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Issue Date: 04 April 2005

In the Matter of:

LORENE BUCKLEN,
(widow of KERMIT BUCKLEN, deceased),
Claimant,

v.

JEWELL SMOKELESS COAL CORPORATION,
Employer,

and

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

CASE NO: 2003 BLA 109
2003 BLA 5467

DECISION AND ORDER – REJECTION OF CLAIMS

Statement of the Case

This case involves two discrete claims for benefits under the Black Lung Benefits Act. They consist of a third request for modification of the now deceased Miner's claim, governed in part by the amended regulations because it was pending on January 19, 2001, but not constrained by the evidentiary limitations in § 725.414, and a Survivor's claim governed by the amended regulations including § 725.414.[1] The Miner, Kermit Bucklen, died on June 18, 2001. The Survivor's claim was filed on October 1, 2001, by the Miner's widow, Lorene Bucklen, now the Claimant in both cases. (D-3, 9) The request for modification of the Miner's claim, alleging a mistake of fact, and accompanied by a death certificate and autopsy report, was filed by Claimant on December 10, 2001, following affirmation by the Benefits Review Board) on October 31, 2001, of Judge Levin's denial of benefits. Judge Levin found that the Miner did not have pneumoconiosis, that the presumption under § 718.305 that the Miner was totally disabled by pneumoconiosis was rebutted, and that there was no indication

[1] All references are to regulations contained in Title 20, Code of Federal Regulations, unless otherwise indicated. The Department of Labor amended the regulations implementing the Federal Coal Mine Health and Safety Act of 1969, as amended, effective on January 19, 2001, and published at 65 Fed. Reg. 80,045-80, 107 (2000)(codified at 20 CFR Parts 718, 722, 725, and 726 (2004)).

that the case was mistakenly decided by Judge Cox in 1991, or by Judge Murty in 1994.[2] Because the Miner was last employed in the coal mine industry in Virginia, the law of the United States Court of Appeals for the Fourth Circuit controls. (D-1[D-2]). *See Shupe v. Director, OWCP*, 12 B.L.R. 1-200, 1-202 (1989)(en banc).

Following an order to show cause and response, the District Director granted the request for modification and awarded benefits in a Proposed Decision and Order issued September 9, 2002, based on a finding of the existence of complicated pneumoconiosis; twenty-nine years of coal mine employment; causality under § 718.203(a)(b); and disability established under § 718.304(a).[3] (D-1) The District Director awarded benefits in the Survivor's claim in a Proposed Decision and Order issued September 11, 2002, based on a finding of complicated pneumoconiosis and twenty-nine years of coal mine employment. (D-20) The Survivor's claim is in pay status from the Black Lung Disability Trust Fund; the Miner's claim is not in pay status. (D-28, 27)

The initial filing of the Miner's claim was on November 4, 1980. It was denied on September 16, 1981, because the Miner did not prove the existence of coalworkers' pneumoconiosis and that he was totally disabled by the disease. The claim has a lengthy history, but the last request for modification was denied because the Miner had not established the existence of coal workers' pneumoconiosis, or that he was totally disabled by pneumoconiosis, and that denial was affirmed by the Benefits Review Board on October 31, 2001. (D-1)

On September 23, 2002, Employer requested a hearing in the Survivor's claim in conjunction with the request for a hearing on the proposed decision and order granting request for modification in the Miner's claim. (D-28) The claims were consolidated for hearing in Abingdon, Virginia, on July 16, 2003, but remain discrete claims subject to different regulations, and were submitted for a decision on the written record pursuant to the Procedural Order Granting Request for Decision on the Written Record and Scheduling Submission of Evidence issued by this tribunal on June 6, 2003. Both the parties offered all of their evidence as proof in both the Survivor's claim and the Miner's claim, but, because of different regulatory constraints, certain of that evidence has been limited by this tribunal as to its admissibility and probative effect.

Employer filed a motion dated July 11, 2003, requesting permission to submit additional evidence pursuant to § 725.456 in excess of the limitations contained in § 725.414, and to submit the deposition of Dr. Tomashefski scheduled on August 29, 2003. Employer contended that, because the medical evidence developed in the Miner's claim "is now

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[2] Judge Levin found the opinions of Drs. Sargent and Castle that pneumoconiosis was not present and had no role in the Miner's disability supported the finding that the presumption of total disability due to pneumoconiosis under § 718.305 had been rebutted, regardless of any weight given to Dr. Garzon's opinion, and contrary to the opinions of Dr. Rasmussen and Dr. Patel, which he discredited. Accordingly he concluded that the Miner had not established a mistake of fact or a change in conditions under § 725.310.

[3] The District Director relied on the medical opinions of Dr. Forehand, 3/23/02, and Dr. Mitchell, 4/3/02; x-ray interpretations by Dr. Chubineh of a film dated 2/28/00, and Dr. Makarewicz, a B-reader, of a film dated 11/09/99; extensive treatment records of Dr. Forehand and Dr. Mitchell from 1991 to 2001; the death certificate; Dr. Turjman's autopsy report dated 6/19/01; and the medical report of Dr. Tomashefski dated 6/13/02. (D-1).

consolidated and intermingled with the survivor's claim," there is "good cause for admitting or allowing evidence in excess of the limitations of § 725.414 to ensure due process and a full and fair adjudication of claimant's entitlement to benefits." In particular, Employer requested that "the medical reports of Drs. Crouch, Tomashefski, and Castle as well as any supplemental reports related thereto" be received as "relevant, material evidence since the evidence and allegations of record are so disparate." Employer also sought to depose Dr. Tomashefski on August 29, 2004, after Dr. Perper's medical report was filed.

In an opposition dated July 15, 2003, Claimant objected to Employer's request on the grounds, in substance, that the limitations on evidence in § 725.414 of the amended regulations are valid and may be exceeded only for good cause; that the two claims and the evidence to support them are clearly distinguishable; that the request is untimely in various respects; and that the issues of admissibility and good cause were not ripe because the Employer had not filed an Evidence Summary Form as required, an omission which precluded categorical evaluation of what evidence would be relied upon by the parties in support of their respective positions. Before this tribunal, Claimant has filed only the single medical report of Dr. Perper. Both parties subsequently filed final Evidence Summary Forms in September 2003 pursuant to the order of this tribunal dated August 7, 2003.

In the order issued August 7, 2003, this tribunal ruled that the medical reports of Dr. Castle, Dr. Crouch, and Dr. Tomashefski submitted by Employer were admissible with respect to the Miner's claim, but that only two were admissible with respect to the Survivor's claim under the constraints of § 725.414 (2003). Claimant's report by Dr. Perper was held admissible as initial or rebuttal evidence under the amended regulations, and Employer was allowed an opportunity to file rehabilitative evidence by Dr. Crouch and Dr. Tomashefski in response to Dr. Perper's critique. Employer was allowed to depose Dr. Tomashefski, but the order limited use of the deposition to rehabilitation of Dr. Tomashefski's initial report in light of Dr. Perper's criticism. Implicitly, it would elaborate Dr. Tomashefski's second report dated July 21, 2003, which was offered by Employer as rehabilitation evidence. The order ruled the deposition untimely as to the Miner's claim. By cover letter dated September 19, 2003, Employer lodged the transcript of Dr. Tomashefski's deposition taken August 29, 2003.

In her Evidence Summary Form dated August 15, 2003, designating the evidence upon which she relies with respect to her Survivor's claim under the amended regulations Claimant identified the medical report of Dr. Forehand dated March 23, 2002, and the medical report of Dr. Mitchell dated April 3, 2002, as her initial evidence under § 725.414(a)(2)(i). She identified the autopsy report of Dr. Turjman dated July 10, 2001, as her affirmative autopsy evidence, and the report of Dr. Perper dated May 10, 2003, as rebuttal evidence directed at the Employer's autopsy report by Dr. Tomashefski dated June 13, 2002. (C-1; D-10, 13) She identified the death certificate signed by Dr. Mitchell as other evidence. (D-9).

In its Evidence Summary Form dated September 18, 2003, Employer identified the medical report of Dr. Castle dated June 19, 2003, as its affirmative evidence under § 725.414(a)(3)(i), the autopsy report of Dr. Tomashefski dated June 13, 2002, the report of

Dr. Tomashefski dated July 21, 2003, as rehabilitative evidence, and the report of Dr. Crouch dated August 12, 2002, as rebuttal evidence, all admissible as such. (E-1; 4; D-13, 21). Employer also identified a substantial number of documents mostly identified with Dr. Mitchell and Dr. Forehand as records of the Miner's hospitalization for a respiratory or pulmonary or related disease, which are admissible pursuant to § 725.414(a)(4). (D-12).

After this tribunal issued its Order - Denying in Part and Granting in Part Respondent's Motion To Submit Additional Evidence on August 7, 2003, Employer requested by letter dated August 21, 2003, the deferral of any final ruling regarding the admissibility of evidence until receipt of written argument on November 3, 2003. Employer asserted that Claimant had exceeded the applicable evidentiary limits with respect to the Survivor's claim, particularly with respect to the reports by Dr. Forehand and Dr. Mitchell, and implied that Dr. Perper's report should be considered a second autopsy report. Employer also complained that because the Director failed to include a copy of Dr. Forehand's report as part of Director's Exhibit No. 1 supplied to Employer, it should be excluded as untimely in the Miner's claim and somehow exceeds the evidentiary limitations. Since the omission by the District Director appears to have been inadvertent and since no prejudice is demonstrated under the circumstances, Employer's claim is unpersuasive, and is overruled.

Issues

1) What submitted evidence is admissible under the pertinent regulations with respect to the Survivor's claim and the Miner's claim?

2) Has Claimant proved the existence of coal workers' pneumoconiosis?

3) Have the presumptions under §§ 718.305 that the Miner was totally disabled by pneumoconiosis at the time of his death, and that his death was due to pneumoconiosis been properly invoked? If invoked, have the pertinent presumptions been rebutted?

4) Has Claimant proved the existence of complicated pneumoconiosis so that the irrebuttable presumption under § 718.304 that the Miner was totally disabled due to pneumoconiosis at the time of death, and that his death was due to pneumoconiosis can be invoked?

5) If Claimant has proved the existence of pneumoconiosis, was it due in whole or in part to the Miner's coal mine employment?

6) Has Claimant proved that there has been a change in conditions or a mistake in a determination of fact with respect to the Miner's request for modification pursuant to § 725.310?

7) Has Claimant proved a change in conditions or a mistake in a determination of fact or a change in an element of entitlement previously adjudicated against the Miner with respect to the Miner's claim?

8) Has Claimant proved with respect to her Survivor's claim that coal workers' pneumoconiosis caused, or contributed to, or hastened the Miner's death?

9) Has Claimant proved that the Miner was totally disabled due to coal workers' pneumoconiosis prior to his death?

Admissibility of Evidence

The evidentiary constraints imposed by the applicable amended regulations in Parts 718 and 725 (2003) upon the evidence submitted in the Survivor's claim do not apply, for the most part, to proof related to the request for modification pertinent to the Miner's claim. However, the evidentiary record pertinent to the Miner's claim, which has been transferred from the District Director to the Office of Administrative Law Judges and admitted without objection into the record of the Miner's claim pursuant to § 725.421, is not automatically admitted, and is not necessarily admissible, with respect to the Survivor's claim. § 725.421(b)(4). The parties have identified their evidence to appropriate uses which qualify the designated exhibits for admissibility with respect to the Survivor's claim on the summary of evidence forms issued by this tribunal. The content of certain of their documentary evidence, however, is subject to additional regulatory constraints under § 725.414. Since both claims are decided on the written record, resolution of most evidentiary issues has been deferred until receipt of closing argument as Employer requested, and as is both reasonable and efficient in this case.

With respect to the Survivor's claim, §§ 724.414(2)(i) and (3)(i) permit the parties to submit, as part of their respective affirmative cases, one report of an autopsy. Claimant properly designated the autopsy report of the prosector, Dr. Turjman, as her report of an autopsy. (D-10). Employer designated the report of Dr. Tomashefski dated June 13, 2002, which analyzed the autopsy evidence as its report of an autopsy. (D-13). Claimant designated the report of Dr. Perper dated May 10, 2003, as rebuttal. (C-1). Employer designated the report of Dr. Crouch dated August 12, 2002, as rebuttal (D-21) Claimant did not designate rehabilitative evidence. Employer designated a supplemental report of Dr. Tomashefski dated July 21, 2003, as rehabilitative evidence. (E-4). Dr. Tomashefski's deposition is deemed to qualify as an extension of that report pursuant to § 725.414(c).[4] All of these reports focused primarily upon the autopsy and resulting histologic slides, and were prepared for use in connection with proof of both the Miner's claim and the Survivor's claim. All referred in varying degrees to the very extensive medical records contained in the documentary file admitted to the evidentiary record of the Miner's claim pursuant to § 725.421. That evidence is not admitted or generally admissible in the Survivor's claim, but particular exhibits have been selectively admitted in conformity with applicable regulations with respect to the Survivor's claim. § 725.421(b)(4).

[4] This tribunal's ruling that Dr. Tomashefski's deposition was untimely with respect to the Miner's claim is deemed to have been improvident, in part, because of the changed schedule for submission of evidence. Although of limited probative value, it has been considered as an extension of Dr. Tomashefski's July 21, 2003, supplemental report with respect to both the Survivor's claim and the Miner's claim. Any prejudice to Claimant from this change is deemed to be negligible.

The issues presented by the constraints of § 724.414 are, first, whether each of these autopsy-related reports, i.e. initial, rebuttal, and rehabilitative, qualifies, at least for certain purposes, as a "report of an autopsy," only one of which is allowed as part of each party's affirmative case, as distinguished from a "medical report"; second, whether any or all such reports of an autopsy are subject to the same constraints applicable to "medical reports," two of which are allowed as part of each party's affirmative case; third, whether, in particular, the requirement that laboratory reports and physicians opinions "that *appear in* a medical report must each be admissible under this paragraph" [§ 725.414(a)(2)(i) and (3)(i); (emphasis supplied], or as records pertaining to the Miner's hospitalization or treatment under § 725.414(a)(4), applies equally to a "report of an autopsy"; and, fourth, to what extent the constraint on admissibility of clinical data and medical opinions which "appear in" a medical report includes any reference to medical evidence upon which a primary opinion might to any degree be based.

This tribunal concludes that the two types of reports should be distinguished, and that, in this case, the several reports designated in relation to the autopsy should be considered reports of an autopsy, and not "medical reports" for purposes of evidentiary constraints under the amended regulations. A basis for distinction inheres in the fact that only a single report of an autopsy is allowed for each party as part of its affirmative case, in contrast with two permissible medical reports, quite probably prepared in a litigation context. It is assumed that reports used for rebuttal and rehabilitation with respect to an autopsy or report of an autopsy fall outside the single report of an autopsy constraint, but would otherwise be subject to the same constraints. Also, reports of an autopsy are distinguishable from medical reports under the amended regulations because rebuttal is expressly allowed in the form of one physician's interpretation of the autopsy submitted by the opposing party. Rebuttal of medical reports is not expressly allowed. If a report of an autopsy is distinguishable for evidentiary purposes from a medical report under the governing regulations, a report of an autopsy could be deemed to qualify as a physician's written assessment of "a single objective test" under §§ 725.414(a)(1), (a)(2)(ii), and (a)(3)(ii), since it is primarily based upon the gross and microscopic assessment by a physician of tissue samples, analogous, for example, to analysis of blood samples, taken from the Miner.

Analysis of the initial report of autopsy by Dr. Turjman, and the histolic slides produced by the autopsy and related analysis, was the primary focus of the several reports in question. These reports of autopsy are distinguishable from ordinary medical reports, in part, because, as is evident from the reports themselves, optimal expert analysis of the histolic slides depends not only on what was seen in gross or through a microscope, but on received information regarding the Miner's medical history—for example, the length and character of his exposure to coal mine dust in and around coal mines, his smoking history, and clinical symptoms. The absence of such significant information would reduce the credibility of the expert opinion with respect to the autopsy evidence, as it could with respect to analysis of other types of objective evidence such as blood gas samples or pulmonary function tests.[5]

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[5] Although inapplicable to Black Lung proceedings, the Federal Rules of Evidence, as adapted for administrative proceedings conducted by the Office of Administrative Law Judges provide at 29 CFR § 18.703 with regard to the bases of opinion testimony by expert, "The facts of data in the particular case upon which an expert bases an opinion or inference may be those perceived by or made known to the expert at or before the hearing. If of a type reasonably relied upon by experts in the particular field in forming opinions or inferences upon the subject, the facts of data

Although descriptive detail varied, none of the reports related to the autopsy contained descriptions of laboratory tests or physicians' opinions in such discrete form and substance as to have direct or independent utility as separate affirmative evidence with respect to these claims. They are used extensively, though in varying degree by various doctors, as the basis for the expert opinions regarding the autopsy evidence. References to such evidence would have been necessary and appropriate for credible reports of the autopsy related to the Miner's claim. To expurgate such obviously relevant and essential material to establish the admissibility in evidence of these reports in the Survivor's claim would be unreasonable, inefficient, and destructive of the probity of the reports.

This tribunal concludes, therefore, that the strictures of §§ 725.414(a)(2)(i) and (3)(i) relating to the requisite admissibility in evidence of laboratory material and physicians' opinions that appear in medical reports do not apply to such reports of an autopsy as have been submitted in this case. Since such explicit references are for practical purposes inextricably involved in the reports of Dr. Perper and Dr. Tomashefski, those doctors' reports would otherwise have to be excluded from the evidentiary record of the Survivor's claim. The references to such data in Dr. Turjman's and Dr. Crouch's reports are sufficiently general, vague, and nonspecific, as not to require their exclusion. In any event, there is good cause for allowing such references notwithstanding, because of the burdensomeness and disadvantages of removing them in relation to the Survivor's claim, at least under the circumstances of this case. Finally, good cause exists for permitting such references with respect to the Survivor's claim in this instance, because they may be deemed essential to the probity of the reports of an autopsy submitted by both parties.[6] Thus, the several reports of autopsy as designated by the parties are ruled admissible and have been admitted into evidence in both the Survivor's and the Miner's claims, and have been considered in relation to both claims.

The medical reports of Dr. Forehand dated March 23, 2002, and Dr. Mitchell dated April 3, 2002, submitted by Claimant as initial evidence in the Survivor's claim, are admissible, as they are proper in number, and internal references to clinical data and doctors' opinions are to hospitalization and treatment records which are admissible. These medical reports are admissible with respect to both the Survivor's and Miner's claims. (C-2).

The medical report of Dr. Castle dated June 19, 2003, which was submitted by Employer as initial evidence, however, is not admissible with respect to the Survivor's claim because Dr. Castle refers to laboratory evidence and physicians opinions which are not admissible into evidence with respect to the Survivor's claim. (E-1). The Benefits Review Board has made clear in *Dempsy* with respect to the medical data "that appears in a medical report" which §§ 725.414(a)(2)(i) and (3)(i) require must be admissible in evidence, "appears in" means "refers to." Whether consideration of the medical report is permissible, however, may depend upon the extent to which the reference is "inextricably linked" with the gist of the opinion. *See Dempsey v.*

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need not be admissible in evidence." It is apparent from the experts in this case that such data is of a type reasonably relied upon by such experts.

[6] Although the parties did not draw these distinctions or analyze them, proper application of the applicable regulations is deemed to require such analysis in this case, *sua sponte*.

Sewell Coal Co., B.L.R. , BRB Nos. 03-0615 BLA and 03-0615 BLA-A (June 28, 2004)(en banc), slip. at 12, 15-16.

Dr. Castle's disclaimer that, because he is not a pathologist, and because the pathology findings were in conflict, he relied upon the clinical findings including the lack of radiographic findings in reaching his conclusions, taints hiss report for purposes of admissibility under § 725.414(a)(3)(i). The appearance in his June 19, 2003, opinion of references to the several reports of autopsy, and to the reports of Dr. Forehand and Dr. Mitchell, and their treatment records, is permissible because those data are in evidence. But he identified with particularity his own report dated March 18, 1996, its express findings and conclusions, and the basis therefore. That report was not identified as evidence offered by Respondent. Dr. Castle's declaration that his opinion was based, in part on his earlier report as described, is problematical insofar as it may be deemed to have *appeared in* or been referenced by his report in violation of the strictures of § 725.414(a)(3)(i). However, his general statement that his opinion was based upon a "thorough review of all the data including medical histories, physical examinations, radiographic evaluations, physiologic testing, arterial blood gases, autopsy material, and other data" of undefined scope does not appear to run afoul of the constraint because the references are insufficiently specific and the link to the opinion insufficiently defined.

Dr. Castle also prepared a supplemental report dated July 18, 2003, devoted to a review of Dr. Perper's report of autopsy, and recording an unchanged opinion, but declaring that the unchanged opinion was based on his review of "all the submitted medical data." This opinion was not identified by Employer as evidence submitted for any specified purpose on the evidence summary form consistent with § 725.414. Dr. Castle's July 18, 2003, supplemental opinion, though undesignated by Employer, could be considered "admissible" because Employer designated only one medical opinion as initial evidence. Alternatively, it could be treated as a supplemental part of Dr. Castle's original July 19, 2003, opinion whose scope in the context of this case requires it to be treated as an integral part of the original report. Most interests appear to be better served if Dr. Castle's report were treated as a single report submitted in two sections, rather than creating an incentive for submission of such a consolidated report to be deferred until the doctor has had access to all of the evidence reasonably intended and projected to be within the scope of his evidentiary review.

This tribunal concludes that a preponderance of the attributes of Dr. Castle's initial and supplemental reports weigh against their admissibility into evidence, and against their having substantial probative weight if they were admissible. Although not designated by Employer as one of its medical reports, Dr. Castle's 1996 medical report might be deemed "admissible" because Employer designated only one medical report as its initial evidence. The propriety of a proxy designation of such status by this tribunal, however, even if hypothetical, seems dubious. The extent to which Dr. Castle's opinion depended upon his earlier referenced opinion cannot be discounted.

Dr. Castle's supplemental report, however, is also tainted by references to inadmissible evidence such as the clinical findings which expressly included radiographic findings not in evidence in the Survivor's claim, and perhaps not "admissible" because not so designated and of excessive number. It is also tainted by Dr. Castle's express disclaimer of reliance upon the

conflicting pathological findings, which shifted his reliance from the reports of autopsy to clinical findings not clearly "admissible" in evidence. This tribunal concludes that the references are sufficiently particular and the dependence so sufficiently and substantially established to require a ruling that they "appeared in" Dr. Castle's opinion for purposes of the constraints in § 725.414(a)(3)(i). This tribunal concludes that it is not possible to ascertain whether Dr. Castle's opinion as stated was developed independently of inadmissible evidence in such a manner that the supplemental report can be properly admitted into evidence in the Survivor's claim, or, in the alternative, given significant probative weight with respect to that claim. Dr. Castle's supplemental report is therefore ruled inadmissible as to the Survivor's claim. It is admissible, as is the June 19, 2003, report, however, with respect to the Miner's claim.

New Evidence Related to the Survivor's Claim and the Miner's Claim

Dr. Forehand

Dr. Forehand is a B-reader and is board-certified in internal medicine and allergy and immunology, and board-eligible in pediatric pulmonary medicine. He also has published during the 1990's a number of articles regarding the effects of coal dust exposure in miners. He recorded in a medical report and opinion dated March 23, 2002, that he had participated in the Miner's medical care for ten years beginning on July 31, 1991. The Miner indicated at that time that he had smoked a pack of cigarettes per day for fifteen years and had worked on a coal mine tipple for about fifteen years. (C-2) This medical report was offered by Claimant and has been admitted as initial evidence with respect to the Survivor's claim and as evidence with respect to the Miner's claim. § 725.414(a)(2)(i). The doctor's declaration and the extensive treatment records in evidence tend to prove that he qualifies as a treating physician. § 725.414(a)(4).

The Miner had told Dr. Forehand that his work in extremely dusty conditions required him to climb repeatedly seven flights of stairs per day, until the dusty conditions and physical demands exceeded his ability to breathe. Dr. Forehand noted that the Miner's respiratory condition had so deteriorated by 1992, that he required continuous oxygen and was required to use inhaled bronchodilators and corticosteroids "to avoid incapacitating shortness of breath and inability to care for himself." Dr. Forehand recorded that at that time the Miner's arterial blood gas in room air had a pO_2 of 55, that the Miner had an FEV_1 of less than one liter, and that those values continued to decline, further impairing lung function and restricting the Miner's activities. Dr. Forehand reported that despite ongoing care the Miner's condition worsened so dramatically that he required hospitalization at least four times each year until he died. Dr. Foreman observed that Dr. Turjman's June 19, 2001, autopsy findings of complicated coal workers' pneumoconiosis correlated with the Miner's work history and decline in respiratory health over the last ten years. As a result, Dr. Forehand opined that the Miner had coal workers' pneumoconiosis which prevented him from working and which contributed to and hastened his death. Dr. Foreman's diagnosis of coal workers' pneumoconiosis appears to have depended virtually entirely upon Dr. Turjman's autopsy findings, and his conclusion that the pneumoconiosis was disabling and hastened the Miner's death is otherwise unreasoned.

Dr. Mitchell

Dr. Mitchell, who is board-certified in family practice, provided a self-styled reasoned opinion in a medical report dated April 3, 2002, that "black lung (complicated coal workers' pneumoconiosis) did contribute to and hasten Mr. Bucklen's death." (C-2). This medical report was offered by Claimant and has been admitted as initial evidence with respect to the Survivor's claim and as evidence with respect to the Miner's claim. The doctor's declaration, like that of Dr. Forehand's, tends to qualify him as a treating physician. He based his opinion on many years of treatment of the Miner prior to his death on June 18, 2001, reflected in charts reflecting outpatient and inpatient care after 1996. He indicated that charts reflecting earlier treatment were on microfilm. He noted that the Miner had a long history of underground coal mining prior to his retirement. Dr. Mitchell reported that he had treated the Miner over the years for COPD, cor pulmonale, and coal workers' pneumoconiosis; that the Miner had required multiple hospitalizations related to these pulmonary problems over the years; that the Miner had been diagnosed as having complicated coal workers' pneumoconiosis; and that he had been followed closely by himself, Dr. Forehand, and during the hospitalizations by Dr. German Iosif.

Dr. Mitchell indicated that the Miner had been subjected to a large number of medical tests performed over the years, and alluded specifically, but without descriptive comment, to a chest x-ray dated June 1, 2001, as the last before the Miner's death; the autopsy report "demonstrating the presence of advanced coal workers' pneumoconiosis with macronodular and micronodular and diffuse pulmonary emphysema"; and the final hospital discharge summary. These items were not attached to his report, as he said they were, in Claimant's submission under cover letter of August 12, 2003, and, except for the autopsy report, the effect which they might have had upon Dr. Mitchell's opinion is indeterminate. The chest x-ray referred to was not separately identified as Claimant's evidence with respect to the Survivor's claim, but, presumably, would be admissible had it been formally offered under the constraints of § 725.414, since Claimant identified no others. He appears to have accepted the results of Dr. Turjman's autopsy report, rather than independent testing, as the basis for a received diagnosis of complicated coal workers' pneumoconiosis, which he opined, without explicit rationale, contributed to and hastened the Miner's death.

Death Certificate

The death certificate recording the Miner's death on June 18, 2001, at the Clinch Valley Medical Center, Richlands, Virginia, was signed by Larry G. Mitchell, MD. Dr. Mitchell identified the immediate cause of death as chronic obstructive pulmonary disease with coalworkers' pneumoconiosis, noting an interval of "years" between onset and death. As "[o]ther significant conditions leading to immediate cause" of death, Dr. Mitchell identified cor pulmonale; arrhythmias; history of GI bleeding. He recorded that an autopsy was authorized. (D-9)

Autopsy Report of Dr. Turjman

Dr. Dorid K. Turjman, who is board-certified in anatomic and clinical pathology and the subspecialty of cytopathology, performed an autopsy on the Miner on June 19, 2001, which was

restricted to his lungs for black lung purposes.[7] (D-10) Dr. Turjman's autopsy protocol was offered by Claimant and is admitted as evidence with respect to both the Survivor's and the Miner's claims. Dr. Turjman's final diagnosis was "Coal workers' pneumoconiosis, advanced, with many macronodular and micronodular lesions and with diffuse pulmonary emphysema. A) Secondary progressive massive fibrosis (PMF). B) Secondary pulmonary hypertension and cor pulmonale. C) Marked congestion and moderate edema, both lungs." The prosector's final note indicated that the Miner "was a 78-year-old, nonsmoker man with a history of underground coal mining occupation for many years. He clinically developed coal workers' pneumoconiosis with secondary chronic obstructive pulmonary disease and secondary cor pulmonale. Examination of the lungs at autopsy confirmed the presence of advanced coal workers' pneumoconiosis including the presence of progressive pulmonary fibrosis (coal macronodules of 2 cm or larger) and the presence of diffuse emphysematous changes and thickened blood vessel walls consistent with clinical pulmonary hypertension and cor pulmonale. The cause of death in this patient is related to his advanced coal workers' pneumoconiosis and its complications mentioned above."

In his autopsy report, Dr. Turjman noted in pertinent part a clinical history from unidentified sources reflecting a nonsmoker with a long history of chronic obstructive pulmonary disease (COPD) which was considered endstage and had required many hospitalizations, coal workers' pneumoconiosis, and cor pulmonale secondary to COPD. Dr. Turjman noted supraventricular arrhythmia, related, and other apparently serious medical problems. After his hospitalization in early June, 2001, the Miner's respiratory status deteriorated until his death on June 18. Significantly, contrary to Dr. Turjman's received information, the Miner had a long and significant smoking history, and was not a nonsmoker.

Dr. Turjman's internal examination of the chest cavity and lungs disclosed, in pertinent part, that both lungs were congested; that serial sectioning through both lungs revealed diffuse emphysematous changes with the presence of confluent areas in both upper lobes forming multiple pulmonary bullae, the presence of multiple fibrotic and darkly pigmented nodules, the largest of these nodes in the right upper lobe measuring 2.5 cm in the greatest dimension. Dr. Turjman recorded observation of smaller coal macules diffusely scattered throughout both lungs, and the right lower lobe markedly congested, without consolidation. Dr. Turjman listed the sections of lung identified to the several histolic slides.

The microscopic description disclosed, in pertinent part, lung sections with diffuse emphysematous changes more pronounced in the upper lobes, and comprised of destruction of alveolar spaces forming a pattern of mainly panlobular emphysema. Dr. Turjman recorded that the tissue sections from where the pulmonary nodules were grossly visible "show[ed] large areas of fibrosis morphologically typical of nodular lesions of coal workers' pneumoconiosis and were characterized by dense fibrosis of lung parenchyma with heavy deposition of carbon pigment. There are a few markedly large nodules [which] are more than 2 cm in diameter, confirming the gross observation. The large nodules are best manifested in the slide labeled #4. The size of these nodules meets the criteria for coal workers' progressive massive fibrosis (PMF). On high-power field, these large nodules show proliferation of fibroblasts with the presence of coarse and fine collagen fibers and with the heavy dark pigment deposition. The lung tissue adjacent to

[7] Judicial notice has been taken of Dr. Turjman's relevant professional credentials not otherwise of record by reference to www.abms.org. *See Maddaleni v. Pittsburgh & Midway Coal Mining Co.*, 14 B.L.R. 1-135 (1990).

these large nodules continues to show emphysematous changes with variable number of pneumocyte type II deposited in the dilated alveolar spaces.

In one of these nodules, central area of necrosis is present with accumulation of pure population of neutrophils. This area of central necrosis is manifested in the slide labeled #4. No association of the accumulated neutrophile with bronchial spaces can be appreciated, therefore, a diagnosis of bronchopneumonia cannot be established. In addition to the large nodules, there are scattered small coal macules noted, especially close to pleural spaces with involvement of subpleural spaces by these macules. . . [T]he medium- and small-sized vessels show moderate thickening of vascular walls, consistent with an element of pulmonary hypertension. The thickening of vascular walls is best manifested in the slide labeled #12. The areas of fibrosis of lung tissue also show proliferation of small blood vessels. In addition, lung parenchyma, especially adjacent to medium-sized bronchioles, shows multiple patches of chronic inflammatory infiltration, mainly lymphocytes. . . . ."

Dr. Tomashefski's Initial Report

In his medical report dated June 13, 2002, offered by Employer and admitted as Employer's initial autopsy evidence, Dr. Tomashefski indicated review of specified medical records and documents pertaining to the Miner spanning a period of April 1962 until 2001, including the death certificate, and twenty-one autopsy slides of satisfactory technical quality representing eighteen blocks of the Miner's lung tissue. (D-13) Dr. Tomashefski is board-certified in anatomic and clinical pathology. In material part he noted that the last entry reviewed was dated May 14, 2001, and indicated in part that the Miner's lungs were clear to auscultation, but that the Miner was diagnosed by Dr. Forehand as having end-stage, chronic obstructive pulmonary disease and coalworkers' pneumoconiosis and was receiving hospice care. Dr. Tomashefski noted that the Miner's medical history included a host of significant medical problems, and that during his final hospitalization he was nonambulatory and dependent upon oxygen. He noted with respect to the Miner's respiratory history that the Miner had complained of exertional dyspnea, cough and wheezing since approximately 1965.

After 1965 physical abnormalities and diminished breath sounds and rales were apparent, as well as progressively severe obstructive lung disease improved slightly after bronchodilators from testing between 1981 until 1994. Dr. Tomashefski noted apparently adverse changes in arterial blood gas test results, x-rays that were predominantly negative for coal workers' pneumoconiosis, but with 26% of the interpretations indicating s and t opacities equivocal for pneumoconiosis, 11% identifying small round (p, q) or irregular (s, t) opacities of low or moderate profusion, 1/2 or 2/1 from 1977 to 1994, and no large opacities by any observer. Dr. Tomashefski noted, however, that chest x-ray reports consistently documented emphysematous changes and bullae. He noted an opinion from Dr. Wiot that the chest x-ray of June 6, 1994, exhibited prominent markings within the bases, but of a character "totally against coalworkers' pneumoconiosis." A chest CT scan on January 20, 1998, showed extensive emphysema with bullae and dense consolidation of the dependent portion of the left base. Treatment for the obstructive lung disease was recorded as having included steroids, supplemental oxygen, and inhaled bronchodilators.

Dr. Tomashefski also noted a thirty year coal mine employment history, one or two years underground as a coal loader, but the rest outside on the tipple as a mechanic and maintenance foreman, ending with retirement in 1980. He noted an indeterminate but long and substantial smoking history probably of a pack a day for between twenty and forty-four years.

Dr. Tomashefski recorded only a very limited description of the autopsy report, noting in pertinent part the description of the lungs as congested and diffusely emphysematous with confluent bullae in the upper lobes, and "[m]ultiple fibrous and darkly pigmented nodules, measuring up to 2.5 cm in maximal dimension, are present along with smaller 'coal macules.'" Dr. Tomashefski recorded the results of his own review of the slides of the Miner's lung as disclosing diffuse airspace enlargement with "apparently detached" septa and subpleural bullae, moderately severe interstitial fibrosis throughout all sections, much of it of longstanding duration and characterized by irregular foci of mature collagen, or alveolar septal fibrosis. He noted multiple areas of organizing fibroblastic tissue within airspaces, a mild degree of fine black pigment and sparse birefringent crystals deposited in the interstitium, "but no coal macules, micronodules, or lesions of progressive massive fibrosis (PMF) are identified." He also noted "a small, fibrous-encapsulated, calcified granuloma in the right upper lobe (slide #4), mucopurulent exudates in the lumens of small bronchi, and organizing hemorrhage, especially in the sections from the left lower lobe."

Dr. Tomashefski opined, based on his review of the medical records, autopsy report, and slides of the lung tissue, that the Miner "had severe panacinar bullous emphysema and interstitial fibrosis. The degree of interstitial fibrosis is probably overestimated in the sampling at autopsy, since Mr. Bucklen showed no obvious evidence of fibrosis in his pulmonary function tests. The cause of interstitial fibrosis is not completely discerned; however, in my opinion, within reasonable medical certainty, a major component is due to organizing pneumonia. Active organizing pneumonia is present in many slides from Mr. Bucklen's autopsy. In the right upper lobe, focal fibrosis may have been the result of an infectious granulomatous process. The histologic pattern of the fibrosis in Mr. Bucklen's lung is not that of progressive massive fibrosis (PMF) (1). Furthermore, fibrosis is not particularly associated with the mild degree of black pigment and mineral dust that is present in Mr. Bucklen's lung tissue."

Dr. Tomashefski continued, "Since I did not observe coal macules or micronodules, it is also my opinion that Mr. Bucklen did not have simple coalworkers' pneumoconiosis. This opinion is consistent with the radiographic data, which is largely negative or equivocal for features consistent with coalworkers' pneumoconiosis." He recorded his disagreement with the diagnosis of PMF in the autopsy report. He reasoned that the largest discrete area of fibrosis described as black and measuring 2.5 cm and located in the right upper lobe as described in the autopsy report and identified on slides #1 and #2 had "neither the configuration of a PMF lesion nor the degree of black pigment typically seen in PMF." He declared that parenchymal scars in the lung tend to concentrate black pigment, regardless of cause, so that the presence of black pigment is not exclusive proof of PMF. He declared that PMF uniformly occurs in a background of simple coalworkers' pneumoconiosis, which was not present in the Miner's lung tissue. Dr. Tomashefski also declared that PMF was not established clinically, since it was not diagnosed on any of his x-rays, and the pulmonary function tests indicated obstructive lung disease, rather than the restrictive or mixed restrictive/obstructive ventilatory impairment characteristic of PMF. Dr.

Tomashefski also opined that the Miner's diffuse panacinar emphysema and diffuse interstitial fibrosis were not due to coal dust exposure or coalworkers' pneumoconiosis because in either case "there should be a spatial relationship with dust deposition and lesions of coalworkers' pneumoconiosis" if either disease were due to coal dust.

Dr. Tomashefski opined that severe emphysema and interstitial fibrosis were the underlying cause of the Miner's death, and the immediate cause of death was respiratory failure precipitated by purulent bronchitis, organizing pneumonia, and lung hemorrhage, and that no impairment experienced by the Miner, or his death, was related either to coalworkers' pneumoconiosis or to coal dust. Dr. Tomashefski attributed the Miner's emphysema to his "long, sustained exposure to cigarette smoke."

Dr. Crouch

In her pulmonary pathology consultation report dated August 12, 2002, which was based upon her examination of twenty glass slides of the Miner's lung tissue, Dr. Crouch, who is board-certified in anatomic pathology, and Professor of Pathology and Immunology at Washington University in St. Louis School of Medicine, diagnosed "emphysema, mixed patterns, severe with non-specific fibrosis[;] no evidence of coal workers' pneumoconiosis." (D-21) Employer offered this report as rebuttal of Dr. Turjman's autopsy report, and it is admitted as such with respect to both the Survivor's and the Miner's claims. Dr. Crouch had received the autopsy report, death certificate, "and miscellaneous occupational and medical records concerning Kermit Bucklen." She did not refer directly or specifically to these records, however, except to disagree with the conclusions in the death certificate and autopsy report premised on the existence of pneumoconiosis, and except by reference to "the history and severity of the emphysema" as a possible contributor to the Miner's death. Dr. Crouch declared categorically that "there is no histologic evidence to suggest that the Miner's chronic lung impairment related to coal mine dust employment," and that, although there was evidence of mild coal dust deposition, "there is no histologically discernable coal workers' pneumoconiosis." She observed no coal dust macules, micronodules nodules, no larger lesions of massive fibrosis, no silicotic nodules and no evidence of complicated silicosis, and no focal emphysema.

Dr. Crouch noted that many areas of severe emphysema were almost totally devoid of identifiable dust particles, and that, although coal dust was found in some areas of fibrosis associated with areas of emphysema, this was commonly seen in severe emphysema, probably reflecting prior episodes of lung infection, as the result of deposition and trapping of inhaled particles in those regions. She characterized the emphysema as severe, with mixed centriacinar, panacinar, and distal acinar patterns with extensive bullous emphysema, and associated fairly typically with non-specific fibrosis. She noted "generally mild deposition of rounded black particles consistent with carbonaceous materials derived from cigarette smoke, as well as small amounts of irregular dark brown to black particles consistent with coal dust…non-specifically trapped in some areas of fibrosis. She suggested that areas of dust deposition in areas of non-specific scarring were misinterpreted as dust related lesions.

Dr. Crouch opined, "The observed emphysema is severe and consistent with a history of chronic obstructive pulmonary disease. The mixed histologic patterns with marked bullous

changes, the absence of associated changes of even mild simple coal workers' pneumoconiosis, and the lack of concordance between the amount of deposited dust and the severity of emphysema indicate that coal dust did not contribute to the alveolar destruction. The major risk factor was cigarette smoking.…Thus, occupational coal dust exposure could not have caused any degree of respiratory impairment or disability and could not have caused, contributed to, or otherwise hasten[ed] this patient's death." Declining to opine definitively as to the immediate cause of death because of the autopsy limitations, Dr. Crouch declared that the history and severity of the emphysema suggested that the severe emphysema and/or associated secondary cardiac disease contributed to the Miner's death.

Progress Reports

The progress records of Dr. Forehand and Dr. Mitchell which cover the decade from July 31, 1991, to May 14, 2001, are notable for impressions by Dr. Mitchell in the most recent notes of "Chronic obstructive pulmonary disease and coal workers' pneumoconiosis, endstage," and cor pulmonale, dated May 14, 2001, which is the first notation of coal workers pneumoconiosis contained in these records. Prior to that report he referred, for example in the record of March 28, 2001, to "exacerbation of chronic obstructive pulmonary disease" usually with recent exacerbation and cor pulmonale. Dr. Forehand's typical assessment was of oxygen and steroid dependent, end-stage chronic obstructive pulmonary disease, or chronic bronchitis. There is no mention, typically and for example in the record dated February 22, 2001, of coal workers' pneumoconiosis or of emphysema, specifically. There is no apparent focus on etiology or causation. In a notation dated August 3, 1992, Dr. Forehand noted an impression of "Emphysema with reversible component," in assessing a problem of "COPD.' But this is the only reference to emphysema in the progress notes. In the note dated August 24, 1992, Dr. Forehand refers to the problem of exacerbation of COPD without further mention of emphysema. Dr. Mitchell's is the only mention of pneumoconiosis. (D-10) These records, rather than supporting the conclusory opinions of Dr. Forehand and Dr. Mitchell, suggest the opposite by the conspicuous absence of supporting data.

Dr. Perper

Dr. Perper's medical opinion and review of the autopsy slides dated May 10, 2003, was submitted by Claimant as rebuttal evidence related to Dr. Tomashefsky's initial report in support of both the Survivor's claim and the Miner's claim. (C-1) Dr. Perper is a lawyer and forensic pathologist and medicolegal consultant with a distinguished resume. His qualifications include public service and various academic appointments related to his field and, although unmentioned in his curriculum vitae accompanying his report, board-certification in anatomic pathology with a subspecialty in forensic pathology.[8] He referred to Dr. Ranavaya's report dated September 10, 1991, Dr. Rasmussen's report dated July 12, 1994, and Dr. Sargent's report dated November 18, 1994, which referred to thirty-one to thirty-five years of coal mine employment, mostly at the tipple, and prior legal decisions establishing 27½ years of coal mine employment. None of these medical reports is admissible with respect to the Survivor's claim under the strictures of § 725.414, which do not apply to the Miner's claim. He also referred in those reports to a

[8] Judicial notice is taken of these qualifications based upon www.abms.org, last consulted January 31, 2005. *See Maddaleni v. Pittsburgh & Midway Coal Mining Co.*, 14 B.L.R. 1-135 (1990).

smoking history, variously reported, but indicating over forty pack years ending about 1988. He reviewed the Miner's clinical history as disclosed by examinations and testing by the same three doctors. He recorded Dr. Ranavaya's conclusion, "Mr. Bucklen has coal workers' pneumoconiosis most likely due to his occupational exposure to dust in the coal mines and that he has pulmonary insufficiency as described which arose primarily from his coal mining dust exposure."

Dr. Perper took cognizance of Dr. Rasmussen's observations resulting from his July 12, 1994, examination that the '[p]ulmonary functions studies showed very severe obstructive ventilatory impairment and a restrictive component could not be excluded. The maximum breathing capacity, single breath carbon monoxide diffusing capacity and the DL/VA were very markedly decreased. Resting arterial oxygen was moderately impaired. On exercise the EKG and blood pressure response were normal but his heart rate increased very markedly, his physiologic dead spaces were elevated, and his oxygen transfer remained markedly abnormal and the patient was markedly hypoxic." He noted Dr. Rasmussen's conclusion "that the patient had very severe and totally disabling pulmonary insufficiency, and was unable to return to his former coal mining job or any gainful employment, and that in view of his long standing occupational exposure to coal mine dust it is "medically reasonable to conclude that he has coalworkers' pneumoconiosis which arose from his coal mine employment." He also noted Dr. Rasmussen's statement that while "the two apparent risk factors for the patient's disabling respiratory insufficiency are, of course his previous cigarette smoking and his coal mine dust exposure with its resultant pneumoconiosis. The latter must be considered at least a major contributing factor to this disabling pulmonary disease." There was no recorded reference to emphysema.

Dr. Perper observed, following a review of Dr. Sargent's November 18, 1994, report of examination and laboratory tests, that, in pertinent part, Dr. Sargent's "diagnostic impressions were: 1. Thirty-five years of coal mine employment, rule out pneumoconiosis[;] 2. History of cigarette abuse with probable chronic obstructive pulmonary disease." Dr. Perper noted that Dr. Sargent's x-ray interpretation as a B-reader, showed bullous changes consistent with emphysema, but no pneumoconiosis opacities (0/0). The pulmonary functions tests showed "severe obstructive ventilatory impairment with hyperinflation, air trapping, and decreased diffusion consistent with pulmonary emphysema." He noted Dr. Sargent's conclusions that: "The patient had a severe totally and permanently disabling purely obstructive respiratory impairment based on both his blood gases and pulmonary functions tests[;] The obstructive respiratory impairment was exclusively due to cigarette smoking[;] Coal Workers' pneumoconiosis was not present based on the negative radiological findings and the absence of mixed obstructive/restrictive pulmonary defects."

Dr. Perper also noted the history of the claim, including that Judge Levin found, in pertinent part, 27 1/2 years of coal mine employment ending in 1980; all but one of sixteen chest x-ray interpretations of x-rays taken between 1986 and 1994 negative for pneumoconiosis, but positive for emphysema; with respect to Dr. Wiot's testimony that all the x-rays were negative for pneumoconiosis, and showed only bullous emphysema; no proof of pneumoconiosis by x-ray, but a presumption of disability due to pneumoconiosis based on pulmonary function and arterial blood gas test results. Dr. Perper noted that Judge Levin had considered Dr.

Rasmussen's report of June 6, 1994, Dr. Sargent's report of November 17, 1994, and Dr. Garzon's report based on reviews of medical documentation in 1991 and March 14, 1996, that the Miner had no evidence of pneumoconiosis, had a clinical picture "entirely consistent with pulmonary emphysema caused by smoking"; that the Miner's obstructive pulmonary disease was exclusively due to smoking with some progression of the emphysema since 1986, and pointed out Dr. Garzon's statement that simple coal workers pneumoconiosis does not progress after cessation of exposure, 1980, in the Miner's case, which is deemed antithetical to the Act and regulations.

Dr. Perper also noted Dr. Castle's consultative review of medical evidence and opinion that the Miner was totally disabled due to emphysema from smoking as confirmed by the clinical symptoms and manifestations, and that the Miner had no evidence of coal workers' pneumoconiosis or disability related to coal mine employment; that Dr. Rasmussen's opinion relied on a single "discredited" chest x-ray; that the opinions of Drs. Sargent, Castle, and Garzon, based on reviews of medical evidence and negative conclusions regarding the existence of pneumoconiosis or related total disability rebutted the presumption of disability due to pneumoconiosis under the presumption under § 718.305. Dr. Perper noted that Judge Levin found an absence of material change in the Miner's pulmonary condition, though the previously established disability had increased in severity, and that Judge Levin had ultimately denied the claim.

Dr. Perper noted that a chest x-ray of March 4, 2001, reread by Dr. Taylor as negative for pneumoconiosis disclosed emphysema and ill defined lower lung markings compatible with pulmonary vascular prominence accentuated by underexposure or early interstitial infiltrate or fibrosis such as usual interstitial pneumonitis or collagen vascular disease.

Dr. Perper referred to the findings in the death certificate issued by Dr. Mitchell and detailed the findings and conclusions in the report of the autopsy prosector, Dr. Turjman, regarding the June 19, 2001, limited autopsy, including Dr. Turjman's notation that the Miner was a nonsmoker and an underground miner. Dr. Perper also detailed pertinent findings of Dr. Tomashefski and Dr. Crouch. With respect to Dr. Crouch's opinion, however, although he recorded Dr. Crouch's observation, "No coal dust macules micronodules or nodules are observed and there is no evidence of massive fibrosis," but inexplicably, other than as a clerical error, added two comments from his own findings as though attributed to Dr. Crouch but which were inconsistent with Dr. Crouch's findings and not in Dr. Crouch's report: "Multiple anthracotic macules are present around blood vessels, airways and within inter-alveolar septa," and "In places clusters of deeply pigmented anthracotic macrophages are seen in the alveoli."

Dr. Perper's microscopic findings noted pervasive and severe destruction of the normal pulmonary architecture; moderate to marked fibroanthracosis in the pleura with areas of subpleural fibroanthracosis dipping into the adjacent lung parenchyma and containing multiple birefringent silica crystals purporting to be shown in Fig. 11 of the photomicrograph of pleural/subpleural area from slide #2. He recorded, "Multiple anthracotic macules are present around blood vessels, airways and within inter-alveolar septa," and that, "[i]n places clusters of deeply pigmented anthracotic macrophages are seen in the alveoli." He observed further, "There is marked fibro-anthracosis with dense and rich anthracotic pigmentation in the shape of: i.

Micro and macronodules, with the macronodules measuring up to 10 mm in maximal dimension. Scar (focal emphysema) is present around the nodules," as purportedly shown on the photomicrographs and descriptive legends on the photomicrographs attached to his report of mixed coal dust type macronodule present in slide #1, Figs. 10 and 9. "ii. Severe interstitial fibro-anthracosis with dense and rich anthracotic pigmentation, as purported shown in Fig. 7 photomicrograph and legend of lung section from slide #5." Dr. Perper noted that this section showed also aggregates of densely anthracotically pigmented macrophages which were not photographed. "iii. Solid areas of fibro-anthracosis, with one area shown in slide #4 measuring up to 1.7 cm or larger, with central necrosis, consistent with a lesion of complicated coal workers' pneumoconiosis (Progressive Massive Fibrosis or PMF)." He noted that the anthracotic pigmentation is extremely rich and dense, and birefringent crystals are present, as purportedly shown in Figs. 2, 3, and 4 of the photomicrograph legends of lesion present in slide #4. He recorded that the pneumoconiotic fibroanthracotic process involved 50 to 75% of the lung sections, and that some of the fibroanthracotic areas contained aggregates of lymphocytic cells. He noted sclerosis of the walls of intrapulmonary blood vessels, severe centriacinar (centrilobular) and panacinar (panlobular) emphysema, a few small foci of organizing bronchopneumonia, and a few alveoli with blood. His microscopic diagnoses were:

1. Progressive Massive Fibrosis (Complicated Coal Workers' Pneumoconiosis) on the background of micronodular, macronodular and interstitial type of coal workers' pneumoconiosis
2. Centrilobular and panlobular (centriacinar and panacinar) emphysema
3. Sclerosis of intrapulmonary blood vessels consistent with pulmonary hypertension and cor pulmonale
4. Foci of organizing bronchopneumonia, few, small
5. Chronic bronchitis, mild[.]

Dr. Perper provided descriptive legends purporting to explain the appearance of the specimens in the fourteen photomicrographs attached to his report.

Dr. Perper than answered seven medicolegal questions, in part affirmatively and in part by critiquing the reports and conclusions of the other pathologists, Dr. Tomashefski and Dr. Crouch. Dr. Perper found the existence of coal workers' pneumoconiosis, based on the exposure to coal dust-containing silica for twenty-seven to thirty-five years, chronic clinical symptomatology of shortness of breath, cough, occasional expectoration of mucus, abnormal pulmonary findings on examination, severely abnormal pulmonary function tests, severely deficient pulmonary diffusion and abnormal arterial blood gases indicative of hypoxia, and the need for therapeutic bronchodilators and supplemental oxygen. He explained the consensus of x-ray interpretations recognizing the presence of COPD but not lesions of coal workers' pneumoconiosis, by asserting that his own experience and medical literature have found that radiologists may miss or underestimate significant simple or complicated coal workers' pneumoconiosis on diagnosis. He declared that the autopsy, as the "gold standard" clearly demonstrated the presence of severe coal workers' pneumoconiosis.

Dr. Perper cited the autopsy findings and diagnosis of the prosector, and his own microscopic pulmonary examination and diagnoses as verifying that the Miner had complicated

coal workers' pneumoconiosis of Progressive Massive Fibrosis (PMF). Dr. Perper asserted that the fourteen photomicrographs illustrating the pathological findings of the lung biopsy observed by him "should be more than sufficient to demonstrate the presence of complicated coal workers pneumoconiosis to any reasonable and unbiased observer." He noted the presence of complicated coal workers' pneumoconiosis at autopsy with a fibroanthracotic mass of 2.0 cm. or more, with necrosis, on the background of marked coal workers' pneumoconiosis with macules, micronodules and macronodules and an interstitial type of coal workers' pneumoconiosis as "clearly evident." He argued that, although the conventional pathological standard requires a complicated pneumoconiosis lesion to be at least 2 cm. in diameter, the standard was arbitrary and that there was no reason why 1.0 cm. lesions, of which the Miner allegedly had several, should not be considered diagnosable as complicated pneumoconiosis or be deemed to correspond to radiological lesions of that same size. He argued that the background of severe pulmonary interstitial fibrosis with presence of marked deposition of anthracotic pigment and birefringent silica crystals consistent with the atypical pattern of interstitial type of coal workers' pneumoconiosis present in this case has been well recognized in recent medical literature which he identified and discussed. His argument seems to be that identification of such interstitial fibrosis-type pulmonary fibrosis sometimes occurs with coal miners who have coal miners' pneumoconiosis, or sometimes is itself a type of coal workers' pneumoconiosis, to provide the necessary background for a finding of complicated pneumoconiosis.

Dr. Perper also asserted that the Miner's centrilobular emphysema was not only a known complication of the Miner's significant smoking stopped thirteen years prior to his death, but "as abundantly substantiated in reliable scientific literature in the last decades, centrilobular emphysema is also a direct result of exposure to mixed coalmine containing silica and coal workers' pneumoconiosis (sic)," and "[w]hile it is legitimate to recognize in general the role of smoking in producing centrilobular emphysema, it is equally legitimate to recognize the significant role of exposure to coal mine dust and coal workers' pneumoconiosis, and there is no logical reason to exclude it." He asserted that the 27.5 or more years of exposure was sufficient to develop coal workers' pneumoconiosis, and the microscopic autopsy evidence revealed severe coal workers pneumoconiosis with marked fibroanthracosis and abundant anthracotic deposits with birefringent silica crystals, indicative of exposure to mixed coal dust containing silica. He also suggested that the scientific literature had recognized a progressive effect of emphysema complications related to coal mine dust because of entrapped and retained intrapulmonary fibrogenic crystalline silica. He cited recognition by WHO, OSHA, and NIOSH, international governmental bodies, and vast medical literature, of the causal connection between exposure to coal and silica in regard to emphysema and chronic obstructive lung disease. In one authority he cited, the severity of the emphysema accompanying patients with coal workers' pneumoconiosis correlates with the degree of exposure to coal dust and is positively related to the dust content of the macules.

Dr. Perper sought to refute the conclusion in the reports of Dr. Tomashefski and Dr. Crouch that the Miner's autopsy lung sections showed no evidence of coal workers' pneumoconiosis and found only emphysema and associated nonspecific interstitial fibrosis. He argued that they ignored the gross pathological findings of the autopsy report; that their characterization of the anthracotic pigmentation in the area of fibrosis as slight and insignificant, was contrary to the showing in the photomicrographs that the anthracotic deposits were, as he

characterized them, "very marked and associated with both nodular, fibroanthracotic massive fibrosis and interstitial coal workers' pneumoconiosis." Dr. Perper asserted that Dr. Crouch ignored what he characterized as a complicated fibro-anthracotic lesion with dense and marked anthracotic deposits and the presence of multiple birefringent silica crystals disclosed on slide #4, and that Dr. Tomashefski misinterpreted this complicated pneumoconiotic lesion measuring more than 2 cm. as being "a small fibrous-encapsulated, calcified granuloma in the right upper lobe." Dr. Perper asserted *ex cathedra* that the photomicrographs, Figs. 2, 3, and 4, clearly substantiate that the lesion is not a granuloma, but as he characterized it. He asserted that both doctors ignored the well substantiated causal relation between mixed coal dust and coal workers' pneumoconiosis to centriacinar emphysema which may progress to panacinar emphysema, and "ignored or were unaware of the interstitial type-fibrosis of coal workers' pneumoconiosis." He also asserted that they ignored the vascular sclerosis of intrapulmonary blood vessels consistent with pulmonary hypertension and cor pulmonale.

Dr. Perper asserted that coal workers' pneumoconiosis progresses in severity after the end of occupational exposure to coal mine dust, as is now ensconsed in the applicable regulations, suggesting that such progression would be attributable to inhaled silica trapped in the lungs. He asserted that the Miner's coal workers' pneumoconiosis was caused by his long occupational exposure to coal dust. And Dr. Perper opined that coal workers' pneumoconiosis caused, substantially contributed to, or accelerated the death of the Miner based on the autopsy findings indicative of marked coal workers pneumoconiosis qualifying as complicated or progressive massive fibrosis with associated centriacinar emphysema "and the other occupational history, non-smoking history and clinical documentation" which he discussed in his report. He opined that coal workers' pneumoconiosis was a significant contributory cause of death and hastened his death both directly and though direct replacement of normal lung tissue by pneumoconiotic lesions and associated centrilobular chronic emphysema which caused pulmonary insufficiency from loss of breathing tissues; the resulting severely depressed pulmonary diffusion of gases and hypoxemia; the sclerotic vascular changes in the intrapulmonary vessels indicative of pulmonary hypertension associated with cor pulmonale. Dr. Perper contended that the hypoxemia could precipitate or aggravate a cardiac arrhythmia in an individual with marked coronary arteriosclerosis, as described in the scientific literature, and he cited the phenomenon of unexplained sudden death of patients with complicated coal workers' pneumoconiosis.

<u>Dr. Tomashefski's Report dated July 21, 2003 in Response to Dr. Perper</u>

Dr. Tomashefski's report dated July 21, 2003, was offered as rehabilitation evidence by Employer, and admissible as such with respect to the Survivor's claim as well as the Miner's claim. It responded specifically to the critique of his report by Dr. Perper, whose report purported to undermine Dr. Tomashefski's opinion. (E-4) Dr. Tomashefski agreed with Dr. Perper's diagnoses of mixed panacinar and centriacinar emphysema, and focal organizing pneumonia, but disagreed with his diagnosis of complicated coal workers' pneumoconiosis. In his opinion the Miner had neither clinical evidence nor pathological features in his lung at autopsy to support a diagnosis of either simple or complicated coalworkers' pneumoconiosis. He pointed to overwhelming negative x-ray interpretations, roughly two-thirds of which identified no small opacities, and roughly a quarter of which identified s and t small irregular opacities of

low profusion, which are not indicative of simple coalworkers' pneumoconiosis which he declared is expressed radiographically as small round opacities of the p, q, or r type. He noted B-reader unanimity negating the existence of large opacities, and a more sensitive chest CT scan taken in January 1998, which showed emphysema but no coal workers' pneumoconiosis. Dr. Tomashefski also cited the Miner's pulmonary function tests indicating severe obstructive changes consistent with emphysema, but no indication whatsoever of a restrictive abnormality, because, he declared, significant interstitial fibrosis, including PMF, causes a restrictive, or mixed restrictive and obstructive ventilatory impairment.

Dr. Tomashefski opined that coal macules are not present in the tissue slides, or in Dr. Perper's photomicrographs which negates a diagnosis of simple coalworkers' pneumoconiosis. He declared categorically that simple coalworkers' pneumoconiosis was not present clinically or histologically in the Miner's lung tissue. What Dr. Perper labeled a coal macule in Fig. 1, Dr. Tomashefski opined was a nonspecific deposit of black pigment in perivascular connective tissue. What Dr. Perper labeled "fibroanthracosis" in Fig. 5, Dr. Tomashefski opined was a small nonspecific peribronchiolar scar with minimal black pigment deposition on one edge of the car as confirmed in Fig. 6. What Dr. Perper labeled a coal micronodule in Fig. 8, Dr. Tomashefski opined was several nonspecific scarred areas within an emphysematous bulla, and a type of fibrosis frequently associated with bullous disease which is not indicative of coalwoworkers' pneumoconiosis. Dr. Tomashefski opined that the larger fibrotic lesions disclosed on slides #2, #3, and #4 were nonspecific interstitial fibrosis without the histological features of PMF or complicated pneumoconiosis. He opined that Figs. 2, 10, 11, and 12 clearly indicate a mild degree of black pigment, irregularly distributed in the fibrotic lesions, in contrast to PMF lesions which would show much more uniform, intense deposition of black pigment than that in the Miner's lung. He dismissed the high-magnification images of focal deposits of black pigment as unrepresentative of the overall degree of black pigment in the Miner's lung, noting that lung scars tend to concentrate black pigment even without exposure to coal dust.

Dr. Tomashefski also pointed to particular histologic features not typical of the coarse collagenous fibrosis seen in PMF which were ignored by Dr. Perper. The nodular necrotic lesion Dr. Perper labeled as a necrotic area of PMF in Fig. 4, Dr. Tomashefski opined was an old fibrocaseous granuloma, probably caused by tuberculosis. Dr. Tomashefski opined that the nonspecific findings in Fig. 14 of a bronchiole with minimal chronic inflammation and mucus stasis was a nonspecific finding which did not support Dr. Perper's diagnosis of mild chronic bronchitis, a clinical diagnosis without histological support because of the limited scope of the autopsy. Because the autopsy did not include the heart, Dr. Tomashefski disagreed with Dr. Perper's diagnosis of sclerosis of intrapulmonary blood vessels consistent with pulmonary hypertension and cor pulmonale, and opined that the tangentially sectioned vessel in Fig. 13 overestimates the degree of mural fibrosis that might be present. He attributed the interstitial fibrosis in the Miner's lung to causes other than coal dust exposure, including TB, organizing pneumonia, and nonspecific fibrosis associated with advanced emphysema He disagreed with Dr. Perper's opinion that coal dust exposure caused or contributed to the Miner's severe, end-stage emphysema because of the absence of simple coal workers' pneumoconiosis "or a spatial association between emphysematous lesions and coal dust deposits.

Dr. Tomashefski responded specifically to Dr. Perper's critique of his prior conclusions. He did not discuss the gross pathological findings of the autopsy report because the multiple darkly pigmented nodules up to 2.5 cm. were not specific for complicated coal workers' pneumoconiosis, reflected the tendency of scars to concentrate black pigment, and because histological analysis and clinical data did not support a diagnosis of PMF. He reiterated his opinion, contrary to Dr. Perper's insistence that the photomicrographs show very marked anthracotic deposits associated with both nodular, fibroanthracotic massive fibrosis and interstitial coal workers' pneumoconiosis, that the photomicrograph show only a mild overall degree of black pigment, that the magnified images are misleading and unrepresentative of the overall mild distribution of black pigment, and the presence of black pigment in the lung of a coal miner does not constitute a histological diagnosis of coalworkers' pneumoconiosis. The two doctors fundamentally disagreed as to whether the extent of the pigmentation deposits and proximate fibrosis were slight or significant. Dr. Tomashefski suggested that Dr. Perper had misunderstood his interpretation of slide #4 and Fig. 4 which he said showed a granuloma localized in the center of a larger area of nonspecific fibrosis probably caused by a mycobacterial infection, but which Dr. Perper had identified as not a granuloma, but a complicated fibro-anthracotic lesion with dense and marked anthracotic deposits and the presence of multiple birefringent silica crystals as shown in Figs. 2, 3, and 4.

Noting the controversial relation between cold dust exposure and the development and degree of emphysema, Dr. Tomashefski declared that there was no evidence of a relationship of the Miner's severe emphysema to coal workers' pneumoconiosis or coal dust deposits as contended by Dr. Perper. He also contradicted Dr. Perper's statement that centriacinar emphysema progresses to panacinar emphysema, and attributed the Miner's severe mixed emphysema exclusively to heavy and sustained smoking. Dr. Tomashefski opined that the mild degree of pigment deposition in the areas of fibrosis, which he opined were overrepresented by Dr. Perper, and the total lack of a restrictive ventilatory defect was inconsistent with a diagnosis of coal dust-induced interstitial fibrosis purportedly identified by Dr. Perper. Dr. Tomashefski reiterated his opinion that the Miner did not have coalworkers' pneumoconiosis of any type, but had severe emphysema and interstitial fibrosis unrelated to coal dust exposure, and that the Miner's respiratory impairment and death were unrelated to coal mine employment or coal dust exposure.

Dr. Tomashefski's Deposition

Dr. Tomashefski's testimony on deposition, August 29, 2003, largely reiterated his conclusions stated in his report of July 21, 2003, and elaborated somewhat on his professional qualifications. (E-6) He testified that he is chairman of the department of pathology of MetroHealth Medical Center in Cleveland, Ohio, with which he has been associated since 1982. He described his broad range of responsibilities with the hospital, including oversight of departmental residency program teaching activities, and active service work in both surgical pathology and autopsy pathology. He also serves as a professor of pathology at Case Western Reserve University School of Medicine with which the hospital is affiliated. (E-6 at 5-7) He testified that as a pathologist he diagnoses coal workers' pneumoconiosis by examining lung tissue, both in gross and by microscopic slides and applies the standard criteria established by the pneumoconiosis panel of the College of American Pathologists regarding coal workers'

pneumoconiosis published in the Archives of Pathology in the late 1970's. He described those guidelines applicable to simple coal workers' pneumoconiosis as including the coal macule with surrounding focal emphysema, the coal micronodules and macronodules, which he described as small pigmented areas of fibrosis that can measure up to 2 centimeters. Diagnosis of progressive massive fibrosis, he testified, requires fibronodular lesions 2 centimeters in size or greater. (E-6 at 8-9)

Based on his review of the autopsy slides and available medical records including Dr. Perper's medical report, Dr. Tomashefski opined that the Miner had severe panacinar bullous emphysema and interstitial fibrosis, but no evidence of coal workers' pneumoconiosis of either the simple or complicated type, and not one coal macule necessary to diagnose coal workers' pneumoconiosis.[9] (E-6 at12) He also identified, in addition to emphysema, organizing pneumonia, a necrotizing granuloma consistent with a remote infectious process, as disclosed on Fig. 4. Although he conceded that coal workers' pneumoconiosis is a type of fibrosis, especially at the micronodular, macronodular and PMF states, he opined that the Miner's interstitial fibrosis was not caused by his prior coal mine dust exposure and was probably attributable to an infectious cause in the vicinity of the granuloma, and to recurring episodes of organizing pneumonia, despite an adequate exposure history to have developed coal workers' pneumoconiosis (E-6 at 13-14). He also opined, after describing the several types of emphysema, that the Miner's emphysema was attributable to smoking more than 20 years and that it was not related in any degree to coal mining. He also testified that he had not found any focal emphysema which he characterized as part of the definition of the early lesion of simple coal workers' pneumoconiosis on the histolic slides. (E-6 at 14-17)

Also in his deposition Dr. Tomashefski addressed several of the 14 photomicrographs attached to Dr. Perper's report, declaring that they did not support a finding of simple coal workers' pneumoconiosis because of the absence of even a single coal macule, considered indispensable for diagnosing simple coal workers' pneumoconiosis. He declared that the photomicrographs depict black pigment in relation to the extensive interstitial fibrosis somewhat attributable to inhaled coal dust, some representing anthracotic pigment from cigarette smoking and correlated with the extent of such smoking.

Dr. Tomashefski opined, in addition, that the photomicrographs did not support a finding of progressive massive fibrosis because the areas of interstitial fibrosis shown do not have the typical nodular look of a PMF lesion or the degree of black pigment typical of such lesions, which are usually nodular or stellate. (E-6 at 19) He also opined that the magnified photomicrographs such as Fig. 4 caused a seriously distorted impression of much more black pigment than the actually minimal amount in imaged lesions as a whole. He repeated his assessment that the lesion in Fig. 4 represents a granuloma because of the visible encapsulated necrotic area with cholesterol spicules which looks like an old, partially healed caseous granuloma derivative of prior tuberculosis or a fungal infection and typical of those in persons unrelated to coal mining. (E-6 at 21-23)

Dr. Tomashefski took issue with Dr. Perper's diagnosis of chronic bronchitis and cor pulmonale as devoid of the essential clinical diagnostic basis and lacking the essential tissue

[9] The reference in the transcript to "vacuole" was obviously an erroneous substitute for "macule."

sections in the autopsy slides for analysis. (E-6 at 23-25) He maintained that he had summarized and considered the gross pathological findings of the autopsy prosector. He also declared his inability to determine what criteria Dr. Perper had used to make his diagnoses, though he was certain that the results of his review of the autopsy did not meet the criteria for simple or complicated coal workers' pneumoconiosis established in the Archives of Pathology and Laboratory Medicine in July 1979. He opined categorically, lacking identification of coal workers' pneumoconiosis, and having concluded that neither the emphysema nor interstitial fibrosis were attributable to coal dust exposure, that the Miner did not have a chronic dust disease of the lung related to coal mine employment, or that coal workers' pneumoconiosis was in any way related to the Miner's death, which was caused by respiratory failure due to severe emphysema and interstitial fibrosis, and precipitated by acute inflammation of the airways and organizing pneumonia unrelated to coal dust exposure.

Dr. Castle

Respondent Employer relies upon Dr. Castle's report dated June 19, 2003, based upon a review of specified medical evidence as its initial medical report. Dr. Castle's opinion was based upon a review of Dr. Crouch's pulmonary pathology consultation report dated August 12, 2002; Dr. Tomashefski's autopsy report dated June 13, 2002; Dr. Turjman's pathology consultation report dated June 19, 2001; the death certificate signed by Dr. Mitchell on July 9, 2001; three x-ray interpretations of films dated February 28, 2000, November 9, 1999, and August 26, 1996; numerous progress notes by Dr. Mitchell and Dr. Forehand covering approximately the decade form 1991 to 2001; and his own medical report dated March 18, 1996, which had concluded that there had been no substantial change in the Miner's condition since the denial of benefits on March 2, 1994, but that the Miner continued to have evidence of severe obstructive airway disease; that he did not suffer from coal workers' pneumoconiosis, though he had sufficient exposure if susceptible; that he had a forty-five pack year smoking history; a severe pulmonary impairment unrelated to coal mine employment and coal dust exposure that was due to the extensive history of tobacco abuse; and no chronic dust disease of the lungs caused by, contributed to, or substantially aggravated by coal mine dust exposure.

In his June 19, 2003, medical report, Dr. Castle, who is board-certified in internal medicine and pulmonary disease and a B-reader, opined that the Miner did not have coal workers' pneumoconiosis; was not permanently and totally disabled as a result of coal workers' pneumoconiosis; and that his death was neither caused by, contributed to, or hastened by coal workers' pneumoconiosis or his coal mine dust exposure. His opinion was declared to be based upon review of the additional medical data submitted, which he identified as the treatment records of Drs. Forehand and Mitchell and others, the autopsy reports, his own earlier medical report dated March 18, 1996, and a "thorough review of all the data including medical histories, physical examinations, radiographic evaluations, physiologic testing, arterial blood gases, autopsy material, and other data," the scope of which is undefined. He resolved the conflicting conclusions of Drs. Crouch and Tomashefski versus Dr. Turjman, the prosector, by relying upon the qualifications of Drs. Crouch and Tomashefski as "academic professors with great experience in pathology and teaching pathology." He concurred with their opinions that there was no simple or complicated pneumoconiosis, and that the severe emphysema was attributable to tobacco smoking and not coal dust exposure. He opined that, with autopsy being the most

reliable basis for diagnosis, coal workers' pneumoconiosis could have played no role in the Miner's death in any way, or in any impairment, identified as severe pulmonary emphysema with bullae resulting in partially reversible severe airway obstruction, or disability during life.

In a supplemental report dated July 18, 2003, Dr. Castle recorded an unchanged opinion which he said was based on his review of "all the additional medical data submitted" in the claim, which apparently referred to Dr. Perper's pathology consultation dated May 10, 2003, and "all the submitted medical data," including his own previous report dated June 19, 2003. (E-3) He noted that Dr. Perper had listed the data he had reviewed, and noted Dr. Perper's opinions that the Miner had evidence of complicated coal workers' pneumoconiosis clearly substantiated at autopsy, which "was a substantial cause of his death and a hastening substantial factor in his death." Dr. Castle also cited Dr. Perper's conclusions that the Miner's coal workers' pneumoconiosis derived from 27 ½ to 35 years or more of occupational exposure as a coal miner to coal dust containing silica, more than enough to develop coal workers' pneumoconiosis.

Dr. Castle concluded that Dr. Crouch's and Dr. Tomashefski opinions, which conflicted with Dr. Perper's, as well as the clinical findings of record, including the physical findings, the physiologic findings of changes consistent with bullous emphysema, and the radiographic findings consistent with bullous emphysema, supported a most likely etiology of tobacco smoke induced bullous emphysema. Because the pathology findings were in conflict, and because he was not a pathologist, Dr. Castle cited the clinical findings, including the lack of radiographic findings to substantiate a diagnosis of either simple or complicated coal worker's pneumoconiosis, to support a conclusion that the most likely diagnosis was that of tobacco smoke induced pulmonary emphysema and an opinion that the Miner's death was unrelated to coal workers' pneumoconiosis.

Other Medical Evidence

The evidence that was before Judge Levin in rendering his September 11, 1996, decision with respect to the request for modification following the denial by Judge Cox on July 2, 1991, and the denial of the ensuing request for modification by Judge Murty on March 2, 1994, consisted of the then recent report of examination and tests by Dr. Rasmussen which accompanied the request for modification; x-ray interpretations submitted subsequent to the previous denial which consisted of thirteen negative interpretations by Dr. Wiot of x-rays taken between 1986 and 1994; negative readings of the June 6, 1994, x-ray by Drs. Spitz and Francke; a positive 1/2 reading by Dr. Patel, relied upon by Dr. Rasmussen; and an 0/1 interpretation by Dr. Castle, which is not evidence of pneumoconiosis under pertinent regulations. Dr. Wiot had also testified that there was absolutely no evidence of pneumoconiosis, but there was evidence of emphysema attributable to cigarette smoking increasing over the years.

Discussion and Conclusions of Law

The Merits of the Survivor's Claim

The conflicting analyses of the autopsy evidence provided by the several physicians are the primary basis for determining the merits of both the Survivor's claim and the Miner's claim. Claimant has the burden of proof with respect to the existence of coal workers' pneumoconiosis, its cause by coal mine employment, the Miner's total disability and death due to pneumoconiosis pursuant to applicable law, notwithstanding the potential invocation of certain presumptions defined in the applicable regulations. This tribunal finds that she has not carried her burden of proof as to all the elements of entitlement as to either claim. Because § 718.305 by its terms does not apply to any claim filed after January 1, 1982, the rebuttable presumption provided in that regulation is not applicable to the Survivor's claim.

Certain premises, however, are not in substantial dispute. Employer has explicitly conceded that the Miner suffered from a totally disabling respiratory impairment. (Resp. Brief at 17). All of the physicians opining with regard to the autopsy and related evidence recognized that the Miner had severe disabling emphysema, and thus a pulmonary impairment, at his death and for many years before his death. All of those physicians have credentials as pathologists which qualify them to provide their expert opinions in evidence. Their opinions were reasoned and based upon extensively described objective evidence. All of the physicians considered and referred to the Miner's medical records with varying degrees of particularity. The prosector's report, however, reflected a fundamental misapprehension of the Miner's significant smoking history, because the prosector expressly assumed that the Miner was a nonsmoker, an error which seriously impairs the credibility of his assessment. The prosector and Dr. Crouch referred to the Miner's medical records only generally and without particularity. The references to medical record by Dr. Perper and Dr. Tomashefski, which were for the most part not admissible with respect to the Survivor's claim, were extensive.

Entitlement to survivor's benefits pursuant to claims filed on or after January 1, 1982, is contingent upon Claimant's proof that the miner had pneumoconiosis which arose out of coal mine employment and which caused or hastened Miner's death. *See Shuff v. Cedar Coal Co.*, 969 F.2d 977, 16 B.L.R. 2-90 (4th Cir. 1992), *cert. denied*, 506 U.S. 1050 (1993); *Boyd v. Director, OWCP*, 11 B.L.R. 1-39 (1988); § 718.1. In a survivor's claim under Part 718, the administrative law judge must make a threshold determination as to the existence of pneumoconiosis under § 718.202(a) prior to considering whether the miner's death was due to pneumoconiosis. *Trumbo v. Reading Anthracite Co.*, 17 B.L.R. 1085 (1993).

Death will be considered to be due to pneumoconiosis where medical evidence establishes that the miner's death was due to pneumoconiosis, where pneumoconiosis was a substantially contributing cause of death, or where death was caused by complications of pneumoconiosis, or where the miner suffered from pneumoconiosis which satisfied the requirements of § 718.304 (complicated pneumoconiosis). § 718.205(c). Pneumoconiosis is a substantially contributing cause of death if a claimant demonstrates that the disease "serve[d] to hasten [the miner's] death in any way." *See Richardson v. Director, OWCP,* 94 F.3d 164, 167, 21 B.L.R. 2-373 (4th Cir.

1996); *Shuff*, *supra* at 1006. However, the standard is not satisfied if pneumoconiosis contributed to the miner's death to a "negligible" degree. *See Grizzle v. Pickands Mather & Co.*, 994 F.2d 1093, 17 B.L.R. 2-123 (4th Cir. 1993).

The evidence derived from an autopsy is recognized as the most reliable diagnostic evidence of the existence of coal workers' pneumoconiosis. *See Terlip v. Director, OWCP*, 8 B.L.R. 1-363 (1985). However, the United States Court of Appeals for the Fourth Circuit has held that chest x-ray evidence is determinative of the existence of complicated pneumoconiosis. *Eastern Assoc. Coal Corp. v. Director, OWCP* [Scarbro], 220 F.3d 250, B.L.R. (4th Cir. 2000). The existence of both simple and complicated pneumoconiosis are in issue with respect to both the Survivor's and the Miner's claims. It is error in the Fourth Circuit to credit a prosector's opinion over the opinions of reviewing pathologists who relied on slides solely on the basis that the prosector examined the miner's whole body at the time of autopsy. *Bill Branch Coal Corp. v. Sparks*, 213 F.3d 186, B.L.R. (4th Cir. 2000). In this case, the autopsy was limited to the lungs.

The several physicians whose opinions are in evidence categorically disagreed as to the existence of simple and complicated coal workers' pneumoconiosis based on the medical evidence they examined. Claimant's witnesses, the prosector, Dr. Turjman, and Dr. Perper, who reviewed the histologic slides of lung tissue and medical records, found substantial evidence which they interpreted to establish the existence of both simple and complicated pneumoconiosis in addition to emphysema and other observed maladies. They identified with particularity macules, nodules, and other lesions in support of their conclusions. The prosector cited what he characterized as indicia of simple and complicated pneumoconiosis in both his gross and microscopic findings.

Dr. Perper's tissue analysis purported to disclose what he referred to as extensive fibroanthracosis, with what he characterized as dense and rich pigmentation in the form of micro- and macronodules of up to one centimeter and associated with severe interstitial fibroanthracosis, as well as anthracotically pigmented macrophages. He described one area of fibroanthracosis measuring approximately 1.7 cm. as consistent with complicated coal workers' pneumoconiosis. He described extensive anthracotic pigment associated with various types of lesions, but the extent to which the anthracotic pigment was interactive with the lung tissue or caused the lesions in question is not clear from his descriptive analysis. Nevertheless, the gist of Dr. Perper's assessment seems to create a sufficient association between the pigmentation and fibrosis to reflect a plausible diagnosis by him of coal workers' pneumoconiosis. A diagnosis of anthracosis may be deemed equivalent to a diagnosis of pneumoconiosis, although proof of anthracotic pigmentation is not enough by itself to establish the existence of pneumoconiosis. *See Dagnan v. Black Diamond Coal Mining Co.*, 994 F.2d 1536 (11th Cir. 1993); *Hapney v. Peabody Coal Co.,* 22 B.L.R. 1-106 (2001)(en banc); *Bueno v. Director, OWCP*, 7 B.L.R. 1-337 (1984). Dr. Perper appears to suggest, however, that the background of severe pulmonary interstitial fibrosis with the presence of the anthracotic pigment and birefringent silica crystals reflected an atypical pattern of coal workers' pneumoconiosis, though allegedly recognized in the pertinent medical literature.

Dr. Perper's diagnosis of complicated pneumoconiosis, however, is more problematical, because it provides little basis, if any, for establishing equivalency with opacities measurable on x-ray of larger than one centimeter as required by applicable regulations and *Scarbro*. The record discloses no x-ray readings identifying large opacities. In addition to his assessment of the tissue in the histologic slides, Dr. Perper placed extensive reliance on medical literature which he cited as supporting a causal relationship between coal mine dust exposure, and related exposure to silica, and pneumoconiosis, chronic obstructive pulmonary disease, and various kinds of emphysema, bronchitis, and cor pulmonale, the indicia of which he purportedly observed. There does not appear to be any direct evidence of silica exposure in the record. Dr. Perper also referred extensively to fourteen photomicrographs attached to his report. This tribunal finds that it lacks the medical expertise or the practical ability to discern the relative merits and resolve the fundamentally conflicting assessments of the particular lesions dependent upon conflicting interpretations of the several photomicrographs. As a result, this tribunal must rely primarily upon the relative credibility of the opining physicians deriving from their credentials.

Employer's experts, Dr. Tomashefski and Dr. Crouch, concluded categorically on the basis of substantially the same histologic slides and medical records that there was no evidence of either simple or complicated pneumoconiosis, bronchitis, or cor pulmonale. As to the findings of bronchitis and cor pulmonale, they declared that the scope of the autopsy, which was limited to the Miner's lungs, did not produce tissue samples which would reliably support such findings. Where Dr. Turjman and Dr. Perper said that the deposits of coal dust were extensive and dense, Dr. Tomashefski and Dr. Crouch said they were sparse, and that the enlargement of certain of the photomicrographs tended to distort or misrepresent the extent and intensity of the deposits. Significantly, both Dr. Tomashefski and Dr. Crouch opined in explicit contradiction of Dr. Perper and Dr. Turjman that there were no coal macules, nodules, or other lesions necessary to a pathological diagnosis of coal workers' pneumoconiosis, and disagreed also as to the quantity, character, and relationship of visible pigmentation to proximate tissue and lesions. A comparison of the extent and quality of the reasoning by which these experts supported their opinions does not readily resolve the conflicts as to credibility, because, ultimately, the opinions depend upon the professional judgment and experience of the individual physicians in a highly technical context, and they purport to see wholly inconsistent phenomena. Dr. Castle even professed an inability to resolve the conflicting pathologic assessments.

Under such circumstances, this tribunal has relied upon a comparison of the credentials of the four pathologists to ascertain which expertise is most likely to be reliable. In that regard, the record discloses little of Dr. Turjman, except that he is a board-certified pathologist and was engaged to perform a limited autopsy by the Claimant. His opinion was erroneously premised on the Miner's being a nonsmoker. Dr. Perper's credentials are extensive, but those of record indicate that he is essentially an independent pathologist devoting much of his time to providing expert opinions in connection with litigation. He has published extensively, and much of his career has been in public service as coroner or in other roles employing his forensic pathologic expertise. It is unclear what effect, if any, his legal training might have had upon his medical opinions in this case.

Dr. Tomashefski, on the other hand, has had a continuing association with a large medical center as chief of pathology and with teaching responsibilities at Case Western Reserve Medical School as well as a medical practice treating patients. Dr. Crouch has high ranking academic responsibilities at the Washington University in St. Louis Medical School, as well as a teaching and treating practice. Both physicians are extensively published. There is no apparent geographic or other association between the two, other than their specialty of pathology, which might engender coordinated or similar conclusions, and their categorically negative conclusions appear to be wholly independent.

This tribunal concludes that the extent of the associations with the academic and large medical institutions in positions of high responsibility would tend to create incentives for greater reliability in the circumstance of starkly inconsistent or opposing positions with which this tribunal is confronted. In addition, Dr. Tomashefski and Dr. Crouch relied on somewhat different approaches in reaching the same conclusion that the Miner did not have coal workers' pneumoconiosis of any kind. Whereas Dr. Tomashefski articulated extensive reliance upon specified medical records relating to the Miner, Dr. Crouch's references were general and limited, and her rationale focused almost exclusively upon her assessment of the histolic slides. This tribunal therefore concludes, based upon the reasoned opinions of Dr. Tomashefski and Dr. Crouch that the evidence available to them, primarily that derived from the autopsy of the Miner, does not establish the existence of either simple or complicated coal workers' pneumoconiosis, and that the Miner did in fact not have coal workers' pneumoconiosis in any of its forms including complicated pneumoconiosis. It follows, as those physicians opined, that coal workers' pneumoconiosis was not a contributing cause of the Miner's death or disability prior to his death.[10]

Recognizing that autopsy evidence is deemed to be the most reliable basis for a diagnosis of pneumoconiosis, the result would be the same if the reports of Dr. Tomashefski and Dr. Perper were excluded from the evidentiary record pertaining to the Survivor's claim under the strictures of §725.414. Dr. Crouch's credentials are substantially more distinguished than Dr. Turgman's, and so, given the stark disparity of the conclusions of the two doctors, this tribunal would adopt Dr. Crouch's opinion as the more credible based upon the superior credentials. Consequently, under that scenario, proof of pneumoconiosis would fail. The opinion of Dr. Castle, even if admissible, and the opinions of Dr. Forehand and Dr. Mitchell, because of their deficient reasoning, would not have significant probative weight in this context. But the evidentiary record on which the determination would be made would be seriously truncated in a context of profound evidentiary conflict and inconsistency of Dr. Tomashefski's and Dr. Perper's reports were not considered.

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[10] Both Dr. Turjman and Dr. Perper diagnosed complicated coal workers' pneumoconiosis in the form of progressive massive fibrosis (PMF). In light of this tribunal's conclusion that proof of pneumoconiosis has failed, it is unnecessary to reach the issue of complicated pneumoconiosis with respect to the Survivor's claim, or, indeed, the Miner's claim. It should be noted, however, that there is no credible x-ray evidence of record which proves the existence of large x-ray opacities greater than one centimeter, and there is no evidence which proves the equivalence of any PMF lesions disclosed by autopsy and any large opacity of greater than one centimeter on x-ray pursuant to § 718.304. Consequently, a failure of proof in regard to complicated coal workers' pneumoconiosis is manifest without further elaboration. *See Scarbro*.

The Miner's Request for Modification: Change in Conditions or Mistake of Fact

In the absence of proof of the existence of coal workers' pneumoconiosis, there has been no change in conditions or mistake in a determination of fact which would support a modification of the last denial of the Miner's claim. Modification of a previous denial pursuant to § 725.310 requires Claimant to establish at the outset that there has been a material change in the Miner's pulmonary condition, or that there has been a mistake in a determination of fact. In order to establish entitlement to benefits with respect to the Miner's claim filed pursuant to Part 718, Claimant must establish the existence of pneumoconiosis, that the pneumoconiosis arose out of coal mine employment, and that the pneumoconiosis was totally disabling, or that the Miner was totally disabled by pneumoconiosis at the time of death. §§ 718.3, 718.202, 718.203, 718.204; *Gee v. W.G. Moore & Sons*, 9 B.L.R. 1-4 (1986) (en banc). Failure to establish any one of these elements precludes entitlement. *Trent v. Director, OWCP*, 11 B.L.R. 1-26 (1987); *Perry v. Director, OWCP*, 9 B.L.R. 1-1 (1986)(en banc).

Section 202(a) prescribes four bases for finding the existence of pneumoconiosis: (1) a properly conducted and reported chest x-ray, (2) a properly conducted and reported biopsy or autopsy, (3) reliance on the presumptions set forth in §§ 718.304, 718.305, and 718.306, or (4) a physician's finding of pneumoconiosis as defined by § 718.201 that is based on objective evidence and a reasoned medical opinion. The record has no evidence of a biopsy, and the presumption under §718.306 does not apply because the Miner died after March 1, 1978. Pneumoconiosis is defined in § 718.201 as a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments arising out of coal mine employment. Any chronic pulmonary disease which results in a respiratory or pulmonary impairment significantly related to dust exposure in coal mines comes within this definition of pneumoconiosis. Thus, "legal pneumoconiosis" exceeds the scope of "medical" or "clinical" pneumoconiosis.

Although the District Director awarded benefits to the Survivor by the proposed decision and order issued September 11, 2002, the procedural process which led to the award of benefits with respect to the request for modification in the Miner's claim is unclear. (D-20). Even if a claimant suffers from a totally disabling respiratory condition, he would not be eligible for benefits if he would have been totally disabled to the same degree because of his other health problems. *See Dehue Coal Co. v. Ballard,* 65 F.3d 1189, 1196 (4th Cir. 1995); *see also Shelton v. Director, OWCP,* 899 F.2d 690, 693 (7th Cir. 1990)(holding miner not entitled to benefits if he would have become disabled by reason of heavy smoking or other activity or condition). A medical opinion that acknowledges a miner's respiratory or pulmonary impairment, but concludes that an ailment other than pneumoconiosis caused the miner's total disability is relevant because it directly rebuts the miner's evidence that pneumoconiosis contributed to his disability. *See Dehue*; *Milburn Colliery Co. v. Hicks*, 138 F.3d 524, 21 B.L.R. 2-323 (4th Cir. 1998)

Claimant's request for modification is governed by § 725.310 of the preamendment regulations, which provides that any party may request modification of an award or denial of benefits if such request is filed within one year of the denial alleging a change in conditions or mistake in determination of fact. Where mistake of fact forms the grounds for the modification request, new evidence is not a prerequisite, and a mistake of fact may be corrected whether

demonstrated by new evidence, cumulative evidence, or further reflection on evidence initially submitted. *Kovac v. BCNR Mining Corporation,* 16 B.L.R. 1071 (1992), *modifying* 14 B.L.R. 1-156 (1990). If no specific mistake is alleged, but the ultimate determination entitlement is challenged, the entire record must be examined for a mistake in a determination of fact. *See Jessee v. Director, OWCP*, 5 F.3d 723, 18 B.L.R. 2-26 (4th Cir. 1993)

In determining whether the requesting party has established modification pursuant to § 725.310, the administrative law judge is obligated to perform an independent assessment of the newly submitted evidence, considered in conjunction with the previously submitted evidence, to determine if the weight of the new evidence is sufficient to establish the element or elements of entitlement which defeated entitlement in the prior decision. *Nataloni v. Director, OWCP*, 17 B.L.R. 1-82 (1993); *Kovac v. BCNR Mining Corp*., 14 B.L.R. 1-156 (1990), *modified on recon.,* 16 B.L.R. 1-71 (1992); *Wojtowicz v. Duquesne Light Co*., 12 B.L.R. 1-162 (1989); *O'Keeffe v. Aerojet-General Shipyards, Inc.*, 404 U.S. 254 (1971) The administrative law judge, as trier-of-fact, has the authority, and the duty, to review the record evidence *de novo* and is bound to consider the entirety of the evidentiary record, and not merely the newly submitted evidence, in determining whether a mistake in a determination of fact has been made in relation to a request for modification. *See Nataloni v. Director, OWCP*, 17 B.L.R. 1-82, 1-84 (1993); *Kovac v. BCNR Mining Corp.*, 14 B.L.R. 1-156 (1990); *see also Jessee*, 5 F.3d at 725, 18 B.L.R. at 2-28.

In dealing with the request for modification related to the Miner's claim, the Claimant must show a change in one of the elements previously decided against the Miner. Those elements were the absence of pneumoconiosis, and the failure of proof that pneumoconiosis was a causative factor in the Miner's disability. The Miner's death is not *per se* a change in conditions, although, as in this case, it makes available new evidence related to the autopsy, and, in that sense, could be deemed to reflect a change in conditions, as well as the possibility of establishing a mistake in a determination of fact. But the rubric in *Jessee* requires an examination of the entire record for a mistake in a determination of fact, in any event, since the Claimant, in effect, contends that the prior denial of the claim, though affirmed by the Benefits Review Board, was wrongly decided, and Claimant has offered the autopsy related evidence, as well as other evidence, in support of that contention.

The Merits of the Miner's Claim

The Miner's claim was filed in 1980, prior to January 1, 1982, and the Miner was employed for fifteen years or more in underground coal mines.[11] It is essentially undisputed, and this tribunal finds, that the Miner suffered from a totally disabling respiratory or pulmonary impairment at and before his death on June 18, 2001. There was a substantial preponderance of

[11] In his March 27, 1987, decision Judge Cox credited the Miner with twenty-seven and a half years of coal mine employment. (D-1[DX-48]) That finding has not been modified and has record support. Judge Levin adopted the unchallenged finding of twenty-seven and a half years of qualifying coal mine employment as the law of the case in his denials dated September 11, 1996, and July 17, 2000, ultimately affirmed by the Benefits Review Board on October 31, 2001. (D-1) Employer has contended that the Miner did not have sufficient underground coal mine employment to support invocation of the presumption under § 718.305, since he worked almost exclusively as a surface worker in strip mines or preparation facilities, which Employer contends would not be substantially similar to underground mine conditions. Since the contention is unsupported by persuasive evidence, it is deemed to be without merit, but the issue need not be resolved in light of the ultimate findings in this case.

negative x-ray interpretations by qualified readers regarding the existence of pneumoconiosis. Thus, the rebuttable presumptions that the Miner was totally disabled due to pneumoconiosis, that the Miner's death was due to pneumoconiosis, and that at the time of death the Miner was totally disabled by pneumoconiosis, are properly invoked with respect to the Miner's claim. § 718.305(a) and (c). The § 718.305 presumption can be rebutted by proof that the Miner's total disability was not caused in whole or in part from dust exposure in the miner's coal mine employment, or by proof that the Miner does not have pneumoconiosis. § 718.305(d). *See Mitchell v. Director, OWCP*, 25 F.3d 500 (7th Cir. 1994); *Summers v. Freeman United Coal Mining Co*., 14 F.3d 1220 (7th Cir. 1994).

Employer erroneously contends that there is no evidence sufficient to invoke any presumption provided by § 718.305. Because there were more than fifteen years of coal mine employment proved, and because the claim was filed in 1980, the rebuttable presumption under § 718.305 has been properly invoked. Early in the history of the case, when the Benefits Review Board vacated Judge Cox's finding that total disability was established under § 718.204(c)(2000) and remanded, the Board noted that, if on remand, Judge Cox were to find total disability established under §718.204(c)(2000), Claimant would be entitled to the rebuttable presumption that his total disability is due to pneumoconiosis at §718.305(2000) and that Judge Cox should consider rebuttal of the presumption pursuant to §718.305(d). The evidence which was before Judge Levin pursuant to that remand was essentially an accumulation of the most probative evidence in the record prior to the introduction of the new evidence in this case. There was substantial evidence which persuaded Judge Levin that the Miner had not proved the existence of pneumoconiosis. Judge Levin looked to that evidence in finding that the presumption had been rebutted. Essentially it consisted of medical opinions by Dr. Sargent and Dr. Castle that denied the existence of pneumoconiosis, the opinion of Dr. Garzon, which was challenged as inconsistent with the requirements of *Warth* and *Stilton*, and which, therefore, was safely disregarded when Judge Levin relied upon the opinions of Dr. Sargent and Dr. Castle.[12] Judge Levin considered the opinion of Dr. Rasmussen, that there was pneumoconiosis and that it contributed to the Miner's disability, less probative than the conflicting opinions of Dr. Sargent and Dr. Castle, because it relied upon a positive x-ray of Dr. Patel which was contradicted by the interpretations of better qualified readers.

Judge Levin found, therefore, that Employer had rebutted the presumptions invoked under § 718.305 by establishing by a preponderance of the evidence that the Miner did not have coal workers' pneumoconiosis as defined under the Act. There is no mistake of fact apparent in that finding, or in earlier findings that were not resolved on appeal or by Judge Levin. The conclusion which this tribunal reaches based on the relative credibility of the reports relating to the autopsy by Dr. Turjman, Dr. Perper, Dr. Tomashefski, Dr. Crouch, and Dr. Castle, presuming that all of those reports are admissible evidence with respect to the Miner's claim, is, as previously discussed, that the Miner did not have coal workers' pneumoconiosis of any kind, or a totally disabling pulmonary or respiratory impairment attributable to pneumoconiosis. The reports of Dr. Forehand and Dr. Mitchell, for reasons discussed previously, do not provide convincing proof to the contrary. Thus, the rebuttable presumptions under § 718.305, that the Miner was totally disabled by pneumoconiosis at the time of his death and that his death was

[12] *Warth v. Southern Ohio Coal Co*., 60 F.3d 173, 19 B.L.R. 2-265 (4th Cir. 1995); *Stiltner v. Island Creek Coal Co*., 86 F.3d 337, 20 B.L.R. 2-246 (4th Cir. 1996).

caused by pneumoconiosis have been rebutted by proof that he did not have pneumoconiosis. § 718.305(d). Claimant having failed to prove a change in conditions, a mistake in a determination of fact, or all the elements of entitlement under the Act with respect to the Miner's claim, benefits must be denied.

ORDER

The Survivor's claim for black lung benefits of Lorene Bucklen, and the request for modification and claim for black lung benefits of Lorene Bucklen on behalf of the deceased miner, Kermit Bucklen are denied.

A

Edward Terhune Miller
Administrative Law Judge