U.S. Department of Labor

Office of Administrative Law Judges
800 K Street, NW, Suite 400-N
Washington, DC 20001-8002

(202) 693-7300
(202) 693-7365 (FAX)

Issue Date: 25 August 2005

In the Matter of:
EARL EVANS Claimant

v. Case No. 2001-BLA-00269

DIXIE PINE COAL CO.
Employer

and

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

DECISION AND ORDER ON REMAND
Denying Benefits

The case arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 et seq. (the "Act"). This matter is before me on remand from the Benefits Review Board ("BRB" or "Board").

CLAIM HISTORY

The Claimant first filed for benefits on July 24, 1978, while working as a miner. Director's Exhibit ("DX") 31: 131-4. After the Department of Labor ("DOL") found that the evidence did not establish any element of entitlement, the case proceeded to the Office of Administrative Law Judges ("OALJ") for a hearing, but Mr. Evans failed to attend. DX 3:17, DX 31; Transcript ("Tr.") at 2-3. The claim was therefore dismissed for failure to prosecute. DX 31:41. Under 20 C.F.R. § 725.465, that dismissal operated as a denial of the claim on the merits.

On October 22, 1992, the Claimant filed a second claim. DX 32:1. He reported that he had not worked in the mines since 1981. *Id*. DOL denied the claim because the proof still failed to establish any element of entitlement. DX 32:10. The Claimant did not request a hearing, but nine months later, he filed another application, which DOL treated as a request for modification. DX 32:11, 13. DOL denied modification on April 28, 1994. DX 32:18. Although the Claimant neither requested a hearing nor pursued the claim, on April 16, 1996, he filed for modification again. DX 32:20. DOL denied the request as untimely. DX 32:21. The Claimant did not pursue the matter further.

On November 19, 1997, the Claimant filed a third application. DX 33:1. DOL denied the claim on February 11, 1998. DX 33:10. The Claimant took no action on the claim for nearly fourteen (14) months, then on April 2, 1999, he sought modification. DOL denied the request on April 6, 1999, as untimely. DX 33:15.

On May 14, 1999, the Claimant filed a fourth application. DX 1. DOL repeatedly denied the application. *See* DX 15, 25, 29. This time, the Claimant requested a formal hearing,

which was held on August 20, 2002, before me. Although I issued a Decision and Order awarding benefits on December 12, 2002, the Board vacated that Decision on February 24, 2004. Following the Board's Decision, the record was reopened, at the Claimant's request, to allow the parties to submit additional medical reports. The Claimant submitted a September 2, 2004 examination by Dr. D.L. Rasmussen. The Employer submitted a May 2, 2005 medical report by Dr. James Castle. Both reports are hereby admitted into evidence. In addition, both parties filed briefs. The matter is now ripe for adjudication.

MANDATE ON REMAND

In a Decision and Order dated February 25, 2004, the Board instructed me to address the threshold issue of whether the Claimant's 1999 duplicate claim was timely filed. BRB slip op. at 4-6. If considered timely filed, I am to further consider whether the evidence of record is sufficient to establish a material change in condition. *Id*. at 7. Should I reach the merits of the claim, I am to provide separate and distinct findings with respect to the x-ray evidence at 20 C.F.R. § 718.202(a)(1), and the medical opinion evidence at 20 C.F.R. § 718.202(a)(4). *Id*. at 7-8. The negative x-ray readings by Drs. Rodgers and Pendergrass should not be characterized as "not conclusive." *Id*. at 8. I am to weigh the conflicting interpretations of the February 14, 2001 x-ray and reconsider all the x-ray evidence at 20 C.F.R. § 718.202(a)(1). *Id*. at 9-11.

In addition, I am to weigh all of the relevant medical opinions in considering the merits of Claimant's 1999 claim. *Id*. at 12. Dr. Rasmussen's opinion is not to be accorded dispositive weight. *Id*. at 12-14. Dr. Branscomb's opinion is not to be characterized as indicating that tobacco and pneumoconiosis are mutually exclusive. *Id*. at 14 n.13. The opinions of Drs. Fino and Branscomb are not to be "selectively analyzed." *Id*. at 14-15. I am to refrain from according greater weight to Dr. Rasmussen's opinion than to the contrary opinions of Drs. Fino and Branscomb on the basis that Dr. Rasmussen examined the Claimant. *Id*. at 15. I am to weigh all of the arterial blood gas study evidence at 20 C.F.R. § 718.204(b)(2)(ii) and weigh together all of the contrary probative evidence of disability, like and unlike, to determine whether the evidence is sufficient to establish total disability. *Id*. at 15-16. Dr. Fino's causation opinion is not to be discredited. *Id*. at 17. I am to consider the Claimant's entitlement to the payment of augmented benefits on behalf of his grandchild in accordance with the Administrative Procedure Act. *Id*.

BURDEN OF PROOF

"Burden of proof," as used in this setting and under the Administrative Procedure Act ("APA")[1] is that "[e]xcept as otherwise provided by statute, the proponent of a rule or order has the burden of proof." "Burden of proof" means burden of persuasion, not merely burden of production. 5 U.S.C.A. § 556(d).[2] The drafters of the APA used the term "burden of proof" to mean the burden of persuasion. ***Director, OWCP, Department of Labor v. Greenwich Collieries***

---

[1] 33 U.S.C. § 919(d) ("[N]otwithstanding any other provisions of this chapter, any hearing held under this chapter shall be conducted in accordance with [the APA]"); 5 U.S.C. § 554(c)(2). Longshore and Harbor Workers' Compensation Act ("LHWCA"), 33 U.S.C. §§ 901-950, is incorporated by reference into Part C of the Black Lung Act pursuant to 30 U.S.C. §§ 932(a).

[2] The Tenth and Eleventh Circuits held that the burden of persuasion is greater than the burden of production, ***Alabama By-Products Corp. v. Killingsworth***, 733 F.2d 1511, 6 BLR 2-59 (11th Cir. 1984); ***Kaiser Steel Corp. v. Director, OWCP*** [*Sainz*], 748 F.2d 1426, 7 BLR 2-84 (10th Cir. 1984). These cases arose in the context where an interim presumption is triggered, and the burden of proof shifted from a claimant to an employer/carrier.

[Ondecko], 512 U.S. 267, 114 S.Ct. 2251 (1994).[3]

MEDICAL EVIDENCE[4]

X-Ray Reports

A February 15, 1999 x-ray was interpreted as positive for pneumoconiosis by Dr. Robinette.[5] DX 33:14. However, Drs. Spitz, Wiot, and Perme all read the film as negative; Drs. Spitz and Perme are B-readers and board certified radiologists; Dr. Wiot is a board certified radiologist.[6] EX 1-3.

A May 26, 1999 x-ray was interpreted as negative for pneumoconiosis by Dr. Cohen, who is board certified in radiology; Dr. Sargent, who is a B-reader and board certified in radiology; and Dr. Dahhan, who is a B-reader. DX 11:12, 23.

A December 3, 1999 x-ray was also interpreted as negative for pneumoconiosis by Dr. Perme, a B-reader and board certified radiologist; Dr. Dahhan, a B-reader; Dr. Wiot, a board certified radiologist;[7] and Dr. Spitz, a B-reader and board certified radiologist. DX 12, 23, 25.

A February 14, 2001 x-ray was taken and read as positive for simple coal workers' pneumoconiosis by Dr. Patel, who is a B-reader and board certified in radiology (*See* Dr. Patel's *curriculum vitae*, CX 1). However, Drs. Spitz, Wiot, and Perme all read the film as negative; Drs. Spitz and Perme are B-readers and board certified radiologists; Dr. Wiot is a board certified radiologist.[8] EX 1, 4, 7.

A September 2, 2004 chest x-ray was taken and interpreted again by Dr. Patel, a B-reader and board certified radiologist; however, this time, he found no classifiable pneumoconiosis, bilateral basal scarring. Dr. Castle, a B-reader, agreed.

Numerous re-readings of prior films were submitted into the record in the instant claim, and are set forth in chart form on pages 4-6 of my prior Decision and Order. None of these films were read as positive for pneumoconiosis.

Pulmonary Function Tests

Pulmonary function tests ("PFTs") were taken on February 5, 1999, May 26, 1999, June 30, 1999, December 3, 1999, February 14, 2001, and September 2, 2004. DX 33:14; DX 4; DX 7; DX 23. The first four studies were each invalidated by Drs. Fino, Branscomb and Michos. DX 33: 8, EX 8, EX 9. The February, 2001 study was valid but did not satisfy DOL's disability criteria. CX 1; ALJ slip op. at 21. The final study, conducted in September, 2004, produced qualifying values, and according to Dr. Rasmussen, revealed a "severe reversible obstruction."

---

[3] Also known as the risk of nonpersuasion, *see* 9 J. Wigmore, ***Evidence*** § 2486 (J. Chadbourn rev.1981).

[4] All of the medical evidence in this case, with the exception of the two medical reports submitted post-remand, was summarized in my December 12, 2002 Decision and Order, and is fully incorporated into this Decision and Order. However, to the extent appropriate, I have re-summarized the medical evidence below.

[5] Although Dr. Robinette lists his qualifications on the report as a B-reader, Dr. Robinette's *curriculum vitae* is not of record.

[6] Dr. Wiot lists his qualification on the report as a B-reader, but this is not indicated on his *curriculum vitae*.

[7] Dr. Wiot lists his qualification on the report as a B-reader, but this is not indicated on his *curriculum vitae*.

[8] Dr. Wiot lists his qualification on the report as a B-reader, but this is not indicated on his *curriculum vitae*.

Arterial Blood Gas Studies

Five arterial blood gas studies were conducted during the same period. The February 2001 test satisfied DOL's disability guidelines (CX 1), and blood gases in the September 2004 test were normal at rest but showed a marked impairment in gas exchange with exercise.

Medical Opinions

1. Dr. M. Kabir

The Claimant's treating physician, Dr. Humayun M. Kabir, had been treating Mr. Evans from May, 1997 through June, 2002. CX 2. Since the early 1980's, Mr. Evans had complained to Dr. Kabir of problems breathing, exacerbated by exertion, but present even when sleeping. Over time, Dr. Kabir diagnosed the Claimant as having a combination of medical impairments, including heart disease, chronic obstructive pulmonary disease and pneumoconiosis. With other medications, Mr. Evans tried courses of prednisone, a steroid. Sleep apnea had been considered.

By June, 2002, the Claimant reported progressive increased shortness of breath. According to the reports (CX 2), he had severe chronic obstructive pulmonary disease with a "good" reversible component. This was documented in a January, 1999 pulmonary function test where the FEV1 was noted as "only" 39% of the predicted amount and was noted to have improved by 65% after administration of a bronchodilator. He was initially prescribed bronchodilators, such as oral Theophylline, oral Singulair and an Aerobid inhaler. For a time, the Claimant improved, but he regressed. As of the last office visit to Dr. Kabir, in June, 2002, the Claimant had been taking Theodur, a Serevent inhaler, with Albuterol and Atrovent. His symptoms were reported as "not under control rather progressively going downhill." He had both daytime as well as nocturnal symptoms. He could do his day to day work but gets out of breath easily. He had some cough which is nonproductive. At night he would wake up once or twice with some shortness of breath. On examination, bilateral rhonchi were noted, but the chest appeared to be soft, but not tender, non-distended, without signs of organomegaly. No clubbing, edema or cyanosis was noted. Dr. Kabir found:

1. Uncontrolled chronic obstructive pulmonary disease
2. Severe obstructive airway disease with significant reversible component
3. Coronary artery disease
4. Coal workers pneumoconiosis

Dr. Kabir recommended a repeat pulmonary function test. Anti-inflammatory therapy was prescribed, in addition to the bronchodilators. Dr. Kabir stated: "The Claimant did not want to take "to (sic) many pills. I will review his pulmonary function test and will talk to him again about the maintenance medications."

Apparently, the only x-ray relied upon by Dr. Kabir was one performed in 1997 (CX 2, *see* notation dated November 11, 1999). The test referred to above was probably the testing performed by Emory Robinette, M.D., who examined the Claimant on February 25, 1999 (*see* below).

2. Dr. E. Robinette

Dr. Emory Robinette examined the Claimant on February 25, 1999. DX 26/DX 33:14. He recounted Mr. Evans' mining history and clinical symptoms, including shortness of breath, cough and dyspnea. The Claimant reported to Dr. Robinette that he had quit smoking fifteen (15) years prior to the examination. Dr. Robinette assumed that the Claimant had a 15 pack year smoking history that had ended about 1984. DX 26/DX 33:14. Mr. Evans measured 5'6" and weighed 181 pounds. His chest revealed diminished breath sounds with bilateral expiratory

wheezes. A chest x-ray showed hyperinflation, emphysema and scattered opacities. PFTs invalidated on expert review, showed reduced flow rates and decreased FVC. Lung volumes were normal, but diffusing capacity was 50% of predicted. According to Dr. Robinette, these findings were consistent with moderate obstructive lung disease, which both responded and did not respond to bronchodilator therapy.

Dr. Robinette's diagnoses included:

1. Simple coal worker s pneumoconiosis
2. Moderate obstructive lung disease with response to bronchodilator therapy
3. Moderate impairment of the diffusion capacity consistent with underlying emphysema and dust reticulation
4. ASCVD with a history of a previous myocardial infarction
5. Prostate asymmetry with a nodular density in the superior aspect of the left prostate lobe

According to Dr. Robinette, Mr. Evans complained about increasing shortness of breath and dyspnea. Dr. Robinette assumed a "substantial dust exposure" from 23 years of coal mine employment. Evidence of diminished breath sounds in both lung fields with bilateral expiratory wheezes present was reported:

> There was moderate prolongation of the expiratory phase ... X-rays were felt to be consistent with dust reticulation with a profusion abnormality of 1/0, predominant S/T abnormalities. There was bilateral apical pleural scarring present and emphysema present. Functionally, there is evidence of airflow obstruction with an FEV1 of 1.67 or 59% of predicted and an FVC of 3.86 or 97% of predicted. There was response to bronchodilator therapy. The diffusion capacity was reduced at 59% of predicted. These findings would suggest that Mr. Evans does have evidence of interstitial pulmonary process that is associated with airflow obstruction. Although is [sic] response to bronchodilator therapy there is is little historical evidence to suggest a clinical diagnosis of asthma. Obvious [sic], Mr. Evans probably needs a bronchodilator on a regular basis. Based on his pulmonary reserve with an FEV1 of 1.67 or 59% of predicted, Mr. Evans is disabled from working as an underground coal miner. In view of his minimal smoking history, I feel the majority of his pulmonary impairment has occurred as a consequence of his prior coal dust exposure and his radiographic abnormalities. Certainly, this is contributing to his impairment of the diffusion capacity and airflow obstruction.

DX 26/DX 33:14.

3. Dr. L. Seargeant

Dr. Lee Seargeant examined Mr. Evans on May 26, 1999, and completed a standard DOL pneumoconiosis form. DX 9. Although Dr. Seargeant found that Mr. Evans had chronic obstructive pulmonary disease, he did not find that pneumoconiosis was present. DX 4, DX 9, DX 10. X-rays taken that day were read by Thomas Cohen and J. Nicholas Sargent, M.D. for the Department of Labor. DX 11 and DX 12. Neither reader found any evidence of pneumoconiosis.

4. Dr. D.L. Rasmussen

The Claimant was examined by D.L. Rasmussen, M.D. on February 14, 2001. CX 1. Mr. Evans told Dr. Rasmussen that he began to work in small truck mines in the 1950's; then:

> He shot and loaded coal for about 8 [or more] years. He was then employed between

> 1960 and 1984 regularly. He worked as a hand loader, shot firer, pulled coal by ponies, rock dusted. He worked underground a total of 8+ years. He then worked 14-15 years on surface mining primarily as a drill operator. His last job was that of general laborer on surface mining. He used jack hammers. He loaded holes with 50 pound bags of explosives, some 10-15 per hole. He had to carry some 60 feet. He shoveled holes. He pushed equipment and dragged lines. Thus, he did considerable heavy manual labor. He states he has a total of 25 years of coal mine employment and has been given credit for 23 years.

The Claimant informed Dr. Rasmussen that he first began to smoke regularly at age twenty five (25) in 1961, and that he smoked an average of one (1) pack of cigarettes a week, until he quit smoking cigarettes in 1986. He admitted that he continues to smoke a pipe occasionally. Mr. Evans told Dr. Rasmussen that his father had emphysema, black lung, and a stroke. No history of hypertension, heart disease, tuberculosis, diabetes, cancer, asthma, or allergies was reported. Mr. Evans told Dr. Rasmussen that he had become progressively short of breath over the past eight or nine years, and that he had a heart attack in 1997.

On physical examination, breath sounds were markedly reduced with prolonged expiratory phase and wheezing on forced expiration, but without rales or rhonchi.

Dr. Rasmussen relied on a positive chest x-ray interpretation, dated February 15, 2001, by Dr. Manu N. Patel, a B-reader and board certified radiologist. Dr. Patel read the x-ray as indicating pneumoconiosis s/s with a profusion of 1/1 throughout all lung zones.[9]

Pulmonary function studies revealed severe, partially reversible obstructive insufficiency. Maximum breathing capacity was noted as "markedly reduced. Single breath carbon monoxide diffusing capacity markedly reduced. There was minimal resting hypoxia." Dr. Rasmussen noted that Mr. Evans underwent an incremental treadmill exercise study:

> [B]eginning at 1.5 mph at a 0% grade. This level was maintained for 3 minutes. Thereafter, the grade of the treadmill was increased 2% per minute. He exercised for 6 minutes and reached a maximum of 1.5 mph at a 6% grade. He achieved an oxygen uptake of only 11.3 cc/kg/mm. which is 38% of his predicted maximum oxygen uptake. He denied chest pain. His EKG and blood pressure responses were normal. He did not exceed his anaerobic threshold at this light exercise level. Heart rate was at least minimally excessive. Volume of ventilation was markedly increased. He retained a breathing reserve of only 27 L/min. There was significant increase in VD/VT ratio and arterial to end tidal PC~2 of +5.9. There was marked impairment in oxygen transfer. He was at least minimally hypoxic.

Id.

Dr. Rasmussen noted that an electrocardiogram was consistent with a previous heart attack.

Dr. Rasmussen determined that "coal mine dust exposure" must be considered a "major contributing factor" to the respiratory insufficiency. He concluded that the studies indicate marked loss of respiratory function as reflected by the significant ventilatory impairment, the marked reduction in diffusing capacity, and the marked impairment in oxygen transfer during very light exercise. Dr. Rasmussen opined that this degree of impairment would render the

---

[9] I note that the x-ray read as positive by Dr. Patel was read as negative by Drs. Spitz, Wiot, and Perme. EX 1, 4, 7. Drs. Spitz and Perme are B-readers and board certified radiologists; Dr. Wiot is a board certified radiologist.

Claimant totally disabled from resuming his last regular coal mine job with its attendant requirement for [sic] heavy manual labor. He opined: "The patient has a significant history of exposure to coal mine dust. He has x-ray changes consistent with pneumoconiosis. It is medically reasonable to conclude that the patient has coal workers' pneumoconiosis." CX 1.

With respect to the cause of the Claimant's impairment, Dr. Rasmussen noted that there were three risk factors: a history of cigarette smoking; a history suggestive of hyperactive airways disease (asthma); and coal mine dust exposure. He determined, though, that the bulk of the Claimant's impairment:

> [Was] secondary to his occupational dust exposure with resultant pneumoconiosis. Any component of asthma would not produce a marked reduction in single breath carbon monoxide diffusing capacity and would not be expected to produce marked remodeling of the airways. Both cigarette smoking and coal mine dust exposure could produce ventilatory impairment and impairment in oxygen transfer.

CX 1:3.

On September 2, 2004, the Claimant returned to Dr. Rasmussen for another examination, as I allowed the parties to submit additional medical reports after the Board's February 25, 2004 Decision. The Claimant was sixty seven (67) years of age at the time of that examination. Dr. Rasmussen noted that the Claimant had first experienced shortness of breath with exertion some twenty (20) or more years ago. He was currently "quite short-winded bending over." The Claimant was out of breath after climbing a flight of stairs; however, he denied chronic cough or sputum production. The Claimant wheezes in the morning and with exertion and he "smothers down" when he is exposed to hair sprays, perfumes and other strong odors. The Claimant sleeps on two (2) pillows. He sometimes wakens and has to go out on the porch for breathing. He denies ankle swelling, hemoptysis, respiratory illness, or any known cardiovascular illness.

Dr. Rasmussen noted that the Claimant began to smoke regularly at age sixteen (16) in 1952, and smoked about ½ pack of cigarettes a day until he quit smoking cigarettes in 1984. Thereafter, to the present, he smoked a pipe occasionally. He was on Advair, Combvient, Singulair, Theophylline, Albuterol by nebulizer and Atrovent inhaler. Dr. Rasmussen noted that the Claimant's father had had emphysema and black lung. There was no family history of hypertension, heart disease, tuberculosis, diabetes, cancer, asthma, allergies or strokes.

The Claimant had been employed in the coal mining industry between about 1962 and 1984, for a total of about 19+ years. He was first a hand loader then loading machine operator, cutting machine operator, shuttle car operator. He worked underground for eight (8) years. Then he worked on surface mining operating a high wall drill for about six (6) years. He operated a jackhammer. He cleaned coal with a mechanical broom. He loaded holes for making shots. His last job was that of general laborer on surface [mines]. He helped load holes carrying 50# explosives some 50 feet. He shoveled to fill up the holes. He also operated a jackhammer at times. Thus, he did considerable heavy and some very heavy manual labor.

The Claimant was 65 ¾ inches tall and 185 pounds at the time of the physical examination. His B/P was 145/81; his heart rate was 56; respirations 20. He had no abnormalities of eyes, ears, nose or pharynx. Neck veins flat. Carotid arteries are palpable. No bruits were heard. No adenopathy or thyroid enlargement. The Claimant had increased AP diameter due partly to a dorsal kyphosis. Chest expansion seemed reduced. Breath sounds were very markedly reduced. There were rare rales. There was prolonged expiratory phase and expiratory wheezing with forced expirations. Heart tones are quite reduced. Rhythm regular. No murmurs, gallops or clicks were heard. Abdomen is soft, non-tender. No liver, spleen or

other organ or masses were felt. Rectal deferred. Peripheral pulses are intact. No edema. No clubbing. Deep tendon reflexes physiologic. No cervical, axillary, epitrochlear or inguinal adenopathy. No gross skin lesions.

A September 2, 2004 chest x-ray interpreted by Dr. Manu Patel, a board-certified radiologist and B-reader, indicated no classifiable pneumoconiosis, bilateral basal scarring.[10]

Electrocardiogram revealed sinus bradycardia and nonspecific ST-T wave changes.

Ventilatory function studies revealed severe, significantly reversible obstructive ventilatory impairment. The singe breath carbon monoxide diffusing capacity was markedly reduced.

Blood gases were normal at rest, but showed a marked impairment in gas exchange with exercise.

Dr. Rasmussen noted that Mr. Evans underwent an incremental treadmill exercise study:

> [B]eginning at 1.5 mph at 0% grade. This level was maintained for 3 minutes, at which time arterial sample was obtained. The patient achieved an oxygen uptake of only 10.4 cc/kg/min., which was 41% of his predicted maximum oxygen uptake. He denied chest pain. His EKG and blood pressure responses were normal. His anaerobic threshold was not identified. His heart rate was somewhat excessive at 58% of predicted maximum. His volume of ventilation was markedly increased and he retained a breathing reserve of only 25 liters. (Ventilatory limitation is reached within liters of breathing reserve.) There was marked increase in VD/VT ratio and marked impairment in oxygen transfer and he was at least minimally hypoxic.

Dr. Rasmussen noted that these studies indicate loss of lung function as reflected by his moderate post-bronchodilator ventilatory impairment, but mainly by his marked impairment in gas exchange during light exercise. He achieved an oxygen uptake of only 10.4 cc/kg/min. His previous coal mine employment required oxygen consumption of 20-30 cc/kg/min.

The Claimant has a significant history of exposure to coal mine dust. However, on this occasion he had no x-ray changes consistent with pneumoconiosis. He previously had an x-ray interpreted by Dr. Patel as showing pneumoconiosis s/s with a profusion of 1/1 throughout all lung zones on 2/1/2001.

Dr. Rasmussen stated that there are three apparent risk factors for this patient's disabling lung disease. These include his significant cigarette smoking, his coal mine dust exposure and asthma. Asthma is not known to be caused by coal mine dust exposure, however, asthma or hyperactive airways disease renders one more susceptible to the adverse effects of environmental exposures. Dr. Rasmussen cited medical literature to substantiate this assertion. Cigarette smoke and coal mine dust exposure both cause chronic obstructive lung disease including emphysema and bronchitis and small airways disease. Dr. Rasmussen cited medical literature to substantiate this assertion. Coal mine dust also causes diffuse interstitial fibrosis and coal miners often exhibit impairment in oxygen transfer absent or exceeding the degree of airways obstruction. Dr. Rasmussen cited medical literature to substantiate this assertion. Coal mine dust can cause loss of lung function absent x-ray changes of pneumoconiosis. Dr. Rasmussen cited medical literature to substantiate this assertion. In addition, the x-ray may fail to reveal significant pneumoconiosis when present. Dr. Rasmussen cited medical literature to substantiate this assertion. Dr. Rasmussen concluded that Mr. Evans' coal mine dust exposure is a major contributing factor to his disabling chronic lung disease. He cited medical literature to substantiate this assertion.

---

[10] Dr. Castle, a B-reader, agreed.

5. Dr. A. Dahhan

The Claimant was examined Dr. A. Dahhan on December 3, 1999. DX 23. The Claimant was sixty-three years old at the time. He informed Dr. Dahhan that he had worked as a miner for twenty (20) years, and smoked one (1) package of cigarettes per day from age twenty (20) to age forty-eight (48). The Claimant still smoked pipe tobacco at the time of the examination. The history reported by Dr. Dahhan noted frequent wheezing, treatment with two bronchodilators, and occasional non-exertional chest pain.

Physical examination revealed good air entry into both lungs with a few scattered expiratory wheezes. Resting blood gases showed minimal hypoxia, and exercise values were normal. A pulmonary function test was attempted, but the Claimant showed less than optimum effort. The study revealed normal FVC and a "mild" reduction in FEV1 on tests that were considered as sub-optimal with invalid components. A chest x-ray showed hyper-inflated lungs with emphysema, but not pneumoconiosis.

Dr. Dahhan rendered a diagnosis of mild chronic obstructive pulmonary disease but with no dust related disease or pneumoconiosis. Rather, Dr. Dahhan opined that the Claimant's mild obstructive lung disease was due to cigarette smoking. Notwithstanding his obstructive impairment, Dr. Dahhan found that the Claimant remained capable of performing his prior coal mine work or any other job of similar physical demand. DX 23.

6. Dr. G. Fino

Dr. Gregory Fino, board certified in internal medicine, subspecialty in pulmonary disease, performed a review of the Claimant's medical records and provided a medical opinion. EX 8. He concluded that the Claimant did not suffer from an "occupationally acquired pulmonary condition as a result of coal mine dust exposure." Dr. Fino stated that he based this conclusion on the following findings: 1) The majority of chest x-ray readings are negative for pneumoconiosis; 2) The acceptable spirometric evaluations are normal with no obstruction, restriction, or ventilatory impairment; 3) The diffusing capacity values are normal. A normal diffusing capacity rules out the presence of clinically significant pulmonary fibrosis … pneumoconiosis is an example of a pulmonary fibrosis; 4) Lung volumes are used to "look" at the consistency of lung tissue … they are a measure of whether the lung is of normal consistency, whether it is over-inflated, or whether it is under-inflated. Over-inflated conditions are due to obstructive lung disease. Under-inflated conditions are due to contraction due to fibrotic scarring as is seen in pulmonary fibrosis. The Claimant has normal lung volumes. There is no over-distention or over-inflation consistent with an obstructive condition nor is there any evidence of under-inflation due to fibrosis of which pneumoconiosis is an example; 5) There is no impairment in oxygen transfer as the Claimant does not become hypoxic with exercise.

Dr. Fino made the following conclusions: 1) There is insufficient objective medical evidence to justify a diagnosis of simple coal workers' pneumoconiosis; 2) The Claimant does not suffer from an occupationally acquired pulmonary condition; 3) There is no respiratory impairment present; 4) From a respiratory standpoint, the Claimant is neither partially nor totally disabled from returning to his last coal mining job or a job requiring similar effort; 5) Even assuming the Claimant did have medical or legal pneumoconiosis, it has not contributed to his disability. He would be disabled had he never stepped foot in the mines.

7. Dr. B. Branscomb

Dr. Benjamin Branscomb reviewed the Claimant's medical records. EX 8. He determined that there was no objective evidence or reasonable medical basis documented in the record to diagnose pneumoconiosis, nor any other condition or impairment caused or aggravated by CWP [i.e. coal workers' pneumoconiosis] or by exposure to coal mine dust. Mr. Evans may be disabled by heart disease and other non-pulmonary problems although the extent of such a disability is not documented. He probably does have mild COPD. If he has it [, it] is neither caused nor aggravated by coal mine dust. It is caused by cigarette smoking in a susceptible subject. Dr. Branscomb stated: "Taken in whole, and bearing in mind the smoking history obtained in a non-compression setting and also my comments concerning cigar and pipe smoke there is ample indication of a sufficient tobacco exposure to result in mild (or even severe) COPD. There is an insufficient objective basis for concluding any COPD is more than mild. There is no pulmonary impairment sufficient to prevent his previous coal mine jobs or similar work demands."

Some fourteen (14) attempts at simple pulmonary function studies have been made without obtaining a valid test. There is no valid objective medical evidence of any pulmonary impairment of any etiology. I am cognizant of the obstructive manifestations which have been associated with coal mine dust and with CWP. Mr. Evans' diagnosed symptoms and findings are not consistent with coal dust related obstructive impairment. He has no illness or impairment caused or significantly aggravated by coal mine dust. EX 8.

8. Dr. J. Castle

On May 2, 2005, Dr. James Castle, board certified in internal medicine and pulmonary disease, reviewed the Claimant's medical records, including Dr. Rasmussen's most recent examination and tests. Based on a "thorough review of all the data, including medical histories, physical examinations, radiographic evaluations, physiologic testing, arterial blood gas studies, and other data," Dr. Castle opined that the Claimant does not suffer from coal workers' pneumoconiosis. Dr. Castle admitted that the Claimant "certainly worked in or around the underground mining industry for a sufficient enough time to have developed coal workers' pneumoconiosis if he were a susceptible host." In that regard, Dr. Castle noted that the Claimant had worked for approximately twenty (20) years in the mining industry and spent approximately eight (8) years working underground. He had last worked in the mining industry in1984, and his last job was that of general laborer on a surface mine. The Claimant had had no coal mine dust exposure since that time.

Dr. Castle stated that another risk factor for the development of pulmonary disease is that of tobacco abuse. According to Dr. Castle, Mr. Evans gave a "remarkably variable history of tobacco abuse." On some occasions, he indicated that he had never been a cigarette smoker. Moreover, during a non-disability evaluation, he was noted to have a 30 or 40 pack year smoking history. On the occasion of Dr. Dahhan's evaluation in 1999, he was noted [to] have a 28 pack-year smoking history. Dr. Castle wrote: "It is also important to note that [the Claimant] did not stop smoking, but only switched to a pipe which he continued to smoke at the time of Dr. Rasmussen's evaluation on September 2, 2004." The Claimant's tobacco smoking history was extensive and certainly significant enough to have caused him to develop chronic obstructive pulmonary disease, chronic bronchitis/emphysema and/or lung cancer and/or atherosclerotic cardiovascular disease.

Dr. Castle noted that another risk factor for the development of pulmonary symptoms is that of bronchial asthma. In that regard, Dr. Castle stated that Claimant was noted as having a

significant history of paroxysmal wheezing associated with exposure to various hair sprays, perfumes, and other strong odors. The Claimant's treating physicians had noted that he has hyperactive airways disease responsive to steroid or anti-inflammatory therapy. When valid pulmonary function studies were performed, the Claimant had demonstrated very markedly reversible airway obstruction indicating the presence of bronchial asthma. According to Dr. Castle, the Claimant had findings entirely consistent with bronchial asthma.

Dr. Castle stated that another risk factor for the development of pulmonary symptoms is that of coronary artery disease. In that regard, Dr. Castle noted that the Claimant had an abnormal electrocardiogram as well as a history of previous myocardial infarction, which can result in significant shortness of breath. The Claimant was also "suspected of having obstructive sleep apnea syndrome," though Dr. Castle noted this had not been documented.

Dr. Castle pointed out that the Claimant had not demonstrated the consistent physical findings of an interstitial pulmonary process in that he did not have the consistent findings of rales, crackles, or crepitations. Dr. Castle noted that the Claimant had "intermittently demonstrated wheezing and occasional rhonchi consistent with a diagnosis of bronchial asthma."

Dr. Castle read the September 2004 x-ray film as negative for pneumoconiosis. This, Dr. Castle recognized, was consistent with the interpretations of the vast majority of B-readers and board certified radiologists. According to Dr. Castle's report, "the vast majority of radiologists and B-readers felt that there was no evidence of coal workers' pneumoconiosis radiographically." In that regard, Dr. Castle noted that Dr. Patel had "also recently indicated that the Claimant had 'no classifiable pneumoconiosis.'" Dr. Castle stated that x-rays interpreted as positive had "generally showed evidence of linear, irregular type opacities," which according to Dr. Castle are "not the typical finding of coal workers' pneumoconiosis." By contrast, he stated that coal workers' pneumoconiosis causes the presence of small, round, regular opacities in the upper lung zones classified as p, q, or r, which had not been the finding in this case. Dr. Castle opined that there were no changes of coal workers' pneumoconiosis radiographically.

With regard to pulmonary function testing, Dr. Castle stated that most of the studies conducted in the past were invalid due to lack of tracings or less than maximal effort. Dr. Castle noted that when the Claimant was examined by Dr. Robinette in 1999, valid pulmonary function studies showed evidence of moderate airway obstruction with a very significant degree of reversibility consistent with bronchial asthma. Dr. Castle also noted that a reduction in diffusing capacity did indicate some degree of pulmonary emphysema due to tobacco smoking. Dr. Castle stated that the recent valid studies obtained by Dr. Rasmussen "clearly showed evidence of moderately severe airway obstruction with a very marked degree of reversibility after bronchodilators." In that regard, the FEV1 had improved by 39% from 1.14 l. to 1.59 l., which "clearly indicates the presence of bronchial asthma."

Dr. Castle opined that, while there had been some decline in the Claimant's FEV1 since 1999, this decline was unrelated to coal workers' pneumoconiosis or coal mine dust exposure. Instead, he believed that the change was clearly due to the Claimant's ongoing tobacco smoking habit as well as a progression of his bronchial asthma. Dr. Castle stated that the Claimant had not demonstrated a worsening in his condition due to a coal mine dust induced lung disease. He opined that the reduction in diffusing capacity experienced by the Claimant was due to tobacco smoke induced pulmonary emphysema, since coal workers' pneumoconiosis "does not generally cause a reduction in the diffusing capacity," and when it does cause a reduction in the diffusing capacity, "it is associated with a high degree of profusion of either p or r type opacities on the chest x-ray." Dr. Castle noted that the latter had not been the finding in this case at any time.

Finally, he stated that when coal workers' pneumoconiosis causes impairment, it generally does so by causing a mixed, irreversible obstructive and restrictive ventilatory defect, which had not been the finding in this case at any time.

Dr. Castle observed that the recent arterial blood gas studies obtained by Dr. Rasmussen demonstrated a mild degree of hypoxemia associated with exercise, which had occurred since 1998. He noted, however, that this occurred "in the presence of [the Claimant's] continued smoking and in the absence of coal mine dust exposure." Therefore, he opined that this finding was due to tobacco smoke induced pulmonary emphysema and loss of defective surface area for gas transfer due to that process.

For the foregoing reasons, Dr. Castle opined that the Claimant did not suffer from coal workers' pneumoconiosis. While he noted that the Claimant had had an adequate enough history and exposure to coal mine dust to have developed coal workers' pneumoconiosis, he believed that the Claimant did not have the physical findings, the radiographic findings, or the physiologic findings to indicate the presence of coal workers' pneumoconiosis.

Dr. Castle noted that he disagreed mostly with Dr. Rasmussen's September 2, 2004 report, though he concurred that the Claimant demonstrated evidence of bronchial asthma and tobacco smoke induced airway obstruction with some degree of pulmonary emphysema. Dr. Castle observed that while Dr. Patel had indicated the Claimant did not have evidence of "classifiable pneumoconiosis," Dr. Rasmussen nevertheless opined that coal mine dust exposure played a role in the Claimant's impairment. Dr. Castle then reemphasized that "besides the negative chest x-ray, the physiologic changes noted by Dr. Rasmussen are clearly those of markedly reversible airway obstruction due to bronchial asthma," which are "not the findings one would expect with coal workers' pneumoconiosis." He reemphasized that the "reduction in diffusing capacity is not typical of a coal mine dust induced lung disease in the absence of rather severe radiographic findings." He stated that these findings were typical of tobacco smoke induced bullous emphysema. Further, Dr. Castle stated that "[e]ven if one were to assume that Mr. Evans does have pneumoconiosis, it is playing no more than a very minimal or *de minimus* role in any respiratory impairment for the reasons stated above."

Dr. Castle opined that the Claimant was permanently and totally disabled from a pulmonary point of view to such an extent that he would be unable to return to his previous coal mine employment. However, he further opined that this disability was unrelated to coal mine dust exposure and coal workers' pneumoconiosis. Rather, he believed it to be due to tobacco smoke induced pulmonary emphysema and bronchial asthma.

Dr. Castle opined that any decline in the Claimant's physiologic function since 1998 had occurred as a result of a progression of his bronchial asthma and tobacco smoke induced airway obstruction associated with the Claimant's ongoing tobacco smoking habit. He also noted that some of the decline in the Claimant's "absolute physiological values [was] due to the aging process." Dr. Castle stated that, while coal workers' pneumoconiosis can be a progressive disease, there was "no specific evidence in this case to implicate [sic] or indicate that coal mine dust exposure [] played any role in his circumstance." Dr. Castle stated that the Claimant had "clearly continued to smoke tobacco products and he clearly ha[d] evidence of bronchial asthma." Moreover, the fact that he has such a marked degree of reversibility with bronchodilator administration demonstrates that he was not well-controlled with regard to his bronchial asthma. Dr. Castle believed it "entirely possible that with appropriate and aggressive therapy, [the Claimant's] ventilatory function may be significantly improved."

Finally, Dr. Castle stated that, "although pneumoconiosis may be a latent and progressive

disease, the evidence in this case clearly indicates that [the Claimant] suffers from bronchial asthma[,] which is a disease of [the] general public[,] … as well as tobacco smoke induced bullous emphysema." Dr. Castle wrote that as the Claimant had retired from mining in 1983 or 1984, it was "extraordinarily unlikely that he would have developed disabling lung disease related to his coal mining exposure some 15 or more years after leaving the mining industry." Moreover, if this had occurred, it is expected that the Claimant would have had "physiologic changes indicating the presence of that process as well as radiographic changes and physical findings." However, these were not the findings in this case.

ISSUES ON REMAND

Timeliness of Duplicate Claim

The first issue before me on remand involves the timeliness of the duplicate claim. Citing ***Tennessee Consolidated Coal Co. v. Kirk***, 264 F.3d 602, 22 BRB 2-288 (6th Cir. 2001), the Employer argued before the Board that the Claimant's duplicate claim was untimely, since the instant claim was barred by 20 C.F.R. §725.308, having not been filed within three years of a communication to the Claimant of a medical determination that he was totally disabled due to pneumoconiosis.[11] BRB slip op. at 4. The Employer argued that the medical evidence submitted in the 1992 claim rebuts the presumption of timeliness in 20 C.F.R. §725.308, since ***Kirk*** holds that the Employer need only show that a medical determination of total disability due to pneumoconiosis was communicated to the Claimant more than three years prior to the duplicate claim filing. *Id*. at 5.

To demonstrate that such a communication was made, the Employer relied on Dr. Hudson's February 19, 1993 report wherein he diagnosed chronic obstructive bronchitis related to smoking and coal dust exposure and opined that the Claimant was "disabled for mining." *Id*. at 5-6 citing DX 32[:6]. In assessing the extent to which the diagnosed condition contributed to the Claimant's impairment, Dr. Hudson stated: "[c]an't partition amount from smoking [and] mining." *Id*. at 6 citing DX 32[:6]. Notwithstanding that the prior evidence favorable to the Claimant was not credited, the Employer argued that Dr. Hudson's February 19, 1993 opinion still qualifies as a communication to the Claimant of a medical determination of total disability due to pneumoconiosis. To that end, the Employer contended that under Section 725.308, the clock begins to run the first time that a miner is told that he is totally disabled by pneumoconiosis, even if the diagnosis is found to be unsupported by the weight of the medical evidence.

On remand, I am to make a factual determination as to whether Dr. Hudson's February 19, 1993 opinion is sufficient to rebut the presumption that the Claimant's 1999 duplicate claim was timely filed. In other words, I must determine whether the opinion qualifies as a communication to the Claimant of a medical determination of total disability due to pneumoconiosis. Were it to qualify, the Claimant's 1999 duplicate claim would be untimely. However, I find that it does not so qualify. While Dr. Hudson did state that the Claimant was totally disabled (*i.e.* "disabled for mining"), he was vague as to whether the total disability was caused by pneumoconiosis. In that regard, he simply stated: "[c]an't partition amount from smoking [and] mining." DX 32:6. In describing the etiology of the cardiopulmonary diagnosis, he also stated: "Smoking – on the light side of exposure to cause this much impairment but feasible. Coal dust exposure – adequate duration of exposure [] evidence of CWP." DX 32:6.

[11] The Director argued that the Employer had waived its right to raise the issue of timeliness, however, the Board rejected this argument. *See* BRB Slip op. at 4-5.

These phrases, which represent the extent of Dr. Hudson's remarks regarding etiology, are equivocal. They merely show that Dr. Hudson was unable to determine with any degree of certainty the relative contributions of coal dust exposure and smoking to the Claimant's impairment. Accordingly, his medical opinion cannot qualify as a communication to the Claimant of a medical determination of total disability *due to pneumoconiosis*. Therefore, I find that the Claimant's 1999 duplicate claim was timely filed.

Material Change in Condition

The second issue before me on remand involves whether the evidence of record is sufficient to establish a material change in condition. In my December 12, 2003 Decision and Order, I found that prior to the submission of Dr. Rasmussen's report, the evidence clearly showed that the Claimant either did not suffer from pneumoconiosis, and even if he had, any respiratory problems were produced by tobacco usage. ALJ slip op. at 12-13 citing DX 33. I noted that Dr. Robinette's report was the only "new" evidence submitted by the Claimant, and that, in fact, Dr. Robinette's report was not really new as his ventilatory studies had been discredited by Dr. Fino's evaluation of them. ALJ slip op. at 13. I also found that the Department of Labor examinations did not add anything of value to the Claimant's case with regard to his proving a change of circumstances. *Id*. However, I concluded that Dr. Rasmussen's report provided "new and material" evidence, and that combined with new evidence from Dr. Kabir, it was reasonable to conclude that the new evidence differed "qualitatively" from evidence submitted with the prior claim. *Id*. Accordingly, I determined that the Claimant had established a material change in condition.[12]

On remand, I am to consider all of the new evidence, favorable and unfavorable to the Claimant, to determine whether he has proven at least one of the elements of entitlement previously adjudicated against him. As noted in the claim history of this case, the most recent determination on the merits was issued on February 11, 1998. DX 33:10. In that determination, which pertained to the Claimant's third application for black lung benefits, DOL denied the claim, concluding that Mr. Evans failed to prove any element of entitlement.[13] DX 33:10.

---

[12] In making this finding, I cited ***Coleman v. Ramey Coal Co.***, 18 B.L.R. 1-9 (1993), to establish that I had discretion to accord more weight to the results of a recent ventilatory study over those of an earlier study. ALJ slip op. at 13. I cited ***Gillespie v. Badger Coal Co.***, 7 B.L.R. 1- 839 (1985), ***Bates v. Director, OWCP***, 7 B.L.R. 1-113 (1984), and ***Kendrick v. Kentland-Elkhorn Coal Co.***, 5 B.L.R. 1-730 (1983), to establish that a medical report containing the most recent physical examination of a miner may be properly accorded greater weight as it is likely to contain a more accurate evaluation of the miner's current condition. Finally, I cited ***Wolf Creek Collieries v. Director, OWCP [Stephens],*** 298 F.3d 511 (6th Cir. 2002), to establish that the opinions of treating physicians are not "presumed" to be entitled to greater weight, but must be "properly weighed and credited."

[13] I note that DOL stated Mr. Evans had been required to prove three elements of entitlement: (1) the existence of coal workers' pneumoconiosis; (2) that the disease was caused, at least in part, by coal mine work; and (3) that the disease caused total disability. DX 33:10. However, in so doing, DOL combined two elements of entitlement into one. Specifically, DOL described the third element as the burden of proving that the disease caused total disability. However, according to the regulations, the Claimant must first prove that he is totally disabled, and then prove that his total disability was caused by coal workers' pneumoconiosis. Since DOL combined these two elements of proof into one, it is not entirely clear whether the Claimant proved total disability but failed to prove causation, or whether the Claimant failed to prove that he even suffers from a totally disabling respiratory impairment in the first place. I infer, however, from the totality of DOL's statements, that the Claimant failed to prove that he even suffers from a totally disabling respiratory impairment in the first place. Accordingly, in assessing whether the Claimant has proven a material change in condition, I will assume that he failed to prove all four elements of entitlement.

Therefore, at this stage, the Claimant may meet his burden of proving a material change in condition if he can prove such a change in condition with regard to any one of the four elements of entitlement.

I find that the Claimant has proven he suffers from a totally disabling respiratory impairment, and therefore has proven a material change in condition with regard to that element of entitlement. One manner of establishing total disability is to show a pulmonary or respiratory impairment that prevents a miner from performing his usual coal mine employment or comparable gainful employment, 30 U.S.C. § 902(f), 20 CFR § 718.204(b). Relevant to this case, the regulations provide three methods of proving total disability: (1) pulmonary function studies; (2) blood gas studies; (3) reasoned medical opinions. 20 CFR § 718.204(b). In this regard, I have examined the pulmonary function studies, blood gas studies, and medical opinions submitted since the February 11, 1998 denial, and have determined that the Claimant is able to establish a totally disabling respiratory impairment. A detailed explanation of this finding is set forth below.

As noted above, pulmonary function studies were taken on February 5, 1999, May 26, 1999, June 30, 1999, December 3, 1999, February 14, 2001, and September 2, 2004. DX 33:14; DX 4; DX 7; DX 23. The first four studies were each invalidated by Drs. Fino, Branscomb and Michos. DX 33: 8, EX 8, EX 9. The February 2001 study was valid but did not satisfy DOL's disability criteria. CX 1; ALJ slip op. at 21. The September 2004 study produced qualifying values, and according to Dr. Rasmussen, revealed a "severe reversible obstruction." As also noted above, five arterial blood gas studies were conducted during the same period. The February 2001 test satisfied DOL's disability guidelines (CX 1), and blood gases in the September 2004 test were normal at rest but showed a marked impairment in gas exchange with exercise. In sum, of the two valid PFTs, only one of them, the September 2, 2004 test, satisfied DOL's disability criteria. Moreover, while two ABG studies produced qualifying results, several more did not. According to ***Coleman v. Ramey Coal Co.***, 18 B.L.R. 1-9 (1993), I have discretion to accord more weight to the results of a recent ventilatory study over those of an earlier study, and therefore is compelling that the more recent studies demonstrate a totally disabling respiratory impairment. However, I still find that neither the PFT nor ABG evidence is persuasive enough to establish total disability.

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

An opinion may be given little weight if it is equivocal or vague. ***Griffith v. Director, OWCP***, 49 F.3d 184, 186-187 (6th Cir. 1995); *Justice* ***v. Island Creek Coal Co.***, 11 B.L.R. 1-91, 1-94 (1988); ***Parsons v. Black Diamond Coal Co.***, 7 B.L.R. 1-236, 1-239 (1984).

Furthermore, I note that the qualifications of the physicians are relevant in assessing the respective probative values to which their opinions are entitled. ***Burns v. Director, OWCP***, 7 B.L.R. 1-597, 1-599 (1984). More weight may be accorded to the conclusions of a treating physician as he or she is more likely to be familiar with the miner's condition than a physician who examines him episodically. ***Onderko v. Director, OWCP***, 14 B.L.R. 1-2, 1-6 (1989). However, a judge "is not required to accord greater weight to the opinion of a physician based solely on his status as claimant's treating physician. Rather, this is one factor which may be taken into consideration in … weighing … the medical evidence ..." ***Tedesco v. Director, OWCP***, 18 B.L.R. 1-103, 1-105 (1994).

Bearing these principles in mind, I will first turn to Dr. Kabir's medical treatment notes. Dr. Kabir's notes do not explicitly indicate that the Claimant is totally disabled. On one hand, he notes the Claimant's "progressive increased shortness of breath" and "severe chronic obstructive pulmonary disease with a 'good' reversible component." According to Dr. Kabir, this was documented in a January, 1999 pulmonary function study. On the other hand, Dr. Kabir recommended a repeat pulmonary function test, suggesting that he was still unclear as to the extent of the Claimant's respiratory disability. Dr. Kabir's diagnoses included "uncontrolled chronic obstructive pulmonary disease" and "severe obstructive airway disease with significant reversible component." I note that, while Dr. Kabir's comments seem to suggest a totally disabling respiratory impairment, he never explicitly states that the Claimant would be prevented from returning to his previous coal mine employment. That is, however, the implication. Moreover, as the Claimant's treating physician, I note that Dr. Kabir is more likely to be familiar with his condition than those physicians who examined him episodically. Thus, I take Dr. Kabir's status as the treating physician as one factor to be considered in weighing the medical evidence.

Next, I turn to Dr. Robinette's opinion. Dr. Robinette determined that the Claimant is totally disabled. He diagnosed moderate obstructive lung disease, stating that the findings suggest Mr. Evans does have evidence of an interstitial pulmonary process that is associated with airflow obstruction. Dr. Robinette opined that the Claimant was disabled from working as an underground coal miner; however, this conclusion was based on an invalidated PFT.

Next, I turn to Dr. Seargeant's opinion. Dr. Seargeant does not state whether he believed the Claimant to be totally disabled. He opined that the Claimant had chronic obstructive pulmonary disease. However, as stated in my previous Decision and Order, his examination "did not add anything of value" with regard to the issue of material change in condition as he did not state whether the Claimant suffers from a totally disabling respiratory impairment.

Next, I turn to Dr. Rasmussen's opinions. Dr. Rasmussen found the Claimant to be totally disabled. In his first opinion, Dr. Rasmussen noted that pulmonary function studies "revealed severe, partially reversible obstructive insufficiency." He concluded that the studies indicate marked loss of respiratory function as reflected by the significant ventilatory impairment, the marked reduction in diffusing capacity, and the marked impairment in oxygen transfer during very light exercise. Dr. Rasmussen opined that this degree of impairment would render the Claimant totally disabled from resuming his last regular coal mine job with its attendant requirement for [sic] heavy manual labor.

In his second opinion, Dr. Rasmussen similarly stated that "[v]entilatory function studies

revealed severe, significantly reversible obstructive ventilatory impairment. The singe breath carbon monoxide diffusing capacity was markedly reduced." He further stated that "[b]lood gases were normal at rest, but showed a marked impairment in gas exchange with exercise." Dr. Rasmussen also noted that Mr. Evans underwent an incremental treadmill exercise study:

> [B]eginning at 1.5 mph at 0% grade. This level was maintained for 3 minutes, at which time arterial sample was obtained. The patient achieved an oxygen uptake of only 10.4 cc/kg/min., which was 41% of his predicted maximum oxygen uptake. He denied chest pain. His EKG and blood pressure responses were normal. His anaerobic threshold was not identified. His heart rate was somewhat excessive at 58% of predicted maximum. His volume of ventilation was markedly increased and he retained a breathing reserve of only 25 liters. (Ventilatory limitation is reached within liters of breathing reserve.) There was marked increase in VD/VT ratio and marked impairment in oxygen transfer and he was at least minimally hypoxic.

Dr. Rasmussen noted that these studies indicate loss of lung function as reflected by the Claimant's moderate post-bronchodilator ventilatory impairment, but mainly by his marked impairment in gas exchange during light exercise. He achieved an oxygen uptake of only 10.4 cc/kg/min. His previous coal mine employment required oxygen consumption of 20-30 cc/kg/min.

Next, I turn to Dr. Dahhan's opinion. Dr. Dahhan did not find the Claimant to be totally disabled. He rendered a diagnosis of mild chronic obstructive pulmonary disease but found the Claimant capable of performing his prior coal mine work or any other job of similar physical demand. He noted that physical examination revealed good air entry into both lungs with a few scattered expiratory wheezes. Resting blood gases showed minimal hypoxia, and exercise values were normal. A pulmonary function test was attempted, but the Claimant showed less than optimum effort. The study revealed normal FVC and a "mild" reduction in FEV1 on tests that were considered as sub-optimal with invalid components.

Next, I turn to Dr. Fino's opinion. Dr. Fino did not find the Claimant to be totally disabled. He stated that there was "no respiratory impairment present" and that "from a respiratory standpoint, [the Claimant] is neither partially nor totally disabled from returning to his last coal mining job or a job requiring similar effort." He noted that the "acceptable spirometric evaluations were normal with no obstruction, restriction, or ventilatory impairment" and that the "diffusing capacity values were normal," which "rules out the presence of clinically significant pulmonary fibrosis" of which pneumoconiosis would be an example. Dr. Fino noted that the Claimant had normal lung volumes and that there was "no over-distention or over-inflation consistent with an obstructive condition" nor any evidence of under-inflation due to fibrosis of which pneumoconiosis would be an example. He also noted that there was "no impairment in oxygen transfer as the Claimant did not become hypoxic with exercise."

Next, I turn to Dr. Branscomb's opinion. Dr. Branscomb did not find the Claimant to be totally disabled. He opined that the Claimant may be disabled by heart disease and other non-pulmonary problems, although the extent of such a disability was not documented. Dr. Branscomb further stated that the Claimant probably did have mild COPD, and possibly even severe COPD, although there was an insufficient objective basis for concluding any COPD was more than mild. Moreover, there was no pulmonary impairment sufficient to prevent his previous coal mine jobs or similar work demands. Dr. Branscomb noted that "some fourteen attempts at simple pulmonary function studies" had been made without obtaining a valid test, and that there was no valid objective medical evidence of any pulmonary impairment of any

etiology.

Finally, I turn to Dr. Castle's opinion. Dr. Castle opined that the Claimant was permanently and totally disabled from a pulmonary point of view to such an extent that he would be unable to return to his previous coal mine employment. He further believed that the Claimant's disability was due to tobacco smoke induced pulmonary emphysema and bronchial asthma. In that regard, he stated that the Claimant was noted as having a significant history of paroxysmal wheezing associated with exposure to various hair sprays, perfumes, and other strong odors. The Claimant's treating physicians had noted that he has hyperactive airways disease responsive to steroid or anti-inflammatory therapy. When valid pulmonary function studies were performed, the Claimant had demonstrated very markedly reversible airway obstruction indicating the presence of bronchial asthma. Dr. Castle stated that another risk factor for the development of pulmonary symptoms is that of coronary artery disease. In that regard, Dr. Castle noted that the Claimant had an abnormal electrocardiogram as well as a history of previous myocardial infarction, which can result in significant shortness of breath. The Claimant was also "suspected of having obstructive sleep apnea syndrome," though Dr. Castle noted this had not been documented.

With regard to pulmonary function testing, Dr. Castle stated that most of the pulmonary function studies conducted in the past were invalid due to lack of tracings or less than maximal effort. Dr. Castle noted that when the Claimant was examined by Dr. Robinette in 1999, valid pulmonary function studies showed evidence of moderate airway obstruction with a very significant degree of reversibility consistent with bronchial asthma. Dr. Castle noted that a reduction in diffusing capacity did indicate some degree of pulmonary emphysema due to tobacco smoking. He stated that the recent valid studies obtained by Dr. Rasmussen "clearly showed evidence of moderately severe airway obstruction with a very marked degree of reversibility after bronchodilators." In that regard, the FEV1 had improved by 39% from 1.14 l. to 1.59 l., which "clearly indicates the presence of bronchial asthma." Dr. Castle opined that, while there had been some decline in the Claimant's FEV1 since 1999, this decline was unrelated to coal workers' pneumoconiosis or coal mine dust exposure. Instead, he believed that the change was clearly due to the Claimant's ongoing tobacco smoking habit as well as a progression of his bronchial asthma. Dr. Castle also noted that some of the decline in the Claimant's "absolute physiological values [was] due to the aging process."

Dr. Castle observed that the recent arterial blood gas studies obtained by Dr. Rasmussen demonstrated a mild degree of hypoxemia associated with exercise, which had occurred since 1998. He noted, however, that this occurred "in the presence of [the Claimant's] continued smoking and in the absence of coal mine dust exposure." Therefore, he opined that this finding was due to tobacco smoke induced pulmonary emphysema and loss of defective surface area for gas transfer due to that process.

On balance, I find that the medical opinions show that the Claimant suffers from a totally disabling respiratory impairment. Although three physicians, Drs. Dahhan, Fino, and Branscomb, conclude that the Claimant is not totally disabled, I am not ultimately persuaded by their opinions. Preliminarily, I note that both Drs. Dahhan and Branscomb admitted the Claimant suffers from mild COPD, and Dr. Branscomb went even further, stating that the Claimant may suffer from severe COPD, but that there was no objective basis for concluding any COPD was more than mild. Thus, while these two physicians declined to diagnose total disability, they nevertheless admit to at least some degree of disability. By contrast, Dr. Fino concluded that there was no respiratory impairment present at all, and that from a respiratory

standpoint, the Claimant was neither partially nor totally disabled from returning to his last coal mining job or a job requiring similar effort. As Dr. Fino was the only physician of record who found no respiratory impairment whatsoever, I find his opinion suspect despite his credential of subspecialty in pulmonary disease. Accordingly, I decline to accord it any weight.[14] I do accord some weight to the opinions of Drs. Dahhan and Branscomb, and duly note their belief that the Claimant suffered from at least mild COPD, and potentially severe COPD, in the case of Dr. Branscomb's opinion. However, I do not find their opinions as comprehensive and well-reasoned as those of some of the physicians who did diagnose a totally disabling respiratory impairment.

In that regard, I find Dr. Castle's opinion particularly thorough and well-reasoned, and note that while certainly not a controlling factor, the May 2, 2005 opinion based on a review of Dr. Rasmussens's most recent examination suggests an up-to-date knowledge of the state of the Claimant's respiratory disability. A medical report containing the most recent physical examination of the miner may be properly accorded greater weight as it is likely to contain a more accurate evaluation of the miner's current condition. ***Gillespie v. Badger Coal Co***., 7 B.L.R. 1-839 (1985). See *also* ***Bates v. Director, OWCP***, 7 B.L.R. 1-113 (1984) (more recent report of record entitled to more weight than reports dated eight years earlier); ***Kendrick v. Kentland-Elkhorn Coal Co.***, 5 B.L.R. 1-730 (1983). Dr. Castle's analysis of the Claimant's disability was logical and well-documented. He clearly explained how the testing substantiated his opinion that the Claimant was totally disabled. Also, I note that Dr. Castle is board certified in internal medicine and pulmonary disease, which lends even more credibility to his opinion. Moreover, his opinion is supported by Dr. Rasmussen's two opinions wherein a totally disabling respiratory impairment is diagnosed. Like Dr. Castle, Dr. Rasmussen also had the opportunity to provide a recent opinion, dated September 2, 2004, which suggests an up-to-date knowledge of the Claimant's respiratory disability. The fact that the physicians who provided the most recent reports agree that the Claimant is totally disabled lends credibility that this is indeed the case. The opinions of Drs. Kabir, a treating physician, and Robinette further support this finding, though I recognize that Dr. Kabir did not explicitly diagnose a totally disabling respiratory impairment, and Dr. Robinette based his conclusion, at least in part, on an invalidated PFT.

In sum, every single physician who provided a medical opinion in this case, with the exception of Dr. Fino, acknowledged at least some degree of respiratory impairment.[15] Even Dr. Branscomb, who concluded against a finding of total disability, admitted that severe COPD may be present, but that there was no objective basis for concluding that to be the case. The two physicians who provided the most recent opinions, and reviewed the most recent evidence, Drs. Castle and Rasmussen, agreed that the Claimant is totally disabled. Their opinions are substantiated by the opinions of Drs. Kabir and Robinette. Accordingly, I find that the Claimant suffers from a totally disabling respiratory impairment, and that he has therefore proven a material change in condition.

Existence of Coal Workers' Pneumoconiosis

Since I have concluded that Mr. Evans established a material change in condition, I must

---

[14] The Board instructed that Dr. Fino's opinion on causation was not to be discredited. BRB slip op. at 17. Here, however, I am discrediting Dr. Fino's opinion strictly as it relates to the element of total disability, not causation.

[15] I note that I decline to assess Dr. Seargeant's opinion with regard to the issue of total disability as he is silent on the issue.

consider all the record evidence, including evidence submitted with the previous claims, to determine whether it supports a finding of entitlement to benefits. Thus, I turn to the first element of entitlement, whether Mr. Evans is able to prove the existence of coal workers' pneumoconiosis. Pneumoconiosis is a progressive and irreversible disease. ***Woodward v. Director, OWCP***, 991 F.2d 314, 320 (6th Cir. 1993). As a general rule, therefore, more weight is given to the most recent evidence. *See* ***Mullins Coal Co. of Virginia v. Director, OWCP***, 484 U.S. 135, 151-152 (1987); ***Crace v. Kentland-Elkhorn Coal Corp.***, 109 F.3d 1163, 1167 (6th Cir. 1997); ***Stanford v. Director, OWCP***, 7 B.L.R. 1-541, 1-543 (1984); ***Tokarcik v. Consolidated Coal Co.***, 6 B.L.R. 1-666, 1-668 (1983); ***Call v. Director, OWCP***, 2 B.L.R. 1-146, 1-148-1-149 (1979). This rule is not to be mechanically applied to require that later evidence be accepted over earlier evidence. ***Woodward***, 991 F.2d at 319-320; ***Burns v. Director****, OWCP*, 7 B.L.R. 1-597, 1-600 (1984).

20 CFR § 718.202(a) (2003) provides that a finding of the existence of pneumoconiosis may be based on (1) chest x-ray; (2) biopsy or autopsy; (3) application of the presumptions described in §§ 718.304, 718.305 or 718.306; or (4) a physician exercising sound medical judgment based on objective medical evidence and supported by a reasoned medical opinion. There is no evidence that Mr. Evans has had a lung biopsy, and, of course, no autopsy has been performed. None of the presumptions apply in this case. Thus, in order to determine whether the evidence establishes the existence of pneumoconiosis, I must consider the chest x-rays and medical opinions.

The record contains a considerable amount of x-ray evidence. Specifically, from 1969 until the September 2, 2004 examination, the record contains eleven (11) x-rays, bearing forty five (45) negative readings and four (4) positive readings.[16] The four positive x-rays are all marked as 1, 0. Dr. Cole, who determined that there was pneumoconiosis on an x-ray dated August 1, 1979, is not board certified (DX 31:12). The same x-ray was read as negative by five (5) board certified radiologists, who are also certified "B" readers and by another physician who is board certified and by a seventh who is not board certified, but is a "B" reader (DX 14, DX 31:13, DX 31:14, DX 31:16, and DX 23). Likewise, Dr. Hudson, who read x-rays taken February 19, 1993 (DX 32:8) and January 21, 1998 (DX 33:7), is not board certified. Five physicians who are both board certified and are also "B" readers, and a sixth physician who is certified, read the 1993 x-ray as negative (DX 14, DX 32:9, DX 33:17, and DX 23). And four board certified "B" readers read the 1998 x-ray as negative (DX 33:8 and DX 14). The other positive x-ray was taken February 5, 1999 by Dr. Robinette, who read it as 1, 0 (DX 33:14, DX 26). Although Dr. Robinette lists his qualifications on the report as a board certified "B" reader, Dr. Robinette's *curriculum vitae* is not of record. Three (3) board certified radiologist "B" readers read the same x-ray as negative (EX 1, EX 2, EX 3) and a fourth found no pneumoconiosis, but did diagnose chronic obstructive pulmonary disease (Dr. Mullens, DX 26). Subsequent to the February 5, 1999 x-ray, Mr. Evans was given x-rays on May 26, 1999, November 18, 1999 and December 3, 1999; all readings from these were negative (DX 11:12, DX 23, EX 5, EX 6). The May 26, 1999 x-ray was read by two consultants to the Department of Labor.

---

[16] In my December 12, 2002 Decision and Order, I had classified the "0, 1 readings" of the February 19, 1993 x-ray by Dr. Rogers (DX 31:21) and Dr. Pendergrass (DX 31:17) as not conclusive. However, the Board indicated that these readings must be classified as negative in accordance with the ILO-U/C Classification. *See* BRB slip. op. at 8 citing 20 CFR § 718.102(b); ***Tackett v. Director, OWCP***, 7 BLR 1-703 (1985). Thus, these readings have been counted among the forty five (45) negative readings.

A February 14, 2001 x-ray was taken and read as positive for simple coal workers' pneumoconiosis by Dr. Patel, who is a B-reader and board certified in radiology (*See* Dr. Patel's *curriculum vitae*, CX 1). However, Drs. Spitz, Wiot, and Perme all read the film as negative. EX 1, 4, 7. Drs. Spitz and Perme are B-readers and board certified radiologists; Dr. Wiot is a board certified radiologist.[17] Greater weight may be accorded the x-ray interpretation of a dually qualified (i.e. B-reader and board-certified) physician over that of a board-certified radiologist. ***Herald v. Director, OWCP***, BRB No. 94-2354 BLA (Mar. 23, 1995)(unpublished). Drs. Patel, Spitz, and Perme are all equally qualified in that they are all B-readers and board certified radiologists. Two of those physicians, Drs. Spitz and Perme, read the film as negative while only one, Dr. Patel, read it as positive. Moreover, the negative readings by Drs. Spitz and Perme are substantiated by the reading of Dr. Wiot, who is a board certified radiologist. Accordingly, I find that the February 2001 x-ray must be resolved as negative for pneumoconiosis. Moreover, Dr. Patel, who also took and read the September 2, 2004 x-ray, found that it indicated no classifiable pneumoconiosis, bilateral basal scarring. Dr. Castle, a B-reader, agreed. This is the most recent evidence and I find it is entitled to significant weight. As a result, I find that the Claimant has failed to prove the existence of pneumoconiosis through x-ray evidence.

Although the Claimant has failed to demonstrate the existence of pneumoconiosis through x-ray evidence, he may nevertheless establish pneumoconiosis through medical opinion evidence. Thus, I turn to the medical opinions of record on this issue.

Dr. Kabir diagnosed coal workers' pneumoconiosis. Apparently, the only x-ray relied upon by Dr. Kabir was one performed in 1997 (CX 2, *see* notation dated November 11, 1999).

Dr. Robinette diagnosed simple coal workers' pneumoconiosis, noting that the Claimant's chest revealed diminished breath sounds with bilateral expiratory wheezes, and that a chest x-ray showed hyperinflation, emphysema and scattered opacities. Dr. Robinette assumed a "substantial dust exposure" from 23 years of coal mine employment. Evidence of diminished breath sounds in both lung fields with bilateral expiratory wheezes present was reported:

> There was moderate prolongation of the expiratory phase ... X-rays were felt to be consistent with dust reticulation with a profusion abnormality of 1/0, predominant S/T abnormalities. There was bilateral apical pleural scarring present and emphysema present.

DX 26/DX 33:14.

Dr. Seargeant did not find that pneumoconiosis was present. DX 4, DX 9, DX 10.

In his first report, Dr. Rasmussen found that the Claimant had x-ray changes consistent with pneumoconiosis and that it was medically reasonable to conclude that he had coal workers' pneumoconiosis. Dr. Rasmussen noted that breath sounds were markedly reduced with prolonged expiratory phase and wheezing on forced expiration, but without rales or rhonchi. He relied on a positive chest x-ray interpretation, dated February 15, 2001, by Dr. Manu N. Patel, a B-reader and board certified radiologist. Dr. Patel read the x-ray as indicating pneumoconiosis s/s with a profusion of 1/1 throughout all lung zones.[18] In his second report, Dr. Rasmussen noted that Claimant's chest expansion seemed reduced. Breath sounds were very markedly reduced. There were rare rales. There was prolonged expiratory phase and expiratory wheezing with forced expirations. He further noted that a September 2, 2004 chest x-ray interpreted by Dr.

---

[17] Dr. Wiot lists his qualification on the report as a B-reader, but this is not indicated on his *curriculum vitae*.

[18] I note that the x-ray read as positive by Dr. Patel was read as negative by Drs. Spitz, Wiot, and Perme. EX 1, 4, 7. Dr. Spitz is a B-reader and board-certified radiologist (EX 1); Drs. Wiot and Perme are B-readers (EX 4, 7).

Manu Patel, a board-certified radiologist and B-reader, indicated no classifiable pneumoconiosis, bilateral basal scarring.[19] He stated that the Claimant had a significant history of exposure to coal mine dust, however, on this occasion he had no x-ray changes consistent with pneumoconiosis. Dr. Rasmussen noted that the Claimant had previously had an x-ray interpreted by Dr. Patel as showing pneumoconiosis s/s with a profusion of 1/1 throughout all lung zones on 2/1/2001. He also stated that coal mine dust can cause loss of lung function absent x-ray changes of pneumoconiosis and cited medical literature to substantiate this assertion. Furthermore, he stated that the x-ray may fail to reveal significant pneumoconiosis when present, citing medical literature to substantiate this assertion.

Dr. Dahhan noted good air entry into both lungs with a few scattered expiratory wheezes. A chest x-ray showed hyper-inflated lungs with emphysema, but not pneumoconiosis.

Dr. Fino concluded that the Claimant did not suffer from an "occupationally acquired pulmonary condition as a result of coal mine dust exposure." He noted that a majority of the chest x-ray readings were negative for pneumoconiosis. He concluded that there was insufficient objective medical evidence to justify a diagnosis of simple coal workers' pneumoconiosis.

Dr. Branscomb determined that there was no objective evidence or reasonable medical basis documented in the record to diagnose pneumoconiosis, nor any other condition or impairment caused or aggravated by CWP [i.e. coal workers' pneumoconiosis] or by exposure to coal mine dust.

Dr. Castle determined that the Claimant does not suffer from coal workers' pneumoconiosis, even though he "certainly worked in or around the underground mining industry for a sufficient enough time to have developed coal workers' pneumoconiosis if he were a susceptible host." Dr. Castle pointed out that the Claimant had not demonstrated the consistent physical findings of an interstitial pulmonary process in that he did not have the consistent findings of rales, crackles, or crepitations. Dr. Castle noted that the Claimant had "intermittently demonstrated wheezing and occasional rhonchi consistent with a diagnosis of bronchial asthma." Dr. Castle read the September 2004 x-ray film as negative for pneumoconiosis. This, he recognized, was consistent with the interpretations of the vast majority of B-readers and board - certified radiologists. According to Dr. Castle's report, "the vast majority of radiologists and B-readers felt that there was no evidence of coal workers' pneumoconiosis radiographically." In that regard, Dr. Castle noted that Dr. Patel had "also recently indicated that the Claimant had 'no classifiable pneumoconiosis.'" Dr. Castle stated that x-rays interpreted as positive had "generally showed evidence of linear, irregular type opacities," which according to Dr. Castle are "not the typical finding of coal workers' pneumoconiosis." By contrast, he stated that coal workers' pneumoconiosis causes the presence of small, round, regular opacities in the upper lung zones classified as p, q, or r, which had not been the finding in this case. Dr. Castle opined that there were no changes of coal workers' pneumoconiosis radiographically.

On balance, I find that the medical opinions do not support a finding of pneumoconiosis. Again, I find Dr. Castle's opinion particularly thorough and well-reasoned. He noted that the most recent x-ray, taken September 2004, was negative for pneumoconiosis, which was consistent with the interpretations of the vast majority of B-readers and board certified radiologists. Moreover, he stated that x-rays interpreted as positive had "generally showed evidence of linear, irregular type opacities," which according to Dr. Castle are "not the typical finding of coal workers' pneumoconiosis." By contrast, he stated that coal workers' pneumoconiosis causes the presence of small, round, regular opacities in the upper lung zones

[19] Dr. Castle, a B-reader, agreed.

classified as p, q, or r, which had not been the finding in this case.

Dr. Castle's opinion is substantiated by the opinions of Drs. Seargant, Dahhan, Fino, and Branscomb, all of whom agree that there is no pneumoconiosis in this case. Although I do not find any of these opinions as detailed or clear as that of Dr. Castle, I nevertheless accord them some weight as supporting opinions. By contrast, none of the physicians who diagnosed pneumoconiosis in this case provided adequate analysis to back up the assertion that Mr. Evans does indeed have pneumoconiosis. Particularly damaging to the Claimant's case is the fact that Dr. Rasmussen, who diagnosed pneumoconiosis in both of his opinions, does not adequately explain why he believes the Claimant suffers from pneumoconiosis in spite of Dr. Patel's recent negative x-ray, dated September 2, 2004. In setting forth the assertions that "coal mine dust can cause loss of lung function absent x-ray changes of pneumoconiosis" and "x-ray may fail to reveal significant pneumoconiosis when present", Dr. Rasmussen does make passing references to some medical literature. However, as he only made passing references to the medical literature, and did not expound upon the relevancy of those references, his opinion is lacking in this regard. The opinions of Drs. Kabir and Robinette, the other physicians who diagnosed pneumoconiosis, were equally scant and unavailing on the issue of pneumoconiosis.

Accordingly, I find that the Claimant has failed to prove the existence of pneumoconiosis through medical opinion evidence, and has therefore failed to prove this element of entitlement altogether.

CONCLUSION

Based on the above, I find that the Claimant's 1999 duplicate claim was timely filed. I also find that the Claimant proved a material change in condition with regard to the issue of total disability. However, I further find that he failed to prove the existence of pneumoconiosis, one of the four elements of entitlement to benefits. Accordingly, his claim must be denied.

ATTORNEY'S FEES

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

ORDER

It is hereby ordered that the claim of Earl Evans is **DENIED**.

A

DANIEL F. SOLOMON
Administrative Law Judge

Notice of Appeal Rights: Pursuant to 20 C.F.R. §725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date this decision if filed with the District Director, Office of Worker's Compensation Programs, by filing a notice of appeal with the Benefits Review Board, ATTN: Clerk of the Board, Post Office Box 37601, Washington, DC 20013-7601. See 20 C.F.R. §725.478 and §725.479. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2605, 200 Constitution Avenue,

NW, Washington, DC 20210.