more coal mines, there is a rebuttable presumption that the pneumoconiosis arose out of such employment. 20 C.F.R. § 718.203(b). Pursuant to § 718.203(b), claimant is entitled to invocation of the rebuttable presumption that his pneumoconiosis arose from his coal mine employment.

The employer presented rebuttal evidence, all of which was considered under § 718.202(a)(4) and mentioned under the positive x-ray interpretations of Dr. Broudy and West under § 718.202(a)(3). Dr. Broudy and West interpreted the respective x-rays as being "Not CWP" or "Not characteristic of CWP. If due to pneumoconiosis, more likely to be related to asbestosis." (DX 24, 42). Dr. White offered sarcoidosis as a possible reason for the abnormal x-rays ("I think this might well be sarcoidosis") while Dr. Colby offered cigarette abuse as the cause of the abnormal pulmonary function. Dr. Broudy blamed Mr. Holbrook's interstitial fibrosis, not on cigarettes or coal dust exposure but on an unknown etiology.

Dr. Dineen summarily dismissed coal dust exposure as the source of Claimant's pulmonary condition because it was not evident from the biopsy. He did not offer any other cause of his conditions. Dr. Westerfield also dismissed coal dust as an etiology and related the lung condition to smoking, emphysema, and scarring from pneumonia. However, on cross-examination he conceded that some of his statements regarding coal dust as a causal entity were not entirely correct. Dr. Branscomb believes that Mr. Holbrook suffers from a rare pulmonary disease and acknowledged that there exists speculation and disagreement about how to classify Mr. Holbrook's disorders. He stated that coal dust exposure does not contribute in any way to

Mr. Holbrook's condition but did not offer an alternative etiology for his condition. Dr. Kleinerman concluded that combinations of certain pathological findings in heavy smokers were absent, thus casting doubt on smoking as a cause, however, he also ruled out coal dust exposure without offering a substitute offending agent or cause.

I find that the Employer's rebuttal evidence offers inconsistent alternatives to a diagnosis of pneumoconiosis. Some rebuttal evidence fails to address an etiology to rebut the presumption that Mr. Holbrook's pneumoconiosis arose from his underground coal mine employment. No two of Employer's rebuttal opinions offer are consistent on this issue. While smoking is offered as the exclusive cause by one physician, smoking is excluded by another. To the contrary, the Claimant has the benefit of the consistent and credible opinions of Dr. Baker and of Claimant's treating physician who both attributed some of Claimant's condition to smoking but believed that his pulmonary impairments were also caused, in part, by coal dust exposure.

. While there has been much debate as to the cause of claimant's interstitial fibrosis and chronic bronchitis, Dr. Caudill's opinion on causation deserves the probative weight where his findings and opinions are the most thorough and complete. He related the interstitial fibrosis to coal dust exposure, thereby meeting the definition of pneumoconiosis at § 718.201 and establishing the requisite causation. Dr. Baker also found a moderate restrictive impairment by pulmonary function studies, pneumoconiosis by x-ray and chronic bronchitis (by cough, sputum production, wheezing) and opined that all of these are due in part to coal mine dust exposure. Thus Dr. Baker's opinion, reliant on objective testing and examination, is both credible and supportive of Dr. Caudill's. While I accorded Dr. Myers' opinion less weight than Dr. Baker's or Dr. Caudill's I also note that his findings were consistent with those opinions. I, therefore, find that claimant's pneumoconiosis arose from his coal mine employment

Cause of Total Disability

A miner shall be considered totally disabled if he has complicated pneumoconiosis (§ 718.304) or if pneumoconiosis prevents him from doing his usual coal mine employment or comparable and gainful employment (§ 718.204(b)). Additionally, total disability causation is defined as follows:

> (c)(1) Total disability due to pneumoconiosis defined. A miner shall be considered totally disabled due to pneumoconiosis if pneumoconiosis, as defined in Sec. 718.201, is a substantially contributing cause of the miner's totally disabling respiratory or pulmonary impairment. Pneumoconiosis is a ``substantially contributing cause" of the miner's disability if it:
>
> (i) Has a material adverse effect on the miner's respiratory or pulmonary condition; or
> (ii) Materially worsens a totally disabling respiratory or pulmonary impairment which is caused by a disease or exposure unrelated to coal mine employment. 20 C.F.R. § 718.205 (2001).

The administrative law judge must consider all the evidence of record and determine whether the record contains "contrary probative evidence." If so, the administrative law judge must assign this evidence appropriate weight and determine "whether it outweighs the evidence supportive of a finding of total respiratory disability." *Troup v. Reading Anthracite Coal Co.*, 22 B.L.R. 1-11 (1999) (en banc); *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).

The regulations at § 718.204(b) provide the following five methods to establish total disability: (1) pulmonary function (ventilatory) studies; (2) blood gas studies; (3) evidence of cor pulmonale; (4) reasoned medical opinions; and (5) lay testimony. 20 C.F.R. § 718.204(b). Here, the arterial blood gas studies and pulmonary function tests did not produce qualifying results.

Under § 718.204(c)(4), "all the evidence relevant to the question of total disability due to pneumoconiosis is to be weighed, with the claimant bearing the burden of establishing by a preponderance of the evidence the existence of this element." *Mazgaj v. Valley Camp Coal Co.*, 9 B.L.R. 1-201, 1-204 (1986). In reviewing the medical opinion evidence regarding etiology, I note that those opinions that did not diagnose Mr. Holbrook as suffering from pneumoconiosis may be accorded little probative value. In *Hobbs v. Clinchfield Coal Co.*, 45 F.3d 819 (4th Cir. 1995), the court held that the administrative law judge's finding that the miner's total disability was not due to pneumoconiosis was supported by substantial evidence as "[t]he medical opinions upon which he relied most strongly were not tainted by underlying conclusions of no pneumoconiosis pursuant to the broad legal definition contained in 20 C.F.R. § 718.201."

On the other hand, in *Toler v. Eastern Assoc. Coal Co.*, 43 F.3d 109 (4th Cir. 1995), the court held that, where the administrative law judge determines that a miner suffers from pneumoconiosis or is totally disabled or both, then a medical opinion wherein the miner is determined not to suffer from pneumoconiosis or is not totally disabled "can carry little weight" in assessing the etiology of the miner's total disability "unless the ALJ can and does identify specific and persuasive reasons for concluding that the doctor's judgment on the question of disability causation does not rest upon her disagreement with the ALJ's finding as to either or both of the predicates (pneumoconiosis and total disability) in the causal chain."

As noted in my previous Decision and Order, the medical opinion evidence is undisputed that claimant is totally disabled due to his interstitial fibrosis and chronic bronchitis, whether or not the physician considered the fibrosis to be coal workers' pneumoconiosis. The opinions of Drs. Baker, Dineen, Caudill, Westerfield, Branscomb, and Kleinerman establish this link. Dr. Broudy believed that poor effort may have contributed to his finding of restriction and that obesity may have contributed to the hypoxemia, but he, nevertheless found the interstitial fibrosis to be disabling. Dr. Baker found that the chronic bronchitis contributed to claimant's disability; however, he related the chronic bronchitis to coal dust exposure, bringing it within the definition of pneumoconiosis at § 718.201.

The Sixth Circuit requires that total disability be "due at least in part" to pneumoconiosis. *Adams v. Director, OWCP*, 886 F.2d 818, 825 (6th Cir. 1989); *Zimmerman v. Director, OWCP*, 871 F.2d 564, 566 (6th Cir. 1989); *Roberts v. Benefits Review Board*, 822 F.2d 636, 639 (6th Cir.

1987). However, in *Peabody Coal Co. v. Smith*, 127 F.3d 504, 507 (6th Cir. 1997), the Sixth Circuit held that, although pneumoconiosis need only be a "contributing cause" to the miner's total disability, a claimant must demonstrate that the disease was more than a *de minimus* or "infinitesimal" factor in the miner's total disability.

As such, the opinions of Mr. Holbrook's treating physician and the reports of Dr. Baker support a finding that the chronic bronchitis, the moderate restrictive impairment due to pneumoconiosis and the fibrosis contribute to Mr. Holbrook's total disability and inability to perform his previous coal mine employment. I find that the credible evidence pre-sented by Dr. Caudill and Baker, and supported by Dr. Myers, establishes that Mr. Holbrook is totally disabled due to pneumoconiosis. I reaffirm my previous award of benefits.

Attorney's Fee

Claimant's counsel has thirty days to submit an application for an attorney's fee. The application shall be prepared in strict accordance with 20 C.F.R. §§ 725.365 and 725.366. The application must be served on all parties, including the claimant, and proof of service must be filed with the application. The parties are allowed thirty days following service of the application to file objections to the fee application.

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JOSEPH E. KANE
Administrative Law Judge

NOTICE OF APPEAL RIGHTS:

Pursuant to 20 CFR § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this Decision and Order by filing a Notice of Appeal with the Benefits Review Board at Post Office Box 37601, Washington, D.C. 20013-7601. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Room N-2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.