RECEIVED

MAR 0 2 2018

S.C. SUPREME COURT

THE STATE OF SOUTH CAROLINA
In The Supreme Court

APPEAL FROM SUMTER COUNTY
Court of Common Pleas

The Honorable W. Jeffrey Young, Circuit Court Judge

Appellate Case No. 2017-001955

Arrowpoint Capital Corporation/Arrowood Indemnity Co., Carrier .....Respondent,

v.

South Carolina Second Injury Fund, ...Petitioner.

[In re: C.L. Williams, Employee/Claimant v. Yuasa Exide, Incorporated, Employer]

APPENDIX – VOLUME I

Latonya Dilligard Edwards, Esquire
Dilligard Edwards, LLC
3790 Fernandina Road, Suite 103
Columbia, South Carolina 29210
(803) 750-2214

ATTORNEY FOR PETITIONER

Vernon F. Dunbar, Esquire
Stephanie L. Pugh, Esquire
McAngus Goudelock & Courie, LLC
Post Office Box 2980
East Camperdown Way, Suite 300 (29601)
Greenville, South Carolina 29602
(864) 239-4000

ATTORNEYS FOR RESPONDENT

INDEX

SC Court of Appeals Record on Appeal 1

THE STATE OF SOUTH CAROLINA
In The Court of Appeals

APPEAL FROM SUMTER COUNTY
Court of Common Pleas

The Honorable W. Jeffrey Young, Circuit Court Judge

Case No. 2013-CP-43-02286
Appellate Case No. 2014-002214

Arrowpoint Capital Corporation/Arrowood Indemnity Co., ...Appellant,

v.

South Carolina Second Injury Fund.....Respondent,

[In re: C.L. Williams, Employee/Claimant

v.

Yuasa Exide, Incorporated, Employer]

RECORD ON APPEAL

Vernon F. Dunbar, Esquire
Stephanie Lamb Pugh, Esquire
McAngus, Goudelock & Courie LLC
Post Office Box 2980
55 East Camperdown Way, Suite 300 (29601)
Greenville, South Carolina 29602
Attorneys for Appellants
(864) 239-4000

Latonya D. Edwards, Esquire
3790 Fernandina Road, Suite 103
Columbia, South Carolina 29210
Attorney for Respondent
(803) 750-2214

INDEX

Decision and Order of Circuit Court (September 16, 2014) 1-7

Appellate Panel Decision and Order (August 13, 2013) 8-15

Hearing Commissioner's Decision and Order (November 27, 2012) 16-24

Settlement Agreement, Release and Order and Form 19 (October 25, 2010) 25-36

Consent Scheduling Order (August 13, 2009) 37-43

Letter to Circuit Court Judge Enclosing Supreme Court Decision (July 31, 2014) . 44-54

Form 54, Employer's Request for Hearing (January 27, 2012) 55-56

Form 55, Second Injury Fund's Answer to Employer's Request for Hearing (February 27, 2012) 57-58

Form 58, Employer's Pre-Hearing Brief (July 27, 2012) 59-60

Form 58, Second Injury Fund's Pre-Hearing Brief (August 3, 2012) 61-63

Form 30, Employer's Request for Commission Review (December 7, 2012) 64-65

Transcript of Hearing Before Circuit Court (June 3, 2014) 66-130

Transcript of Hearing Before Full Commission (September 16, 2013) 131-159

Transcript of Hearing Before Hearing Commissioner (August 13, 2012) 160-174

Employer's APA Submissions (July 27, 2012) 175-269

Second Injury Fund's APA Submissions (August 3, 2012) 270-272

"Overview of Lead Cases" Binder (August 13, 2012) 273-444

Memorandum of Law (August 10, 2012) 445-456

Certificate of Compliance 457

RECORDED
2014 SEP 16 PM 1:21
JAMES C. CAMPBELL
CLERK OF COURT
SUMTER COUNTY, S.C.

STATE OF SOUTH CAROLINA)	IN THE COURT OF COMMON PLEAS
)	OF THE THIRD JUDICIAL CIRCUIT
COUNTY OF SUMTER)	
)	
Arrowpoint Capital Corporation,)	C/A CASE NO.: 2013-CP-43-02286
)	
Plaintiff/Appellant,)	
)	
v.)	
)	
South Carolina Second Injury Fund,)	
)	
Defendant,)	
)	
[In Re: C.L.Williams,)	
)	
Employee/Claimant,)	
)	
v.)	
)	
Yuasa Exide, Incorporated,)	
)	
Employer.])	

CERTIFIED TRUE COPY
OF ORIGINAL FILED
DEPUTY CLERK OF COURT
SUMTER COUNTY
SOUTH CAROLINA

ORDER

STATEMENT OF THE CASE

This is a claim for partial reimbursement from the South Carolina Second Injury Fund ("the Fund") by Yuasa Exide Incorporated, Employer, and Arrowpoint Capital Corporation, Carrier (collectively "Carrier"), pursuant to S.C. Code Ann. § 42-9-400. The underlying case was settled by Employer/Carrier and Claimant without a hearing. In this reimbursement claim, Carrier alleged that they incurred substantially greater liability for compensation and medical benefits when employee C.L. Williams' ("Claimant") preexisting hypertension, diabetes, chronic obstructive pulmonary disease, cardiomyopathy, pulmonary disease, cardiovascular disease, arterial sclerosis, and renal insufficiency were either aggravated by or combined with his February 1, 1999 work

related occupational exposure. Carrier further alleged that Claimant's preexisting conditions were permanent and serious enough to constitute a hindrance or obstacle to employment. The Fund asserted that Claimant did not have any of the alleged conditions prior to the heavy metal exposure and if these conditions preexisted, they were not permanent and serious enough to constitute a hindrance or obstacle to Claimant's employment as per S.C. Code Ann. § 42-9-400. The Fund also asserted that the claim was barred by S.C. Code Ann. § 42-7-320(B) and its subsections.

The Hearing Commissioner denied Carrier's reimbursement request pursuant to S.C. Code Ann. § 42-9-400 and the Full Commission, En Banc, affirmed.

STANDARD OF REVIEW

The standard of review for decisions of the Workers' Compensation Commission is established in the Administrative Procedures Act. South Carolina Second Injury Fund v. Liberty Mutual Insurance Co., 353 S.C. 117, 576 S.E.2d 199 (S.C. App. 2003). A reviewing court must not disturb the Workers' Compensation Commission's findings if those findings are supported by substantial evidence in the record. Pearson v. JPS Converter & Indus. Corp., 327 S.C. 393, 489 S.E.2d 219 (S.C. App. 1997). The fact that reasonable minds may differ or that there is the possibility of drawing inconsistent conclusions does not prevent an agency's findings from being supported by substantial evidence. Grant v. South Carolina Coastal Council, 319 S.C. 348, 461 S.E. 2d 388 (S.C. 1995).

DISCUSSION

After reviewing the evidence, and hearing the oral arguments of the parties, I affirm. Carrier argued that the decision below should be reversed. I disagree.

The statutory reimbursement scheme requires that Carrier establish several elements to receive reimbursement recovery. Section 42-9-400 requires that (1) Claimant have a permanent preexisting impairment (§42-9-400(a)); (2) that the permanent preexisting impairment create substantially greater liability for medical costs and permanent disability than that which would have resulted from the work injury alone (§42-9-400(a)); (3) that Carrier had knowledge of the permanent preexisting impairment (§42-9-400(c)); (4) that the permanent preexisting impairment was permanent and serious enough to constitute a hindrance or obstacle to employment or reemployment (§42-9-400(d)); and, (5) that Carrier provide the Fund with notice of a potential claim prior to the payment of seventy-eight (78) weeks of compensation (§42-9-400(f)). Furthermore, case law instructs that a Carrier's right to receive reimbursement from the South Carolina Second Injury Fund depends upon complete compliance with the requirements for recovery. South Carolina Second Injury Fund v. American Yard Products, 496 S.E.2d 862 (S.C. 1998).

The substantial evidence in the record supports the fact that Claimant did not have the alleged conditions prior to his occupational exposure. On December 12, 1983, Claimant's physical examination indicated that there were no findings of lead involvement and that he could continue working in his area. ROA p.97. On January 11, 1984, Claimant's physical examination revealed a past history of hypertension and mild cirrhosis. ROA p.98. In February, April and September 1984, Claimant's physical examinations revealed that he could function safely in his present job without complications. ROA p.100. Claimant's subsequent physical examinations from 1988 to

1997 revealed hypertension, and a history of elevated lead levels. ROA pp. 103-104, 106, 108, 110, 112, 114 and 116.

In January 1994, Claimant presented to the Davis Clinic for evaluation of fluid retention, congestive heart failure, fever and cough. ROA pp. 133-134. Claimant was diagnosed with bronchitis and his treatment plan included prescription medication for his bronchitis and congestive heart failure and a two (2) week follow up. ROA p.134. Claimant continued to follow up with the Davis Clinic for his hypertension, and congestive heart failure. ROA pp. 135-140.

Although there is evidence Claimant had a history of high blood pressure in 1982, the records indicate lead exposure dating back to 1976. There is no evidence that the high blood pressure preexisted the lead exposure. The evidence actually indicates that the lead exposure may have caused the elevated blood pressure. ROA pp. 129-132. Furthermore, Claimant did not develop any of the conditions alleged until after his occupational exposure. ROA pp. 141-145. The Commission's decision was supported by substantial evidence in the record.

Substantial evidence also supported the Commission's decision that Claimant's occupational exposure was not his first and subsequent injuries. The Commission determined that Claimant was exposed to lead over a twenty–five (25) year period of employment and that exposure constituted one (1) injury. That decision is supported by the plain meaning of the statute. Section 42-9-400(a) references "subsequent disability," "preexisting impairment," and "subsequent injury." Claimant's alleged preexisting conditions include hypertension, diabetes, chronic obstructive pulmonary disease,

cardiomyopathy, pulmonary disease, cardiovascular disease, arterial sclerosis, and renal insufficiency.

The plain meaning of the statute requires that these conditions predate the subsequent injury, which is the occupational exposure and is cumulative. In other words, the cumulative exposure is one (1) injury; it is not the first and subsequent injuries, nor is it the preexisting condition and the subsequent injury. Claimant sustained one (1) injury that culminated on February 1, 1999. The medical evidence and the statutory language support the Commission's determination that Claimant's conditions did not exist prior to his occupational exposure.

Carrier cited various cases to support its position that the Commission's decision was not supported by substantial evidence. In State Workers' Compensation Fund v. SC SIF, 313 S.C. 536, 443 S.E.2d 546 (1994), the South Carolina Supreme Court decided the issues partly based on a statute that applied specifically to law enforcement officers, including firefighters, which presumes that heart disease arises out of and in the course of a firefighter's employment. 313 S.C. 536 at 539; 443 S.E.2d at 548 (S.C. 1994). See, S.C. Code Ann. § 42-11-30. In that case, Claimant had a definitive diagnosis of coronary artery disease prior to a subsequent injury. In this case, Claimant did not have a diagnosis prior to the date of his alleged total disability.

In Springs Industries, Inc. v. S.C. Second Injury Fund, the issue was whether Claimant sustained a subsequent injury. 296 S.C. 359, 372 S.E.2d 915 (S.C.App. 1988). In Springs, Claimant worked in the cotton industry for thirty-one (31) years and then became totally disabled after working for another employer for nine (9) months. The Court determined that Claimant's thirty-one (31) years of prior cotton dust exposure was

the "but for" cause of the subsequent injury pursuant to S.C. Code Ann. § 42-9-400(g). This case did not involve the same issue or analysis. In both State Workers' Compensation Fund and Springs, the underlying cases were actually adjudicated and Carriers actually paid the claimants' compensation and medical benefits, unlike Carrier in this case.

The Commission's decision is also supported recent case law. In Carolinas Recycling, the South Carolina Court of Appeals reversed the decision of the Commission because the Appellate Panel "relied exclusively upon an evaluation by a non-treating physician who only met with the Claimant on one occasion." 398 S.C. 480, 485, 730 S.E.2d 324, 327 (Ct.App. 2012). Here, like Carolinas Recycling, the hindrance element was not an issue. Also, while two (2) physicians supported reimbursement in this case, neither of those physicians provided treatment to Claimant. Consistent with Carolinas Recycling, the Commission refused to rely on physicians who did not provide any treatment to Claimant.

Carrier also cited Burnette v. City of Greenville to support its assertion that the Commission's decision was not supported by substantial evidence in the record. 401 S.C. 417, 737 S.E.2d 200 (2012). I disagree. In Burnette, the South Carolina Court of Appeals reversed and remanded the case to the Commission with instructions to either reconsider or enter findings of fact concerning Claimant's injuries because the Commission's decision was based on the medical opinion of a single commissioner rather than a medical provider. Id. Burnette is clearly distinguished from this case because it was decided solely on medical issues that required an interpretation of diagnostic tests such as MRIs and CT scans. Here, the issues appear to be one of statutory interpretation.

It is well settled that an agency's interpretation of its own statutes will be given great deference unless compelling reasons require otherwise. CFRE, LLC v. Greenville County Assessor, 395 S.C. 67, 77, 716 S.E.2d 877, 882 (S.C. 2011). While Carrier submitted medical certificates supporting the elements of reimbursement, the Commission is not required to give medical questionnaires conclusive effect to the exclusion of other more compelling medical evidence. Ballenger v. Southern Worsted Corporation, 209 S.C. 463, 467, 40 S.E.2d 681, 682-83 (S.C. 1946).

ORDER

IT IS HEREBY ORDERED, ADJUDGED, AND DECREED that the Order of the South Carolina Workers' Compensation Commission denying Carrier's claim for reimbursement from the South Carolina Second Injury Fund pursuant to S.C. Code Ann. § 42-9-400 was supported by substantial evidence and is AFFIRMED.

AND IT IS SO ORDERED this 5 day of Sept 2014.

W. Jeffrey Young, Presiding Circuit Court Judge

SCANNED

E-MAILED

EN BANC

APPELLATE PANEL

DECISION AND ORDER

OF THE

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION

W.C.C. FILE NO. 9930399

ARROWPOINT CAPITAL CORPORATION,..... CARRIER/APPELLANT,

- V -

SC SECOND INJURY FUND,..... DEFENDANT/RESPONDENT.

[In Re: C.L. Williams v. Yuasa-Exide, Incorporated et. al]

Appellate Panel Review held in Columbia, South Carolina on September 16, 2013, per notices timely and properly served on all parties.

Appellate Panel Decision and Order filed 11-21, 2013.

APPEARANCES: Carrier/Appellant represented by Stephanie L. Pugh, Esquire, of Turner Padget, Greenville, South Carolina.

Defendant/Respondent represented by Latonya Dilligard Edwards, Esquire, of Dilligard Edwards, LLC, Columbia, South Carolina.

Appeal to Circuit Due 12/20/13

NOV 21 2013

3180.167

STATEMENT OF CASE

On August 13, 2012, Commissioner Andrea C. Roche held a hearing on this matter in Sumter, South Carolina and issued the following Order:

> "Based upon the foregoing Findings of Fact and Conclusions of Law, IT IS SO ORDERED that Carrier's claim for reimbursement pursuant to S.C. Code Ann. § 42-9-400 is DENIED and DISMISSED with prejudice subject to Carrier's statutory right to appeal."

Within the statutory period, counsel for the Employer and Carrier filed a Request for Commission Review setting forth the grounds for appeal with copies furnished to all interested parties prior to being presented to the Appellate Panel on September 16, 2013. All proffered testimony has been taken and together with all documentary evidence has been delivered to the individual members of the Full Commission and has since been under advisement and consideration.

By appeal, Employer and Carrier respectfully submitted the following:

1. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that Claimant did not have preexisting hypertension, cardiovascular disease, heavy metal poisoning, chronic obstructive pulmonary disease (COPD), pulmonary hypoventilation syndrome, rectal bleeding, coronary artery disease, pulmonary edema, tinnitus, loss of hearing and vertigo prior to his February 1, 1999 work injury.

2. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that Claimant did not have any impairment that preexisted the lead exposure.

3. The Hearing Commissioner abused her discretion in finding as a fact and

concluding as a matter of law that a finding that hypertension predated the lead exposure would be speculative.

4. The Hearing Commissioner abused her discretion in finding that the medical questionnaires submitted were not supported by the medical evidence.

5. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the lead exposure may have actually caused Claimant's high blood pressure.

6. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the Claimant's heavy metal exposure over the twenty-five (25) year period constitutes one injury.

7. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that Claimant did not suffer from any preexisting conditions and thus there was no combination with or aggravation of preexisting conditions to create substantially greater medical costs and permanent disability.

8. The Hearing Commissioner erred in failing to find as a fact and conclude as a matter of law that Carrier proved that Claimant had preexisting conditions that were permanent and serious enough to be a hindrance or obstacle to employment.

9. The Hearing Commissioner legally erred and abused her discretion in denying Employer and Carrier's claim in view of the recently promulgated cases of Carolinas Recycling Grp. v. SC Second Injury Fund, 398 S.C. 480, 485, 730 S.E.2d 324, 327 (Ct. App. 2012) and Burnette v. City of Greenville, 401 S.C. 417, 428, 737 S.E. 2d 200, 206 (S.C. Ct. 2012).

10. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that Claimant did not suffer an injury by accident or failing to find such with regard to Claimant's exposure to lead beginning in 1976 and ending on February 1, 1999.

11. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that Carrier has not met all necessary requirements for reimbursement pursuant to §42-9-400.

In accordance with S.C. Code Ann. § 42-17-50, the Appellate Panel shall review the award, and if good and proper grounds are shown, may reconsider evidence, receive further evidence, rehear the parties and amend the award. Furthermore, the Appellate Panel may make its own findings of fact and conclusions of law. Lowe v. Am-Can Transp. Serv., Inc., 283 S.C. 534, 324 S.E.2d 87 (Ct. App. 1984). After careful review in the instant case, the Commission has determined that all of the Hearing Commissioner's Findings of Fact and Conclusions of Law are correct as stated. Accordingly, they shall become the law of the case, and are sustained in their entirety.

FINDINGS OF FACT

Based upon the foregoing, to include review of the Commission's file and all evidence submitted, we make the following findings of fact based upon the preponderance of the reliable, probative, and substantial evidence:

1. On February 1, 1999, Claimant sustained an occupational exposure to heavy metal/lead during the course of his employment with Yuasa-Exide, Incorporated. This finding is based upon the evidence in the record.

2. The South Carolina Second Injury Fund was placed on notice of this potential reimbursement claim on April 17, 2008. This is based on the parties' stipulations.

3. Claimant did not have preexisting hypertension, cardiovascular disease, heavy metal poisoning, chronic obstructive pulmonary disease, pulmonary hypoventilation syndrome, rectal bleeding, coronary artery disease, pulmonary enema, tinnitus, loss of hearing and vertigo prior to his occupational exposure. This finding is based upon the totality of the medical evidence.

4. Section 42-9-400(a) references a "subsequent disability," "preexisting impairment," and "subsequent injury." The injury for which reimbursement is sought is lead exposure. There is no evidence that Claimant had any impairment that preexisted the lead exposure. This finding is based upon S.C. Code Ann. § 42-9-400(a) and all evidence in the record.

5. Claimant was exposed to lead over a twenty–five (25) year period of employment; and, as such, we find that the heavy metal exposure is one injury. This finding is based upon all evidence in the record.

6. Although there is evidence Claimant had a history of high blood pressure in 1982, Carrier submitted records showing lead exposure back in 1976. To find that high blood pressure preexisted the lead exposure would be speculative. The evidence in the record indicates that the lead exposure may have actually caused the elevated blood pressure. Thus, we further find that the questionnaires submitted by Carrier are not supported by the other evidence in the record. This finding is based on Carrier APA pp. 40-43 and all evidence in the record.

7. Since we find that Claimant did not have any preexisting conditions, we also find that there was no combination or aggravation of the preexisting conditions by the subsequent injury to create substantially greater medical costs and permanent disability. This finding is based on the totality of the evidence in the record.

8. Carrier failed to meet its burden in establishing all necessary elements for partial reimbursement pursuant to S.C. Code Ann. § 42-9-400. Accordingly, Carrier is not entitled to reimbursement from the South Carolina Second Injury Fund.

CONCLUSIONS OF LAW

Based upon the foregoing and applicable statutory and case law, we conclude as a matter of law:

1. Reimbursement from the South Carolina Second Injury Fund is governed by S.C. Code Ann. § 42-9-400.

2. The right of Carrier to receive reimbursement from the South Carolina Second Injury Fund depends upon complete compliance with the requirements for recovery. S. C. Second Injury Fund v. Amer. Yard Prod., 330 S.C. 20, 496 S.E.2d 862 (1998).

3. To qualify for reimbursement, Carrier had the burden to prove that Claimant had a preexisting condition that was permanent and serious enough to be a hindrance or obstacle to his employment. Carrier failed to meet its burden of proof on this issue; and, therefore, failed to meet all requirements for reimbursement pursuant to S.C. Code Ann. § 42-9-400.

4. To qualify for reimbursement Carrier had the burden to prove that Claimant's preexisting condition was either aggravated by or combined with his work related injury to create substantially greater medical costs and permanent disability. Carrier failed to meet its burden of proof on this issue; and, therefore, failed to meet all requirements for reimbursement pursuant to S.C. Code Ann. §42-9-400.

5. Since Carrier did not meet all requirements for reimbursement pursuant to S.C. Code Ann. § 42-9-400, it is not entitled to reimbursement from the South Carolina Second Injury Fund, and its claim is denied.

ORDER

IT IS THEREFORE ORDERED that the Order of the Single Commissioner filed on November 27, 2012, is hereby affirmed by the Appellate Panel, and constitutes the Decision and Order of the Full Commission.

IT IS FURTHER ORDERED that Carrier's claim for reimbursement pursuant to S.C. Code Ann. § 42-9-400 is DENIED and DISMISSED with prejudice subject to Carrier's statutory right to appeal.

S.C. WORKERS' COMPENSATION COMMISSION

Gene McCaskill, Commissioner

FULL AFFIRMATION

CONCUR:

Avery B. Wilkerson, Jr., Commissioner

Melody James, Commissioner

Susan S. Barden, Commissioner

Aisha Taylor, Commissioner

T. Scott Beck, Commissioner

CERTIFICATE OF SERVICE

This is to certify that the undersigned has on this date served a copy of this order in the above entitled action upon all parties to this case by sending an electronic copy hereof by electronic mail addressed to the attorneys for said parties; or if there is an unrepresented party(ies), by depositing a copy hereof, postage paid in the United States mail, first class, addressed to the unrepresented party(ies) and to the attorney(s) for the represented party(ies).

By Valerie Deller on November 21, 2013

DECISION AND ORDER

OF THE

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION

SCWCC NO. 9930399; SCSIF NO. 141031

ARROWPOINT CAPITAL CORPORATION

V.

SOUTH CAROLINA SECOND INJURY FUND

[In Re: C.L. Williams, Employee v. Yuasa Exide, Incorporated, Employer]

HEARING:	August 13, 2012, in Sumter, South Carolina.
APPEARANCES:	Yuasa Exide, Incorporated, Employer, and Arrowpoint Capital Corporation, Carrier, represented by Vernon F. Dunbar, Esquire, of Turner Padget Graham & Laney, P.A., Greenville, South Carolina. The South Carolina Second Injury Fund represented Latonya Dilligard Edwards, Esquire, of Dilligard Edwards, LLC, Columbia, South Carolina.
PURPOSE OF HEARING:	To determine issues as set forth in the Forms 54 and 55.
OPINION AND AWARD:	By Andrea C. Roche, Commissioner
FILED:	November 27, 2012

MAILED

E-MAILED

ADMINISTRATIVE PROCEDURE ACT (APA) SUBMISSIONS

APA NO.	ITEM	DATE	PAGES
1	Second Injury Fund Questionnaire of Dr. Edward L. Baker, Jr.	06/26/11	1-3
2	Second Injury Fund Questionnaire of Dr. Eugene Shippen	05/31/11	4-6
3	Yuasa Exide Health History documents	01/20/82 - 08/12/98	7-31
4	Yuasa Exide Periodic Health History Questionnaire	05/24/93	32-35
5	Yuasa Exide Occupational Health History Questionnaires	08/31/86	36-39
6	Claimant's Blood Lead Levels	03/15/76 – 07/02/85	40-43
7	Davis Clinic	01/19/84 – 01/26/99	44-59
8	Post Yuasa Exide Medical Records	02/15/02 – 11/14/07	60-88
EXHIBITS			
A	Second Injury Fund Correspondence	09/20/11	89
B	Claimant's Knowledge Affidavit	10/20/10	90-92

ADDITIONAL APA SUBMISSIONS BY CARRIER:

1. Article regarding lead

   a) Health Effects of Chronic Lead Exposure among Employees of Exide Battery Production Plant – Sumter SC

   b) Lead-related nephrotoxicity: A review of the epidemiologic evidence

   c) Literature Review – Lead and Health Effects

   d) Statistical Data Concerning Hypertension, Diabetes, Renal Kidney Failure, and Memory Loss in Society

   e) Case Studies in Environmental Medicine – Lead Toxicity

2. OSHA and Yuasa-Exide Regulations

   a) Electronic Code of Federal Regulations

   b) Lead Control Program – Yuasa-Exide Regulations

3. Commission and Court cases involving *Lee Ernest Franklin*

   a) Employee's Notice of Claim and/or Request for Hearing dated July 3, 2001

   b) Decision and Order of Commissioner George N. Funderburk dated June 10, 2005

   c) Appellate Panel Decision and Order of the South Carolina Workers' Compensation Commission dated May 3, 2006

   d) Decision and Order of Commissioner George N. Funderburk dated January 31, 2008

   e) Decision of the Supreme Court of South Carolina, Opinion No.: 26880 dated September 13, 2012

STIPULATIONS

The parties stipulated to the following:

1. The South Carolina Workers' Compensation Commission had jurisdiction to hear the case.

2. Venue was proper in Sumter County.

3. Notice was timely and proper.

4. The Commission's file became a part of the record with the exception of self-serving declarations and unstipulated medical reports.

STATEMENT OF THE CASE

This is a claim for partial reimbursement from the South Carolina Second Injury Fund ("the Fund") by Yuasa Exide Incorporated, Employer, and Arrowpoint Capital Corporation, Carrier (collectively "Carrier"), pursuant to S.C. Code Ann. § 42-9-400. Carrier alleged that they incurred substantially greater liability for compensation and medical benefits when employee C.L. Williams' ("Claimant") preexisting diabetes, hypertension, heavy metal poisoning, chronic obstructive pulmonary disease, pulmonary hypoventilation syndrome, rectal bleeding, coronary artery disease, pulmonary enema, tinnitus, loss of hearing and vertigo were either aggravated by or combined with his February 1, 1999 work related occupational exposure. Carrier further alleged that Claimant's preexisting conditions were permanent and serious enough to constitute a hindrance or obstacle to employment. The Fund asserted that Claimant did not have any of the alleged conditions prior to the heavy metal exposure and if these conditions preexisted, they were not permanent and serious enough to constitute a hindrance or obstacle to Claimant's employment as per S.C. Code Ann. § 42-9-400. The Fund also asserted that the claim was barred by S.C. Code Ann. § 42-7-320((B) and its subsections.

EVIDENCE OF THE CASE

On February 1, 1999, Claimant sustained alleged injuries to his brain, kidneys, cardiovascular and vestibular systems due to heavy metal/lead exposure during the course of his employment with Yuasa Exide Incorporated.

On December 12, 1983, Claimant's physical examination indicated that there were no findings of lead involvement and that he could continue working in his area. Carrier APA p.8. On January 11, 1984, Claimant's physical examination revealed a past history of hypertension and mild cirrhosis. Carrier APA p.9. In February, April and September 1984, Claimant's physical examinations revealed that he could function safely in his present job without complications. Carrier APA p.11. Claimant's subsequent physical examinations from 1988 to 1997 revealed hypertension, and a history of elevated lead levels. Carrier APA pp.14-15, 17, 19, 21, 23, 25, and 27.

In January 1994, Claimant presented to the Davis Clinic for evaluation of fluid retention, congestive heart failure, fever and cough. Carrier APA pp.44-45. Claimant was diagnosed with bronchitis and his treatment plan included prescription medication for his bronchitis and congestive heart failure and a two (2) week follow up. Carrier APA p.45. Claimant continued to follow up with the Davis Clinic for his hypertension, and congestive heart failure. Carrier APA pp.46-51.

On March 13, 1998, Claimant was diagnosed with severe depressed left ventricular function and was prohibited from engaging in heavy and repetitive lifting. Carrier APA p.30. On August 12, 1998, Claimant was terminated for health related problems and began receiving social security disability benefits. Carrier APA p.31. Claimant continued to follow up with his hypertension, diabetes, chronic obstructive pulmonary disease and cardiomyopathy. Carrier APA pp.60-80. On January 1, 2005,

Claimant presented to Tuomey Healthcare System for consultation regarding his congestive heart failure, where his medication was modified and medical therapy was offered. Carrier APA pp.81-84.

On June 13, 2006, Claimant followed up regarding his congestive heart failure and hypertension. Carrier APA p.85. Claimant's treatment plan included a thyroid profile, lipid panel, a continuation of his present medication and follow up. Carrier APA p.85.

On May 31, 2011, Dr. Eugene Shippen, of Wyomissing, Pennsylania, completed a questionnaire supporting reimbursement with respect to Claimant's alleged preexisting conditions. Carrier APA pp.4-6. On June 26, 2011, Dr. Edward L. Baker, Jr., completed a questionnaire supporting Second Injury Fund reimbursement with respect to Claimant's alleged preexisting conditions. Carrier APA pp.1-3. On October 20, 2010, Claimant completed an affidavit indicating that he was unaware that he had any preexisting conditions. Carrier APA pp.90-93.

FINDINGS OF FACT

Based upon the foregoing, to include review of the Commission's file and all evidence submitted, I make the following findings of fact based upon the preponderance of the reliable, probative, and substantial evidence:

1. On February 1, 1999, Claimant sustained an occupational exposure to heavy metal/lead during the course of his employment with Yuasa Exide, Incorporated. This finding is based upon the evidence in the record.

2. The Fund was placed on notice of this potential reimbursement claim on April 17, 2008. This is based on the parties' stipulations.

3. Claimant did not have preexisting hypertension, cardiovascular disease, heavy metal poisoning, chronic obstructive pulmonary disease, pulmonary hypoventilation syndrome, rectal bleeding, coronary artery disease, pulmonary enema, tinnitus, loss of hearing and vertigo prior to his occupational exposure. This finding is based upon the totality of the medical evidence.

4. Section 42-9-400(a) references a "subsequent disability," "preexisting impairment," and "subsequent injury." The injury for which reimbursement is sought is lead exposure. There is no evidence that Claimant had any impairment that preexisted the lead exposure. This finding is based upon S.C. Code Ann. § 42-9-400(a) and all evidence in the record.

5. Claimant was exposed to lead over a twenty-five (25) year period of employment; and, as such, I find that the heavy metal exposure is one injury. This finding is based upon all evidence in the record.

6. Although there is evidence Claimant had a history of high blood pressure in 1982, Carrier submitted records showing lead exposure back in 1976. To find that high blood pressure preexisted the lead exposure would be speculative. The evidence in the record indicates that the lead exposure may have actually caused the elevated blood pressure. Thus, I further find that the questionnaires submitted by Carrier are not supported by the other evidence in the record. This finding is based Carrier APA pp.40-43 and all evidence in the record.

7. Since I find that Claimant did not have any preexisting conditions, I also find that there was no combination or aggravation of the preexisting conditions by the subsequent injury to create substantially greater medical costs and permanent disability. This finding is based on the totality of the evidence in the record.

8. Carrier failed to meet its burden in establishing all necessary elements for partial reimbursement pursuant to S.C. Code Ann. § 42-9-400. Accordingly, Carrier is not entitled to reimbursement from the South Carolina Second Injury Fund.

CONCLUSIONS OF LAW

Based upon the foregoing and applicable statutory and case law, I conclude as a matter of law:

1. Reimbursement from the South Carolina Second Injury Fund is governed by S.C. Code Ann. § 42-9-400.

2. The right of Carrier to receive reimbursement from the South Carolina Second Injury Fund depends upon complete compliance with the requirements for recovery. South Carolina Second Injury Fund v. American Yard Products, 496 S.E.2d 862 (S.C. 1998).

3. To qualify for reimbursement, Carrier had the burden to prove that Claimant had a preexisting condition that was permanent and serious enough to be a hindrance or obstacle to Claimant's employment. Carrier failed to meet its burden of proof on this issue; and, therefore, failed to meet all requirements for reimbursement pursuant to S.C. Code Ann. § 42-9-400.

4. To qualify for reimbursement Carrier had the burden to prove that Claimant's preexisting condition was either aggravated by or combined with his work related injury to create substantially greater medical costs and permanent disability. Carrier failed to meet its burden of proof on this issue; and, therefore, failed to meet all requirements for reimbursement pursuant to S.C. Code Ann. §42-9-400.

5. Since Carrier did not meet all requirements for reimbursement pursuant to S.C. Code Ann. § 42-9-400, it is not entitled to reimbursement from the South Carolina Second Injury Fund, and its claim is denied.

AWARD

Based upon the foregoing Findings of Fact and Conclusions of Law, IT IS SO ORDERED that Carrier's claim for reimbursement pursuant to S.C. Code Ann. § 42-9-400 is DENIED and DISMISSED with prejudice subject to its statutory right to appeal.

Commissioner Andrea C. Roche

CERTIFICATE OF SERVICE

This is to certify the undersigned has this date served this order in the above entitled action upon all parties to this cause by sending an electronic copy hereof by electronic mail addressed to the attorney or attorneys for said parties or by depositing a copy hereof, postage paid, in the United States certified mail addressed to any unrepresented party.
November 27, 2012

By: Barbara Cheeseboro, Administrative Assistant to Commissioner Roche

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION

W.C.C. FILE NO: 9930399

C. L. Williams,

Employee,

Claimant,

vs.

Yuasa-Exide, Inc.,

Employer,

AND

Arrowpoint Capital Corporation,

Carrier,

Defendants.

SETTLEMENT AGREEMENT, RELEASE AND ORDER

APPROVED

OCT 26 2010

S. C. Workers' Comp. Comm.

WHEREAS, the undersigned, C.L. Williams, hereinafter referred to as Claimant, allegedly sustained injuries to his brain, kidneys, liver, musculoskeletal system, cognitive, pulmonary, and neuropathic systems, eyes, hearing, and genitourinary systems; or contracted an occupational disease as a consequence of his employment duties, on or about February 1, 1999. Claimant allegedly sustained these aforesaid injuries because of performing employment duties during the course and scope of his employment with Yuasa-Exide, Inc., hereinafter referred to as Employer.

Claimant asserts that occupational exposure to lead, toxins, and other chemicals; exposure to loud noises; specific work-related accident(s); and by virtue of repetitive trauma, he sustained injuries to the aforesaid body parts and aggravations of

Path received 11/3/10 TNP 03180.00167

pre-existing conditions and maladies. Claimant's primary complaints are as follows: hypertension, diabetes, cardiomyopathy, high cholesterol, COPD, pulmonary hypoventilation syndrome, pulmonary edema, coronary artery disease, hyperlipidemia, renal insufficiency, and blurred vision.

The Claimant has a history of diabetes, hypertension, cardiomyopathy, bladder outlet obstruction, COPD, pulmonary hypoventilation syndrome, rectal bleeding, bronchitis, coronary artery disease, pulmonary enema, obstructive pulmonary disease, loss of vision, tinnitus, loss of hearing, and vertigo. Claimant underwent an medical evaluation with Drs. Edward L. Baker, J. Routt Reigart, and Barry Weissglass on May 22, 2008. Claimant was assigned the following impairment ratings: 49% medical impairment to the heart; 8% medical impairment to the cardiovascular system due to hypertension; 4% medical impairment to the vestibular system due to vertigo; 5% medical impairment to the ears; and 29% medical impairment to the brain due to dementia and toxic encephalopathy.

WHEREAS, Arrowpoint Capital Corporation, hereinafter referred to as Carrier, is the Insurance Carrier for the Employer; and

WHEREAS, all references to Yuasa-Exide, Inc. and Arrowpoint Capital Corporation shall include said entities and their respective predecessors, successors, assigns, parent companies, subsidiaries and/or related companies; and

WHEREAS, the average weekly wage is $177.75 and the compensation rate is $118.50. The parties admit that during the year 1997, Claimant's gross wages were $29,624.27. The Employer and Carrier deny Claimant's allegations that he has suffered

permanent disability because of the aforesaid alleged injuries; is entitled to payment of past medical treatment; payment of present and future medical treatment; payment of temporary total disability compensation benefits; and payment for medications, counseling and therapy; and

WHEREAS, all parties are operating under and are subject to the provisions of the South Carolina Workers' Compensation Act; and

WHEREAS, Claimant contends that he is in need of an additional medical examination and further medical treatment; that he has sustained injuries to the brain, cardiovascular system, musculoskeletal system, liver, kidneys, pulmonary system, and neuropathic system; and the Employer and Carrier expressly deny that Claimant suffered any disability and assert that there is no need for past, present or future medical treatment. The Employer and Carrier also deny Claimant sustained compensable injuries as a consequence of his employment duties either by accident, repetitive trauma, or exposure to lead, known and unknown chemicals and toxins. The Employer and Carrier deny Claimant's allegations and rely upon the report of Dr. Nicholas Lind and other medical providers to refute Claimant's allegations.

WHEREAS, there is a bona fide dispute between the Claimant and the Employer and Carrier as set forth above. Further disputes have arisen as to whether the Claimant suffered any other work related injuries during the period of his employment with Yuasa-Exide, Inc. either as a consequence of repetitive trauma or injury by accident or by virtue of contraction of an occupational disease; and

WHEREAS, on account of the doubts that exist as to what benefits, if any, the Claimant would be adjudged to be entitled to recover under the Workers' Compensation Act, the Claimant and the Employer and Carrier, with the approval of the South Carolina Workers' Compensation Commission, have deemed it advisable, proper and in the best interests of all parties to compromise and settle all possible liabilities and controversies between them, now and in the future. It is also the express intent of the parties to fully and finally end not only the aforementioned claims, but also any and all worker's compensation claims that the Claimant may have had as a result of his employment with Yuasa-Exide, Inc., pursuant to the terms of the agreement. The basis and consideration of such settlement being as follows:

WHEREAS, in consideration of compliance with the terms of this agreement and in consideration of the lump sum payment of One Hundred Eighty-Five Thousand Dollars and 00/100 ($185,000.00), being made to the Claimant upon the approval of this agreement by the South Carolina Workers' Compensation Commission, being paid on a controverted, disputed, and denied basis, the undersigned, C.L. Williams, does hereby release and forever discharge Yuasa-Exide, Inc. and Arrowpoint Capital Corporation from any and all claims, demands, actions or causes of action under the South Carolina Worker's Compensation Act, and otherwise on account of any and all injuries, disability, disfigurement, specific loss, mental/psychological injuries, depression, death, operations, medical, hospital or like expense, continuances, recurrences, aggravations, changes of condition, ailments, illnesses, and diseases or other damages, consequences or results, past, present or future in any way connected with, or arising from the injuries allegedly sustained by Claimant on or about February

1, 1999, as well as any and all other injuries by accident, including occupational diseases, sustained by Claimant during his employment tenure. The payment shall be allocated as follows:

> Sixty-Nine Thousand Four Hundred Five and 12/100 ($69,405.12) Dollars towards attorneys fee and costs; and One Hundred Fifteen Thousand Five Hundred Ninety-Four and 88/100 ($115,594.88) Dollars in compromise settlement of disputed past and future disability compensation benefits at the rate of Four Hundred Eighty-Five and 28/100 ($485.28) Dollars per month commencing on the date of this agreement for a period of One Hundred Twenty-Two and Sixteen (238.20) months thereafter, which is the life expectancy of the Claimant under the Mortuary Table pursuant to Section 19-1-150 of the 1976 Code of Laws of South Carolina, as amended, and pursuant to Section 42-9-10 and 42-9-20 of the 1976 Code of Laws of South Carolina, as interpreted by the South Carolina Supreme Court decision of Utica-Mohawk Mills v. Orr, 227 S.C. 226, 87 S.E.2d 589.

Claimant does hereby acknowledge that the Defendants compliance with the terms of this agreement that the Employer, Carrier and successor companies and entities have fully, finally and completely paid and discharged each and every of their obligations, liabilities and responsibilities on a doubtful and disputed basis under the South Carolina Workers' Compensation Act and that the sum set forth above is being paid to, and received by, the undersigned, C.L. Williams, in full and final satisfaction of all claims whatsoever and that the Employer, Carrier and successor companies and

entities shall not henceforth be liable for the payment of any amount whatsoever except as set forth herein; and

WHEREAS, the Claimant, Employer and Carrier agree that this Settlement Agreement, Release and Order shall not be construed or considered to be an admission by Employer and Carrier of guilt or noncompliance with any federal, state or local statute, public policy, tort law, contract law, common law, or any other wrongdoing; and

WHEREAS, the Employer and Carrier expressly deny responsibility and liability for any incurred medical treatment and expenses allegedly incurred as a result of the alleged accident described above. The parties further agree that although the Employer and Carrier deny that they are responsible for providing any past, additional or future medical treatment or for payment for any purported disability for causes known and unknown; and for payment of any conditional payments made by Medicaid/Medicare or any governmental entity, the parties agree that a Medicare Set Aside (MSA) may be required since Claimant is a Medicare beneficiary and receives Social Security disability benefits. Thus, the Employer and Carrier agree to obtain a Medicare Set Aside (MSA) analysis and to submit such for approval by the Center for Medicaid and Medicare Services (CMS) and the Employer and Carrier is to be solely responsible to fund any MSA account as required by the Center for Medicaid and Medicare Services (CMS) so as to ensure the Claimant's Medicare entitlement is not delayed, jeopardized or stopped. In addition, the Employer and Carrier are solely responsible to determine, negotiate, and if necessary, repay any amounts owed to Medicaid, Medicare or any governmental

entity for past or conditional Medicare or other payments, so as to ensure that the Claimant's Medicare or other entitlements are not delayed, jeopardized or stopped.

WHEREAS, it is not the intention of the Employer and Carrier, nor the Claimant, to shift the responsibility for paying any purported future medical expenses related to the Claimant's admitted injuries to the Federal Government. As a consequence of Claimant's status as a Medicare and Social Security disability beneficiary, this settlement does meet the criteria for review and approval by the Centers for Medicare and Medicaid Services (CMS). The parties have considered Medicare's interest in this case and is of the opinion that an MSA may be necessary and that (CMS) approval of such may be needed, despite the fact that this claim is being resolved on a disputed, controverted, and denied basis with respect to liability.

WHEREAS, this Settlement Agreement and Release is full, final and complete regardless of whether Claimant is able to keep any employment whatsoever, or is able to earn any wages at any time in the future; and is contingent upon the express willingness of Employer and Carrier to fund, if such is required, an MSA; and

WHEREAS, full and complete medical reports are on file with the South Carolina Workers' Compensation Commission and these are duly considered by it in approving this Settlement Agreement and Release.

WHEREAS, Claimant represents that he has been fully advised of his rights under the South Carolina Workers' Compensation Act and that he is of the opinion that the proposed settlement is reasonable and fair, and requests that the South Carolina Workers' Compensation Commission approve this settlement as set forth in this

Settlement Agreement, Release and Order. The parties acknowledge that the Commission relies upon the representation of counsel for the claimant that the Claimant has been fully apprised of his rights under the laws of the South Carolina Workers' Compensation Act and that he believes the settlement is reasonable and fair and thus requests that the South Carolina Workers' Compensation Commission approve this settlement as set forth herein.

NOW THEREFORE, upon approval of this settlement by the South Carolina Workers' Compensation Commission and upon compliance by the Employer and Carrier of the terms of the agreement, Claimant hereby relinquishes and releases each and every claim against Employer and Carrier related to this accident or any other accident, injury or disease, which he now has, or may hereafter have, so that he shall not henceforth have any other or future claim or demand related to this accident, nor shall anyone on his behalf or claiming by, through or under him or as dependent, have any claim or demand on account of this accident, or any other accident; and

The undersigned further acknowledges that the consideration herein expressly recited is the sole and only consideration for the execution hereof, and that no promise, agreement or suggestions of any other or additional consideration has been made to, or received by, me with respect to the worker's compensation settlement.

IN WITNESS WHEREOF, I have hereunto set my hand and seal at Sumter, South Carolina, this 20th day of August 2010.

C.L. Williams

ATTORNEY'S CERTIFICATE

I, _____, Esquire, a practicing attorney of Sumter, South Carolina, do hereby certify that I represent C.L. Williams and as his attorney and with his approval, I negotiated and secured the foregoing Agreement for the payment of the amount recited therein, on the conditions and terms and for the reasons therein stated, and that as attorney for C.L. Williams it is my opinion that such disposition is for the best interest of C.L. Williams, under all circumstances and that the consideration as recited in Agreement is full and adequate under the circumstances, and that as his attorney, I approve the Agreement, and I further certify that, before C.L. Williams signed and executed the above Agreement, the same was read over and explained to him and understood by him, and was executed freely and voluntarily on his part, and that the consideration therein recited is the sole and only consideration for the execution thereof.

Dated at Sumter, South Carolina, this 20th day of Oct 2010.

Attorney for Claimant

ORDER AND AWARD

Upon examination and consideration of the foregoing Agreement and Release, the Attorney's Certificate, and the Workers' Compensation Commission File No. 9930399, and it appearing that the settlement set forth in the Settlement Agreement and Release and Order is proper and complies lawfully with the applicable and governing laws of South Carolina;

Further, upon representation of Claimant and his counsel that his rights have been fully explained to him and that he understands them, that the settlement is reasonable and fair, and that it is the Claimant's desire that the settlement as set forth herein be approved;

IT IS ORDERED AND AWARDED that the Settlement Agreement and Release be, and the same hereby is, approved and the terms of the settlement agreement are adopted herein and made the Order and Award of the South Carolina Workers' Compensation Commission, and that upon the payment of the amount recited and compliance with the terms set forth in the Settlement Agreement and Release, the Employer, Yuasa-Exide, Inc., and the Carrier, Arrowpoint Capital Corporation, be, and are forever discharged from each and every liability or responsibility under the South Carolina Workers' Compensation Act on account of the Claimant's injuries on or about February 1, 1999, whether for compensation, disfigurement or for medical or related services or for any account whatsoever and each and every known, unknown or undiscovered consequence or result thereof, past, present or future, whether for continuation, aggravation, recurrence or otherwise as a result of this accident.

The sum set forth in the Settlement Agreement and Release shall be paid to the Claimant in a lump sum without commutation; whereupon, the Employer and Carrier are authorized to close their files in this matter and the Commission's files shall be, and they are hereby finally closed.

All orders, awards, and opinion heretofore issued by the South Carolina Workers' Compensation Commission in this case are hereby set aside, abrogated, and nullified.

This voluntary settlement entered into by the parties constitutes a final and binding settlement document; and it is not subject to review under any circumstances or conditions.

Dated at Columbia, South Carolina on this 21st day of Oct 2010.

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION

BY: _____
Commissioner

I certify this to be a true and correct copy.

I CONSENT:

Vernon F. Dunbar
Attorney for Employer and Carrier

FEE SUBJECT TO FORM 61

South Carolina Workers' Compensation Commission
1613 Marion St.
P.O. BOX 1715
Columbia, SC 29202-1715
(803) 737-5773

WCC File #: 9930399
Carrier File #: 715001233000
Carrier Code #:
Employer FEIN #:

Claimant's Name: C. L. Williams SSN: Employer's Name: Yuasa-Exide, Inc.

Address: 55 Haxwell Avenue Address: 1990 Corporate Blvd.

City: Sumter State: SC Zip: 29150 City: Sumter State: SC Zip: 29150

Home Phone: (803) 934-1273 Work Phone: Insurance Carrier: Arrowpoint Capital Corporation

Preparer's Name: Vernon F. Dunbar, Esq. Law Firm: Turner Padget Graham & Laney P.A. Preparer's Phone #: (864) 552-4601

Compensation Paid:	Number of Weeks	From (m/d/yyyy)	To (m/d/yyyy)	Amount
1. Number of Weeks T.T.				$ 0.00
2. Number of Weeks T.P.				$ 0.00
3. Number of Weeks P.P.				$ 0.00
4. Disfigurement				$ 0.00
5. Agreement and Final Release				$ $185,000.00
Total Compensation Paid				$ $185,000.00
6. Total Medical Benefits* Paid				$ 0.00
7. Funeral Benefits				$ 0.00

☐ Case Denied

Date of Injury: February 1, 1999 (m/d/yyyy)

By signing this receipt, I acknowledge that I have received the compensation shown above.

By: C. L. Williams
Claimant

By: Vernon F. Dunbar
Employer's Representative

August 13, 2010
Date (m/d/yyyy)

C. L. Williams
Print or type the name of the person, other than the claimant, receiving benefits and sign below.

By:

Report of Additional Fees and Recoupment

A. Carrier Reimbursement by Third Party		$
B. Attorney's Fee Paid by Employer		$
C. Attorney's Fee Paid by Claimant (non-contingent fees only)		$

File this form with the Claims Department according to R.67-411 and R.67-1204. A person, other than the claimant, receiving benefits should sign on the line provided. * Do not include as medical costs fees paid for expert testimony, fees for determining carrier's liability, costs of autopsy, birth and death certificates and impartial examination. Form 19 must be filed within 16 days of final payment of compensation. Form 19 must be filed when a claim is denied.

WCC Form # 19
Rev. Date 3/00

19

STATUS REPORT AND COMPENSATION RECEIPT

Received
11/2/10 TNR
03180.00167

SCANNED

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION
W.C.C. FILE NOS: 0128469, et. al.

James Anderson, et. al.,

Employees,

Claimants

vs.

Enersys; Yuasa, Inc.; Yuasa-Exide Inc.; Yuasa Battery of America, Inc.; Exide Corporation; and Exide Alkaline Battery Division; et al.,

Employers,

AND

Connecticut Indemnity Company/ Arrowpoint Capital; Great American Insurance Company of New York; Mitsui Sumitomo Insurance Company; Wausau Underwriters Insurance Company; Liberty Insurance Corporation; and Employers' Insurance of Wausau; et al.,

Carriers,

Defendants.

CONSENT SCHEDULING ORDER

At the request of the parties, a scheduling conference was held on the above referenced claims on Friday, April 22, 2009 in Columbia, South Carolina. The Claimants are represented by David T. Pearlman, Esquire. The Defendants are represented by Cynthia C. Dooley, Esquire; Robert P. Gruber, Esquire; and Vernon F. Dunbar, Esquire. Each Defense attorney represents the Employer and the Carrier for a specific period of time and will be responsible to handle the claim based on the Claimant's last day of

employment as follows: Cynthia C. Dooley, Esquire: 01/01/70 - 06/10/91; Robert P. Gruber, Esquire: 06/10/91 - 06/10/98; Vernon F. Dunbar, Esquire: 06/18/98 - 06/10/08. Latonya Edwards, Esquire appeared on behalf of the South Carolina Second Injury Fund (SIF).

The parties discussed the most efficient method for discovery, exchange of information and mediation prior to adjudicatory Hearings. Attorney Pearlman states there are approximately three hundred (300) Claimants who are former Employees of the above named Employers and who have or will be filing Workers' Compensation claims with the Commission. The list of these Claimants will be provided to the Defendants. Furthermore, it is anticipated that approximately fifty (50) Claimants will initially file WCC Form No. 50s, Requests for Hearings. Due to the large number of Hearing requests, the parties agree it is necessary and proper to enter into a Scheduling Order to resolve current discovery issues and to set time limitations for the exchange of information, for discovery, and for mediations prior to Hearings. Pursuant to the South Carolina Rules of Civil Procedure and the Regulations of the South Carolina Workers' Compensation Commission, the following Scheduling Order is established by agreement of the parties:

First: The Defendants agree to provide to the Claimants within ten (10) days of the signing of this Order information or documents in compliance with the prior Subpoena requesting "ambient air testing or air quality testing reports records and documents" and with the prior Subpoena requesting "work job descriptions or work instructions for the following jobs: oxide manufacturing, grid casting, pasting, formation, tub formation, and distribution." Each Defense attorney shall comply with the Subpoena for the periods of time of their respective representation of the Employer and Carrier. If the information is not obtainable, the Defense attorney shall provide a letter or affidavit certifying the efforts made and the reasons why the information is not able to be produced.

Second: The Claimants attorney will provide the names, or appropriate WCC Forms No. 50s, of the approximately fifty (50) Claimants who will be initially requesting Hearings to the Defendants on or before June 30, 2009.

Third: The Claimants who will be requesting Hearings as set forth in paragraph Second above shall be made available to the Defendants attorney handling their claim for depositions to be completed by September 1, 2009. Prior to the scheduling of the depositions, the Claimants will be provided with a copy of their personnel or employment file, including all medical records in the possession of the Defendants at least two (2) weeks prior to their deposition.

Fourth: The Claimants shall complete medical or other expert evaluations on or before September 26, 2009, at which time the Claimants will provide to the Defendants reports from their respective experts and evaluators. The Defendants shall complete medical or other expert evaluations on or before September 31, 2009, at which time the Defendants will provide to the Claimants reports from their respective experts and evaluators.

Fifth: The parties shall complete any additional evaluations as the parties deem necessary after receipt of the reports set forth in paragraph Fourth above, on or before October 21, 2009, at which time the parties will exchange these additional reports from their respective experts and evaluators.

Sixth: The parties shall complete the taking of depositions and exchange rebuttal reports if obtained from witnesses or experts on or before November 20, 2009.

Seventh: Mediation conferences regarding the Claimants identified in Second above shall be scheduled for the week of December 7, 2009. The parties agree to utilize Earl Ellis, Esquire as a mediator.

Eighth: If claims are not resolved by mediation or otherwise, adjudicatory Hearings shall be scheduled by the South Carolina Workers' Compensation Commission not before January 31, 2010.

Ninth: The Claimants attorneys will provide to the Defendants attorneys a list of all the individuals presently

represented by the Claimants attorneys for injuries arising out of their employment with their Employers on or before July 30, 2009.

Tenth: Between January 1, 2010 and February 28, 2010, the parties shall participate in a conference to determine the discovery schedule concerning the remaining claimants not identified as the initial group and to implement a Scheduling Order to ensure the expeditious handling of the remaining claims.

Eleventh: The terms of this scheduling Order can only be extended, modified, or amended by the consent of all parties involved.

NOW THEREFORE, on joint motion of the attorneys for the parties, it is hereby;

ORDERED, ADJUDGED, AND DECREED the above scheduling agreement shall be adopted as an Order of this Commission and shall be binding as an Order upon all parties.

AND IT IS SO ORDERED!

Commissioner

Aug. 13, 2009
Columbia, SC

WE JOINTLY MOVE AND AGREE:

David T. Pearlman, Esquire
Malcolm M. Crosland, Jr., Esquire
Attorneys for the Claimants

Robert P. Gruber, Esquire
Attorney for the Defendants

Vernon F. Dunbar, Esquire
Attorney for the Defendants

Latonya Edwards, Esquire
Attorney for the Second Injury Fund

Cynthia C. Dooley, Esquire
Attorney for the Defendants

CERTIFICATE OF SERVICE

This is to certify that the undersigned has this date served this order in the above entitled action upon all parties to this cause by depositing a copy hereof, postage paid, in the United States mail addressed to the attorney or attorneys for said parties.

This 1st day of August, 2009

By Valerie D. Dodds
Administrative Assistant to the Commissioner

Latonya Edwards / David T. Pearlman / Cynthia C. Dooley

represented by the Claimants attorneys for injuries arising out of their employment with their Employers on or before July 30, 2009.

Tenth: Between January 1, 2010 and February 28, 2010, the parties shall participate in a conference to determine the discovery schedule concerning the remaining claimants not identified as the initial group and to implement a Scheduling Order to ensure the expeditious handling of the remaining claims.

Eleventh: The terms of this scheduling Order can only be extended, modified, or amended by the consent of all parties involved.

NOW THEREFORE, on joint motion of the attorneys for the parties, it is hereby:

ORDERED, ADJUDGED, AND DECREED the above scheduling agreement shall be adopted as an Order of this Commission and shall be binding as an Order upon all parties.

AND IT IS SO ORDERED!

_____
Commissioner

_____, 2009
Columbia, SC

WE JOINTLY MOVE AND AGREE:

_____
David T. Pearlman, Esquire
Malcolm M. Crosland, Jr., Esquire
Attorneys for the Claimants

_____
Robert P. Gruber, Esquire
Attorney for the Defendants

_____
Vernon F. Dunbar, Esquire
Attorney for the Defendants

_____
Latonya Edwards, Esquire
Attorney for the Second Injury Fund

_____
Cynthia C. Dooley, Esquire
Attorney for the Defendants

Employers on or before July 30, 2009.

Ninth: Between January 1, 2010 and February 28, 2010, the parties shall participate in a conference to determine the discovery schedule concerning the remaining claimants not identified as the initial group and to implement a Scheduling Order to ensure the expeditious handling of the remaining claims.

Tenth: The terms of this scheduling Order can only be extended, modified, or amended by the consent of all parties involved.

NOW THEREFORE, on joint motion of the attorneys for the parties, it is hereby:

ORDERED, ADJUDGED, AND DECREED the above scheduling agreement shall be adopted as an Order of this Commission and shall be binding as an Order upon all parties.

AND IT IS SO ORDERED!

_____
Commissioner

_____, 2009
Columbia, SC

WE JOINTLY MOVE AND AGREE:

_____
David T. Pearlman, Esquire
Malcolm M. Crosland, Jr., Esquire
Attorneys for the Claimants

_____
Robert P. Gruber, Esquire
Attorney for the Defendants

_____
Vernon F. Dunbar, Esquire
Attorney for the Defendants

_____
Latonya Edwards, Esquire
Attorney for the Second Injury Fund

_____
Cynthia C. Dooley, Esquire
Attorney for the Defendants

represented by the Claimants attorneys for injuries arising out of their employment with their Employers on or before July 30, 2009.

Tenth: Between January 1, 2010 and February 28, 2010, the parties shall participate in a conference to determine the discovery schedule concerning the remaining claimants not identified as the initial group and to implement a Scheduling Order to ensure the expeditious handling of the remaining claims.

Eleventh: The terms of this scheduling Order can only be extended, modified, or amended by the consent of all parties involved.

NOW THEREFORE, on joint motion of the attorneys for the parties, it is hereby:

ORDERED, ADJUDGED, AND DECREED the above scheduling agreement shall be adopted as an Order of this Commission and shall be binding as an Order upon all parties.

AND IT IS SO ORDERED!

_____
Commissioner

_____, 2009
Columbia, SC

WE JOINTLY MOVE AND AGREE:

_____
David T. Pearlman, Esquire
Malcolm M. Crosland, Jr., Esquire
Attorneys for the Claimants

_____
Robert P. Gruber, Esquire
Attorney for the Defendants

_____
Vernon F. Dunbar, Esquire
Attorney for the Defendants

_____
Latonya Edwards, Esquire
Attorney for the Second Injury Fund

_____
Cynthia C. Dooley, Esquire
Attorney for the Defendants

mgc | INSURANCE DEFENSE

Reply To
VERNON F. DUNBAR
Direct Dial: (864) 239-6735
vernon.dunbar@mgclaw.com

July 31, 2014

MAILED

MAILED

The Honorable W. Jeffrey Young
Third Circuit Court
141 North Main Street
Sumter, South Carolina 29150

Arrowpoint Capital Corp/Arrowpoint Indemnity Company v. South Carolina Second Injury Fund
IN RE: C.L. Williams v. Yuasa Exide
Date of Accident: February 1, 1999
Civil Action No.: 2013-CP-43-02286
Our File No.: 20113.14062

Arrowpoint Capital Corp/Arrowpoint Indemnity Company v. South Carolina Second Injury Fund
IE RE: Mary McConico v. Yuasa-Exide
Date of Accident: July 31, 1999
Civil Action No.: 2013-CP-43-02283
Our File No.: 20113.14171

Arrowpoint Capital Corp/Arrowpoint Indemnity Company v. South Carolina Second Injury Fund
IE RE: Joe Mathis v. Yuasa-Exide
Date of Accident: July 31, 1999
Civil Action No.: 2013-CP-43-02284
Our File No.: 20113.14073

Arrowpoint Capital Corp/Arrowpoint Indemnity Company v. South Carolina Second Injury Fund
IN RE: James Huggins v. Yuasa Exide
Date of Accident: February 1, 1999
Civil Action No.: 2013-CP-43-02287
Our File No.: 20113.14168

Dear Judge Young:

You heard oral arguments in the above-referenced matters on June 3, 2014, and we are in receipt of your decision denying Arrowpoint Capital Corporation's request for reimbursement from the South Carolina Second Injury Fund.

On July 30, 2014, our Supreme Court issued an opinion in *State Accident Fund v. South Carolina Second Injury Fund [In re: Johnny M. Adger v. City of Manning]*, a copy of which I am enclosing with this letter. The Court unanimously reversed the decision of the South Carolina Workers' Compensation Commission which had denied Second Injury Fund reimbursement for the appellant carrier. We submit that the Supreme Court's analysis is directly on point with regard to the four cases we argued in June. In light of this recent opinion, we would respectfully request that you reconsider your decision issued on July 21, 2014. Please advise if you would prefer for our request to be filed as a formal motion.

By copy of this letter to Latonya Edwards and Timothy Killen, counsel for the Second Injury Fund, we are notifying them of our communication with you concerning these matters.

Thank you very much for your time and consideration.

With kind regards, I remain

Very truly yours,

Ashley Forbes for VFD

Vernon F. Dunbar

VFD/bhg
Enclosure

cc: Latonya Edwards, Esquire
Timothy B. Killen, Esquire
Stephanie Pugh, Esquire (sent via email)
Ashley Forbes, Esquire (sent via email)
Mr. Ronald Mazariegos (sent via email)
Mr. Eric Rowell (sent via email)

THE STATE OF SOUTH CAROLINA
In The Supreme Court

State Accident Fund, Appellant,

v.

South Carolina Second Injury Fund, Respondent,

In Re: Johnny M. Adger, Employee,

v.

City of Manning, Employer,

and

State Accident Fund, Carrier.

Appellate Case No. 2012-212722

Appeal from the South Carolina Workers' Compensation Commission

---

Opinion No. 27424
Heard February 6, 2014 – Filed July 30, 2014

REVERSED AND REMANDED

F. Earl Ellis, Jr., of Adams and Reese, LLP, of Columbia, for Appellant.

Latonya Dilligard Edwards, of Dilligard Edwards, LLC, of Columbia, for Respondent.

CHIEF JUSTICE TOAL: The State Accident Fund (Appellant) appeals an order from the Appellate Panel of the South Carolina Workers' Compensation Commission (the Commission) denying Appellant's request for reimbursement from the South Carolina Second Injury Fund (Respondent) for benefits paid to Johnny Adger (Claimant), who suffered from preexisting diabetes when a work-related injury occurred. We reverse and remand.

FACTS/PROCEDURAL BACKGROUND

Claimant suffered an accidental injury to his left knee on August 17, 2007, while working as a police officer with the Manning Police Department. As a result, Claimant was treated using various non-operative methods, including steroid injections. In January 2008, Claimant reached maximum medical improvement (MMI) and was assigned a 32% permanent impairment rating to his lower left extremity. However, in April 2008, Claimant returned to the doctor because he continued to experience swelling and pain in his left knee. Ultimately, Claimant underwent knee replacement surgery. Claimant continued to experience swelling and pain in his left knee, and Claimant followed up with the orthopaedic center for several months after the surgery.

At the time of his injury, Claimant suffered from preexisting diabetes, which Claimant's employer was aware of prior to the injury. Claimant experienced problems with his diabetes for years before the accident and required medication to control the condition.[1] Claimant's diabetes was medically controlled around the time of the injury; however, Claimant's diabetes was uncontrolled on several occasions during the course of his knee treatment.

Claimant's primary care physician, Dr. William Aldrich, opined that Claimant's injury "most probably aggravate[d] the diabetes" and resulted in "substantially greater medical costs" than would have occurred from the injury alone. However, in Dr. Aldrich's opinion, the aggravation of Claimant's diabetes "most probably" did not result in "substantially greater time lost from work" or "substantially greater disability" than would have occurred from the injury alone.

[1] For example, Claimant suffered tingling in his right foot which only subsided after removing his shoe. In addition, Claimant was prescribed shoes made especially for diabetics.

The workers' compensation carrier, Appellant, filed a claim for partial reimbursement from Respondent, alleging that it incurred substantially greater liability for compensation benefits and medical benefits because Claimant's preexisting diabetes was aggravated by his work-related injury. Respondent denied that the claim met the requirements of the statute governing reimbursement. *See* S.C. Code Ann. § 42-9-400 (Supp. 2013).[2]

South Carolina Workers' Compensation Commissioner T. Scott Beck (the Single Commissioner) held a hearing on this matter on September 27, 2011. Thereafter, the Single Commissioner filed an order denying Appellant's claim for reimbursement and dismissing the claim with prejudice. The Single Commissioner concluded that Appellant "had the burden to prove that Claimant's preexisting diabetes was permanent and serious enough to constitute a hindrance or obstacle to Claimant's employment," but also found that "the evidence in the record rebuts the presumption that Claimant's diabetes was a hindrance to his employment." In addition, the Single Commissioner determined that Appellant did not prove that "Claimant's preexisting diabetes created substantially greater lost time from work and permanent disability than would have resulted from the work injury alone" based upon his findings that "[t]he evidence in the record does not indicate a fluctuation in blood sugars, a modification in diabetic medication or delayed post-surgical healing" and that "the medical records reveal that Claimant was written out of work for only three (3) days immediately after the work injury." Therefore, the Single Commissioner denied Appellant's claim for reimbursement, concluding that it did not meet the standard of reimbursement in section 42-9-400.

On appeal, the Commission affirmed the Single Commissioner's order in its entirety.[3] Appellant appealed the Commission's order to the court of appeals. This Court certified the appeal pursuant to Rule 204(b), SCACR.

---

[2] Subject to detailed provisions of the statute, section 42-9-400 provides that if an employee with a permanent physical impairment suffers a subsequent injury by accident arising out of and in the course of his employment, the Second Injury Fund must reimburse the employer or his insurance carrier for compensation and medical benefits. S.C. Code Ann. § 42-9-400.

[3] One commissioner dissented, asserting that she would find the evidence did not rebut the presumption that Claimant's diabetes was a hindrance or obstacle to employment and that instead, the evidence supported the conclusion that Claimant's injury aggravated his diabetes and resulted in increased medical costs.

ISSUE

Whether the Commission erred in denying Appellant's claim for reimbursement for Claimant's medical payments under section 42-9-400?[4]

STANDARD OF REVIEW

The South Carolina Administrative Procedures Act (the APA) sets forth the standard for judicial review of decisions by the Commission. *See* S.C. Code Ann. § 1-23-380 (Supp. 2012); *Grant v. Grant Textiles*, 372 S.C. 196, 200, 641 S.E.2d 869, 871 (2007); *Lark v. Bi-Lo*, 276 S.C. 130, 136, 276 S.E.2d 304, 307 (1981). Although the Court may not substitute its judgment for the judgment of the agency as to the weight of the evidence on questions of fact, the Court may reverse a decision of the Commission if it is affected by an error of law or is clearly erroneous in view of the substantial evidence on the record as a whole. S.C. Code Ann. § 1-23-380. The Court may find the Commission's findings clearly erroneous if they are based on a mistaken view of the evidence. *Grayson v. Carter Rhoad Furniture*, 312 S.C. 250, 252, 439 S.E.2d 859, 860 (Ct. App. 1993).

LAW/ANALYSIS

The Second Injury Fund was established in 1972 to encourage employers to hire disabled or handicapped persons. *Liberty Mut. Ins. Co. v. S.C. Second Injury Fund*, 318 S.C. 516, 518, 458 S.E.2d 550, 551 (1995); *Springs Indus. v. S.C. Second Injury Fund*, 296 S.C. 359, 361, 372 S.E.2d 915, 916 (Ct. App. 1988). If a disabled or handicapped employee's work-related injury results in the award of workers' compensation benefits, the employer is entitled to partial reimbursement if it satisfies the statutory requirements of section 42-9-400. *Springs Indus.*, 296 S.C. at 361, 372 S.E.2d at 916. Therefore, the Second Injury Fund serves to fully compensate disabled employees for work-related injuries without penalizing an employer if the employer is subject to greater liability because of the employee's preexisting condition. *Liberty Mut.*, 318 S.C. at 518, 458 S.E.2d at 551; *Springs Indus.*, 296 S.C. at 361, 372 S.E.2d at 916.

Section 42-9-400(a) of the South Carolina Code provides:

---

[4] Appellant does not challenge the Commission's decision to deny reimbursement for Claimant's compensation benefits.

> If an employee who has a permanent physical impairment from any cause or origin incurs a subsequent disability from injury by accident arising out of and in the course of his employment resulting in compensation and medical payments liability or either, for disability that is substantially greater and is caused by aggravation of the preexisting impairment than that which would have resulted from the subsequent injury alone, the employer or his insurance carrier shall pay all awards of compensation and medical benefits provided by this title; but such employer or his insurance carrier shall be reimbursed from the Second Injury Fund . . . for compensation and medical benefits . . . .

S.C. Code Ann. § 42-9-400(a).

"[P]ermanent physical impairment" is defined as a "permanent condition, whether congenital or due to injury or disease, of such seriousness as to constitute a hindrance or obstacle to obtaining employment or to obtaining reemployment if the employee should become unemployed." *Id.* § 42-9-400(d). When an employer establishes prior knowledge of the employee's permanent condition, "then *there shall be a presumption* that the condition is permanent and that a hindrance or obstacle to employment or reemployment exists," but only if the condition is one of thirty-two conditions listed in the statute. *Id.* (emphasis added). Diabetes is one such condition. *Id.* § 42-9-400(d)(2).

I. Presumption Under Section 42-9-400(d)

First, Appellant argues that the Commission erred by incorrectly applying the presumption found in section 42-9-400(d). In its findings of fact, the Commission stated that "[t]hough [Appellant] is entitled to a presumption that Claimant's preexisting diabetes was a hindrance to employment, the medical evidence in the record rebuts this presumption." However, inconsistent with this finding of fact, the Commission stated in its conclusions of law that "[t]o qualify for reimbursement, [Appellant] had the burden to prove that Claimant's preexisting diabetes was permanent and serious enough to constitute a hindrance or obstacle to Claimant's employment." Appellant further contends that the Commission erred in concluding that Appellant had the burden of proof because it was entitled to a presumption that Claimant's preexisting diabetes constituted a "permanent physical impairment." We agree.

A presumption is a "legal inference or assumption that a fact exists, based on the known or proven existence of some other fact or group of facts." Black's Law Dictionary 1304 (9th ed. 2009). "A presumption shifts the burden of production or persuasion to the opposing party, who can then attempt to overcome the presumption." *Id.* Appellant presented facts sufficient to support the application of the presumption under section 42-9-400(d).

Therefore, we find that Appellant was entitled to a presumption that Claimant's diabetes was permanent and constituted a hindrance or obstacle to his employment or reemployment. Because the Commission's order incorrectly sets forth the burden of proof in its law section, we hold that the Commission's order is erroneous as a matter of law. *See* S.C. Code Ann. § 1-23-280 (providing that the Court may reverse a decision of the Commission if it is affected by an error of law).

II. Rebuttal of the Presumption

Because Appellant was entitled to the presumption, the burden to rebut the presumption then shifted to Respondent. The Commission concluded that "the evidence in the record rebutted the presumption that Claimant's preexisting diabetes was a hindrance to his employment." Therefore, we must determine whether Respondent, in fact, presented evidence which rebutted the presumption.

Appellant argues that the Commission erred in concluding that Respondent rebutted the presumption provided under section 42-9-400(d). We agree.

When a presumption shifts the burden of production to the opposing party, that party must present substantial evidence in order to rebut the presumption. *See* 100A C.J.S. *Workers' Compensation* § 1029 (2014) ("Because a fact must be proved with substantial evidence in a workers' compensation proceeding, a rebuttable presumption must be met with substantial evidence.").

In deciding whether there was evidence to rebut the presumption that the Claimant suffered a "permanent physical impairment," the Commission focused on medical evidence indicating that prior to the injury, Claimant's diabetes was medically managed and was not uncontrolled or problematic. The Commission relied on this evidence in discussing whether Claimant's preexisting diabetes was a hindrance to Claimant's employment at the time of his injury, *not* whether it was a hindrance to *obtaining* employment, as the statute requires. *See* S.C. Code Ann. § 42-9-400(d) (defining "permanent physical impairment" as a permanent condition

constituting "a hindrance or obstacle to *obtaining* employment or to obtaining reemployment if the employee should become unemployed" (emphasis added)); *Springs Indus.*, 296 S.C. at 362, 363–64, 372 S.E.2d at 917 (concluding that an employee's chronic cough and breathing difficulties caused by exposure to cotton dust would be a hindrance to obtaining employment); *see also State v. Scott*, 351 S.C. 584, 588, 571 S.E.2d 700, 702 (1993) ("What a legislature says in the text of a statute is considered the best evidence of the legislative intent or will." (citation omitted)).

The Commission also relied on medical evidence suggesting that post-surgery, Claimant's diabetes was successfully managed with medication and that his left knee was responding well to treatment. This view of the evidence fails to consider the substantial evidence in the record indicating the significant symptoms and problems Claimant experienced after his work-related injury. According to the medical records, Claimant's diabetes caused tingling in his foot which did not subside until he removed his shoe, and Claimant required special shoes and medication to control his diabetes. The evidence in the record actually *supports* the presumption that Claimant's diabetes constituted a hindrance or obstacle to obtaining employment or reemployment as a police officer. Accordingly, the Commission's conclusion that the evidence rebutted the presumption is clearly erroneous in view of the substantial evidence on the record. *See* S.C. Code Ann. § 1-23-380 (providing that the Court may reverse a decision of the Commission if it is clearly erroneous in view of the substantial evidence on the record as a whole).

III. Increased Medical Costs

Finally, Appellant argues it satisfied section 42-9-400 by proving that the aggravation of Claimant's preexisting diabetes resulted in substantially increased medical costs, and therefore, the Commission erred in denying Appellant reimbursement under section 42-9-400(a). We agree.

Section 42-9-400(a) provides that the Second Injury Fund must reimburse a carrier for medical payments when an employee with a preexisting condition incurs a subsequent disability from a work-related injury "resulting in compensation and medical payments liability, or either, for disability that is substantially greater and is caused by aggravation of the preexisting impairment than that which would have resulted from the subsequent injury alone . . . ." S.C. Code Ann. § 42-9-400(a). Subsection (a)(2) specifies the details of reimbursement of medical payments, stating that "an employer or carrier must establish that his *liability for medical payments is substantially greater by reason of the aggravation of the preexisting*

impairment than that which would have resulted from the subsequent injury alone." S.C. Code Ann. § 42-9-400(a)(2) (emphasis added).

In conforming to the purpose of the Second Injury Fund, section 42-9-400(a) provides for reimbursement from the Second Injury Fund when --subsequent to a work-related injury—the aggravation of a preexisting permanent impairment causes substantially greater liability for compensation *or* medical payments than would have resulted from the work-related injury alone. *See* S.C. Code Ann. § 42-9-400(a); *Liberty Mut.*, 318 S.C. at 519, 485 S.E.2d at 551–52.

Section 42-9-400 provides for reimbursement of medical payments even when there is not increased liability for compensation. *See* S.C. Code Ann. § 42-9-400(a); *Liberty Mut.*, 318 S.C. at 519, 485 S.E.2d at 551–52. In *Liberty Mutual*, we held that a carrier was entitled to reimbursement for medical payments where the carrier was liable for greater medical payments—but not greater compensation payments—because of an employee's preexisting diabetes. 318 S.C. at 519, 458 S.E.2d at 551. The employee was paralyzed by a work-related accident and was rendered totally and permanently disabled for workers' compensation purposes. *Id.* at 517, 458 S.E.2d at 551. The employee's preexisting diabetes aggravated his paralysis, causing him to undergo a double amputation. *Id.* The Second Injury Fund argued that section 42-9-400(a) required the employee to suffer greater *disability*, in addition to increased medical payments, for the carrier to receive any reimbursement. *Id.* at 518, 458 S.E.2d at 551. Although we found that the carrier was not entitled to reimbursement of compensation payments because the accident rendered the employee totally disabled and thus the employee's diabetes did not increase the compensation payments, we held that the carrier incurred greater liability for the employee's medical payments because of the diabetes, and was therefore entitled to reimbursement for medical payments under section 42-9-400. *Id.* at 519, 485 S.E.2d at 551–52.

The Commission denied Appellant's claim for reimbursement in full. Without mentioning medical payments, the Commission stated that "Claimant's preexisting diabetes did not create substantially greater liability for permanent disability nor did it result in substantially greater lost time from work." However, these facts fall under the compensation liability prong of the statute. In its order, the Commission ignored Dr. Aldrich's expert opinion that Claimant's injury most probably aggravated his diabetes and resulted in substantially greater medical costs than would have resulted from his work-related injury alone. Respondent presented no evidence or expert opinion that contradicted Dr. Aldrich's statement concerning medical costs. Therefore, based on the fact that the medical evidence

supports the conclusion that the Claimant's work-related injury aggravated his diabetes and resulted in increased medical costs, we hold that Appellant satisfied the requirements of section 42-9-400(a), and the Commission's decision to deny Appellant's claim for reimbursement of medical payments is clearly erroneous based on the evidence.[5]

CONCLUSION

For the foregoing reasons, the Commission's decision denying Appellant reimbursement for Claimant's medical payments is reversed and remanded for the Commission to determine the amount of reimbursement to which Appellant is entitled.

REVERSED AND REMANDED.

KITTREDGE, HEARN, JJ., and Acting Justice James E. Moore, concur. PLEICONES, J., concurring in result only.

---

[5] Although Dr. Aldrich opined that aggravation of Claimant's diabetes most probably did not result in substantially greater time lost from work or substantially greater disability than would have occurred from the injury alone, reimbursement for increased medical payments is an important aspect of the Second Injury Fund and section 42-9-400, regardless of a preexisting condition's effect on compensation payments. *See Liberty Mut.*, 318 S.C. at 519, 485 S.E.2d at 551–52.

South Carolina Workers' Compensation Commission
1612 Marion St.
P.O. BOX 1715
Columbia, SC 29202-1715
(803) 737-5723

WCC File #: 9930399
Carrier File #: 715001233000
Carrier Code #:
Employer FEIN #:

Claimant's Name: C. L. Williams SSN:
Address: 55 Maxwell Avenue
City: Sumter State: SC Zip: 29150
Home Phone: (803) 934-1273 Work Phone:

Employer's Name: Yuasa-Exide, Inc.
Address: 1960 Corporate Blvd.
City: Sumter State: SC Zip: 29150
Insurance Carrier: Arrowpoint Capital Corporation

Preparer's Name: Vernon F. Dunbar, Esq. Law Firm: Turner Padget Graham & Laney P.A. Preparer's Phone #: (864) 552-4601

Check applicable claims and complete all blanks.

1. The employee sustained a compensable accidental injury to the Muscular skeletal, pulmonary, neuropathic, genitourinary systems and brain on 02/01/1999 in Sumter (county), State of South Carolina (state).
2. That the Second Injury Fund was put on notice of the claim on July 21, 2008 (date).
3. That the carrier concluded the disability claim by ☐ Award ☒ Agreement on October 25, 2010 (date).
4. That the subsequent injury combined with or was aggravated by the below-named permanent impairment under S.C. Code Section 42-9-400 (d):
   a. Listed Impairment – (1) – (33) Cardiovascular disease, arterial sclerosis, kidney failure/renal insufficiency, diabetes, and chronic obstructive pulmonary disease (COPD)
   b. (34) (a) Hypertension, pulmonary edema, cardiomyopathy, hyperlipidemia, Cardiovascular disease, arterial sclerosis, kidney failure/renal insufficiency, diabetes, and chronic obstructive pulmonary disease (COPD)
   c. (34) (b) Hypertension, pulmonary edema, cardiomyopathy, hyperlipidemia, Cardiovascular disease, arterial sclerosis, kidney failure/renal insufficiency, diabetes, and chronic obstructive pulmonary disease (COPD)
5. ☒ a. That the impairment preexisted;
   ☒ b. That the impairment was permanent; and
   ☒ c. That the impairment is a physical condition.
6. ☒ That the prior impairment combined with or was aggravated by the subsequent injury.
7. ☒ That the combination/aggravation substantially increased the liability of the carrier for: ☐ disability ☐ medical or ☒ both.
8. ☒ That the impairment was a hindrance or obstacle to employment or re-employment.
9. ☒ a. That the employer has knowledge of the prior impairment;
   ☒ b. That the impairment was unknown to the employee and the employer; or
   ☒ c. That the employee concealed the prior impairment from the employer.
10. ☒ That the subsequent injury would not have occurred "but for" the prior impairment.
11. That the above claim qualifies for reimbursement under S.C. Code Section 42-9-410 because: South Carolina Supreme Court's holding in the case of *Ellison v. Frigidaire Home Products, 271 S.C. 159, 638 S.E.2d 664 (Ct. of App. 2004).*
12. Other grounds for claim: Plaintiff reserves the right to amend its Form 54 to allege additional grounds for recovery/ reimbursement.

RECEIVED
FEB - 1 2012
3180.167
SCW66
JAN 30 2012
JUDICIAL
SCANNED

Signature

January 27, 2012
Date (m/d/yyyy)

WCC Form # 54
Rev. 6/90

54

Employer's Notice of Claim and/or Request for Hearing

PROOF OF SERVICE

The undersigned certifies that she is an employee at TURNER, PADGET, GRAHAM & LANEY, P.A., and that the attached ***Form 54*** was served upon Michael T. Harris, Deputy Director for the South Carolina Second Injury Fund, this 27th day of January 2012 in accordance with Regulations 67-210, 67-211, 67-213, 67-214, and 67-215 and such other law as may be applicable, via U.S. Mail:

Michael T. Harris, Esquire
Deputy Director
South Carolina Second Injury Fund
100 Executive Center Drive
Santee Building, Suite 101
Columbia, SC 29210

Jeanie McDade
Secretary to Vernon F. Dunbar

South Carolina Workers' Compensation Commission
P.O. Box 1715
Columbia, South Carolina 29202-1715

WCC File No. 9930399
Carrier File No. 7150012330000
Carrier Code No. 00500
Employer FEIN

YUASA / EXIDE, INC.	1990 CORP. BLVD., SUMTER, SC 29150
Employer's Name	Address
~~STATE ACCIDENT FUND~~ ARROWPOINT CAPITAL	
Insurance Carrier's Name	Address
C L. WILLIAMS	
Employee's Name	Address

The Second Injury, in answer to the claim, respectfully shows:

1. It is denied that the employee sustained a compensable accident:

2. It is denied that the notice was given to the Second Injury Fund:

3. It is denied that the disability claim has been concluded.

4. It is denied that the impairment is:
as alleged or a hindrance or obstacle to employment.

5a. It is denied that the impairment pre-existed.
b. It is denied that the impairment was permanent.
c. It is denied that the impairment is physical.

6. It is denied that the impairment combined with or was aggravated by the subsequent injury.

7. It is denied that the combination/aggravation substantially increased the carrier's liability for disability, medical, or both:

8. It is denied that the impairment was a hindrance or obstacle to employment or re-employment.

9a. It is denied that the employer had knowledge of the impairment.
b. It is denied that the impairment was unknown to the employee and employer.
c. It is denied that the employee concealed the impairment.

10. It is denied that the subsequent injury would not have occurred 'but for' the prior impairment.

11. It is denied that the claim qualifies for reimbursement under S.C. Code Section 42-9-410;

12. Other grounds for denial:
State of limitations (42-15-40, 15-3-600); failure to timely submit file materials (42-7-320(B)(2)); all affirmative defenses; laches; estoppel; res judicata; fraud; intervening trauma; negligence; injuries neither compensable nor work-related; failure to pay assessment (42-7-310).

Signature on behalf of the Second Injury Fund — Date 2/27/12

STATE OF SOUTH CAROLINA *
* CERTIFICATE OF SERVICE
COUNTY OF RICHLAND *

The undersigned employee of the South Carolina Second Injury Fund, 100 Executive Center Drive, Suite 101, Columbia, South Carolina, 29210, does hereby certify that he or she has served the following named individual(s) with a copy of the pleading indicated below by mailing a copy of same to them in the United States mail, with sufficient postage affixed thereto and return address clearly marked on the date indicated below:

COUNSEL SERVED:

VERNON F. DUNBAR
TURNER, PADGET
PO BOX 1509
GREENVILLE, SC 29602

PLEADING: FORM 55 - SECOND INJURY FUND'S ANSWER TO REQUEST FOR HEARING

TIMOTHY B. KILLEN

February 27, 2012

Columbia, South Carolina

South Carolina Workers' Compensation Commission
1612 Marion St.
P.O. BOX 1715
Columbia, SC 29202-1715
(803) 737-5723

PRE-HEARING BRIEF
WCC File No: 9930399
SIF No.: 141031

Claimant's Name: C. L. Williams
Address: 55 Maxwell Avenue
City Sumter State: SC Zip: 29150
Home Phone: (803) 934-1273 Work Phone:

Employer's Name: Yuasa-Exide, Inc.
Address: 1990 Corporate Blvd.
City Sumter State: SC Zip: 29150
Carrier: Arrowpoint Capital Corporation

Preparer's Name: Vernon F. Dunbar, Esquire
Preparer's Phone #: (864) 552-4601

A claim for workers' compensation benefits is made based on the following grounds:
☒ Injury ☒ Illness ☒ Repetitive Trauma

1. Compensation Rate: $569.70 2. AWW: $379.82 **Date of Injury:** 02/01/1999
3. Type of injury and body part(s): Brain, kidneys, muscular skeletal, cognitive, neuropathy, cardiovascular and vestibular systems caused by heavy metal poisoning.
4. Facts in controversy: The amount of reimbursement owed and due to Yuasa-Exide, Inc. from the Second Injury Fund.
5. Legal issues involved: None. Fund has accepted case for reimbursement. Parties disagree on the amount of reimbursement owed and due pursuant to Section 42-9-400 of the S.C. Code of Laws.
6. Unusual aspects: Fund takes the position that it cannot resolve case after December 31, 2011. Hence, an adjudicatory hearing is required according to the Funds interpretation of the statute.
7. Witnesses (designate if expert):* Ron Whetsel. Plaintiff reserves the right to add additional witnesses.
8. Exhibits: SIF Claimant's Knowledge Affidavit. Court and Commission Decisions regarding the case of Lee Ernest Franklin. Last, literature regarding permissible exposure limits to lead in an occupational setting.
9. Medical evidence (indicate report pursuant to R.67-612; deposition or appearance): See APA Submissions
10. Name, address, and specialty, if any, of the treating physician: Yuasa-Exide's in-house medical department. Medical treatment supplied by Drs. Eric Byrd and A. W. Hursey.
11. Impairment rating(s); body part(s); physician and date of opinion: On May 22, 2008, Drs. Edward L. Baker and J. Routt Reigart, assigned claimant 49% medical impairment to the heart; 8% medical impairment to the cardiovascular system; 4% medical impairment to the vestibular system due to vertigo; and 29% medical impairment to the brain due to dementia and toxic encephalopathy.
12. I am amending my Form 50/51 in the following manner: N/A.

I verify the contents of this form are accurate and true to the best of my knowledge.

Signature: Vernon F. Dunbar
Vernon F. Dunbar, Esq.
Email: VDunbar@TurnerPadget.com
Date of hearing: August 13, 2012
Time needed for hearing: 1 hour

On behalf of ☐ Claimant ☒ Employer / Plaintiff

File this form and proof of service on the opposing party according to R.67-611 and R.67-212. Do not send medical reports.
* Commissioners reserve the right to admit expert witnesses at hearings.

PROOF OF SERVICE

The undersigned certifies that she is an employee at TURNER, PADGET, GRAHAM & LANEY, P.A., and that the attached Pre-Hearing Brief and Notice of Witnesses and Written Medical Reports to be Introduced as Direct Evidence on Behalf of Employer / Plaintiff were served upon Timothy B. Killen, Esquire, the attorney for defendant, South Carolina Second Injury Fund, this 27th day of July, 2012, in accordance with Regulations 67-210, 67-211, 67-213, 67-214, and 67-215 and such other law as may be applicable, by depositing a copy of same in the United States Mail, postage prepaid, addressed to:

Andrea P. Roche, Commissioner
SC Workers' Compensation Commission
P. O. Box 1715
Columbia, SC 29202-1715

Timothy B. Killen, Esquire
SC Second Injury Fund
100 Executive Center Drive
Santee Building, Suite 101
Columbia, SC 29210

Jeanie McDade
Secretary to Vernon F. Dunbar

South Carolina Workers' Compensation Commission
1333 Main Street, Suite 500
P.O. BOX 1715
Columbia, SC 29202-1715
(803) 737-5739
www.wcc.sc.gov

PRE-HEARING BRIEF
WCC File No:9930399
SIF File No: 141031

Employer/Carrier: Yuasa-Exide, Inc./Arrowpoint Capital Corporation

(In Re: Joe Mathis, Jr. v. Yuasa-Exide, Inc.)

Address: 1990 Corporate Blvd.

City: Sumter State: SC Zip: 29150

Employer Phone:

Preparer's Name: Latonya Dilligard Edwards

Defendant's Name: S.C. Second Injury Fund

Address: 100 Executive Center Drive, Suite 101

City: Columbia State: SC Zip: 29210

Carrier: Arrowpoint Capital Corporation

Preparer's Phone #: (803) 750-2214

A claim for workers' compensation benefits is made based on the following grounds:
x Injury ☐ Illness ☐ Repetitive Trauma

1. Compensation Rate: $ 2. AWW: $ **Date of Injury:** February 1, 1999
3. Type of injury and body part(s): Claimant sustained injuries to his brain, kidneys, skeletal, cognitive, neuropathy, cardiovascular and vestibular systems.
4. Facts in controversy: None.
5. Legal issues involved: The Fund offered to settle this case in 2011 but rescinded the offer when Carrier did not accept.
6. Unusual aspects: None.
7. Witnesses (designate if expert):* None.
8. Exhibits: Letter from S.C. Second Injury Fund dated November 28, 2011.
9. Medical evidence (indicate report pursuant to R.67-612; deposition or appearance):
The Fund will object to any of Carrier's APA submissions that were not submitted by June 30, 2011 per 42-7-320(B)(2).
10. Name, address, and specialty, if any, of the treating physician: Dr. C. West Jacobs, IV, Sumter Medical Consultants, P.A., 250 W. Wesmark Boulevard, Sumter, SC 29150; Tuomey Healthcare System , 129 N. Washington Street, Sumter, SC 29150; Dr. Marion Davis, Tumoney Regional Medical Center, 129 North Washington Street, Sumter, SC 29150;
11. Impairment rating(s); body part(s); physician and date of opinion: 49% impairment to the heart; 8% impairment to the whole person; 4% impairment to the vestibular system; 5% impairment to the auditory system; and, 29% to the brain by Dr. Edward L. Baker, Dr. J. Routt Reigart and Dr. Barry Weissglass dated May 22, 2008.
12. I am amending my Form 50/51 in the following manner: N/A

Signature: Latonya Edwards Email: latonya@dilligardedwards.com

Date of hearing: August 13, 2012 Time needed for hearing: _____

On behalf of ☐ Claimant ☐ Employer X SC Second Injury Fund

File this form and proof of service on the opposing party according to R.67-611 and R.67-212. Do not send medical reports.
* Commissioners reserve the right to admit expert witnesses at hearings.

WCC Form # 58
Rev. 9/07

58

PRE-HEARING BRIEF

STATE OF SOUTH CAROLINA)
)
COUNTY OF LEXINGTON)

CERTIFICATE OF SERVICE

The undersigned employee of Dilligard Edwards, LLC, 3790 Fernandina Road, Suite 103, Columbia, South Carolina 29210, does hereby certify that she has served the following named individual(s) with a copy of the pleading indicated below by mailing a copy of the same to them in the United States mail, with sufficient postage affixed thereto and return address clearly marked on the date indicated below:

COUNSEL SERVED:

Vernon F. Dunbar, Esquire
Turner Padget Graham & Laney, P.A.
Post Office Box 1509
Greenville, South Carolina 29602

PLEADING: FORM 58 – SECOND INJURY FUND'S ANSWER TO REQUEST FOR HEARING

Erica M. Samuel

August 3, 2012

Columbia, South Carolina

South Carolina Workers' Compensation Commission
1612 Marion St.
P.O. BOX 1715
Columbia, SC 29202-1715
(803) 737-5723

WCC File #: 9930399
Carrier File #:
Carrier Code #:
Employer FEIN #:

Claimant's Name: C. L. Williams SSN: Employer's Name: Yuasa-Exide, Inc.
Address: 55 Maxwell Avenue Address: 1990 Corporate Blvd.
City: Sumter State: SC Zip: 29150 City: Sumter State: SC Zip: 29154
Home Phone: (803) 934-1273 Work Phone: Insurance Carrier: South Carolina Second Injury Fund
Preparer's Name: Vernon F. Dunbar, Esq. Law Firm: Turner Padget Graham & Laney P.A. Preparer's Phone #: (864) 552-4601

REQUEST FOR COMMISSION REVIEW

Request for Commission Review by ☐ claimant ☒ employer (check one) Date of injury: 02/01/1999

The undersigned makes application for review of the findings of the Commissioner in the above-captioned case. The request for review is based on the following grounds: (State the grounds of your appeal in the form of questions presented. Each question presented must contain a concise statement of one proposition of law or fact. Refer to evidence by title and exhibit number. Use additional pages if necessary).

1. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that claimant did not have preexisting conditions of hypertension, cardiovascular disease, heavy metal poisoning, chronic obstructive pulmonary disease (COPD), pulmonary hypoventilation syndrome, rectal bleeding, coronary artery disease, pulmonary enema, tinnitus, loss of hearing and vertigo prior to his date of accidental injury of February 1, 1999, the error being that the aforesaid preexisting conditions predated claimant's date of accident of February 1, 1999.

2. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that claimant did not have any impairment that preexisted the lead exposure, the error being that the lead exposure continued during the entire course of claimant's employment from 1976 to 1999, with the last injurious exposure occurring on February 1, 1999.

(Check one) Oral argument ☒ is ☐ is not requested. Appellant's request for oral argument is waived if not indicated on this form.

I certify that I have served this document pursuant to R.67-211 by delivering a copy to:

David T. Pearlman, Esquire
The Steinberg Law Firm
P.O. Box 9
Charleston, SC 29402-0009

Ms. Virginia Crocker, Judicial Director
South Carolina Workers' Compensation Commission
P.O. Box 1715
Columbia, SC 29202-1715

Latonya Edwards, Esquire
Dilligard Edwards, LLC
3790 Fernandina Road, Suite 103
Columbia, SC 29210

Timothy Killen, Esquire
SC Second Injury Fund
100 Executive Center Drive
Santee Building, Suite 101
Columbia, SC 29210

E-MAILED

on the 7th day of December, 2012 by ☒ first class mail ☐ personal service ☐ certified mail.

Stephanie L. Pugh for VFD
Preparer's Signature

Attorney
Title

MAILED
December 7, 2012
Date

Check this box if you are not represented by an attorney. ☐

If the claimant appeals and is representing himself or herself, the Judicial Department will prepare the additional copies of this form and serve this form on the opposing party. R.67-701B. Otherwise, file the original and four copies of this form with the Judicial Department. The appeal must be postmarked no later than 14 days from the date of service of the Hearing Commissioner's decision. R.67-701 and R.67-205. Attach the filing fee to this form. Attach a Form 32 if you are unable to pay the filing fee. Refer to R.67-701 through R.67-711 for additional information.

3. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law and abused her discretion that even though evidence supports claimant having a preexisting condition of hypertension dating to 1982, finding that high blood pressure preexisted the lead exposure would be speculative, the error being that claimant's high blood pressure or hypertension continued to worsen after continuous exposure to various levels of lead or aggravated a preexisting and underlying condition.

4. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law and abused her discretion in finding that the medical questionnaire submitted on behalf of the employer and carrier are not supported by medical evidence in the record, the error being that the questionnaires were completed by competent and imminently qualified physicians who opined and concluded that the lead to which claimant was continuously exposed aggravated the hypertension and the other aforementioned health maladies.

5. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the evidence in the record indicates that lead exposure may have actually caused Claimant's high blood pressure, the error being that such a finding is against the greater weight of the evidence.

6. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that Claimant's heavy metal exposure over the twenty-five year period constitutes one injury, the error being that such a finding against the greater weight of the evidence and is contrary to South Carolina law.

7. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that claimant did not suffer with any preexisting conditions and, thus, there was no aggravation of preexisting conditions or combination of any preexisting conditions with the subsequent lead exposure injuries to create substantially greater medical costs and permanent disability, the error being that this finding is totally contrary to the evidence contained in the record and also consitutes an abuse of discretion.

8. The Hearing Commissioner erred in failing to find as a fact and conclude as a matter of law that the carrier proved that claimant suffered with preexisting conditions that were permanent and serious enough to be a hindrance or obstacle to employment or re-employment, the error being that claimant was eventually declared disabled because of the preexisting cardiovascular disease and cardiac problems; and that claimant's aforementioned health maladies are listed conditions under Section 42-9-400, which would therefore constitute a presumptive statutory hindrance or obstacle to employment and re-employment and increased medical costs and increased disability.

9. The Hearing Commissioner legally erred and abused her discretion in denying the employer and carrier's claim for reimbursement in view of the recently promulgated case of *Carolina's Recycling Group and Employers' Insurance of Wausau v. South Carolina Second Injury Fund* and *Burnette v. City of Greenville*, both promulgated by the South Carolina Court of Appeals, the error being that the Commissioner's decision to ignore the only evidence in the case constitutes an abuse of discretion and error of law.

10. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that claimant did not suffer an injury by accident or failed to find such with regards to claimant's exposure to lead beginning in 1976 and ending on February 1, 1999, the error being that the cumulative and cascading effect of lead exposure constituted an injury by accident according to *Grayson v. Gulf Oil, 292 S.C. 528, 357 S.E. 2d 479 (1987).*

11. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the carrier has not met all necessary elements for reimbursement pursuant to § 42-9-400, the error being that such a finding is against the greater weight of the evidence and contrary to South Carolina law.

State of South Carolina

County of Sumter

Arrowpoint Capital Corp./Arrowpoint Indemnity Company

vs. South Carolina Second Injury Fund

Re: C.L. Williams vs. Yuasa Exide 2013-CP-43-02286
Re: Mary McConico vs. Yuasa Exide 2013-CP-43-02283
Re: Joe Mathis vs. Yuasa Exide 2013-CP-43-02284
Re: James Huggins vs. Yuasa Exide 2013-CP-43-02287

Motions
June 3, 2014
Sumter, S.C.

BEFORE THE HONORABLE W. Jeffrey Young, Judge.

A P P E A R A N C E S:

Attorney for Plaintiffs

Mr. Vernon F. Dunbar
Ms. Stephanie L. Pugh
Ms. Ashley R. Forbes
Mr. Timothy B. Killen

Attorney for Defendants
Ms. Latoya D. Edwards

Margaret T. Sullivan,
Court Reporter

THE COURT: Yuasa Exide, Incorporated versus Arrowwood Indemnity Company under South Carolina Second Injury Fund. Mr. Vernon Dunbar is present for the plaintiff. And Ms. Latoya Dillard Edwards is here for the defense. Are you all ready to proceed?

MS. EDWARDS: Yes, Your Honor.

THE COURT: Give me a little background on his somebody.

Ms. EDWARDS: May I approach with the file?

THE COURT: You may, certainly.

MR. DUNBAR: Judge, do you have my brief?

THE COURT: No, I don't.

MR. DUNBAR: I will provide you a copy. May it please the court.

THE COURT: Yes, sir.

MR. DUNBAR: My name is Vernon Dunbar. And I represent Arrowwood Indemnity also known as Arrowpoint South Carolina Second Injury Fund. In this particular case the South Carolina Worker's Compensation Commission denied reimbursement. We're simply here to argue that the commission's order is subject to reversal. Why? They committed an error of law. Two, there is no substantial evidence to support their findings of

fact. Which because there are no evidentiary findings to submit, to support their findings of fact, that also constitutes an error of law.

Let me begin with by providing you an overview of the South Carolina Second Injury Fund, if I may. We have three cases, so what I tell you, will be applicable in all three cases. Or actually four appeals. As you are aware, the South Carolina Worker's Compensation Act is a no fault system. Meaning, if someone gets hurt, no fault, the employer pays. As a consequence of it being a no fault system, employer's were resigned not to hire people with preexisting disabilities, because they didn't want their worker's compensation premiums to go up. So as a precursor to the American Disabilities Act, our State legislature created the Second Injury Fund. Just like many other states across the country.

The Second Injury Fund was designed to encourage employer's to hire people with preexisting conditions with disabilities, so that they could work. Not to be worried about if someone cut their finger and they have diabetes and later they have to amputate their arm and what -- some other body parts, that they would not

be saddled with a permanent and total disability award when it was only a finger cut. Our legislature recognized that, and they provided reimbursement. And they tell us the grounds by which we get can reimbursement. So it's fairly simple and easy. And the bar is low when it comes to getting or obtaining reimbursement from the South Carolina Second Injury Fund.

Well in this particular case, the reason why we say they committed an error of law, the statute provides 42-9-400, which I have placed before you. It talks about the manner in which we can obtain reimbursement. In 42-9-400, the employee has to have preexisting permanent impairment. And if that is shown and that there is a subsequent injury combining with that preexisting condition, the employer is entitled to a reimbursement from the fund. Not only are they entitled to reimbursement from indemnity payments, but also for medical payments in this particular case. And if you look at 42-9-400 in its entirety which I passed up, particularly section D, it talks about the permanent impairment must constitute a hindrance and obstacle to employment and reemployment. But it also goes on further to

talk about certain medical maladies in which a hindrance or obstacle to employment and reemployment is again, there is a presumption. And it lists 33 items, epilepsy, diabetes, cerebral palsy, to name a few. And the one that we're focusing on in this particular case is, heavy metal poisoning.

In this particular case, Mr. Mathis worked at Yuasa Exide. Also previously known as ESB battery plant, also known as the Exide plant. It was later purchased in 1991 by Yuasa. And later became known as Yuasa Exide. Then later become known as Enersys. The statute says that in order for an employer to obtain reimbursement in Section (C)42-9-400 (C), when a claim is made for reimbursement thereunder, that the employer had knowledge of the permanent physical impairment at the time the employee was hired, or at the time the employee was retained in employment after the employer acquired such knowledge.

In this particular case, the plain reading of the statute says, a person doesn't have to walk into a plant, free and clear of any disabilities or have that disability when they walk into the plant. In other words, that person can develop a

medical malady while they're working at a plant, and if the employee and employer is aware of it, the employee is subject or the employer is subject to reimbursement from the fund. The commission held that because Mr. Mathis did not have heavy metal poisoning at the time he walked into Yuasa Exide well an exide plant, I should say, or ESB, in 1975, he is precluded or my client is precluded from reimbursement. That's simply not correct. That's not a correct reading of the law.

The correct reading of the law didn't come from Vernon. I think the plain language of the statute certainly would suggest, certainly implies, certainly explicitly states that Arrowpoint Capital and Yuasa Exide is entitled to reimbursement. But even if that were not the case, the law according to the Supreme Court has already ruled that an individual can obtain or have or acquire a disability or permanent impairment while they're working, and the case would still qualify for a second injury fund reimbursement. The case on point is South Carolina Second Injury Fund, or the State Accident Fund versus The South Carolina Second Injury Fund, which is cited in my brief. And I do have a copy

of that and I will hand it to you later.

In this particular case, Mr. Hunt worked for the Forestry Department, and he had worked there for 30 years. And during the 12th year of his employment, it was discovered that he had a severe cardiac problem. The fund took the position that the cardiac condition did not preexist at the time Mr. Hunt was employed initially with the Forestry Department. Our Supreme Court turns hands on the argument. And that's in saying that the condition of the roads during his employment, the employer became aware of it, and therefore was subject to reimbursement. That argument was made. That argument was made early on, and the court addressed that. And I am trying to find where I cited in my brief. In any event, it is contained somewhere therein. The court said that because he acquired cardiac disease well after he had began working, that he was -- the employee certainly was subject to second injury fund reimbursement. And the court ordered such.

The funds attempted to make the same argument and did make the same argument in the case of the Transportation Insurance Company

versus South Carolina Second Injury Fund. And I will hand you copies of that case as well. I believe I left it on the table there. And in that particular case, and let me hand it to you before I go any further. Your Honor, in that particular order of the the Supreme Court in that decision, which I argued, you will look at Roman Numeral IV, which is on page 6 of 6, Yuasa Exide. The fund made the same argument it did in the Hunt case. And in this particular case they argued, that Great American was not entitled to reimbursement because plaintiff's hypertension and liver failure did not preexist plaintiff's occupational exposure. The court simply said, we disagree. It doesn't t have to preexist. That's not the law. That's not what the statute says. That's not what this case says. So as a matter of law, they fail on that proposition.

Now this was a legal argument. The court had previously ruled on it. And that's the Hunt decision. Additionally, the court went on to say they didn't cite any authority for that position. No authority. You can't cite any authority. That's why I was so amazed by the South Carolina Worker's Compensation Commission, because earlier

I had argued the Lee Ernest Franklin case which is the transportation case, in which Great American was ordered reimbursement from the fund. The commission did that. And that was different makeup of the commission but that commission understood lead exposure. And from that standpoint, they ruled that it was an occupational disease. It was an injury by accident. It was cumulative trauma. And every time Mr. Franklin went into Yuasa Exide, it was a new exposure, exposing him to a dangerous level of lead. Which warranted reimbursement from the fund.

So the fund can state no case law, no statute for its proposition that the person had to have heavy metal poisoning, epilepsy, or any of the conditions before they starting working at the plant. It's simply not there.

Now let me go to the facts of the case. Joe Mathis was 60 or is a 62 year old male. He worked at Yuasa Exide from September 1975 until November 30, 1998. Why is September 75 important? Prior to 1983, there were no governmental regulations with respect to lead exposure. There were no permissible exposure levels whatsoever. It had not been established by OSHA. Records were

kept, but OSHA had not promulgated any rules or regulations. That wasn't until 1993. And OSHA regulations are contained in the record on appeal. So that's why that is important. So from 1975 until 1983, Mr. Mathis' blood lead levels were extremely high. They exceeded OSHA's levels in terms of permissible exposure limits. He worked there for 23 years exposed to lead daily.

On June 3, 2009, he filed a claim because he's having memory problems, cognitive problems. He doesn't know why. Under worker's compensation law when you are exposed to things such as, asbestosis or asbestos and you develop asbestosis, it has a latency period. Lead is similar to asbestos. It has a latency period of about 30 years. And in this particular case, Mr. Mathis filed a claim. And under the occupational disease statute as well as the statute of limitations governing worker's compensation, an individual can file a claim two years from time he is definitively diagnosed, or when he becomes disabled. He was still working some menial jobs, but he was not definitively diagnosed until later on in September of 2009.

Mr. Mathis' blood lead levels are crucial

in determining what constitutes a preexisting condition. We contend that he was exposed to heavy metal poisoning, had heavy metal poisoning when he worked at ESB and Exide before it was sold to Yuasa. And keep in mind, ESB, Exide and Yuasa had an in-house medical department. So when you Yuasa purchased industrial battery division of Exide, they got all those medical records. They knew the blood lead levels. They do every complaint of every employee. So They had the knowledge that these people were suffering with certain maladies; had certain preexisting conditions, yet they retained them, which is what the statute says, 42-9-400, retained them in employment. That is crucial, because once we knew that he was exposed to lead at enormous levels that well exceeded OSHA standards, as a matter of fact, Yuasa Exide's industry standards were actually lower than OSHA standards.

However you look at, this gentleman's exposure to lead exceeded both OSHA's standards, as well as industry standards. Clearly, there was heavy metal poisoning. There was lead exposure that resulted in him being exposed to unhealthy levels of lead. And the only way to treat lead

when you are exposed to it, is to engage or participate in something called the medical removal program, MRP. Basically you have to remove the worker from the lead environment until their lead levels or blood lead levels return to a safe level. Interesting, what is a safe level. If you and I walked into a plant right now and asked or a doctor's office and they measured our blood, most people is between 3 and 7. You will see in this particular case, this gentleman's blood lead level's went up 9 fold from the time that he was hired.

As a matter of fact, he was hired in September. In November it had already increased 5 fold if you go by using the No. 7. It went up 35 in just two months of employment. But this is when it was under the control and ownership of the Exide Corporation. This gentleman may have been placed in the medical removal program on one occasion. It should have been on many occasions, but was not. In any event, his blood lead levels from 79, range from 62 to 64. And from 78 to 79, never went below 50 micrograms per desilter of blood. Clearly in 70's assuming had heavy metal poisoning.

Yuasa Exide purchased the plant in 1991. So with this new employer, they retained previous levels of lead exposure, heavy metal poisoning as the result. They still retained, They still make lead batteries. So he's still exposed to lead even though it's not in the 60's and 50's, he's still exposed to lead on a continuing basis. So this lead that he's exposed to from 1991 on, aggravates, accelerates the underlying condition. So he's exposed to heavy metal poisoning. And there is no dispute about that whatsoever, that he has heavy metal poisoning because of exposure to excessive levels of lead.

The reason why this case is baffling is because the fund put in no evidence. No evidence whatsoever. Even though the law as I read the statute, doesn't support their position. Even though the Supreme Court's holding in the case of State Accident Fund versus Second Injury Fund, In Re: Hunt, doesn't support their position. The commission ruled that Mr. Mathis had to have had heavy metal poisoning before he was ever employed in 1975. There is simply no proposition for that. Let's take a look at the evidence that we submit.

We submitted medical questionnaires as

well as the medical reports from various doctors. And what is interesting in the record on appeal in 204, our experts, their qualifications. Were not refuted one iota. Dr. Baker, very impressive credentials. And if you look at page 204 of the record on appeal in 205, it talks about it. He is an occupational -- he has a degree in occupational neurology. He's a professor, internal medicine doctor. He's a epidemiologist. An expert in toxicologist or toxicology; is a professor at the Harvard University School of Public Health. He worked at the CDC, not OSH, assistant surgeon general.

Dr. Howard Hugh who also examined Mr. Mathis. Harvard University. Medical School at the University of Michigan. And this is on page 206. He's telling that he was on a Harvard Faculty from 1980 to 1985. Dr. Rigor, John Rigor beginning on page 207 in the record. Dartmouth College. Harvard Medical School. Consultant to U.S. Environmental Protection Agency. Also to the CDC. Despite these impressive credentials and their testimony, and their medical opinions, that Mr. Mathis' exposure to lead on November 30, 1998, aggravated the preexisting conditions. Basically

what they're saying from 1998 or from 1975 to 1998, he was exposed to lead. But from 1998, the last time he was at the plant it aggravated an underlying preexisting condition. More importantly, it combined with his present problems to render him unstable.

The reason why November 30, 1998, is so important, the date of the accident, controls in worker's compensation. The last time he was exposed to lead, which is the last injurious exposure, was on November 30, 1998. And that's on the page 92 of the record on appeal. You can see, and it's APA page 1.

THE COURT: And you're referring to this and I have none of this in front of me. You understand.

MR. DUNBAR: I'm sorry, Your Honor. I do have a copy of the record, and I will provide that to you.

THE COURT: And you keep referring to it. I'm looking through your report, and I have nothing to refer to.

MR. DUNBAR: Well let me give it to you. I'm sorry, I thought you had that part. We had overnighted it on Thursday. Last Thursday.

THE COURT: Okay.

MR. DUNBAR: So I apologize. I don't know what happened with it. I think it was received.

THE COURT: All right.

Mr. DUNBAR: I'm sorry. I apologize. The questions were asked, right in the accordance with the statute. Did Joe Mathis have preexisting conditions or heavy metal poisoning and cognitive impairment. Drs. Baker, as well as, Shipman, all said, yes. Were there preexisting conditions. Would they constitute a hindrance or obstacle to employment or reemployment. Again, I am looking at page 92. Yes. They needn't have to ask that question, because the statute says in certain medical maladies and diseases, there is a presumption that there will be a hindrance or obstacle to employment and reemployment. And those conditions are: diabetes, epilepsy, and includes heavy metal poisoning.

So, Your Honor, we believe that based upon the evidence that's been presented, certainly the substantial evidence does not support the commission's decision in the order. In order for this court to affirm the commission's decision, there has to be some evidence in the record. The

fund put in no evidence. They did not challenge the qualifications of our experts, which I think are imminently qualified. They put in no countervening medical or medical report or medical treatise or anything of that nature.

In essence, there is absolutely no evidence to support the fund's position. The question to the fund is, what evidence do you rely on. What expert do you rely on to say that it had it preexist. This is a very technical medically weighted issue. You have to medical evidence. This is heavy metal poisoning. You can't just can't make it up as you go along. They put in no evidence. I submit had they put in evidence, you probably could affirm the commission's decision. But because there is absolutely no evidence whatsoever, this court is precluded from affirming the commission's decision. All the medical evidence in support of reimbursement was proffered and submitted into evidence by Arrowpoint Capital Corporation and Arrowwood Indemnity.

So we take the position that Mr. Mathis' heavy metal poisoning caused his cognitive problems and mental retardation. What is

interesting about this case is, this gentleman worked there for 23 years. And if you look at some of the early performance evaluations, and in particular, the performance evaluations in and he worked at Yuasa Exide. He performed in an outstanding matter. However, towards the end of tenure, he was making mistakes. He could not keep up. He was being chastised for not completing paperwork that he had had prepared in a very exemplary fashion for the past 23 years. He was ultimately terminated. He was not diagnosed as having mental retardation until 2009, when he was seen by two neuropsychologists. One, Dr. Randy Way, whose report is in record. And I also employed a neuropsychologist because this was the plaintiff's expert, Dr. Nicholas Lind, post trama resources. He confirmed it. And they both attributed his mental retardation to his exposure to lead. And attributed to past exposure to lead.

I think the fund's argument is this in a nutshell, and I anticipate it. Why should the industry who manufactures batteries be rewarded by giving them reimbursement. Let me give you the quick and simple answer to that. Arrowpoint Capital Corporation insures Yuasa Exide. Yuasa

Exide did not take control of that plant until 1991. So from 1974, when they started manufacturing lead in Sumter, lead batteries, until 1991, it was all under ESB and Exide. Before there were any OSHA promulgated or OSHA regulations promulgated, before safety was of utmost importance. But in worker's compensation the last carrier, the last employer, stuck with the claim. So no matter what we did to protect workers and I say we, Yuasa Exide and Arrowpoint Capital Corporation, we still have to pay for the sins of the previous employer. And that's the beauty of the Second Injury Fund. Because the Worker's Comp. Compensation when you look at the Franklin case, which is pages 346 through 363, there are a lot of orders there. That was the first case that went up. It ended up being in the transportation case that the Supreme Court decided.

It recognizes the fact that the last employer is on the hook. And as a result of that, the last employer who took safety precautions should be entitled to reimbursement. As a matter of fact, Mr. Mathis said it wasn't until 8 years before he was let go, that they installed showers

in the facility. Yuasa Exide did that. And I submit that because this gentleman did not know that he had mental retardation and the statute that I handed up says that if a condition is not known to the employee, and that's 42-9-400, subsection C, we're still entitled to reimbursement. Shipman he did not know he had mental retardation caused by lead. It's an unknown condition. It was unknown to him, so of course it was unknown to us. It was not discovered until 2009. So the fact that mental retardation was an unknown condition, we take the position that we're certainly entitled to reimbursement.

THE COURT: Thank you.

MR. DUNBAR: Thank you, Your Honor.

THE COURT: Yes, ma'am.

MS. EDWARDS: Good morning, Your Honor. I promise to be brief. The statute that we are talking about here is 42-9-400. That's it. We are not talking about blood lead levels. We're not talking about the various articles Mr. Dunbar cited in his brief. We are not talking about OSHA standards. The statute is 42-9-400. It requires the carriers meet certain elements. Now I don't

know if this is the -- 42-9-400.

In addition to that, what Mr. Dunbar left off, it also requires that the Second Injury Fund have notice of a potential claim for reimbursement prior to the payment of 7 years of benefits. And I say that just for the court's benefit. That's not an issue here. Okay. And that's 42-9-400 (G). But let me go to the statute, because that's what is relevant here. And one of the preexisting conditions that the carrier has alleged -- is alleging, is brain damage.

Now I am going to cite the substantial evidence that Mr. Dunbar says is not in the evidence. And I'm assuming that the record Your Honor has, is has paginated the same way as the pleadings that you submitted. Because we don't have the record here. It was paginated the same way. So I am going to trust that the pages are consistent, because that's how its cited in my brief. The pages that I am going to cite, as it relates to the brain injury, and certainly we believe that this court has all of the evidence to affirm the decision below. And let me also point out, the decision was made by the commission in bulk. And Your Honor knows what that means. It

was all of them, with the exception of the one single commissioner who decided the initial, initially. And so I wanted to point that out. So it was not just a panel of 3 as is customary, it was all 6 of them.

And so the brain damage issue, let me talk about that. The commission found that the claimant did not have brain damage prior to his occupational exposure. And they based it on this, that carrier at pages 30 and 31. This is an initial occupational history form that claimant completed in 84. In 1984. Let me point out that this claimant worked for Yuasa Exide for approximately 23 years. And let me just do an aside. Mr. Dunbar made much ado about the fact that it was -- that they may have been owned by different people or whatever. The commission did not buy that argument.

In fact, yes, we admit that's it a different name. The company had a different name. We doubt that it's a different employer. And we've been back and forth on that, but the commission didn't buy that argument, and I'm requesting that Your Honor not either. In 1986, the claimant's examination at APA page 33,

claimant was healthy. That's not anything that one commissioner decided. That's in the record, the medical records. Carrier APA pages 33 to 36 and 38.

THE COURT: And you are referring to, and I'm looking at the record on appeal.

MS. EDWARDS: I sure hope it's the same, Your Honor. Go ahead. I hope it's the same page. Because that's what I am concerned about, because I didn't get the record either. And so I'm basing the pages on the prior APA submissions. And so that was my main concern when Mr. Dunbar passed out a record.

THE COURT: And I'm looking at 33 is page 3 of the consent scheduling order.

MS. EDWARDS: Your Honor, may I approach, please?

MR. DUNBAR: Your Honor, let me just explain. The APA's that we talked about, is the top page, the top number. Because we put the transcript's record. And it is numbered. See it's at the top.

MS. EDWARDS: Well that necessarily means that my pages are incorrect, because I don't have this. I am sorry, Your Honor, I do feel like I am

at a disadvantage here. But I am going to make my argument. Then if Your Honor will, I can submit them again once I get a copy of his new pages that, you know, I am sorry to have to frustrate the court as frustrated as I am. I am sorry to have to do that. But I will do is, I will just cite the evidence, and I will submit a new record. Do you have a copy for me?

MR. DUNBAR: Yes.

MS. EDWARDS: I can cite and include a new brief---

THE COURT: All right.

MS. EDWARDS: ---with the new pages on it. I'm sorry, Your Honor. Plaintiff's physical examination in 1986 through 95, did reveal elevated cholesterol, but he was otherwise healthy. August 12th 2009, plaintiff testified in the deposition. The commission relied on that also. He testified that he worked at Yuasa Exide for 23 years from 1975 to 1998. And he noted that his most recent physical examination, this is in 2009, years after he stopped working there. His examination was normal. And in the last 25 years he had visited the doctor for a pinched nerve in his back, which required physical therapy and then

he stopped. This is was after he stopped working for Yuasa Exide.

Now there is a vocational assessment because once again, they are alleging brain damage. There is a vocational assessment indicating that this claimant had severely deficient achievement and was functioning to be illiterate. But the records indicate that he was high school graduate. No history of failing grades. Had never been diagnosed with a learning disability, or never had a need for placement in any specialized educational classes.

Now there is resent case law that Mr. Dunbar did not cite. And I think it might be in his brief, Sparks vs. Palmetto Hardwood Incorporated. And that case requires that in order for a carrier to prove brain damage, it has to be both permanent and severe. And the citation for that case is the Southeastern citation is 738, S.E. 2d, 831. And it's a 2013. There's also the case of Crisp vs. SouthCo. The citation for that if 738 S.E. 2d, 835. These cases were decided together. The South Carolina citation for Crisp is 401 South S.C. 627.

Now on that, the medical evidence is

clear. This claimant did not have brain damage. And once again, I believe that Mr. Dunbar is arguing over the weight of the evidence. And we all know that the weight of the evidence is something that's afforded to the commission. They do not have to blindly rely on a questionnaire from two medical physicians, I might add, who never treated this claimant, never. And so they did not rely on those questionnaires. And they were cautioned. They were wise not to do that, because in another case that Mr. Dunbar cited, which I actually argued and was reversed on, Carolina's Recycling.

The reason that that case, the main reason that case was reversed was because, and this is a quote, that case citation is 398, S.C. 480, 73, S.E. 324. That's a 2012 case. The reason that our Court of Appeals reversed that case was because they found the appellant panel relied exclusively upon an evaluation by a non-treating physician who only met with the claimant on one occasion. So certainly, I would say that the commission was well within it's authority and their authority, to not place as much weight on two treating physicians. I admit they have an

exceptional credentials. Exceptional. But they were not treating physicians. They never treated this plaintiff. Or any of these other plaintiffs that we're going to argue about after this case. And so the commission was well within its authority to do that.

One of the other things that I wanted to point out is that, well let me go back, Your Honor, get back to the substantial evidence. One of the other issues in the case was whether the heavy metal poisoning was both the preexisting condition and subsequent injuries. We dispute that that's the case. Now Mr. Dunbar cited Hunt. State Worker's Compensation Fund. I forgot the site. It is in his brief, I think on page 12 at the bottom. It's cited somewhere there, State Worker's Compensation Fund.

And one of the reasons that that case is distinguished is because, the Hunt case also was decided based on a particular statute that applies to two law enforcement individuals and first responders. That statue I believe is 42-30-11. I might be wrong, Your Honor, but I think that's what it is. But and under that statute, firefighters, people like that, police officers, a

heart attack is presumed to arise out of and during the course of employment. That case was decided on something totally different. And once again, we would assert that it is not relevant. Even if it is, it's distinguishable.

As it relates to the once again, the blood lead levels, all of that, that's not relevant to the appeal. The only thing that's relevant is 42-9-400. And the commission found that this carrier did not met those requirements. Now some of the other things that the commission also relied upon well and Mr. Dunbar also cited Burnette. I wanted to distinguish Burnette as well. That's a case that was reversed and remanded. That citation is 737 S.E. 2d. 200, and it's a 2012 case. In this case, the Court of Appeals reversed and remanded the case to the commission because they had a concern that the commission's decision was based on a single commissioner rather than the medical provider. And in this case everything that the commission based it's decision on, was based on the medical evidence in the record.

Now like I said, Mr. Dunbar might be a little concerned that the commission didn't read

the evidence the same way he did. And that's really what the issue is. The commission didn't read the evidence the same way he did. They weighted different evidence other than the way he would. But we all know that that's not the standard. Just because reasonable minds differ, that doesn't mean the case is subject to reversal. It means that even if another commissioner would have decided differently, if there's substantial evidence, the case still is affirmed. And so that's not the case. We don't have any reversible errors here.

We have a carrier who didn't get reimbursement, and they're alleging that there are errors. There is no errors. The substantial evidence in the record supports affirmation. And the beauty of the Second Injury Fund statute is not -- it's that the carrier has to meet the requirements. They're outlined in 42-9-400. And we would assert they don't meet the requirement as the commission found, because this lead poisoning that's alleged is not both the preexisting condition and the subsequent injury.

In addition, the preexisting brain damage that is alleged, has not been proven based on

their own evidence that's in the record. And one of the things I also want to point out is that Mr. Dunbar appears to be arguing the last injuries exposure rule. That's a rule that came out of another case that doesn't -- is not relevant to this case, because that just determines what carrier is responsible for the claim. It doesn't say anything about reimbursement, Your Honor. The Second Injury Fund statute is a reimbursement statute. Even if the carrier pays. And may I also submit or indicate that the carriers didn't pay any of these claims. They settled.

So none of these underlying claims were adjudicated by any tribunal or anything. They settled them to avoid litigation. They settled them on a doubtful industry basis, and now they're coming back trying to get medical costs and temporary total that they did not pay. And so, those are all of the things that the commission took into consideration. They were well within their authority to consider those things. And we would assert that you have all of the evidence that you need and wanted to affirm the decision of the commission involved.

And once again if you have any questions,

Your Honor, I'll be happy to answer. But I will resubmit the brief based on the new record and the new pagination. I apologize for that. But I will submit that ASAP.

THE COURT: Thank you.

MS. EDWARDS: And it's probably going to be the same thing for the other cases too if there is no different record. Do you have any questions, Your Honor.

THE COURT: I do not. Mr. Dunbar, very briefly.

MR. DUNBAR: In response to Ms. Edwards' argument the Form 54, which the fund requires us to file and that's page 40.

THE COURT: Of yours or hers or whose?

MR. DUNBAR: Of mine. Page 40 of the record.

THE COURT: Okay.

MR. DUNBAR: She said that we settled those cases. She's absolutely right, but that does not preclude us from reimbursement. Line 3, states the carrier concluded the disability claim by agreement. It's a settlement agreement and release. It's how most of the worker's compensation claims are resolved. So that's

certainly not a bar to this particular case.

With respect to whether someone had -- whether he had cognitive impairment prior to his employment, that's irrelevant. That is a red herring. Simply if you take a look at page 224 of my record on appeal, and this also indicated in the OSHA statutes. But our experts say, said this: toxic exposures cause delay cognitive impairment. Exposure to a neurotoxic agent such as lead, causes brain damage, which is manifest only later in life, during the time in which the normal aging occurs; thereby, accelerating the aging process. That's page 224 at the very top.

This gentleman did not know the ill effects of lead until a number of years later. It has been corroborated and substantiated by two neuropsychologists. Also, the experts that I cited, Drs. Baker, Hugh, and Rigor. More importantly, these gentlemen have over 80 years combined of clinical research. They treat patients. They saw. They evaluated Mr. Mathis. She said they weren't treating physicians. Mr. Mathis got most of his treatment from Yuasa Exide, the in-house medical department. That's where all the employees went to get their medical.

He didn't go to Urgent Care or have a family doctor. He went there. The record is replete with that.

Your Honor, with respect to the brain damage, Crisp vs. Sparks, those are totally separate issues. We're talking mental retardation. We're talking cognitive impairment. That's Crisp vs. Sparks, stands for a physical brain injury wherein someone gets lifetime indemnity and lifetime medical. In this particular case, there was no evidence of a permanent physical brain injury. Certainly significant mental retardation caused by the impact of lead on his cognitive function.

Also with respect to the weight of the evidence, I find it interesting that she would make an argument. You can only weigh evidence when there's something to weigh. The fund put in no evidence. Where are their expert reports. They did nothing to refute that. With respect to the law itself, the commission has acted in a contradictory fashion.

In the Lee Ernest Franklin case, the same commission, and that's again page 346, look at findings of fact 3 through 6. The commission

found that, and Mr. Franklin also worked at Yuasa Exide. He had renal kidney failure because of high lead levels. The same as Mr. Mathis. And what the commission found was, every time he went into the plant, that was a new, separate and distinct accident. The commission in this case gave no reason for departing from that line of reasoning. The reason why Franklin, which was incapsulated and the transportation insurance case was affirmed because it is on all 4's with the statute, and with the case law that's been promulgated by our courts regarding Second Injury Fund reimbursement.

Finally, Your Honor, this case was settled, but we were ordered to go to mediation. The commission ordered us to go to mediation. That's for purpose of mediation. It helps all parties. So we resolved the case and after we resolved it, we filed our claim for reimbursement. Now, of course, the fund was well within its right to deny it. But if you're to deny it, you've got to put forth some evidence to give you something to weigh. I submit because the lead exposure delays, you don't see, it's a delayed response with respect to the neurocognitive functioning

later. It comes out later. They gave you absolutely nothing to weigh.

It still goes back to that argument that they made in transportation case. I'll say section roman numeral IV, they made that same argument and the Supreme Court said, you're wrong. We disagree. They said it emphatically. That's not the law. Thank you.

THE COURT: Thank you. I will take this one under advisement.

MS. PUGH: My name is Stephanie Pugh and I represent the appellants Yuasa Exide and Arrowwood Indemnity Company. And it's the South Carolina Worker's Compensation Commission. It is our position that the commission created or committed errors of law and incorrectly deciding questions of fact; in that, they concluded that we are not entitled to reimbursement from the South Carolina Second Injury Fund. As has already been discussed today, the purpose of the fund was to encourage employers to both hire and retain disabled employees. This was prior to the advent of the ADA. And just as an example if an employee had a back injury in 2004. And suffered another back injury in 2006, you could seek recovery if you had

knowledge of that 2004 injury.

In this case the injury is lead poisoning. Or the subsequent injury is lead poisoning. This case involves a gentlemen by the name of C.L. Williams. He was employed at the Sumter Battery Plant for 23 years. He was first hired by ESB Incorporated in 1976. Later hired by Exide Corp., and then there was an acquisition purchase, and he was ultimately hired by Yuasa Exide in 1991. As I lay down the chart, this in order to obtain Second Injury Fund reimbursement. You have to have four, you have a set of four elements. Those four are that the claimant had a preexisting condition prior to date of injury; that the employer had knowledge of that preexisting condition; that the preexisting condition was a hindrance or obstacle to employment and that the preexisting condition or impairment combined with or was aggravated by the subsequent injury. In this case, the lead poisoning to result in substantially greater disability.

The main issue in all these cases is the error of law we believe the commission committed, in that they decided for the first time ever, that the preexisting condition had to preexist the

first day these folks were ever exposed to lead at a Sumter Battery Plant. We were shocked when we got this order, because nobody argued it below, so it came out of the blue for us. And it was contrary to law as we knew it at that time. 42-9-400, which is the governing statute, specifically states that the preexisting condition has to preexist the disability suffered by the subsequent injury.

So the question becomes, when did those preexisting conditions have to preexist. What date rules. It's our position that the earliest, the date of injury, rules. In worker's comp. typically that's the case. In an occupational disease, which is what lead poisoning is, does not exist until an employee becomes disabled and discovers that his condition is compensable. It's just like a mesothelioma case. There's a latency period. If you don't know you have mesothelioma until 2012, your time period, your date of injury is going to be until 2012.

In this case these folks didn't they were poisoned. Didn't know they had a lead poisoning condition that was compensable under the act until they were evaluated by a specialist. In my case,

that was in 2008. But even if we use the date of injury as the date, then we can establish that all of preexisting conditions had preexisted at that time. Now as Mr. Dunbar argued earlier, the arguments that were by the commission were what the fund argued in the Lee Ernest Franklin case. And that the Supreme Court shot down the argument in that case. And they said, there's no case law to support such a proposition that a preexisting condition had to preexist, the first ever exposure. Because there was no authority, they shot down the argument. It's the same as the case here. There is no authority. The commission has nothing. Cited no case law upon which to hang its hat for this decision. And as a result of such, we believe they committed an error of law that should be reversed.

Now regarding the errors of fact or let me just say alternatively, there were multiple employers here, because the company changed hands on multiple occasions and was purchased. So by the time Mr. -- well the last employer of Mr. Williams was Yuasa Exide, and that happened in 1991. So even if they say the preexisting conditions had to preexist the day exposure with

the employer, we're not even looking at 1976, we're looking at 91. That's just an alternate argument, because we believe that at the earliest, the date of injury would control here. But regarding the errors of fact, the question becomes, is there substantial evidence in the record to support the commission's findings on these errors of fact.

All the errors of fact surround the four elements for reimbursement. Either the commission found that they didn't exist in the case, or they didn't address them whatsoever. And just as in Mr. Vernon Dunbar's cases, important to point out in this case, there's only one page of evidence submitted by the fund. We submitted nearly 100 pages. They submitted one page. And that one page is a letter.

THE COURT: The one page is pretty on point.

MS. PUGH: Okay.

THE COURT: We don't, as judges and rulers, we don't count pages.

MS. PUGH: Well that one page is a letter resending a settlement offer in this case. It does rebut any of the evidence we set forth

establishing the elements for reimbursement. All it does it is resend a settlement offer. So it does nothing. It certainly does not qualify a substantial evidence in this case regarding any element required for reimbursement. It's just correspondence for sending an offer of settlement. We believe that we did establish all the elements for reimbursement in this case. And the first one being the preexisting condition in my case.

This guy, was -- he had a lot of problems. I mean this case is pretty much a poster child for what a reimbursement case would look like, prior to receiving this decision. He had a terrible heart condition. Cardiac disease. He had was hospitalized. Ultimately rendered disabled and removed from the work force because of heart condition. He had pulmonary problems; hospitalized for those as well. Those are both two hindrances or obstacles to employment, presumptively, hindrances or obstacles establishing another element, element No. 3 under the statute.

And the medical doctors, the experts, Dr. Shipman and Baker the experts, concluded that these were all preexisting conditions. The heavy

metal poisoning. This guy was exposed to heavy metal for 22 years. The doctors said on their questionnaires that the last day the guy was exposed, the last day Mr. Williams was exposed to heavy metal poisoning or to the lead, that was a no injury. Because it aggravated whatever existed the day before. So the last day constituted a new injury. And that's at APA and 3 and 6.

Now you've heard this before but the case law suggests that the heavy metal poisoning could be both the the preexisting and the subsequent injury. Now all I have to do is establish one. It could be the cardiac disease. It could be the pulmonary disease. It could be the hearing loss or the diabetes that's in this case. But in addition to all of those, we also believe we have a heavy metal poisoning as a preexisting and subsequent injury. And in this case the -- well we referred to the Hunt case earlier at 313, S.C. 536.

In that case the claimant who worked for the Forestry Commission and was about for 20 years was then diagnosed with heart or coronary artery disease maybe let's see like 13 years later. He had a worsening of the heart disease. And the

Supreme Court that satisfied the requirements for reimbursement from the Second Injury Fund. The Second Injury Fund said, no, it was just one injury just like they're doing here. The Supreme Court disagreed with that, and felt that it was both a preexisting and subsequent injury. The same thing happened in the Springs Industries case at 296, South Carolina, 359.

In that case, the lady was exposed to cotton dust and was then was diagnosed with cenosis. That was a 1989 case. And in that case the court too found that yes, the cotton dust exposure was a preexisting condition. And that the worsening of that exposure of the cenosis, was the subsequent injury. So we believe the case law clearly illustrates that we are entitled to reimbursement not just on cardiac disease, although you only have to have one. Pulmonary disease, but also heavy metal poisoning. I rely on my brief for the arguments regarding the hearing loss and diabetes. But I just would request that you remember that. I only have to establish one preexisting condition.

Regarding the hindrance or obstacle to employment, I've already mentioned this. But

cardiac disease is presumptive under the statute. There is nothing in the record at all to rebut that presumption. Pulmonary disease is also a presumption under the statute as a hindrance or obstacle to employment, and so is heavy metal disease and diabetes. So we believe that the commission erred in concluding that we did not establish a preexisting condition. Especially since the experts concluded otherwise. And there's no evidence rebutting the presumption and there's no evidence, period.

Now regarding the element of substantially greater disability, which is No. 4 on the chart up there, we have two specialists. Two lead experts. And Mr. Dunbar has already talked about their credentials. And they have both concluded that as a result of this lead exposure, Mr. Williams preexisting conditions, the cardiac disease, the pulmonary disfunction and so forth were in fact aggravated and did result in substantially greater disability. This is also supported by the literature that were presented. The only evidence in the record would establish that we do in fact have -- that we have in fact satisfied this element.

Now knowledge which is element No. 2 listed up on there on the chart is not really issue in this case, because Yuasa Exide had an in-house medical department. And all of their records were accessible to the employers. So really never had been an argument we didn't have knowledge. So that's not really a point. We believe the commission failed to find that we established the element of knowledge. At just the conclusion, I would draw your attention to your Burnette vs. City of Greenville case. That is the case that the South Carolina -- and that's at 401 S.C. 417. 401, S.C. 417.

The Supreme Court actually denied cert. on that case a few weeks ago. I think maybe two weeks ago. And in that case, the Court of Appeals said the commission can only disregard medical evidence when there is other competent evidence in the record. I will submit that there is no competent evidence. There is no evidence, period upon which the commission could have based this decision. So it's our position that they have not only committed a reversal error of law, but have also committed errors of fact as there is no substantial evidence in the record to support

their decision.

Any questions, Your Honor?

THE COURT: Not at this point. Thank you. Yes, ma'am.

MS. EDWARDS: I will bring everything with me, Your Honor. Hopefully this will be the last time, I will have to stand up. Let me start with C.L. Williams once again. All of our arguments are substantially the same. And I've also been advised that the record pages that I've cited are the top numbers and not the bottom. I don't know if that makes a difference for Your Honor. And I told your law clerk just let me know if you still need these submitted with the bottom numbers in them, I will be happy to do that.

On C.L. Williams, we also again believe that there is substantial evidence in the record supporting the commission's decision. And we're asking that it be affirmed. Now just a few of the things, the commission determined that -- I'm sorry, let me start over. The reasons the substantial evidence that we are citing to, and Your Honor is correct, the evidence is the evidence. Once again I think the carrier is asserting that they disagree with the weight of

evidence, that the commission assigned to their records.

As it relates to the C. L. Williams case, this claimant requested reimbursement. They requested reimbursement based on their lives and previous conditions. Now at APA page 8, which I think the top numbers we are talking about, Claimants 1983 physical examination indicated no findings of lead involvement. He can continue working in his area. Page 9, that's 1984. Because he worked for this particular employer for, since 1976. A long time. He worked there for some part of 7 years. I can't put my finger on the date, but it's in my brief, Your Honor. And so that's -- those are some of the medical records.

In addition, page 11 the physical examinations from 84 again, he could function safely at the present job without any complications. A variety of pages, 14 and 15, 17, 19, 21, 23, 25, 27. These are 1988 and 1997 records that revealed hypertension and evaluated lead levels. But once again, the commission found that the exposure was not of the person's subsequent injuries, but one cumulative injury.

The deposition that we took of their medical professional, also indicated that it was one cumulative injury.

And at carrier APA pages 44 and 45, a 1994 record, claimant presented for a variety of other problems that were easily remedied, fever, cough. And at page 46 through 51, claimant continued to follow up. And the medical records indicated that hypertension and congestive heart value were present. But once again these conditions and most of them were caused by the lead, they did not preexist the lead exposure. In 1982, I believe that claimant -- I am sorry, there is a history blood pressure dating back to 1982, and the lead exposure dates all the way back to 1976. And so at pages 52 through 56, these records indicate that the claimant did not develop these conditions until after the occupational exposure.

Now another case that the claimant cited Hunt again, I will just reiterate what I said. That case was decided we believe based on the statute 42-30-11, which applies specifically to first responders; such as, firefighters. Carrier also cited Burnette vs. City of Greenville. And once again, we would assert that that case is

distinguished, because the commission based its decision on the medical evidence. They did not reverse, or did not create conditional medical records. They used the carrier's medical records. Also Burnette was decided solely on medical issues that necessarily required an interpretation of diagnostic testing; such as, MRI's and CT scans.

Here the issue, one of the main issues is statutory interpretation. Whether this is a first and subsequent issue. That's clearly not a medical issue. So those are two major reasons that the case that we're talking about today is distinguished from Burnette. Also as it relates to the competent evidence issue that I believe Ms. Pugh mentioned, the case law doesn't say that medical questionnaires be only competent evidence. If the commission could consider anything in the record. And once again, I'll go back to Anderson, which is an old case, but it's still good law. It's 2002 South Carolina, 54, 24, S.E. 2d. 104. It's a 1943.

It allows the opinion of medical experts to be considered. And it says, may constitute substantial evidence. It does not exclude other sufficiently compelling evidence from being

considered. And we believe that the commission considered the totality of the medical evidence. No, they didn't give it the same weight that the carrier was giving it obviously. But we believe once again, this is case was decided, involved as well. The commission thought it important enough that 6 of the 7 be present with the exception of the commissioner that actually heard the first -- the initially case brought by the carrier. And so we believe that there is substantial evidence in the record to support an affirmation in this particular case.

Now, I can move on to the my next case, if you would like. I don't know the Court Reporter, I don't know how that's going to work.

THE COURT: That's going to make it difficult. Let me get the next case, and then we will allow you.

MS. EDWARDS: Okay. That's fine.

THE COURT: If you want to say ditto.

MS. EDWARDS: That's fine, Your Honor. Whatever works. I'll step aside. Thank you, Your Honor.

THE COURT: And what I am going to need to you all to do is really, on this appellant's

record on appeal, you all got to get on the same page. And she was affirmed. I am going to order that all of your pages now go back to date stamps, because that's what I have got here. You are going to have to go back and redo your briefs based upon date stamp, because I can't go back through and hope that that's the No. 5 that you are talking about, and make a decision. I need to know the exact page you are talking about. Which one is next? This is James Huggins?

MS. FORBES: Yes, Your Honor.

THE COURT: And you are Ms. Forbes.

MS. FORBES: Your Honor, do you have a copy of the brief up there?

THE COURT: I do, I have your appellate's brief to Circuit Court. All right, Ms. Forbes.

MS. FORBES: May it please the court.

THE COURT: Yes, ma'am.

MS. FORBES: My name is Ashley Forbes, and I represent the appellants, Yuasa Exide and Arrowpoint Capital Corporation in this appeal concerning their entitlement to reimbursement from the South Carolina Second Injury Fund. I will discuss the case, the plaintiff's case. We are here today because the commission erred in denying

appellant's members from the South Carolina Second Injury Fund, the appellant's medical ailments for reimbursement with respect to Mr. Huggins, permanent physical impairment of heavy metal poisoning, arthritis, diabetes and hypertension.

The commission's decision was erroneous was a matter of law and was also erroneous in light of reliable, probative and substantial in the record. You of course already heard the elements for reimbursement from the fund. Briefly for the purposes of the preserving the record, South Carolina Code 42-9-400 governs reimbursement for the fund. There are 4 elements that must be met. First, the claimant had a permanent physical impairment. Second, that the employer had knowledge of the preexisting impairment. Third, that the preexisting impairment was a hindrance or obstacle to employment or reemployment. And fourth, that the preexisting impairment combined with, or was aggravated by the work place injury to result in substantially greater disability than that which would have resulted from the subsequent injury alone.

It is with respect to this first element that we submit the commission committed an error

of law. And of course that finding was that Mr. Huggins did not have permanent disability impairments or preexisting conditions. In its fourth and fifth findings of fact, the commission found that heavy metal exposure was one cumulative injury. And there were no conditions that preexisted exposure. So if heavy metal exposure occurred on Huggins first day of employment in the plant, under the commission's reasoning, Mr. Huggins would had to have a condition that preceded his first ever date of work at the Sumter Battery plant in 1981. But this is completely contrary to South Carolina law.

And in fact, this is not even an argument that the fund made before the commissioner. The commissioner issued a decision that was contrary to South Carolina law without either party argued the position that the preexisting condition had to preexist Huggins' the employment with plant.

In worker's compensation cases as you've heard, the date of accident controlled. An accident in occupational disease cases occurs when an employee becomes disabled. Or with reasonable diligence could discover that his condition is compensable. The accident may also occur on the

date of defendant diagnosis. We cited several cases in our brief that concern the date of accident in occupational disease cases.

THE COURT: And all this is in your brief what you are telling me.

MS. FORBES: Yes, Your Honor. For the most part. In this case, date of injury was May 1st 2001. This was Huggins' last day of employment with Yuasa Exide. And it's also important to know, Your Honor, just for the purposes of all these cases, that the dates of accident were actually chosen by the Steinberg law firm for the claimant's date of accident in the underlying case. So these are not dates that we just pulled out of thin air.

Huggins date of definite date of diagnosis may actually have been much later. We have a 2008 health evaluation. But for the purposes of today, the date of disability can be no earlier than May 1st 2001. Huggins testified in his deposition that he suffered from hypertension, diabetes and joint pain or arthritis during his time in the plant. He also suffered from preexisting heavy metal poisoning while he worked at the Yuasa plant. And all of these impairments predate

Mr. Huggins' date of accident on May 1st 2001. And qualify as preexisting conditions.

By deciding that Mr. Huggins had no conditions that preexisted the exposure, the commission's decision was contrary to South Carolina authority that allows for reimbursement where the first and subsequent injuries occur in the same employment. You've heard these cases cited already today. But first in the State Worker's Compensation Fund vs. The South Carolina Second Injury Fund, this is what we've been referring to as the Hunt case, of course.

The Supreme Court granted reimbursement to a carrier. In a case where a firefighter was diagnosed with coronary artery disease; was retained by his employer, and was rendered totally disabled because of his worsening heart disease and arteriosclerosis. I believe that the fund earlier today discussed the fact that this was a firefighter and there was a presumption in the statute for firefighters. That is actually, the argument there, is whether it arose out of or in the course of employment. It does not have actually have to do with the reimbursement.

THE COURT: In the interest of time, do

you have anything different that you're going tell me that is not in your brief?

MS. FORBES: Well, Your Honor, I mean I certainly have argued everything in my brief.

THE COURT: Okay. I will take your brief as your complete argument.

MS. FORBES: All right. Sounds good.

THE COURT: That saves you some time. Yes, ma'am.

MS. EDWARDS: I will be even more brief, Your Honor. Your Honor, I believe that everything that I have said in my entire briefs, applies to a particular case. Yes. Everything that I have said in my prior argument, and have submitted in my brief, applies to this case. And so I would specifically request that that be included in the record on appeal.

THE COURT: Thank you. And now we have got Mary McConico.

MR. DUNBAR: Yes, sir.

THE COURT: Yes, sir.

MR. DUNBAR: May it please the court, Your Honor. I represent Yuasa Exide and Arrowpoint Capital Corporation which respectfully denied the claim for reimbursement from the South Carolina

Injury Second Injury Fund. We appealed the full commission's decision on the basis it constitutes a gross error of law as well as, the fact that their findings of fact are not supported not by substantial evidence, but not supported by any evidence whatsoever. And hence, likewise constitutes an error of law.

Your Honor, it's our position that Ms. McConico worked there for 25 plus years, and during that time she suffered with a myriad of medical maladies. She was ultimately deemed disabled from returning to work, because she suffered a disabling stroke when she worked there in July of 1999. What is interesting about this case, just a year prior in 1998, February of 1998, she had stroke like symptoms. And actually returned to work and had another accident after being exposed to lead. So we take the position that this case clearly stands for the proposition, or our position that Second Injury Fund should have reimbursed the carrier in this particular case.

Now with respect to Ms. McConico, she suffered a disabling stroke. She never returned to work after 1999. This case was settled. We

are only seeking indemnity benefits, not medical. In any of these cases, we're not seeking any medical reimbursement whatsoever. But, the bottom line in this particular case, after she had a disabling stroke, she was diagnosed with a myriad of other maladies for which we do not seek any medical reimbursement or indemnity reimbursement whatsoever. That's not even part of the claim. However, I did submit a disk to the fund containing those post employment records; that disks were submitted on or about June 30th. The fund said they couldn't read certain portions of it. Three days. They didn't notify me of that until September. So the disk was delivered in June. In September they notified me. Three days later, and we rectified that problem.

The commission found that because the disk was not submitted by on or before June 30,th and because they couldn't read it, that the claim should be barred. We take the position that this is totally contrary to the law. The reason why we don't even need the post employment records to prove our entitlement to reimbursement in this particular case. I have briefed the issue, because it's basically a technical error. It

didn't show any prejudice. And they did not even put up any evidence to show that they needed these records.

Well let me tell you why these records are not even relevant. In the post employment records it talks about her diabetes, which again is part of this case. It talks about being treated for bronchitis in 2001 and 2004. A lot of them are chemistry labs from lab corps, with a lot of chemistry analysis. Venus Doppler studies. Also treatment for diabetes, mammograms, right heel pain, right knee pain. This is what those records contain. So we take the position that those records certainly didn't assist us in proving our case for reimbursement.

What does assist us is the fact that blood lead levels were extremely high. Five times normal range when she worked there from 1975 until 1985. She should have been placed in a medical removal program when she worked for the Exide company. A lot of times she was not. But she was placed in the medical removal program at least three times. But her her blood lead levels constantly remained 40 microgram per desilter above.

This is a very technical issue, and that's why the funds argument fails, because they don't have any scientific or medical evidence to refute OSHA's regulations, our expert testimony or the industry regulations. They have not submitted anything to that nature. But her blood lead levels aggravated the hypertension. Everyone agrees that lead exposure causes or aggravates hypertension. Her blood pressure readings were as follows: 190 over 110. 204 over 118. 224 over 120. 204 over 118, just to give you a sampling. She continued to work.

Now when Yuasa Exide purchased this plant in 1991, they knew this lady had a long history of hypertension, which is categorized as cardiovascular problems. She also had a minor stroke in 1998, which is the cerebral accidents, what the fund -- how it's labeled in the statute. She had a cerebral accident in 1998. Yet we retained her in employment. We retained her in employment knowing her history from 1974 to 1991. Kept her in employment even after she had the mild stroke in 1998.

The purpose of the fund is simply that, to retain people in their employment. When I took

her deposition, the best job she ever had, she did not want to leave that job. We kept her in employment. And that is the purpose of the South Carolina Second Injury Fund. That's why they -- this client, my client, is entitled to reimbursement. Your Honor, quickly, with respect to the purchase of the plant, I would like to and you a case, Yuasa Exide vs. NLRB. In which the 4th Circuit actually cites that Yuasa purchased the industrial battery plant in 1991.

So hence, a new owner who had knowledge of her preexisting maladies and conditions, because all of the medical records, all of the medical files from ESB, Exide, transferred over to Yuasa exide. That's why we were able to go back from the beginning of time, and tell you a little bit about her medical history.

Your Honor, with respect to the disk, we still -- we maintain that we have met the elements. We rely upon Hunt vs. State Forestry Commission, because we retained her in employment. And even if we needed to know that she had these preexisting conditions at the time she became employed, we certainly knew it. So even if you brought that legal argument in the case of Mary

McConico, we knew she had some problems in 1991 when we purchased the plant. Hindsight, probably from a business decision, It would have more prudent to dismiss her and fire her. But that's contrary to the South Carolina Second Injury Fund statute.

So, Your Honor, we take the position, that clearly this case is subject to reimbursement, despite the disk issue, because they have proven.

THE COURT: Thank you. Yes, ma'am.

MS. EDWARDS: Very briefly. Your Honor, this is only issue that is substantially different from the other cases. And that's the issue of 42-7-320 (b) 2. I believe that's on page 14 of the record. Because that page 14, has two 14's on it. So I am assuming that that page would be consistent. That statute was created in 2007. And so the statute says it's 42-7-320 (b) 2. And in order of self insurer or insurance carrier, must submit all required information for consideration of its second -- a claim to the Second Injury Fund by June 30, 2011. Failure to submit that information so that the claim is accepted, comprised or denied, shall bar recovery.

Now in finding of fact No. 3, on page 13,

I believe that's the same page, Your Honor. The page before. The commission determined that this disk that the carrier submitted, which was submitted on June 30th 2011, which they technically met the date, but nothing was on the disk. And so the commission found that because nothing was on the disk, they did not submit the material by June 30th 2011. That's not a Second Injury Fund requirement, Your Honor. That's a statutory requirement. It's mandatory. It's not discretionary. They can assert no argument for this court disregarding that statutory requirement. And so I would rely on, and let me just say, I am making an appearance on behalf of Mr. Timothy Killen, esquire, who is not here. But I still wanted to argue his case for him. I don't know if he submitted a brief to Your Honor. We would be happy to do that too, Your Honor. But I didn't want have this case reset, because they substantially involved the same issues. And so they're okay. But everything else that will be in the brief that you get, and that is in the record, is reflected. And I will defer to those, and also just point out the substantially different thing with this case, as opposed to the three others

that I had. Because of the different statute that required them.

And so, the carrier doesn't even get to the other things. I believe that that in particular throws the case out. And we don't even really have to address the other issues. And so I will just complete my argument that way, Your Honor. If you have any questions, I'd be happy to answer those.

THE COURT: Thank you.

MR. DUNBAR: Your Honor, if I may respond. With respect to this issue, this is all acquired information. The fund put up no testimony that the post medical records were required. What was required was the 25 years of medical literature and medical records that we put into evidence. What was required was the settlement agreement and release the fund requires. What was required was those medical questionnaires completed by Dr. Shipman, by Dr. Rigor, by Dr. Baker and Vizelex. Those were all submitted. And what does those -- what do those records suggest? That she had preexisting condition. Cardiac problems, coronary artery disease, that were all preexisting and aggravated by lead exposure. Those post

employment records do none of that. Are they required? We don't know, because the fund put up no testimony to say, we needed those records. We want to accept the case. They don't need those records. They need the records from 1999 back, with respect to her preexisting conditions, because that's what the court, that's what the statute says the court must be concerned with; whether there was a preexisting permanent impairment.

We have proven that ad nauseam with respect to the medical records that were on Yuasa Exide's premises.

THE COURT: Thank you. I look forward to getting your revised briefs.

MS. EDWARDS: Yes.

THE COURT: And then I will give you my ruling.

MS. EDWARDS: Thank you very much.

MR. VERNON: Thank Your Honor.

--End of Requested Transcript of Record--

C-E-R-T-I-F-I-C-A-T-E

I, Margaret T. Sullivan, Court Reporter, for the Third Judicial Circuit of the State of South Carolina, do hereby Certify that the foregoing is a true, accurate and complete Transcript of Record of the proceedings had and evidence introduced on June 3, 2014, in Common Pleas Nonjury Court, Sumter County, Sumter, South Carolina.

I do further that I am neither kin, counsel nor interest to any party hereto.

1-9-15
DATE

Margaret T. Sullivan
COURT REPORTER
My Commission expires: 9/7/2021

COPY

SOUTH CAROLINA WORKERS' COMPENSATION HEARING

FULL COMMISSION APPELLATE HEARING

ARROW POINT CAPITAL INSURANCE,

Appellant

-VS-

SC SECOND INJURY FUND,

Respondent

In Re of C.L. WILLIAMS

-VS-

YUASA-EXIDE, INC.

WCC FILE NO.
9930399

SLP

OCT 8 2013

3180.167

SCANNED

Pursuant to Notice of the Appellate Hearing taken on the 16th day of September 2013, heard before the South Carolina Workers' Compensation Commission Full Commission Appellate Panel including; Honorable Susan S. Barden, Gene McCaskill, Melody James, Aisha Taylor, Avery Wilkerson, and T. Scott Beck, Chair, and commencing at the hour of 1:32 p.m. in Columbia, South Carolina.

C. Tatum Court Reporting
P.O. Box 248
Edgefield, SC 29824
706.207.5483
ctatumcr@gmail.com

APPEARANCES

Stephanie Lamb Pugh, Esq.
Turner Padget Graham & Laney, PA
PO Box 1509
Greenville, SC 29602
843.552.4613
jmcdade@turnerpadget.com
REPRESENTING THE APPELLANT

Latonya D. Edwards, Esq.
Dilligard Edwards, LLC
3790 Fernandina Road, Suite 103
Columbia, SC 29210
803.750.2214
latonya@dilligardedwards.com
REPRESENTING THE RESPONDENTS

INDEX

PROCEEDINGS 4

APPELLANT ARGUMENT BY MS. PUGH 4

RESPONDENT ARGUMENT BY MS. EDWARDS 14

APPELLANT REPLY BY MS. PUGH 20

REPORTER CERTIFICATE 22

OBJECTIONS:

(None)

EXHIBITS

NUMBER	DESCRIPTION	PAGE
	(None Offered)	

LEGEND OF THE TRANSCRIPT

dashes [--]	intentional or purposeful interruption
[ph]	denotes phonetically written
[sic]	written as said

****

This transcript may contain quoted material. Such material is reproduced as read or quoted by the speaker.

PROCEEDINGS

COURT REPORTER: Today is September 16, 2013. This is South Carolina Worker's Compensation Case Number 9930399. This is the case of *Arrow Point Capital Insurance versus South Carolina Second Injury Fund*, in Re of *C.L. Williams versus Yuasa-Exide, Inc.*

The Appellant is the Defendant, represented by Stephanie Pugh. The Respondent is represented by Latonya Edwards.

Each side is allowed ten minutes for oral argument and the Appellant three minutes in reply. You are requested to argue the grounds of exception and stay within the record.

COMMISSIONER BECK: Ms. Pugh.

APPELLANT ARGUMENT

MS. PUGH: May it please the Court. My name is Stephanie Lamb Pugh and I represent Yuasa-Exide and their carrier, Arrow Point, in this Workers' Compensation Reimbursement Case. It's our position that we're entitled to reimbursement pursuant to the preponderance of the evidence that's in the record on the C.L. Williams case, pursuant to 42-9-400.

Just as a background, all of the cases you'll

hear today is with the subsequent injury of lead poisoning or heavy metal poisoning. As you know, the purpose of the Second Injury Fund was to encourage employers to hire or retain disabled or injured workers. That is why 42-9-400 requires knowledge of a preexisting condition either at the time of hire or at some point during the retention of that employee.

Now, we're defer to our Global Report that was handed up to you last time -- I believe we handed you some notebooks -- regarding conditions that lead poisoning causes or aggravates. But just to name a few of those, you have cardiac disease, kidney failure, and I'm sure you all know a lot of these, brain damage, gout, arthritis, and those are in the report.

The finding -- the holding in each of these cases today was not something that was argued below, especially in this case, so the first time we saw it was in the order. But in particular, in my case, it was not argued by the Second Injury Fund that preexisting conditions had to preexist the first day these employees ever stepped foot into the Sumter battery plant. So that was the first time we knew this was going to be a holding

or even argued.

The Second Injury Fund took two positions in my case. One, that the claim was barred by the Sunset Provision, or what they call the Sunset Provision, 42-7-320. And two, that my preexisting conditions did not constitute hindrance or obstacles to employment. Neither of those issues are on appeal.

Now, the typical occupational disease injury, it does not -- an occupational disease injury does not even exist until an employee becomes disabled and could discover that his injury is compensable, and that pursuant to the *Hanks versus Blair Mills* case, *Drake versus Raybestos-Manhattan* case, and the *Muir versus C.R. Bard* case. The compensability is defined by definitive diagnosis. So all of this matters because now we suddenly have this date of injury issue, did the preexisting conditions have to preexist the first day they walked into the plant or did they have to preexist the occupational disease injury. And it would be our position that the injury did not exist until they had disability and knew that their claim was compensable.

Now, this argument was put forth by the Second Injury Fund in the South Carolina Supreme Court

case of Lee Earnest Franklin. And in that case -- you'll see it, we handed it up in the book, but it's under one of the tabs on the last page -- the South Carolina Supreme Court in that case said that the argument was deemed abandoned because no authority was cited for the proposition that these preexisting conditions had to preexist before the -- well, had to preexist the first day of exposure to the chemical ever, which is the first day they stepped into the plant. And again, we handed that up. The Supreme Court deemed it abandoned and in this case -- in these cases, in these holdings, we have no authority cited for the proposition, still, that the preexisting conditions had to preexist the first day these people ever walked into the plant, that could be 20 or 30 years ago.

Alternatively, we have to argue now on appeal, that if, in fact, it is the case that preexisting conditions have to preexist the first day people ever walk into their employer's location, that because there is multiple employers in the chain of -- or in their chain of employment at the Sumter battery plant that they would only -- the preexisting conditions would only have to preexist the first day of their employment of their last

employer.

Now, regarding my particular claim, the substantive elements of this claim, C.L. Williams, is that we believe the we're entitled to reimbursement based on the preexisting conditions of cardiac disease, pulmonary disease, heavy metal poisoning, diabetes, and hearing loss.

This gentleman came in when he started working at the Sumter battery plant. He started having problems with hypertension, of course, the hypertension -- as you see this man, the years pass by, his heart condition gets worse, his dad actually dies from heart disease. He ends up having congestive heart failure, cardiomyopathy. He was hospitalized multiple times, he was placed on medication, which the doctor said, specifically, interfered with his ability to work. He ultimately was taken out of work due to his cardiac condition. He applied for disability because of his cardiac condition. So we don't believe, regarding the heart condition, that there's anyway to say that it wasn't a preexisting condition before the date of injury or that it did not constitute a hindrance or obstacle to employment because of his hospitalizations, the fact that he applied for

disability, and was ultimately terminated, actually, because they couldn't accommodate his heart -- the work restrictions related to his heart. Now, even after he leaves you see his heart condition worsening. He gets a defibrillator, so he keeps worsening after his termination.

In a letter from the Second Injury Fund at APA 89 they admitted that they had -- in my case, in particular, we had a preexisting history of hypertension and congestive heart failure, which led to this claimant being diagnosed with diabetes.

Now, regarding the pulmonary disease. You also see a constant deterioration of this man's condition. It starts off with wheezing and coughing, he has abnormal chest x-rays in 1990, shortness of breathe. He was repeatedly diagnosed with bronchitis, even had plural affusion at one point, was diagnosed in the ER. He was hospitalized with a condition called hemoptysis, which I wasn't familiar with, but it's a lung condition where you're spitting up bloody sputum from his throat, had abnormal lung disease, and then ultimately, even after he leaves, he's diagnosed with COPD.

Now, the third preexisting condition would be

heavy metal poisoning. It's our position that the heavy metal poisoning preexisted the Claimant's ultimate date of injury, and is therefore a preexisting condition. The effects of lead or what lead does to your body -- and again, I'll defer to the Global Report, but just to say it briefly -- the effects are cumulative and incremental. Which we know now means that everyday these people walked into this lead plant, whatever condition they had prior, it was aggravated each day they stepped into it. So that the more lead that was in their systems the worse the effects of lead could be. So what would happen is, it comes into your body through inhalation or -- guess you could actually -- well, it comes in through inhalation, but it goes to your bloodstream, stores in your organs, goes into your bones, and will actually sit in your bones and stay latent for decades. So you may not know the impact of the lead until 20, 30 years later.

We believe that the -- well, the drafters of the lead report -- the Global Report, which are leading experts in the field of lead poisoning, agree that these claimants, the last day of their exposure to lead was actually a new injury because

it aggravated the injury they had the day before. We believe that pursuant to the Hunt case -- and I'll cite that, it's *State Workers' Compensation Fund versus Second Injury Fund*, 313.South Carolina.536. In that case the South Carolina Supreme Court allowed reimbursement for an occupation disease of heart disease with a subsequent injury of heart disease.

You see the same thing taking place in the *Springs Industries Case versus South Carolina Second Injury Fund.* In that case the Claimant was exposed to cotton dust and then later developed byssinosis and the cotton dust exposure was deemed the preexisting condition and the byssinosis was the subsequent injury.

In this case this gentleman had lead levels ranging from twenty-one to ninety-three micrograms. The typical average for you and me is probably a three for a blood lead level, so this man's average was about thirty-one times. Now, it can go all the way up to ten, could be deemed average. But for you and me, most people in the US, as we've gotten better with our environmental regulations and stuff it's gone down to three.

No question about this man's exposure. He was

subject to the medical removal program, where they would actually take him out of the lead environment to try to decrease his lead poisoning. It didn't get better. He even wore a respirator and still had problems.

I'll rely on my brief regarding hearing loss and diabetes. But regarding the hindrance or obstacle to employment element we have presumptive hindrances for cardiac disease, heavy metal poisoning, pulmonary disease, and diabetes. But I'd also defer to the fact that he was hospitalized multiple times for congestive heart failure, multiple emergency room trips, applied for disability, taken out of work because of his heart condition, so we believe that speaks to a hindrance or obstacle to employment. We also have the medical questionnaires.

Now, regarding knowledge, there's no question here -- the Sumter battery plant, at different times, had an in-house medical department, like many big manufacturing companies do. So they cited and noted this guy's preexisting conditions fairly well throughout their records.

So we have --

COMMISSIONER McCASKILL: You say, At times,

they didn't have it continuously?

MS. PUGH: Now, in the earlier days -- I'm not sure if they had it in the earlier days, but we know -- the plant eventually laid off most people around 2001 or 2002. But, now, I'm not sure if they had it continuously, but in all of the records that we have -- or in this case, I have medical records substantiating all of these preexisting conditions, for the most part, in the employers own records in-house.

Now, the last element requiring the increase in disability as a result of the combination of effects or aggravation, I would defer to the questionnaires of the lead experts at APA one through three. The Global Report that we handed up to you also talks about the aggravation of latent conditions or even just genetic conditions that you have that I've already talked about and --

(Interruption)

COMMISSIONER BECK: Finish your thought.

MS. PUGH: -- we believe that the questionnaires from the experts and the literature would definitely provide that evidence.

In summary, we believe that the preponderance of the evidence established, we're entitled for

reimbursement for the conditions of heavy metal poisoning, cardiac disease, pulmonary disease, hearing loss, and diabetes. And we believe that the pursuant to the *Burnett versus City of Greenville* case, the fact that there is no evidence -- in fact, there's only one piece of evidence submitted by the Second Injury Fund in this case -- the fact that there is no evidence to contradict our medical evidence we believe would lend credence to our position.

COMMISSIONER BECK: Thank you. Ms. Edwards.

RESPONDENT ARGUMENT

MS. EDWARDS: Good afternoon. I'm Latonya Edwards, I represent the Second Injury Fund. And let me just start by saying occupational exposure cases, as you all know, they're not the same as other cases. They're not were a claimant comes in with a prior back injury or prior arthritis, it's a cumulative exposure, we admit to that. We also admit there's not a lot of case law on it so we might have to make some new ones. And so we totally admit that and that's probably why this case is so perplexing to everybody involved in it, probably not you all, but to everybody else involved in it -- or maybe so. Maybe so, I'm

sorry.

But the reason we think that the decision below was correct are as follows. And I'll be free to answer any questions -- ask me any questions that you would like. One, we assert that the Claimant didn't have any of these conditions before the occupational exposure. And we assert that because occupational exposure is a cumulative exposure. And I would also defer to my Global brief, as well as to some of the deposition testimony of the doctor that clearly indicates that it's a cumulative exposure. There's not an exposure or an injury everyday that the person goes into the plant. It's a very unique type of injury. And especially for Second Injury Fund reimbursement purposes.

But we first assert that the conditions did not exist prior to the lead exposure. And we assert that that's what it means in an occupational disease case to preexist, the conditions have to preexist the exposure. And in this particular case I believe that the Claimant had worked for this particular employer for 25 years. Certainly, I'd think it would be wrong -- I can't think of a more artful word, but I think it would be wrong to

assert that everyday the Claimant walked into the plant it was an injury. It's not. The exposure was cumulative over 25 years. The Claimant didn't have any of these conditions before that occupational exposure.

And there's no evidence that Claimant had high blood pressure, I think some of the -- it's a whole laundry list of conditions, I'm not going to name all of them, but hypertension, high blood pressure, there's no evidence that that preexisted the lead exposure.

Now, I think one of the things that Carrier said that the first time they heard we were going to argue that was, you know, when we said it somewhere where we weren't supposed to say it, I think we were supposed to say it earlier. We've always asserted that Claimant didn't have a preexisting condition in this case. Now, so I would certainly dispute that they didn't have notice that we were going to assert that there was not a preexisting condition.

As it relates to the issues on the lead exposure being the first and subsequent injury, I do believe that that's what Claimant's have asserted -- and let me just say something about

Springs while I'm thinking about it. That was a separate employer. This Claimant worked one place for this employer and then she worked somewhere else. So it was easy to kind of bifurcate, you know, a preexisting condition versus a second or subsequent injury, one employer, two different employers. So this particular case, I think they've tried to assert that there was two different employers, but I think even at Form 30 cites that the Claimant's employment was from 1974 to 1995, or something like that, 25 years. I think that, at least, is evidence of admission that I think they're asserting that this is one employer, even though there's been some issue in other cases about a merger and whether those documents constitute the same employer or separate. I don't think that's the issue in this case. And the issue in the Springs case was two separate employers, not the same. So easy to make the distinction, I think, in that particular case.

As it relates to, again, the first and subsequent injury, we don't believe that that's what the statute envisions as it relates to Second Injury Fund reimbursement. We believe that lead exposure or occupational exposure in this case in

the form of lead or heavy metal exposure is one continuous injury. And like I said, I'll say it again, we don't have a lot of case law on that, I readily admit that. But I think that's the plain meaning of the statute. And I've cited that in my brief, I'm not going to belabor that at all. And I've also referenced it in my Global brief, as well.

As it relates to -- I can't remember, either Burnett -- I reference Carolina's Recycling in my brief as a reason to affirm this decision. In that decision, Carolina's Recycling, the hearing Commissioner -- I'm sorry -- in that case before the Court of Appeals, the Court of Appeals reversed that decision because the appellant panel, according to them, relied upon an evaluation by a non-treating physician who only met with the Claimant on one occasion. And like that case, that case the -- this case the treating physician was like that one in Carolina's Recycling. He was a one shot guy, he filled out a bunch of questionnaires. I understand he might be the leading professional, I think is what Carrier's attorney says, on some of these lead issues, but it doesn't change the fact that he was a non-treating

physician who completed a questionnaire.

And so we certainly believe that Carolina's Recycling would warrant an affirmation of the decision below, because like that case, the Commissioner did not rely on a non-treating physician's one shot questionnaire. And so we believe that that decision would be consistent with that case.

Also I think Burnett was cited, also. I think Burnett also is distinguished from this case because in Burnett, the issues in that case were what I deem medical issues. They referenced or regarded an interpretation of MRIs and CT scans. This one doesn't. This case is of purely statutory argument, doesn't require a doctor to look at MRIs, just based on statutory interpretation. And we believe that the Commission's decision was in line with that statutory interpretation.

And -- I want to make sure I didn't leave anything out. I think that's it unless your Honors have any questions for me.

COMMISSIONER BECK: Questions?

MS. EDWARDS: No? Thank you.

COMMISSIONER BECK: Ms. Pugh, three minutes, please.

APPELLANT REPLY

MS. PUGH: Just to address some of the issues. First of all, we do have case law. We have the Hanks case, and I have a copy of that case here if you would like for me to hand that up. But we have the *Hanks versus Blair Mills* -- well, I'm sorry, we have the Hanks -- the Hunt case, I'm sorry. Warren Hunt, it's a South Carolina Workers' Compensation Fund case. I'm going to cite it for the record, 313.South Carolina.536. In that case there was a preexisting heart condition subsequent injury, same employer, of a heart condition, and there was reimbursement given for that case, and that was considered an occ disease.

We also have the Springs Industries case. And it doesn't matter if it's the same employer or two separate employers, the whole purpose is to look at retention and employment. So you're looking at -- you're trying to encourage employers to hire or retain, not just to hire.

Next, regarding the preexisting conditions. We believe the Fund admitted there was preexisting conditions in the letter that I cited earlier. The statute itself, 42-9-400, doesn't say exposure, it says subsequent injury. So we are not looking at

exposures to determine when the preexisting conditions had to occur, we're looking at the subsequent injury.

Now, the Carolina's Recycling case. That case doesn't really -- I don't read it the way that the Second Injury Fund reads it in this case. In that case, as some of you probably know, the evaluation that the Court held the Commission should not have relied upon was an evaluation that took place only before the subsequent injury so that was the whole issue with that case.

In these cases, that's just not what happened here. We had lead experts go evaluate these Claimants after their subsequent injuries. In the Carolina's Recycling case there was an evaluation from way back, after the first injury, not the second injury. So we believe that's easily distinguishable from the cases that we have before you today.

Nothing further unless you all have any questions.

COMMISSIONER BECK: Thank you very much. That will conclude this proceeding.

*

(Whereupon, the hearing concluded at 1:32 p.m.)

STATE OF SOUTH CAROLINA

CERTIFICATE

Be it known that I, Christine Tatum, a Professional Court Reporter and Notary Public, did hear the appellate matter of ***Arrow Point Capital Insurance -vs- South Carolina Second Injury Fund, in Re of C.L. Williams -vs- Yuasa-Exide, Inc.***, Workers' Compensation File No. 9930399 on Monday, September 16, 2013, in Columbia, South Carolina, before the South Carolina Workers' Compensation Commission Full Commission Appellate Panel including; Honorable Susan S. Barden, Gene McCaskill, Melody James, Aisha Taylor, Avery Wilkerson, and T. Scott Beck, Chair;

That the foregoing pages constitute a true and accurate transcription of the testimony given at that time and place aforesaid to the best of my skill and ability;

I further certify that I am not counsel or kin to any of the parties to this cause of action, nor am I interested in any matter of its outcome.

In Witness whereof, I have hereunto set my hand this 30th Day of September 2013.

Christine Tatum
Notary Public For South Carolina
My Commission Expires December 29, 2021

INDEX for FC 9-16-13 Williams

A
abandoned [2] 7:5, 11
abnormal [2] 9:15, 22
accommodate [1] 9:2
action [1] 22:19
address [1] 20:2
admission [1] 17:12
admit [4] 14:19, 20, 22; 18:4
admitted [2] 9:8; 20:22
affirm [1] 18:11
affirmation [1] 19:3
affusion [1] 9:17
aforesaid [1] 22:16
afternoon [1] 14:13
aggravated [2] 10:10; 11:1
aggravates [1] 5:12
aggravation [2] 13:13, 16
agree [1] 10:24
aisha [2] 1:16; 22:12
allowed [2] 4:11; 11:6
alternatively [1] 7:17
apa [2] 9:7; 13:14
appeal [2] 6:8; 7:17
appeals [2] 18:14, 14
appearances [1] 2:2
appellant [9] 1:4; 2:9; 3:4, 6; 4:8, 12, 16; 18:15; 20:1
appellate [5] 1:2, 12, 15; 22:5, 11
applied [3] 8:19, 25; 12:13
argue [3] 4:13; 7:17; 16:14
argued [3] 5:18, 21; 6:1
argument [8] 3:4, 5; 4:12, 16; 6:24; 7:5; 14:12; 19:15
arrow [4] 1:3; 4:4, 19; 22:5
artful [1] 15:25
arthritis [2] 5:15; 14:18
assert [7] 15:5, 7, 17, 19; 16:1, 20; 17:8
asserted [2] 16:17, 25
asserting [1] 17:13
attorney [1] 18:24
authority [2] 7:6, 13
average [3] 11:18, 19, 21
avery [2] 1:17; 22:12

B
background [1] 4:25
bard [1] 6:15
barden [2] 1:16; 22:11
barred [1] 6:3
battery [4] 5:24; 7:23; 8:9; 12:19
beck [8] 1:17; 4:15; 13:20; 14:11; 19:22, 24; 21:22; 22:13
becomes [1] 6:11
belabor [1] 18:6
below [3] 5:19; 15:3; 19:4
bifurcate [1] 17:4
big [1] 12:21
blair [2] 6:13; 20:6
blood [3] 11:19; 16:7, 9
bloodstream [1] 10:16
bloody [1] 9:21
body [2] 10:5, 13
bones [2] 10:17, 18
book [1] 7:2
box [2] 1:21; 2:5
brain [1] 5:15
breathe [1] 9:16
briefly [1] 10:6
bronchitis [1] 9:17
bunch [1] 18:21
burnett [5] 14:4; 18:10; 19:9, 10, 11
byssinosis [2] 11:13, 14

C
c.l [5] 1:9; 4:6, 23; 8:3; 22:6
c.r [1] 6:15
call [1] 6:4
called [1] 9:19
capital [3] 1:3; 4:5; 22:5
cardiac [6] 5:13; 8:6, 18, 19; 12:9; 14:2
cardiomyopathy [1] 8:14
carolina [15] 1:1, 14, 19; 4:3, 5; 6:25; 7:4; 11:5, 10; 20:8; 22:1, 6, 9, 9, 24
carolina.536 [2] 11:5; 20:10
carolina's [6] 18:10, 12, 20; 19:2; 21:4, 15
carrier [2] 4:19; 16:12
carrier's [1] 18:23
cases [8] 4:25; 5:18; 7:12; 14:16, 17; 17:14; 21:12, 18
causes [1] 5:12
certainly [3] 15:23; 16:19; 19:2
certificate [2] 3:7; 22:2
certify [1] 22:18
chain [2] 7:21, 22
chair [2] 1:17; 22:13
change [1] 18:25
chemical [1] 7:9
chest [1] 9:15
christine [2] 22:3, 23
cite [2] 11:3; 20:9
cited [6] 7:6, 13; 12:21; 18:5; 19:9; 20:23
cites [1] 17:10
city [1] 14:4

claim [4] 6:3, 23; 8:2, 3
claimant [11] 9:11; 11:11; 14:17; 15:6, 22; 16:1, 3, 6, 17; 17:2; 18:18
claimant's [3] 10:2; 16:24; 17:10
claimants [2] 10:24; 21:14
clearly [1] 15:11
columbia [3] 1:18; 2:13; 22:9
combination [1] 13:12
commencing [1] 1:18
commission [7] 1:2, 14, 15; 21:8; 22:10, 10, 25
commissioner [9] 4:15; 12:25; 13:20; 14:11; 18:13; 19:5, 22, 24; 21:22
commission's [1] 19:17
companies [1] 12:21
compensability [1] 6:15
compensable [2] 6:12, 23
compensation [8] 1:1, 14; 4:3, 20; 11:3; 20:9; 22:7, 10
completed [1] 19:1
concluded [1] 21:25
condition [20] 5:6; 8:12, 18, 20, 21, 22; 9:5, 14, 19, 21, 25; 10:4, 9; 11:14; 12:15; 16:18, 21; 17:5; 20:11, 12
conditions [22] 5:11, 22; 6:6, 18; 7:7, 14, 19, 24; 8:5; 12:22; 13:9, 17, 17; 14:1; 15:6, 17, 20; 16:4, 8; 20:21, 23; 21:2
congestive [3] 8:14; 9:10; 12:12
considered [1] 20:14
consistent [1] 19:7
constant [1] 9:13
constitute [4] 6:6; 8:23; 17:16; 22:14
contain [1] 3:18
continuous [1] 18:2
continuously [2] 13:1, 6
contradict [1] 14:8
copd [1] 9:24
copy [1] 20:4
cotton [2] 11:12, 13
coughing [1] 9:15
couldn't [1] 9:2
course [1] 8:10
credence [1] 14:9
ctatumcr@gmail .com [1] 1:24
cumulative [5] 10:7; 14:19; 15:8, 12; 16:3

D
dad [1] 8:12
damage [1] 5:15
dashes [1] 3:14
decades [1] 10:18
december [1] 22:25
decision [7] 15:2; 18:11, 12, 15; 19:4, 7, 17
decrease [1] 12:3
deem [1] 19:12
deemed [4] 7:5, 11; 11:13, 21
defer [5] 5:9; 10:5; 12:11; 13:13; 15:9
defibrillator [1] 9:5
defined [1] 6:16
definitely [1] 13:23
definitive [1] 6:16
denotes [1] 3:15
department [1] 12:20
deposition [1] 15:10
description [1] 3:11
deterioration [1] 9:13
determine [1] 21:1
developed [1] 11:12
diabetes [5] 8:7; 9:11; 12:7, 10; 14:3
diagnosed [4] 9:11, 16, 18, 24
diagnosis [1] 6:16
didn't [7] 12:3; 13:1; 15:6; 16:3, 17, 19; 19:19
dies [1] 8:13
different [3] 12:19; 17:6, 9
dilligard [1] 2:11
disability [5] 6:22; 8:19; 9:1; 12:14; 13:12
disabled [2] 5:4; 6:11
discover [1] 6:12
disease [18] 5:13; 6:9, 10, 21; 8:6, 6, 13; 9:12, 22; 11:7, 7, 8; 12:9, 10; 14:2, 2; 15:20; 20:14
dispute [1] 16:19
distinction [1] 17:19
distinguishable [1] 21:18
distinguished [1] 19:10
documents [1] 17:15
doesn't [6] 18:25; 19:14, 15; 20:16, 24; 21:5
drafters [1] 10:21
drake [1] 6:14
due [1] 8:18
dust [2] 11:12, 13

E
earnest [1] 7:1
easily [1] 21:17
easy [2] 17:4, 19
edgefield [1] 1:22

edwards [8] 2:10, 11; 3:5; 4:10; 14:11, 13, 14; 19:23
effects [4] 10:4, 7, 12; 13:13
element [2] 12:8; 13:11
elements [1] 8:3
emergency [1] 12:13
employee [2] 5:8; 6:11
employees [1] 5:23
employer [9] 8:1; 15:23; 17:2, 3, 6, 13, 16; 20:12, 16
employers [8] 5:4; 7:21; 13:9; 17:7, 9, 18; 20:17, 19
employer's [1] 7:20
employment [8] 6:7; 7:22, 25; 8:24; 12:8, 16; 17:10; 20:18
encourage [2] 5:4; 20:19
ends [1] 8:13
entitled [3] 4:21; 8:4; 13:25
environment [1] 12:2
environmental [1] 11:23
envisions [1] 17:23
especially [2] 5:19; 15:15
esq [2] 2:3, 10
established [1] 13:25
evaluate [1] 21:13
evaluation [4] 18:16; 21:7, 9, 15
eventually [1] 13:4
everybody [2] 14:23, 24
evidence [10] 4:22; 13:23, 25; 14:5, 6, 8, 9; 16:6, 10; 17:12
exception [1] 4:13
exhibits [1] 3:10
exide [1] 4:7
exist [3] 6:11, 22; 15:18
experts [4] 10:23; 13:14, 22; 21:13
expires [1] 22:25
exposed [1] 11:12
exposure [21] 7:8; 10:25; 11:13, 25; 14:15, 19; 15:7, 8, 9, 12, 13, 18, 21; 16:2, 5, 11, 23; 17:25, 25; 18:1; 20:24
exposures [1] 21:1

F
failure [4] 5:14; 8:14; 9:10; 12:12
familiar [1] 9:20
fernandina [1] 2:12
few [1] 5:13
field [1] 10:23
file [2] 1:4; 22:8
filled [1] 18:21
finding [1] 5:17
finish [1] 13:20
follows [1] 15:3
foot [1] 5:23
foregoing [1] 22:14
form [2] 17:9; 18:1
forth [1] 6:24
franklin [1] 7:1
free [1] 15:3
full [3] 1:2, 14; 22:10
fund [18] 1:7; 4:6; 5:3, 22; 6:2, 25; 9:7; 11:4, 4, 11; 14:7, 14; 15:15; 17:24; 20:9, 22; 21:6; 22:6
further [2] 21:20; 22:18

G
gene [2] 1:16; 22:12
genetic [1] 13:17
gentleman [2] 8:8; 11:16
gets [2] 8:12; 9:5
given [2] 20:13; 22:15
global [6] 5:9; 10:6, 22; 13:15; 15:9; 18:7
goes [3] 10:16, 17; 15:13
gone [1] 11:24
gotten [1] 11:22
gout [1] 5:15
graham [1] 2:4
greenville [2] 2:6; 14:5
grounds [1] 4:13
guess [1] 10:14
guy [1] 18:21
guy's [1] 12:22

H
hand [2] 20:5; 22:22
handed [5] 5:10, 10; 7:2, 10; 13:15
hanks [4] 6:13; 20:4, 6, 7
having [2] 8:9, 14
hearing [8] 1:1, 2, 12; 8:7; 12:6; 14:3; 18:12; 21:25
heart [14] 8:12, 13, 14, 21; 9:3, 4, 4, 10; 11:7, 8; 12:12, 14; 20:11, 12
heavy [7] 5:2; 8:6; 10:1, 2; 12:9; 14:1; 18:1
held [1] 21:8
hemoptysis [1] 9:19
hereunto [1] 22:21
he's [1] 9:23
high [2] 16:6, 9
hindrance [4] 6:6; 8:23; 12:7, 15
hindrances [1] 12:9
hire [4] 5:4, 7; 20:19, 20
history [1] 9:9
holding [2] 5:17, 25
holdings [1] 7:12
honorable [2] 1:15; 22:11
honors [1] 19:20

hospitalizations [1] 8:25
hospitalized [3] 8:15; 9:19; 12:11
hour [1] 1:18
hunt [3] 11:2; 20:7, 8
hypertension [4] 8:10, 11; 9:10; 16:9

I
i'd [2] 12:11; 15:23
impact [1] 10:19
inc [3] 1:11; 4:7; 22:7
including [2] 1:15; 22:11
increase [1] 13:11
incremental [1] 10:7
index [1] 3:2
indicates [1] 15:11
industries [2] 11:10; 20:15
inhalation [2] 10:14, 15
in-house [2] 12:20; 13:10
injured [1] 5:5
injuries [1] 21:14
injury [42] 1:7; 4:6; 5:1, 3, 21; 6:2, 9, 10, 12, 18, 21, 22, 25; 8:23; 9:7; 10:3, 25; 11:1, 4, 8, 11, 15; 14:7, 14, 18; 15:13, 14, 15; 16:2, 23; 17:6, 22, 24; 18:2; 20:12, 25; 21:3, 6, 10, 16, 17; 22:6
insurance [3] 1:3; 4:5; 22:5
intentional [1] 3:14
interested [1] 22:20
interfered [1] 8:17
interpretation [3] 19:13, 16, 18
interruption [2] 3:14; 13:19
involved [2] 14:23, 25
issue [5] 6:18; 17:14, 17, 17; 21:11
issues [6] 6:7; 16:22; 18:24; 19:11, 12; 20:2
it's [13] 4:20; 7:3; 9:20; 10:1; 11:3, 24; 14:18; 15:12, 14; 16:2, 7; 20:8, 16
its [1] 22:20

J
james [2] 1:16; 22:12
jmcdade@turnerpadget.com [1] 2:8

K
keeps [1] 9:6
kidney [1] 5:14
kin [1] 22:19
knew [2] 5:25; 6:23
knowledge [2] 5:6; 12:18
known [1] 22:3

L
laid [1] 13:4
lamb [2] 2:3; 4:18
laney [1] 2:4
latent [2] 10:18; 13:16
later [2] 10:20; 11:12
latonya [3] 2:10; 4:10; 14:13
latonya@dilligardedwards.com [1] 2:15
laundry [1] 16:8
law [3] 14:20; 18:3; 20:3
lead [23] 5:1, 12; 10:4, 5, 9, 11, 12, 19, 22, 23, 25; 11:16, 19; 12:2, 3; 13:14; 15:18; 16:10, 22; 17:24; 18:1, 24; 21:13
leading [2] 10:23; 18:23
least [1] 17:12
leave [1] 19:19
leaves [2] 9:4, 23
led [1] 9:11
lee [1] 7:1
legend [1] 3:13
lend [1] 14:9
letter [2] 9:7; 20:23
level [1] 11:19
levels [1] 11:16
line [1] 19:17
list [1] 16:8
literature [1] 13:22
llc [1] 2:11
location [1] 7:20
looking [3] 20:18, 25; 21:2
loss [3] 8:7; 12:6; 14:3
lot [3] 5:14; 14:20; 18:3
lung [2] 9:20, 22

M
man [1] 8:11
man's [3] 9:13; 11:19, 25
manufacturing [1] 12:21
material [2] 3:18, 19
matter [3] 20:16; 22:5, 20
matters [1] 6:17
mccaskill [3] 1:16; 12:25; 22:12
meaning [1] 18:5
means [2] 10:8; 15:19
medical [6] 12:1, 17, 20; 13:7; 14:9; 19:12
medication [1] 8:16
melody [2] 1:16; 22:12
merger [1] 17:15
met [1] 18:17
metal [7] 5:2; 8:6; 10:1, 2; 12:9; 14:1; 18:1
micrograms [1] 11:17
mills [2] 6:13; 20:6
minutes [3] 4:11, 12; 19:24

monday [1] 22:8
most [3] 11:22; 13:4, 9
mris [2] 19:13, 15
muir [1] 6:15
multiple [4] 7:21; 8:15; 12:12, 13

N
neither [1] 6:7
new [2] 10:25; 14:21
ninety-three [1] 11:17
none [2] 3:9, 12
non-treating [3] 18:17, 25; 19:5
nor [1] 22:19
notary [2] 22:4, 24
notebooks [1] 5:11
noted [1] 12:22
notice [2] 1:12; 16:20
number [2] 3:11; 4:4

O
objections [1] 3:8
obstacle [3] 8:24; 12:8, 16
obstacles [1] 6:7
occ [1] 20:14
occasion [1] 18:18
occupation [1] 11:7
occupational [9] 6:9, 10, 20; 14:15; 15:7, 8, 19; 16:5; 17:25
occur [1] 21:2
offered [1] 3:12
ones [1] 14:21
oral [1] 4:11
order [1] 5:20
organs [1] 10:16
our [7] 4:20; 5:9; 6:21; 10:1; 11:23; 14:9, 10
outcome [1] 22:20
own [1] 13:9

P
p.m [2] 1:18; 21:25
p.o [1] 1:21
padget [1] 2:4
page [22] 2:1; 3:1, 11; 4:1; 5:1; 6:1; 7:1, 3; 8:1; 9:1; 10:1; 11:1; 12:1; 13:1; 14:1; 15:1; 16:1; 17:1; 18:1; 19:1; 20:1; 21:1
pages [1] 22:14
panel [3] 1:15; 18:15; 22:11
part [1] 13:9
particular [7] 5:20; 8:2; 9:9; 15:21, 23; 17:7, 20
parties [1] 22:19
pass [1] 8:11
perplexing [1] 14:23
person [1] 15:13
phonetically [1] 3:15
physician [3] 18:17, 19; 19:1
physician's [1] 19:6
piece [1] 14:6
place [4] 11:9; 17:2; 21:9; 22:16
placed [1] 8:15
plain [1] 18:4
plant [11] 5:24; 6:20; 7:10, 15, 23; 8:9; 10:9; 12:19; 13:4; 15:14; 16:2
plural [1] 9:17
poisoning [10] 5:2, 2, 12; 8:7; 10:1, 2, 23; 12:3, 10; 14:2
position [4] 4:20; 6:21; 10:1; 14:10
positions [1] 6:2
preexist [10] 5:22; 6:19, 20; 7:7, 8, 14, 19, 24; 15:20, 21
preexisted [2] 10:2; 16:10
preexisting [23] 5:6, 22; 6:5, 18; 7:7, 14, 18, 24; 8:5, 22; 9:9, 25; 10:4; 11:14; 12:22; 13:8; 16:18, 21; 17:5; 20:11, 21, 22; 21:1
preponderance [2] 4:22; 13:24
pressure [2] 16:7, 9
presumptive [1] 12:8
problems [2] 8:10; 12:5
proceeding [1] 21:23
proceedings [1] 3:3
professional [2] 18:23; 22:4
program [1] 12:1
proposition [2] 7:6, 13
provide [1] 13:23
provision [2] 6:4, 5
public [2] 22:4, 24
pugh [11] 2:3; 3:4, 6; 4:9, 15, 17, 18; 13:2, 21; 19:24; 20:2
pulmonary [4] 8:6; 9:12; 12:10; 14:2
purely [1] 19:14
purpose [2] 5:3; 20:17
purposeful [1] 3:14
purposes [1] 15:16
pursuant [6] 1:12; 4:21, 23; 6:13; 11:2; 14:4

Q
question [2] 11:25; 12:18
questionnaire [2] 19:1, 6
questionnaires [4] 12:17; 13:14, 22; 18:22
questions [5] 15:4, 4; 19:21, 22; 21:21
quoted [2] 3:18, 19

R
ranging [1] 11:17
raybestos-manhattan [1] 6:14
read [2] 3:19; 21:5
readily [1] 18:4
reads [1] 21:6
reason [2] 15:2; 18:11
recycling [6] 18:10, 12, 20; 19:3; 21:4, 15
reference [1] 18:10
referenced [2] 18:7; 19:12
regarded [1] 19:13
regarding [8] 5:11; 8:2, 20; 9:12; 12:6, 7, 18; 20:21
regulations [1] 11:23
reimbursement [8] 4:20, 21; 8:5; 11:6; 14:1; 15:15; 17:24; 20:13
related [1] 9:3
relates [4] 16:22; 17:21, 23; 18:9
relied [2] 18:16; 21:9
rely [2] 12:6; 19:5
removal [1] 12:1
repeatedly [1] 9:16
reply [3] 3:6; 4:12; 20:1
report [6] 5:9, 16; 10:6, 22, 22; 13:15
reporter [3] 3:7; 4:2; 22:4
reporting [1] 1:20
represent [2] 4:18; 14:14
represented [2] 4:8, 9
representing [2] 2:9, 16
reproduced [1] 3:19
requested [1] 4:13
require [1] 19:15
requires [1] 5:5
requiring [1] 13:11
respirator [1] 12:4
respondent [4] 1:8; 3:5; 4:9; 14:12
respondents [1] 2:16
restrictions [1] 9:3
result [1] 13:12
retain [2] 5:4; 20:20
retention [2] 5:7; 20:18
reversed [1] 18:14
road [1] 2:12
room [1] 12:13

S
scans [1] 19:13
scott [2] 1:17; 22:13
second [17] 1:7; 4:5; 5:3, 21; 6:2, 24; 9:7; 11:4, 11; 14:7, 14; 15:15; 17:5, 23; 21:6, 17; 22:6
separate [4] 17:2, 16, 18; 20:17
september [4] 1:13; 4:2; 22:8, 22
set [1] 22:21
shortness [1] 9:16
shot [2] 18:21; 19:6
sic [1] 3:16
side [1] 4:11
skill [1] 22:16
somewhere [2] 16:15; 17:3
sorry [4] 15:1; 18:13; 20:6, 7
south [15] 1:1, 14, 18; 4:3, 5; 6:25; 7:4; 11:5, 10; 20:8; 22:1, 6, 9, 9, 24
speaker [1] 3:19
speaks [1] 12:15
specifically [1] 8:16
spitting [1] 9:21
springs [4] 11:10; 17:1, 18; 20:15
sputum [1] 9:21
starts [1] 9:14
state [2] 11:3; 22:1
statute [3] 17:23; 18:5; 20:24
statutory [3] 19:14, 16, 18
stay [2] 4:14; 10:18
stephanie [3] 2:3; 4:9, 18
stepped [3] 5:23; 7:10; 10:10
stores [1] 10:16
stuff [1] 11:23
subject [1] 12:1
submitted [1] 14:7
subsequent [11] 5:1; 11:8, 15; 16:23; 17:6, 22; 20:11, 25; 21:3, 10, 14
substantiating [1] 13:8
substantive [1] 8:3
such [1] 3:18
suddenly [1] 6:17
suite [1] 2:12
summary [1] 13:24
sumter [4] 5:24; 7:22; 8:9; 12:19
sunset [2] 6:4, 4
supposed [2] 16:15, 16
supreme [4] 6:25; 7:4, 11; 11:6
susan [2] 1:15; 22:11
systems [1] 10:12

T
tabs [1] 7:3
talked [1] 13:18
tatum [3] 1:20; 22:3, 23
taylor [2] 1:16; 22:12

ten [2] 4:11; 11:21
terminated [1] 9:1
termination [1] 9:6
that's [10] 4:22; 14:22; 15:19; 16:24; 17:17, 22; 18:4; 19:20; 21:12, 17
therefore [1] 10:3
there's [8] 8:21; 12:18; 14:6, 20; 15:12; 16:6, 10; 17:14
they're [3] 14:16, 17; 17:13
they've [1] 17:8
thinking [1] 17:1
third [1] 9:25
thirty-one [1] 11:20
throat [1] 9:22
throughout [1] 12:23
times [5] 8:15; 11:20; 12:12, 20, 25
today [4] 4:2; 5:1, 18; 21:19
totally [1] 14:22
transcript [2] 3:13, 18
transcription [1] 22:15
treating [1] 18:19
tried [1] 17:8
trips [1] 12:13
true [1] 22:14
turner [1] 2:4
twenty-one [1] 11:17
type [1] 15:14
typical [2] 6:9; 11:18

U
ultimate [1] 10:3
ultimately [3] 8:17; 9:1, 23
under [1] 7:3
unique [1] 15:14
upon [2] 18:16; 21:9

V
versus [10] 4:5, 6; 6:13, 14, 15; 11:4, 10; 14:4; 17:5; 20:6

W
walk [1] 7:20
walked [4] 6:19; 7:15; 10:8; 16:1
warrant [1] 19:3
warren [1] 20:8
wasn't [2] 8:22; 9:20
wcc [1] 1:4
weren't [1] 16:15
we've [2] 11:22; 16:16
wheezing [1] 9:14
whereof [1] 22:21
whole [3] 16:7; 20:17; 21:10
wilkerson [2] 1:17; 22:13
williams [5] 1:9; 4:6, 23; 8:3; 22:7
witness [1] 22:21
word [1] 15:25
wore [1] 12:4
work [4] 8:17, 18; 9:3; 12:14
worked [3] 15:22; 17:2, 3
worker's [1] 4:3
workers [8] 1:1, 14; 4:19; 5:5; 11:3; 20:8; 22:7, 10
working [1] 8:8
worse [2] 8:12; 10:12
worsening [2] 9:5, 6
written [2] 3:15, 16
wrong [2] 15:24, 25

X
x-rays [1] 9:15

Y
you'll [2] 4:25; 7:2
yuasa [1] 4:6
yuasa-exide [3] 1:11; 4:18; 22:7

BEFORE THE
SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION
WCC FILE NO. 9930399

ARROWOOD INDEMNITY COMPANY,	HEARING BEFORE COMMISSIONER ANDREA C. ROCHE
v.	**TRANSCRIPT**
THE SOUTH CAROLINA SECOND INJURY FUND,	
IN RE: C. L. WILLIAMS, CLAIMANT,	
v.	
YUASA BATTERY, EMPLOYER,	

THE WORKERS' COMPENSATION HEARING, TAKEN BEFORE CORA ELLIS BRUTON, A NOTARY PUBLIC IN AND FOR THE STATE OF SOUTH CAROLINA, COMMENCING AT THE HOUR OF 12:50 PM., MONDAY, AUGUST 13, 2012, SUMTER CITY HALL/OPERA HOUSE, 21 NORTH MAIN STREET, SUMTER, SOUTH CAROLINA 29151.

CORA ELLIS BRUTON
COURT REPORTER
131 BROWNING COURT
LEXINGTON, SOUTH CAROLINA 29073
803-397-0189

APPEARANCES

FOR THE CARRIER, ARROWOOD INDEMNITY COMPANY
VERNON F. DUNBAR, ESQUIRE
TURNER, PADGET, GRAHAM & LANEY, P.A.
POST OFFICE BOX 1509
GREENVILLE, SOUTH CAROLINA 29602
864-552-4600

FOR THE SOUTH CAROLINA SECOND INJURY FUND
LATONYA D. EDWARDS, ESQUIRE
DILLIGARD EDWARDS, LLC
3790 FERNANDINA ROAD, SUITE 103
COLUMBIA, SOUTH CAROLINA 29210
803-750-2214

REPORTED BY
CORA ELLIS BRUTON
COURT REPORTER
131 BROWNING COURT
LEXINGTON, SOUTH CAROLINA 29073
803-397-0189

INDEX

BY MR. DUNBAR 3
BY MS. EDWARDS. 7
BY MR. DUNBAR 10
BY MS. EDWARDS. 13
BY MR. DUNBAR 13

CERTIFICATE 15

EXHIBITS
(No Exhibits Proffered)

THE COURT: Today's date is August the 13th, 2012. This is WCC File Number 9930399. This is the case of Arrowood Indemnity Company versus The South Carolina Second Injury Fund; In Re: C. L. Williams versus Yuasa Battery.

We're here today to determine the issues as set forth in the Forms 54 and 55.

Are there any objections to APAs, jurisdiction, venue or any other items?

MS. EDWARDS: No, Your Honor.

MR. DUNBAR: None from the Defendants, Your Honor.

THE COURT: Without objection the Commission's File becomes a part of the record with the exception of self-serving declarations and unstipulated medical reports.

I failed to mention that Vernon F. Dunbar is here on behalf of the Carrier and Latonya Edwards is here on behalf of The Second Injury Fund.

Mr. Dunbar, I'll hear from you.

BY MR. DUNBAR: Your Honor, I won't belabor going through the whole history --

THE COURT: Thank you.

MR. DUNBAR: -- of Yuasa Exide, as I did in the previous case, but just for the record I did in the

case of Mary McConico. I'll rely upon those statements in the case of C. L. Williams as well. Your Honor, we take the position that Mr. Williams had preexisting conditions of cardio vascular disease, hypertension, heavy metal poisoning. He also had pulmonary problems and the other is diabetes. He was placed in medical removal program it appears on at least five occasions; meaning that his blood lead levels had reached a certain level that he had to be taken out of the work environment and I have that as pages 15 --

THE COURT: Mr. Dunbar, I'm going to stop you. I have cardio vascular disease, hypertension, diabetes, heavy metal poisoning, which I assume came from this job, --

MR. DUNBAR: Yes.

THE COURT: -- and what was the other one? You mentioned --

MR. DUNBAR: Hypertension?

THE COURT: I've got that one.

MR. DUNBAR: I did not mention neurological --

THE COURT: Pulmonary.

MR. DUNBAR: Pulmonary. Had pulmonary problems.

THE COURT: Those are preexisting this job?

MR. DUNBAR: That's correct.

THE COURT: Okay.

MR. DUNBAR: Or if it did not it certainly combined with the preexisting conditions which rendered him permanently and totally disabled. As a matter of fact he was deemed permanently and totally disabled in 1999 due to severe cardio vascular problems and could no longer work. Tried to work and his doctor actually commended him for continuing to work despite those problems. APA page 36, Your Honor, he completed a health history questionnaire that was required; Yuasa Exide has people complete one of those periodically, sometimes annually, sometimes a little less frequent. But in 1990 he had an abnormal chest x-ray. 1990 he also had an abnormal kidney x-ray and he also had severe breathing problems in 1990 his breathing tests came back abnormal. These tests apparently were given and Mr. C. L. Williams checked off on these preexisting conditions as well.

With respect to the hypertension and cardio vascular problems, Your Honor, APA page 7 shows a hypertension problem which our doctors say -- these are all Yuasa Exide's physician's notes, stating that he has a long history of high blood pressure and also notes that he's had a long history of elevated blood lead levels; and that's APA page 7. APA pages 29

through 31 and other blood hypertension or cardio vascular disease references are on APA pages 9, 12, 17, 21, 23 and 25. 27 through 31 is a discussion of his heart trouble. He was certified disabled on sometime around February 10th, 1998. He had severe problems with hypertension and cardio vascular problems. As a matter of fact, APA page 30 talks about severe depressed left ventricle -- ventricle function and he was unable to do repetitive labor. He was deemed disabled and was terminated.

THE COURT: What page was that?

MR. DUNBAR: APA page 30 from Sumter Medical Consultants. He was still employed at the plant at that particular time; he began receiving disability benefits.

With respect to heavy metal poisoning, pages 7 through 9, 11 of our APA, Your Honor, points that out and also pages 40 through 43. In those particular cases you will see the blood lead levels and again, as in all these other cases the blood lead levels are extremely high. APA page 40 you will see it ranging from no less than 41, as high as 93, sustained levels in the 50s, 60s and 80s during 1978. Never taken out of the environment at that point in time because in 1978 there was no basis by which industry nor the

government required removal. And you'll see elevated blood lead levels well beyond permissible exposure limits also on page 41. So clearly I don't think the Fund can deny heavy metal poisoning; and that continues on page 42 as well as 43. Sustained levels over 50, yet he continued to work.

He also suffered with diabetes and that's APA pages 53 through 56. Heavy metal poisoning and diabetes are both those listed conditions. He had elevated blood sugars on July 2nd, 1998 and we see diabetes -- diabetes for the first time being mentioned in APA page 54 as opposed to just elevated blood sugars. And then throughout he began treatment for his diabetic condition.

Your Honor, we take the position that because this gentleman suffered with those preexisting conditions and the expert reports, medical evidence that we've submitted, the medical questionnaires as well as the Claimant's knowledge affidavit we believe that this case is subject to Second Injury Fund reimbursement.

THE COURT: Okay. Ms. Edwards.

BY MS. EDWARDS: Yes. The Second Injury Fund denied this claim, Your Honor, and I'll just be frank. We did think that this case had some reimbursement

potential, but we also thought that there was evidence in the case that could go either way. And so we offered to accept the case last year at Carrier APA page 89, and I believe Vernon has that offer at his Fund -- I'm sorry, at Fund -- at Carrier APA page 89 -- I think that's -- Vernon entered that into evidence and I'm not going to object to it because I think there were some good points and bad points on each side hence we -- and tried to enter some offer of acceptance with them in September of 2011.

At Fund APA page 1 we finally ended up withdrawing the offer simply because the Carrier -- I don't know if they didn't think that was an acceptable compromise or not, but they did not respond to it. And so we asserted, and we again made some additional movement toward doing an offer and acceptance in December as well. The denial primarily in this case is based on the statutory construction argument at 42-7-320(b) and its subsection which prohibits the Fund from accepting a case after December 31, 2011. But I also want to point out some of the case -- the -- the evidence that I think supports a denial based on the substantive issues. At Carrier APA pages 7 through 9 Mr. Dunbar cites the incidents of lead and high blood pressure -- lead exposure and the -- I'm

sorry, those are the lead levels at 7 through 9. At Carrier APA page 8, in spite of those lead levels that document indicates that the -- that it was felt that the Claimant -- that this did not pose a problem to Claimant's health by continuing to work in the area that he was previously employed and that there was no finding of any lead involvement during that period. And so even with the lead levels that are cataloged at Carrier APA pages 40 through 43 I think that document fully indicates that those lead levels did not pose any problem to his health or continuing to work in that area. And so at Carrier APA page -- I think another page that was cited was -- let me just look through just quickly. Sorry. Okay. And also at Carrier APA page 30 that Mr. Dunbar also cites; this is a 1998 document. I'm not really sure when this gentleman stopped working for Yuasa Exide. I'm not sure when he -- when did he stop working? I'm sorry.

MR. DUNBAR: I think some time in '99.

MS. EDWARDS: '99? Okay. So that -- this was just before he stopped working. It does indicate that he had a problem performing repetitive labor and if other work could be found that doesn't involve heavy lifting he would be able to continue working in that area.

So we believe that those documents, in spite of the fact that we offered to make some kind of acceptance to them, because like we said, in a lot of cases there are good and bad points on all sides. And so the side that I think -- the evidence that I think supports our denial based on the permanent and hindrance issue are the documents indicating that in spite of the lead exposure he was still okay as far as his health was concerned to work in that area and didn't pose a health risk; and that he could still do work as long as it was not repetitive trauma or anything in that sort.

And so in addition to those arguments on the hindrance -- hindrance issue we also would once again assert our argument based on the 42-7-320(b) and the statutory subsections.

THE COURT: Okay. Mr. Dunbar.

BY MR. DUNBAR: Yes, very briefly. With respect to APA page 8 wherein Ms. Edwards argues that the letter shows that he has average 59 blood lead level or micrograms per deciliter of blood in the past six months. This was prior to the promulgation of the OSHA Regulations which we don't believe the Commission has adopted, but even if that were the case according to OSHA's own regulations 1925, I believe 1025-K-1

states that a sustained level of 50 micrograms per deciliter of blood he should have been removed from the environment, so that is certainly harmful. But this is, again, promulgated before OSHA Regulations really came into effect. So we take the position that this document dated December 2nd, 1983 certainly does not comport with OSHA's Guidelines. It certainly doesn't comport with industry guidelines because he would have been removed because of the fear of lead poisoning at sustained levels of 59 micrograms per deciliter of blood.

Secondly, the doctor did indicate that he applauded Mr. Williams for continuing for work, working despite his congestive heart failure.

And with respect to the Commission's ability to hear this case we certainly believe that the Commission by law can hear cases that the Fund does not accept. That particular Statute that -- 42-7-320(b) basically says as I read it, it's an internal document to govern the Fund -- to govern the Fund. Basically tells the Fund, we're going to wind down, you do not accept cases voluntarily after December 2011. However, 42-9-400 is still in effect, which means the Commission has the absolute right to adjudicate these particular proceedings, because in

this particular case the Fund decided not -- the compromise was not accepted in terms of settlement. That's what the Commission is for to resolve those issues. Now it can cut either way and we understand that when we come before the Commission in Second Injury Fund cases it's all or nothing. In this particular case the reason why the compromise figure was rejected was because the clincher documents contained two average weekly wages; one for $29,000, one for like $112,000. I'm sorry, $112 dollars per week and the other compensation rate was around $400 dollars per week the reason why this gentleman was out on disability. But the clincher reflected both average weekly wage and compensation rates. The Fund accepted the lower rate. This case, I believe, was settled for around $175, $185,000 dollars. We did not take into account brain injury so it wasn't settled as a brain or lifetime injury case; it was settled for commuted value or less. So it was our argument that the Fund should have used the higher comp rate in giving us some type of settlement offer and they did not. And that's why -- and we couldn't get -- we couldn't get the clincher amended in time before December 31st, 2011 so the Fund could not go back and re-evaluate. Since that time the clincher has been

amended and there is an amended Order in Mr. Williams' case showing the accurate average weekly wage and compensation rate and was signed by Commissioner Williams.

THE COURT: Okay.

BY MS. EDWARDS: Just briefly, Commissioner, if I can. I promise I will be brief.

On the issue of hindrance, I think that if the Carrier had removed the Claimant from the job that certainly would be the stronger argument. They didn't have to wait for OSHA Regulations to change to remove somebody from an environment where they're being exposed, so they could have done that; they didn't do that. His record said that he was okay, healthy, he could go ahead and work in that arena. Also, I dispute or disagree with Mr. Dunbar's assertion that the Statute is an internal document on the winding down process. We believe the Statute applies to everybody, not just the Second Injury Fund, and certainly anybody who's practicing in the workers' comp arena we certainly would assert that the Statute applies to them as well. And so I would just like to add that and that will be all for me. I'll let Mr. Dunbar have the last word if he so chooses to.

BY MR. DUNBAR: The only thing I would mention is

that the diabetes and the cardiac disease and the heavy metal poisoning are presumed to create hindrance and obstacles to employment or re-employment and I think the Statute speaks to that.

THE COURT: All right. Thank you. That concludes the hearing.

(The hearing concluded at 1:08 p.m.)

STATE OF SOUTH CAROLINA)
)
COUNTY OF LEXINGTON)

CERTIFICATE

BE IT KNOWN THAT I TOOK THE FOREGOING WORKERS' COMPENSATION HEARING;

THAT I WAS THEN AND THERE A NOTARY PUBLIC IN AND FOR THE STATE OF SOUTH CAROLINA-AT-LARGE;

THE FOREGOING TRANSCRIPT CONSISTING OF 14 TYPEWRITTEN PAGES REPRESENTS A TRUE, ACCURATE AND COMPLETE TRANSCRIPTION OF THE TESTIMONY SO GIVEN AT THE TIME AND PLACE AFORESAID TO THE BEST OF MY SKILL AND ABILITY;

THAT I AM NOT RELATED TO NOR AN EMPLOYEE OF ANY OF THE PARTIES HERETO, NOR A RELATIVE OR EMPLOYEE OF ANY ATTORNEY OR COUNSEL EMPLOYED BY THE PARTIES HERETO, NOR INTERESTED IN THE OUTCOME OF THIS ACTION.

WITNESS MY HAND AND SEAL THIS 2ND DAY OF NOVEMBER, 2012.

_____
CORA ELLIS BRUTON
NOTARY PUBLIC FOR SOUTH CAROLINA
MY COMMISSION EXPIRES JANUARY 18, 2015

CORA ELLIS BRUTON - COURT REPORTER
131 BROWNING COURT - LEXINGTON, SOUTH CAROLINA 29073
803-397-0189

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION

W.C.C. FILE NO: 9930399

C. L. WILLIAMS,

Employee,

Claimant,

vs.

YUASA-EXIDE, INC.,

Employer,

AND

ARROWPOINT CAPITAL CORPORATION,

Carrier,

Defendants.

NOTICE OF WITNESSES AND WRITTEN MEDICAL REPORTS TO BE INTRODUCED AS DIRECT EVIDENCE ON BEHALF OF DEFENDANTS

TO: SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION AND G. MURRELL SMITH, JR., ESQUIRE:

YOU ARE NOTIFIED that the Defendants, pursuant to the provisions of the South Carolina Workers' Compensation Act and Section 1-23-330 of the South Carolina Code of Laws (Cum. Supp. 1988) submit the following medical records and other documents as evidence:

APA#	NAME OF PROVIDER/OTHER	DATE(S) OF RECORD(S)	PAGE NUMBERS
1.	Edward L. Baker, Jr., MD – Second Injury Fund Questionnaire	6/26/2011	1-3
2.	Eugene Shippen, MD – Second Injury Fund Questionnaire	5/31/2011	4-6
3.	Yuasa Exide's Health	1/20/1982-	7-31

	History Documents	8/12/1998	
4.	Yuasa Exide's Periodic Health History Questionnaire	5/24/1993	32-35
5.	Yuasa Exide's Occupational Health History Questionnaires	8/31/1986	36-39
6.	Claimant's Blood Lead Levels	3/15/1976-7/02/1985	40-43
7.	Records of the Davis Clinic	1/19/1984-1/26/1999	44-59
8.	Post Yuasa Exide Medical Records	2/15/2002-11/14/2007	60-88
Exhibits:			
A.	Letter from Second Injury Fund	9/20/2011	89
B.	Employee/Claimant Knowledge Affidavit	10/20/2010	90-92

YOU ARE FURTHER NOTIFIED that you have the right to cross-examine or otherwise oppose this evidence and, should you desire to exercise this right, you are to promptly schedule the deposition of any provider whose records are submitted, for the purposes of cross-examination, or otherwise promptly submit opposing medical records into evidence.

YOU ARE FURTHER NOTIFIED that these records, or photocopies of the same, will be provided to the South Carolina Workers' Compensation Commission for insertion in their file and for consideration as evidence on behalf of the Defendants.

YOU ARE FURTHER NOTIFIED that the following witnesses may be called on behalf of the Defendants:

Vernon F. Dunbar, Esq.
TURNER PADGET GRAHAM & LANEY P.A.
P.O. Box 1509
Greenville, SC 29602
(864) 552-4601
Attorneys for the Employer/Carrier

Greenville, SC

July 27, 2012

SECOND INJURY FUND MEDICAL QUESTIONNAIRE

Re: Claimant: C.L.Williams
Employer: Yuasa-Exide, Inc.
WCC File No.: 9930399
Date of Accident: 02/01/1999

1. Did C.L. Williams have preexisting conditions of diabetes, hypertension, heavy metal poisoning, chronic obstructive pulmonary disease, pulmonary hypoventilation syndrome, rectal bleeding, coronary artery disease, pulmonary enema, tinnitus, loss of hearing and vertigo?

X Yes _____ No

Please comment if necessary:

My review of the medical records indicated that he had diabetes, hypertension, heavy metal poisoning, chronic obstructive pulmonary disease, rectal bleeding, coronary artery disease, tinnitus, vertigo and hearing loss.

2. Were the preexisting conditions as stated above serious conditions which would constitute a hindrance or obstacle to employment or reemployment?

X Yes _____ No

Please comment if necessary:

3. Did C.L. Williams' exposure to levels of lead either aggravate or combine with his underlying preexisting conditions and render him permanently disabled?

X Yes _____ No

Please comment if necessary:
He is totally disabled.

4. Because C.L. Williams had the aforesaid preexisting conditions, did these conditions cause him to lose substantially more time from work or to be disabled from work than he would have solely from the last exposure to lead on February 1, 1999.

X Yes _____ No

Please comment if necessary:

5. Because C.L. Williams suffered from preexisting diseases, maladies and conditions as set forth above, did these conditions result in him having a substantially higher percentage of permanent disability than he would have had from the last known exposure to lead? The last exposure to lead occurred on or about February 1, 1999?

X Yes _____ No

Please comment if necessary:

6. Have the medical costs in this case been substantially increased due to the existence of the preexisting factors/conditions as set forth above and in the medical records of C.L. Williams?

X Yes _____ No

Please comment if necessary:

7. Does your review of C.L. Williams' blood lead level and medical files and all other medical reports reflect he had suffered with heavy metal poisoning prior to February 1, 1999?

X Yes ____ No

Please comment if necessary:

8. Because C.L. Williams had suffered with heavy metal poisoning prior to February 1, 1999, did his last exposure to lead on or about February 1, 1999 constitute a new accidental injury by virtue of this last exposure and aggravate the preexisting conditions as set forth above?

X Yes ____ No

Please comment if necessary:

9. Did C.L. Williams' February 1, 1999 work related accident which exposed him to lead result in disability that was substantially greater by reason of the combined effects of the preexisting conditions and heavy metal poisoning or by aggravation of the preexisting conditions including heavy metal poisoning than would have resulted solely from the February 1, 1999 accidental injury?

X Yes ____ No

Please comment if necessary:

By: Edward L. Baker, Jr., MD, MPH, MSc

June 26, 2011

SECOND INJURY FUND MEDICAL QUESTIONNAIRE

Re: Claimant: C.L.Williams
Employer: Yuasa-Exide, Inc.
WCC File No.: 9930399
Date of Accident: 02/01/1999

1. Did C.L. Williams have preexisting conditions of diabetes, hypertension, heavy metal poisoning, chronic obstructive pulmonary disease, pulmonary hypoventilation syndrome, rectal bleeding, coronary artery disease, pulmonary enema, tinnitus, loss of hearing and vertigo?

_X_ Yes ____ No

Please comment if necessary:

2. Were the preexisting conditions as stated above serious conditions which would constitute a hindrance or obstacle to employment or reemployment?

_X_ Yes ____ No

Please comment if necessary:

3. Did C.L. Williams' exposure to levels of lead either aggravate or combine with his underlying preexisting conditions and render him permanently disabled?

_X_ Yes ____ No

Please comment if necessary:

4. Because C.L. Williams had the aforesaid preexisting conditions, did these conditions cause him to lose substantially more time from work or to be disabled from work than he would have solely from the last exposure to lead on February 1, 1999.

__X__ Yes _____ No

Please comment if necessary:

5. Because C.L. Williams suffered from preexisting diseases, maladies and conditions as set forth above, did these conditions result in him having a substantially higher percentage of permanent disability than he would have had from the last known exposure to lead? The last exposure to lead occurred on or about February 1, 1999?

__X__ Yes _____ No

Please comment if necessary:

6. Have the medical costs in this case been substantially increased due to the existence of the preexisting factors/conditions as set forth above and in the medical records of C.L. Williams?

__X__ Yes _____ No

Please comment if necessary:

7. Does your review of C.L. Williams' blood lead level and medical files and all other medical reports reflect he had suffered with heavy metal poisoning prior to February 1, 1999?

__X__ Yes _____ No

Please comment if necessary:

8. Because C.L. Williams had suffered with heavy metal poisoning prior to February 1, 1999, did his last exposure to lead on or about February 1, 1999 constitute a new accidental injury by virtue of this last exposure and aggravate the preexisting conditions as set forth above?

X Yes ____ No

Please comment if necessary:

9. Did C.L. Williams' February 1, 1999 work related accident which exposed him to lead result in disability that was substantially greater by reason of the combined effects of the preexisting conditions and heavy metal poisoning or by aggravation of the preexisting conditions including heavy metal poisoning than would have resulted solely from the February 1, 1999 accidental injury?

X Yes ____ No

Please comment if necessary:

By: _____

Eugene Shippen, M.D.
Lead Toxicology Consultant
1124 Old Mill Road
Wyomissing, PA 19610

May 31, 2011

TO BE COMPLETED BY EXAMINING PHYSICIAN

	YES	NO
1. GENERAL		
Pallor		
Cyanosis		
Jaundice		
Skin Disease		

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
2. EYES				
Pupils - Regular				
Equal				
React to Light				
React to Acc				
Exophthalmus				
Nystagmus				
Strabismus				
Cataract				
Conjunctivae - Normal				
Congested				
Icteric				
Cornea - Normal				
Arcus senilis				
Ulcers				
Retina - Normal				
Hemorrhages				
Exudates				
Disc edema				
Blurring				

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
3. EARS				
Discharge				
Drum intact				
Canals normal				

	YES	NO
4. NOSE		
Perforation of Septum		
Mucous Membranes		
Normal	✓	
Congested		
"Allergic"		
Polyps		

	YES	NO
5. MOUTH		
Good Hygiene		
Dentures - Partial		
Complete		
None		

	YES	NO
6. THROAT		
Tonsils - Absent		
Normal	✓	
Infected		
7. THYROID - Normal		
Enlarged		
Nodular		
8. THORAX		
Chest - Symmetrical		
Shape - Normal		
Breath Sounds - Normal		
Rales (Type and Location)		

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
9. BREASTS				
Normal				
Tumor				
Other				

	SITTING RIGHT	SITTING LEFT
10. CIRCULATORY SYSTEM		
Blood Pressure -		
Systolic	125	
Diastolic	90	
	YES	NO
Apex within M.C.L.	✓	
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal		
Forceful		
Distant		
Murmurs		
Pheripheral Vessels - Normal	✓	

	YES	NO
11. SPINE		
Cervical - Normal	✓	
Thoracic - Normal		
Lumbar - Normal		
Sacral - Normal	✓	
12. ABDOMEN		
Scars		
Masses		
Organomegly		
13. HERNIAL ORIFICES		
Hernia		
Contents - Normal		
14. ANUS		
Ext. Hemorrhoids		
Int. Hemorrhoids		
Fissure		
Prostate - Normal	✓	
15. EXTREMITIES - NORMAL		
Describe deformity or mal function including scars		
16. MISCELLANEOUS		
Adenopathy - Locate		
Varicosities - Locate		
Etc.		
17. OTHER FINDINGS		
Pelvis - if indicated		

LIST ALL POSITIVE FINDINGS OR ANY DIAGNOSIS INDICATED FROM ANSWERS TO THE ABOVE OR PREVIOUS PAGE.

1-07-82 Ref. to Clearance Ch

BLOOD PANEL

HT. 68"

WGT: 184 1/2

VISION 1-20-82

AUDIO

LABSTIX Patient exam pkg 6 4/82

Mtg for Review 1-20-82

HISTORY 1-20-82

H-x ? WBC

Lung Hx ? Sx Hb

Abn Pertuss W/B

C/B Rebuttal

Hx Sx Hb

Hx Partial Bleeding

Hx Visual Lesions

Audiogram 4s

① Repeat WBC 2 wks

② Sic. Dr M. Davis Re: Partial Bleeding

Denied e/p 25 Jan 82

AMT

EXIDE

December 12, 1983

C. L. Williams

34 year old Black Male

Stacker in the Assembly Area of Electric Vehicle

He has averaged 59ug/100 GM in the past six (6) months and his most recent blood lead was reported at 57ug/100. He underwent a complete history and physical exam and in review of past personal and family history in February 1983, at which time there were no findings of any lead involvement period. His interval history has been completely negative from that standpoint and it is felt that it poses no problem to his health by continuing to work in an area that he has previously been employed.

AWH:sw

A Corporation of
Inco Electro-Energy
U.S. Route 15 Sumter, South Carolina 29150 803/481 8511 Twx 810-666-2583

Physician's Examination and Consultation

	YES	NO
1. GENERAL		
Pallor		
Cyanosis		
Jaundice		
Skin Disease		

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
2. EYES				
Pupils - Regular				
Equal				
React. to Light				
React. to Acc.				
Exophthalmus				
Nystagmus				
Strabismus				
Cataract				
Conjunctivae - Normal				
Congested				
Icteric				
Cornea - Normal				
Arcus senilis				
Ulcers				
Retina - Normal				
Hemorrhages				
Exudates				
Disc edema				
Nicking				

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
3. EARS				
Discharge				
Drum intact				
Canals normal				

	YES	NO
4. NOSE		
Perforation of Septum		
Mucous Membranes		
Normal		
Congested		
Atrophic		
Polyps		

	YES	NO
5. MOUTH		
Good Hygiene		
Dentures - Partial		
Complete		
None		
6. THROAT		
Tonsils - Absent		
Normal		
Infected		
7. THYROID - Normal		
Enlarged		
Nodular		
8. THORAX		
Chest - Symmetrical		
Shape - Normal		
Breath Sounds - Normal		
Rales (Type and Location)		

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
9. BREASTS				
Normal				
Tumor				
Other				

	SITTING RIGHT	SITTING LEFT
10. CIRCULATORY SYSTEM		
Blood Pressure		
Systolic	138	
Diastolic	98	

	YES	NO
Apex within M.C.L.		
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal		
Forceful		
Distant		
Murmurs		✓
Peripheral Vessels - Normal	✓	

	YES	NO
11. SPINE		
Cervical - Normal		
Thoracic - Normal		
Lumbar - Normal		
Sacral - Normal		
12. ABDOMEN		
Scars		
Masses		
Organomegaly		
13. INGUINAL REGION		
Hernia		
Genitalia - Normal		
Scrotal contents - Normal		
14. RECTUM		
Pat. Hemorrhoids		
Int. Hemorrhoids		
Masses		
Prostate - Normal		
15. EXTREMITIES - NORMAL		
Describe deformity or mal function including reason		
16. MISCELLANEOUS		
Adenopathy -		
Locate		
Varicosities -		
Locate		
Etc.		
17. OTHER FINDINGS		
Pelvic - If indicated		

LIST ALL POSITIVE FINDINGS OR ANY DIAGNOSIS INDICATED FROM ANSWERS TO THE ABOVE OR PREVIOUS PAGE.

NAME: C. L. Williams age 35

S.S. NO.:

BP 138/98

CHEM. SCREEN: 1-11-84

LABSTIX:

Blood Lead 50 ugm %

HEIGHT: 68½

WEIGHT: 250 ½ 165

Lab → Nurse Asst

AUDIO SCREEN: 1-11-84 CLB

VISUAL SCREEN: 1-11-84 - St Refuses V/A

HISTORY COMPLETED 1-11-84 - Fam. hx. CVA, ↑ BP

Past hx ↑ BP

Past hx renal cysts

Disp Chem B/M-E

Hyperflexia & hyperactive

Nurse called Dr. York MD

Dr. York

Counseled

Physician's Examination and Consultation

1. GENERAL	YES	NO
Pallor		/
Cyanosis		/
Jaundice		/
Skin Disease		/

2. EYES	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Pupils - Regular	/		/	
Equal	/		/	
React to Light	/		/	
React to Acc	/		/	
Exophthalmos		/		/
Nystagmus		/		/
Strabismus		/		/
Cataract		/		/
Conjunctivae - Normal	/		/	
Congested		/		
Icteric				
Cornea - Normal	/		/	
Arcus senilis		/		/
Ulcers		/		
Retina - Normal	/		/	
Hemorrhages		/		/
Exudates		/		/
Disc edema		/		/
Nicking		/		/

3. EARS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Discharge		/		/
Drum Intact	/		/	
Canals normal	/		/	

4. NOSE	YES	NO
Perforation of Septum		/
Mucous Membranes		
Normal	/	
Congested		
"Allergic"		
Polyps		

5. MOUTH	YES	NO
Good Hygiene	/	
Dentures - Partial		/
Complete	/	
None		

6. THROAT	YES	NO
Tonsils - Absent		/
Normal	/	
Infected		

7. THYROID - Normal	YES	NO
	/	
Enlarged		/
Nodular		

8. THORAX	YES	NO
Chest - Symmetrical	/	
Shape - Normal	/	
Breath Sounds - Normal	/	
Rales (Type and Location)		/

9. BREASTS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Normal	/		/	
Tumor		/		/
Other				

10. CIRCULATORY SYSTEM	SITTING RIGHT	SITTING LEFT
Blood Pressure	138	
Systolic	72	
Diastolic		

	YES	NO
Apex within M.C.L.	/	
Rhythm - Normal Sinus	/	
Heart Sounds - Normal	/	
Forceful		
Distant		
Murmurs		/
Peripheral Vessels - Normal	/	

11. SPINE	YES	NO
- Cervical - Normal	/	
Thoracic - Normal	/	
Lumbar - Normal	/	
Sacral - Normal	/	

12. ABDOMEN	YES	NO
Scars		/
Masses		/
Organomegaly		/

13. INGUINAL REGION	YES	NO
Hernia		/
Genitalia - Normal	/	
Scrotal contents - Normal	/	

14. RECTUM	YES	NO
Ext. Hemorrhoids		/
Int. Hemorrhoids		/
Masses		/
Prostate - Normal	/	

15. EXTREMITIES - NORMAL	
Describe deformity or mal function including neuro	

16. MISCELLANEOUS	
Adenopathy - Locate	
Varicosities - Locate	
Etc. -	

17. OTHER FINDINGS	
Pelvic - If indicated	

EXIDE

2-13-84 C. L. WILLIAMS 34 years old Black Male
Stacker - E.V. Assy.

For the past six (6) months his blood leads have averaged 57ug/dl. His most recent blood lead done on January 11, 1984 was 50ug/dl. He has had a 1983 lead-in-air level of 30ug/m^3 and he presently wears a Survivair respirator. He recently underwent a complete physical examination on January 30, 1984 including a review of past personal and family history, all of which were noncontributory to any lead involvement and it is felt that he can continue to function in his present capacity.

:sw A. W. Hursey, Jr., M.D.

4-26-84 C. L. WILLIAMS 35 year old Black Male
Factory Worker - EV Assembly

For the past six (6) months his blood leads have averaged 57ug/dl. His most recent blood lead was 55ug/dl. He presently wears a Survivair respirator. He underwent a complete physical exam on January 30, 1984 and that and his interval history has been negative for any lead involvement. He reportedly works in a low lead area however, I am at lost to explain why his blood leads continue to be elevated. It is felt that he can continue to function in his present capacity and be seen for a review in two (2) months.

:sw A. W. Hursey, Jr. M.D.

9-04-84 C.L. WILLIAMS 35 year old Black Male
Stacker - EV Assembly

For the past six months his blood leads have averaged 43ug/dl. His most recent blood lead was 48ug/dl. He wears a Survivair respirator. He has had a progressive decrease in his blood leads since the spring. Primarily because of increased personal hygiene in the plant. He underwent a complete physical exam on this date with no problems referable to any lead intoxication and it is felt that he can safely function in his present position without any complications.

:sw A. W. Hursey, Jr., M.D.

A Corporation of
Inco Electro Energy
P.O. Box 2408 Sumter, South Carolina 29150 803/773/5467 Twx 810-663-2583

Physician's Examination and Consultation

	YES	NO
1. GENERAL		
Pallor		
Cyanosis		
Jaundice		
Skin Disease		

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
2. EYES				
Pupils - Regular				
Equal				
React to Light				
React to Acc.				
Exophthalmos				
Nystagmus				
Strabismus				
Cataract				
Conjunctiva - Normal				
Congested				
Icteric				
Cornea - Normal				
Arcus senilis				
Ulcers				
Retina - Normal				
Hemorrhages				
Exudates				
Disc edema				
Neuritis				

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
3. EARS				
Discharge				
Drum intact				
Canals normal				

	YES	NO
4. NOSE		
Perforation of Septum		
Mucous Membranes - Normal		
Congested		
"Allergic"		
Polyps		

	YES	NO
5. MOUTH		
Good Hygiene		
Dentures - Partial		
Complete		
None		

	YES	NO
6. THROAT		
Tonsils - Absent		
Normal		
Infected		
7. THYROID - Normal		
Enlarged		
Nodular		
8. THORAX		
Chest - Symmetrical		
Shape - Normal		
Breath Sounds - Normal		
Rales (Type and Location)		

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
9. BREASTS				
Normal				
Tumor				
Other				

	SITTING RIGHT	SITTING LEFT
10. CIRCULATORY SYSTEM		
Blood Pressure		
Systolic	120	
Diastolic	90	
	YES	NO
Apex within M.C.L.		
Rhythm - Normal Sinus		
Heart Sounds - Normal		
Forceful		
Distant		
Murmurs		
Peripheral Vessels - Normal		

	YES	NO
11. SPINE NSERD		
Cervical - Normal		
Thoracic - Normal		
Lumbar - Normal		
Sacral - Normal		
12. ABDOMEN		
Scars		
Masses		
Organomegaly		
13. INGUINAL REGION		
Hernia		
Genitalia - Normal		
Scrotal contents - Normal		
14. RECTUM		
Ext. Hemorrhoids		
Int. Hemorrhoids		
Masses		
Prostate - Normal		

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
15. EXTREMITIES - NORMAL				
Describe deformity or mal function including scars				

6. Velcro
3° Peripheral
Blue Burn

	YES	NO
16. MISCELLANEOUS		
Adenopathy - Locate		
Varicosities - Locate		
Etc.		
17. OTHER FINDINGS		
Pelvic - If indicated		

LIST ALL POSITIVE FINDINGS OR ANY DIAGNOSIS INDICATED FROM ANSWERS TO THE ABOVE OR PREVIOUS PAGE.

NAME: C.L. Williams Age 75 6-month Physical

S.S. NO.:

BP 122/90 Hx HBP

CHEM. SCREEN: 8-13-84 - OK Hx mild diabetes

LABS TX: Blood lead 48 mg% Rx

HEIGHT: 5'8½" Continue with Rx

WEIGHT: 201½ 7 Sept 84

AUDIO SCREEN: 8-13-84 CLB

VISUAL SCREEN: OK

HISTORY COMPLETED: Home - Fam hx CVA - HBP

Physician's Examination and Consultation

	YES	NO
1. GENERAL		
Pallor		✓
Cyanosis		✓
Jaundice		✓
Skin Disease		X

	RIGHT		LEFT	
2. EYES	YES	NO	YES	NO
Pupils - Regular	✓		✓	
Equal	✓		✓	
React to Light	✓		✓	
React to Acc	X		X	
Exophthalmos		✓		✓
Nystagmus		✓		✓
Strabismus		✓		✓
Cataract		✓		✓
Conjunctiva - Normal	✓		✓	
Congested		✓		
Icteric				
Cornea - Normal	✓		✓	
Arcus senilis				
Ulcers		✓		
Retina - Normal	✓		✓	
Hemorrhages				
Exudates		✓		✓
Disc edema		✓		✓
Nicking				

	RIGHT		LEFT	
3. EARS	YES	NO	YES	NO
Discharge		✓		✓
Drums intact	✓		✓	
Canals normal	✓		✓	

	YES	NO
4. NOSE		
Perforation of Septum		✓
Mucous Membranes		
Normal		
Congested		
"Allergic"		
Polyps		

	YES	NO
5. MOUTH		
Good Hygiene		
Dentures - Partial		✓
Complete		
None		

	YES	NO
6. THROAT		
Tonsils - Absent		✓
Normal	✓	
Infected		

	YES	NO
7. THYROID - Normal		
Enlarged		✓
Modular		

	YES	NO
8. THORAX		
Chest - Symmetrical		
Shape - Normal		
Breath Sounds - Normal		✓
Rales (Type and Location)		

	RIGHT		LEFT	
9. BREASTS	YES	NO	YES	NO
Normal				
Tumor			✓	
Other				

	SITTING	
10. CIRCULATORY SYSTEM	RIGHT	LEFT
Blood Pressure		
Systolic	120	
Diastolic	70	
	YES	NO
Apex within M.C.L.	✓	
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal	✓	
Forceful		
Distant		
Murmurs		✓
Peripheral Vessels -		
Normal	✓	

	YES	NO
11. SPINE NSE Rad		
Cervical - Normal	✓	
Thoracic - Normal	✓	
Lumbar - Normal	✓	
Sacral - Normal	✓	

	YES	NO
12. ABDOMEN		
Scars		✓
Masses		
Organomegaly		✓

	YES	NO
13. INGUINAL REGION		
Hernia		✓
Genitalia - Normal	✓	
Scrotal contents - Normal	✓	

	YES	NO
14. RECTUM		
Ext. Hemorrhoids		—
Int. Hemorrhoids		—
Masses		—
Prostate - Normal		—

	YES	NO
15. EXTREMITIES - NORMAL		
Describe deformity or mal function including neuro		

16. MISCELLANEOUS

Adenopathy -

Locate

Varicosities -

Locate

Etc.

17. OTHER FINDINGS

Pelvic - If indicated

SU-04 11/84

Physician's Examination and Consultation

1 GENERAL	YES	NO
Pallor		✓
Cyanosis		✓
Jaundice		✓
Skin Disease		✓

2 EYES	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Pupils - Regular	✓		✓	
Equal	✓		✓	
React to Light	✓		✓	
React to Acc	✓		✓	
Exophthalmus		✓		✓
Nystagmus		✓		✓
Strabismus		✓		✓
Cataract		✓		✓
Conjunctiva - Normal	✓		✓	
Congested				
Icteric				
Cornea - Normal	✓		✓	
Arcus senilis				
Ulcers				
Retina - Normal				
Hemorrhages	✓		✓	
Exudates				
Disc edema				
Nicking				

3 EARS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Discharge		✓		✓
Drum intact	✓		✓	
Canals normal	✓		✓	

4 NOSE	YES	NO
Perforation of Septum		✓
Mucous Membranes		
Normal	✓	
Congested		
Allergic		
Polyps		

5 MOUTH	YES	NO
Good Hygiene	✓	
Dentures - Partial		
Complete		
None	✓	

6 THROAT		
Tonsils - Absent		
Normal	✓	
Infected		

7 THYROID		
Normal	✓	
Enlarged		
Modular		

8 THORAX		
Chest - Symmetrical	✓	
Shape - Normal	✓	
Breath Sounds - Normal	✓	
Rales (Type and Location)		✓

9 BREASTS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Normal	✓		✓	
Tumor		✓		✓
Other				

10 CIRCULATORY SYSTEM	SITTING RIGHT	SITTING LEFT
Blood Pressure		
Systolic	130	
Diastolic	80	
Apex within M C L	YES ✓	NO
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal	✓	
Forceful		
Distant		
Murmurs		✓
Peripheral Vessels - Normal		

140/100 10-12-88
18-24-88

11 SPINE	YES	NO
Cervical - Normal	✓	
Thoracic - Normal		
Lumbar - Normal		
Sacral - Normal	✓	

12 ABDOMEN		
Scars		✓
Masses		
Organomegly		✓

13 INGUINAL REGION		
Hernia		✓
Genitalia - Normal	✓	
Scrotal contents - Normal	✓	

14 RECTUM		
Ext Hemorrhoids		✓
Int Hemorrhoids		✓
Masses		✓
Prostate - Normal	✓	

15 EXTREMITIES - NORMAL	
Describe deformity or mal function including neuro	WNL

16 MISCELLANEOUS	
Adenopathy - Locate	
Varicosities - Locate	
Etc.	

17 OTHER FINDINGS	
Pelvic - If indicated	

LIST ALL POSITIVE FINDINGS OR ANY DIAGNOSIS INDICATED FROM ANSWERS TO THE ABOVE OR PREVIOUS PAGE.

NAME C. L. Williams Age 40

SS NO

B.P. 140/95 10-13-88

CHEM. SCREEN 10-13-88 Hx: Borderline Hypertension

PAPTIX Bldg 40 (9-1-88) Diet 15 lb weight gain

Au Bb 17 (11-87) Epistaxis LFTs -

HEIGHT 69 Hx. Elevated Lipids

WEIGHT 213

AUDIO SCREEN 10-13-88 Clear A. 10-24-88. Ewell Kyzar

VISUAL SCREEN 10-12-88

xC. L. Williams

HISTORY COMPLETED Taken Home ✓ OK -14-

SU-04 11/84

Physician's Examination and Consultation

	YES	NO
1. GENERAL		
Pallor		✓
Cyanosis		
Jaundice		
Skin Disease		✓

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
2. EYES				
Pupils - Regular	✓		✓	
Equal				
React to Light	✓		✓	
React to Acc				
Exophthalmos		✓		✓
Nystagmus		✓		✓
Strabismus		✓		✓
Cataract		✓		✓
Conjunctiva - Normal	—		—	
Congested				
Icteric				
Cornea - Normal	—		—	
Arcus senilis				
Ulcers				
Retina - Normal	—		—	
Hemorrhages				
Exudates				
Disc edema				
Nicking				

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
3. EARS				
Discharge		—		—
Drum intact		—		—
Canals normal	—		—	

	YES	NO
4. NOSE		
Perforation of Septum		—
Mucous Membranes - Normal	—	
Congested		
"Allergic"		
Polyps		

	YES	NO
5. MOUTH		
Good Hygiene	—	
Dentures - Partial		
Complete		
None	—	
6. THROAT		
Tonsils - Absent		
Normal	—	
Inflamed		
7. THYROID - Normal	—	
Enlarged		
Modular		
8. THORAX		
Chest - Symmetrical	—	
Shape - Normal	—	
Breath Sounds - Normal	—	
Rales (Type and Location)		—

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
9. BREASTS				
Normal	—		—	
Tumor		—		—
Other				

	SITTING RIGHT	SITTING LEFT
10. CIRCULATORY SYSTEM		
Blood Pressure		
Systolic	120	
Diastolic	80	

	YES	NO
Apex within M.C.L.	—	
Rhythm - Normal Sinus	—	
Heart Sounds - Normal	—	
Forceful		
Distant		
Murmurs		—
Peripheral Vessels - Normal	—	

	YES	NO
11. SPINE		
Cervical - Normal	✓	
Thoracic - Normal		
Lumbar - Normal		
Sacral - Normal	✓	
12. ABDOMEN		
Scars - ventral hernia		✓
Masses	✓	
Organmegly		✓
13. INGUINAL REGION		
Hernia		✓
Genitalia - Normal	✓	
Scrotal contents - Normal	—	
14. RECTUM		
Ext. Hemorrhoids		—
Int. Hemorrhoids		—
Masses		—
Prostate - Normal	—	
15. EXTREMITIES - NORMAL	WNL	
Describe deformity or mal function including neuro		

	YES	NO
16. MISCELLANEOUS		
Adenopathy - Locate		
Varicosities - Locate		
Etc.		
17. OTHER FINDINGS		
Pelvic - If indicated		

LIST ALL POSITIVE FINDINGS OR ANY DIAGNOSIS INDICATED FROM ANSWERS TO THE ABOVE OR PREVIOUS PAGE.

NAME: C. L. Williams Age 41

S.S. NO.:

BP 120/80

CHEM. SCREEN: 8-16-90

LABSTIX

HEIGHT: 6 9½

WEIGHT: 226 1/4

AUDIO SCREEN 8-16-90 OU's A. OK

VISUAL SCREEN

HISTORY COMPLETED

Hx: 2 ventral leads repaired at WRP
Pt's CNT. on diuretics
& hypertensive Rx.

Emil Kyd 8-30-90

C. L. Williams

SU-04 11/84

Physician's Examination and Consultation

1. GENERAL	YES	NO
Pallor		✓
Cyanosis		✓
Jaundice		✓
Skin Disease		✓

2. EYES	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Pupils - Regular	✓		✓	
Equal	✓		✓	
React to Light	✓		✓	
React to Acc	✓		✓	
Exophthalmus		✓		✓
Nystagmus		✓		✓
Strabismus		✓		✓
Cataract		✓		✓
Conjunctiva - Normal	✓		✓	
Congested				
Icteric				
Cornea - Normal	✓		✓	
Arcus senilis				
Ulcers				
Retina - Normal	✓		✓	
Hemorrhages				
Exudates				
Disc edema				
Nicking				

3. EARS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Discharge		✓		✓
Drum intact	✓		✓	
Canals normal	✓		✓	

4. NOSE	YES	NO
Perforation of Septum		✓
Mucous Membranes		
Normal	✓	
Congested		
"Allergic"		
Polyps		

5. MOUTH	YES	NO
Good Hygiene	✓	
Dentures - Partial		
Complete		
None	✓	

6. THROAT	YES	NO
Tonsils - Absent		
Normal	✓	
Infected		

7. THYROID - Normal	YES	NO
	✓	
Enlarged		
Nodular		

8. THORAX	YES	NO
Chest - Symmetrical	✓	
Shape - Normal	✓	
Breath Sounds - Normal	✓	
Rales (Type and Location)		✓

9. BREASTS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Normal	✓		✓	
Tumor		✓		✓
Other		✓		✓

10. CIRCULATORY SYSTEM	SITTING RIGHT	SITTING LEFT
Blood Pressure		
Systolic	120	
Diastolic	80	

	YES	NO
Apex within M.C.L.	✓	
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal	✓	
Forceful		
Distant		
Murmurs		✓
Pheripheral Vessels - Normal	✓	

11. SPINE	YES	NO
Cervical - Normal	✓	
Thoracic - Normal	✓	
Lumbar - Normal	✓	
Sacral - Normal	✓	

12. ABDOMEN	YES	NO
Scars		✓
Masses		✓
Organomegaly		✓

Ventral hernia

13. INGUINAL REGION	YES	NO
Hernia -		✓
Genitalia - Normal	✓	
Scrotal contents - Normal	✓	

14. RECTUM	YES	NO
Ext. Hemorrhoids		✓
Int. Hemorrhoids		✓
Masses		✓
Prostate - Normal	✓	

15. EXTREMITIES - NORMAL	YES	NO
Describe deformity or mal function including neuro	✓	

WNL

16. MISCELLANEOUS	YES	NO
Adenopathy -		
Locate		
Varicosities -		
Locate		
Etc.		

17. OTHER FINDINGS	YES	NO
Pelvic - If indicated		

SU-84 11/84

Physician's Examination and Consultation

1. GENERAL	YES	NO
Pallor		✓
Cyanosis		
Jaundice		✓
Skin Disease		✓

2. EYES	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Pupils - Regular	✓		✓	
Equal	✓		✓	
React to Light	✓		✓	
React to Acc	✓		✓	
Exophthalmus		✓		✓
Nystagmus		✓		✓
Strabismus		✓		✓
Cataract		✓		✓
Conjunctiva - Normal	✓		✓	
Congested				
Icteric				
Cornea - Normal	✓		✓	
Arcus senilis				
Ulcers				
Retina - Normal	✓		✓	
Hemorrhages				
Exudates				
Disc edema				
Necking				

3. EARS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Discharge		✓		✓
Drum intact	✓		✓	
Canals normal	✓		✓	

4. NOSE	YES	NO
Perforation of Septum		✓
Mucous Membranes		
Normal	✓	
Congested		
"Allergic"		
Polyps		

5. MOUTH	YES	NO
Good Hygiene	✓	
Dentures - Partial		
Complete		
None	✓	

6. THROAT		
Tonsils - Absent		
Normal	✓	
Infected		

7. THYROID - Normal	✓	
Enlarged		
Nodular		

8. THORAX		
Chest - Symmetrical	✓	
Shape - Normal	✓	
Breath Sounds - Normal	✓	
Rales (Type and Location)		✓

9. BREASTS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Normal	✓		✓	
Tumor		✓		✓
Other				

10. CIRCULATORY SYSTEM	SITTING RIGHT	SITTING LEFT
Blood Pressure		
Systolic	130	
Diastolic	82	
	YES	NO
Apex within M.C.L.	✓	
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal	✓	
Forceful		
Diastolic		
Murmurs		✓
Peripheral Vessels - Normal	✓	

11. SPINE	YES	NO
Cervical - Normal	✓	
Thoracic - Normal	✓	
Lumbar - Normal	✓	
Sacral - Normal	✓	

12. ABDOMEN		
Scars		✓
Masses		
Organomegaly		✓

13. INGUINAL REGION		
Hernia		✓
Genitalia - Normal	✓	
Scrotal contents - Normal	✓	

14. RECTUM		
Ext. Hemorrhoids		✓
Int. Hemorrhoids		✓
Masses		✓
Prostate - Normal	✓	

15. EXTREMITIES - NORMAL	
Describe deformity or mal function including neuro	WNL

16. MISCELLANEOUS	
Adenopathy - Locate	
Varicosities - Locate	
Etc.	

17. OTHER FINDINGS	
Pelvic - If indicated	

LIST ALL POSITIVE FINDINGS OR ANY DIAGNOSIS INDICATED FROM ANSWERS TO THE ABOVE OR PREVIOUS PAGE.

NAME: C. L. Williams Age 42

S.S. NO.:

BP 130/82 Hx Chronic Pyelonephritis on Persistent ARF

CHEM. SCREEN: 6-27-91 Hx CHF

CARDIAC: Hypertension

HEIGHT: 6 9 3/4

WEIGHT: 242

AUDIO SCREEN: 6-27-91 Jan B. 6-15-91

VISUAL SCREEN: " OK Dr. B. Wright Jan

HISTORY COMPLETED: C. L. Williams

SU-04 11/84

Physician's Examination and Consultation

1. GENERAL	YES	NO
Pallor		✓
Cyanosis		✓
Jaundice		✓
Skin Disease		✓

2. EYES	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Pupils - Regular	✓		✓	
Equal	✓		✓	
React to Light	✓		✓	
React to Acc	✓		✓	
Exophthalmos		✓		✓
Nystagmus		✓		✓
Strabismus		✓		✓
Cataract		✓		✓
Conjunctiva - Normal	✓		✓	
Congested				
Icteric				
Cornea - Normal	✓		✓	
Arcus senilis				
Ulcers				
Retina - Normal	✓		✓	
Hemorrhages				
Exudates				
Discoloring				
Nicking				

3. EARS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Discharge		✓		✓
Drum Intact	✓		✓	
Canals normal	✓		✓	

4. NOSE	YES	NO
Perforation of Septum		✓
Mucous Membranes Normal	✓	
Congested		
"Allergic"		
Polyps		

5. MOUTH	YES	NO
Good Hygiene	✓	
Dentures - Partial		
Complete		
None	✓	

6. THROAT	YES	NO
Tonsils - Absent		
Normal	✓	
Infected		

7. THYROID - Normal	YES	NO
Enlarged		
Nodular		

8. THORAX	YES	NO
Chest - Symmetrical	✓	
Shape - Normal	✓	
Breath Sounds - Normal	✓	
Rales (Type and Location)		✓

9. BREASTS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Normal	✓		✓	
Tumor		✓		✓
Other		✓		✓

10. CIRCULATORY SYSTEM	SITTING RIGHT	SITTING LEFT
Blood Pressure Systolic	130	
Diastolic	82	
	YES	NO
Apex within M.C.L.	✓	
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal	✓	
Forceful		
Distant		
Murmurs		✓
Peripheral Vessels Normal	✓	

11. SPINE	YES	NO
Cervical - Normal	✓	
Thoracic - Normal	✓	
Lumbar - Normal	✓	
Sacral - Normal	✓	

12. ABDOMEN	YES	NO
Scars		✓
Masses		✓
Organmegly		✓

13. INGUINAL REGION	YES	NO
Hernia		✓
Genitalia - Normal	✓	
Sexual contact - Normal	✓	

14. RECTUM	YES	NO
Ext. Hemorrhoids		✓
Int. Hemorrhoids		✓
Masses		✓
Prostate - Normal	✓	

15. EXTREMITIES - NORMAL	YES	NO
Describe deformity or mal function including neuro	WNL	

16. MISCELLANEOUS	YES	NO
Adenopathy		
Locale		
Varicosities		
Locale		
Etc.		

17. OTHER FINDINGS	YES	NO
Pelvic - If indicated		

SU 04 11/84

Physician's Examination and Consultation

	YES	NO
1. GENERAL		
Pallor		✓
Cyanosis		✓
Jaundice		✓
Skin Disease		✓

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
2. EYES				
Pupils - Regular	✓		✓	
Equal	✓		✓	
React to Light	✓		✓	
React to Acc	✓		✓	
Exophthalmus		✓		✓
Nystagmus		✓		✓
Strabismus		✓		✓
Cataract		✓		✓
Conjunctiva - Normal	✓		✓	
Congested				
Icteric				
Cornea - Normal	✓		✓	
Arcus senilis				
Ulcers				
Retina - Normal	✓		✓	
Hemorrhages				
Exudates				
Disc edema				
Nicking				

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
3. EARS				
Discharge		✓		✓
Drum Intact	✓		✓	
Canals normal	✓		✓	

	YES	NO
4. NOSE		
Perforation of Septum		✓
Mucous Membranes		
Normal	✓	
Congested		
"Allergic"		
Polyps		

	YES	NO
5. MOUTH		
Good Hygiene	✓	
Dentures - Partial		
Complete		
None	✓	
6. THROAT		
Tonsils - Absent		
Normal	✓	
Infected		
7. THYROID - Normal	✓	
Enlarged		
Modular		
8. THORAX		
Chest - Symmetrical	✓	
Shape - Normal	✓	
Breath Sounds - Normal	✓	
Rales (Type and Location)		✓

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
9. BREASTS				
Normal	✓		✓	
Tumor		✓		✓
Other				

	SITTING RIGHT	SITTING LEFT
10. CIRCULATORY SYSTEM		
Blood Pressure		
Systolic	130	
Diastolic	90	

	YES	NO
Apex within M.C.L.	✓	
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal	✓	
Forceful		
Distant		
Murmurs		✓
Peripheral Vessels - Normal	✓	

	YES	NO
11. SPINE		
Cervical - Normal	✓	
Thoracic - Normal	✓	
Lumbar - Normal	✓	
Sacral - Normal	✓	
12. ABDOMEN		
Scars		✓
Masses		✓
Organmegaly		✓
13. INGUINAL REGION		
Hernia		✓
Genitalia - Normal	✓	
Scrotal contents - Normal	✓	
14. RECTUM		
Ext. Hemorrhoids		✓
Int. Hemorrhoids		✓
Masses		✓
Prostate - Normal	✓	
15. EXTREMITIES - NORMAL	✓	
Describe deformity or mal function including paras	WNL	
16. MISCELLANEOUS		
Adenopathy -		
Locate		
Varicosities -		
Locate		
Etc.		
17. OTHER FINDINGS		
Pelvic - If indicated		

LIST ALL POSITIVE FINDINGS OR ANY DIAGNOSIS INDICATED FROM ANSWERS TO THE ABOVE OR PREVIOUS PAGE.

NAME: C.L. Williams Age 42

S.S. NO.:

BP 130/90

CHEM. SCREEN: 5-7-92 — on Premarin MRP

LABSTIX — Hx CA?, Hypertension - on Inderal - Benity

HEIGHT: 69

WEIGHT: 238 3/4 — Em M Hyd 5-21-92

AUDIO SCREEN 5-7-92 Wnl B.

VISUAL SCREEN — C.L. Williams

HISTORY COMPLETED

SU-04 11/84

Physician's Examination and Consultation

1. GENERAL	YES	NO
Pallor		1
Cyanosis		1
Jaundice		1
Skin Disease		1

2. EYES	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Pupils - Regular	1		1	
Equal	1		1	
React to Light	1		1	
React to Acc	1		1	
Exophthalmos		1		1
Nystagmus		1		1
Strabismus		1		1
Cataract		1		1
Conjunctiva - Normal	1		1	
Congested				
Icteric				
Cornea - Normal	1		1	
Arcus senilis				
Ulcers				
Retina - Normal	1		1	
Hemorrhages				
Exudates				
Disc edema				
Nicking				

3. EARS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Discharge		1		1
Drum intact	1		1	
Canals normal	1		1	

4. NOSE	YES	NO
Perforation of Septum		1
Mucous Membranes		
Normal	1	
Congested		
"Allergic"		
Polyps		

5. MOUTH	YES	NO
Good Hygiene	1	
Dentures - Partial		
Complete		
None	1	

6. THROAT	YES	NO
Tonsils - Absent		
Normal	1	
Infected		

7. THYROID - Normal	YES	NO
Enlarged		
Modular		

8. THORAX	YES	NO
Chest - Symmetrical	1	
Shape - Normal	1	
Breath Sounds - Normal	1	
Rales (Type and Location)		1

9. BREASTS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Normal				
Tumor		1		1
Other				

10. CIRCULATORY SYSTEM	SITTING RIGHT	SITTING LEFT
Blood Pressure		
Systolic	130	
Diastolic	58	

	YES	NO
Apex within M.C.L.	1	
Rhythm - Normal Sinus	1	
Heart Sounds - Normal	1	
Forceful		
Distant		
Murmurs		1
Peripheral Vessels - Normal	1	

11. SPINE	YES	NO
Cervical - Normal	1	
Thoracic - Normal	1	
Lumbar - Normal	1	
Sacral - Normal	1	

12. ABDOMEN	YES	NO
Scars		1
Masses		1
Organomegly		1

13. INGUINAL REGION	YES	NO
Hernia		1
Genitalia - Normal	1	
Scrotal contents - Normal	1	

14. RECTUM	YES	NO
Ext. Hemorrhoids		1
Int. Hemorrhoids		1
Masses		1
Prostate - Normal	1	

15. EXTREMITIES - NORMAL	YES	NO
Describe deformity or anal function including neuro	WNL	

16. MISCELLANEOUS	YES	NO
Adenopathy - Locate		
Varicosities - Locate		
Etc. -		

17. OTHER FINDINGS	YES	NO
Pelvic - If indicated		

SU-64 11/84

Physician's Examination and Consultation

	YES	NO
1. GENERAL		
Pallor		✓
Cyanosis		
Jaundice		
Skin Disease		✓

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
2. EYES				
Pupils - Regular	✓		✓	
Equal				
React to Light				
React to Acc	✓		✓	
Exophthalmus		✓		✓
Nystagmus				
Strabismus				
Cataract	✓		✓	
Conjunctiva - Normal	✓		✓	
Congested				
Icteric				
Cornea - Normal	✓		✓	
Arcus senilis				
Ulcers				
Retina - Normal	✓		✓	
Hemorrhages				
Exudates				
Disc edema				
Nicking				

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
3. EARS				
Discharge		✓		✓
Drum intact	✓		✓	
Canals normal	✓		✓	

	YES	NO
4. NOSE		
Perforation of Septum		✓
Mucous Membranes		
Normal	✓	
Congested		
"Allergic"		
Polyps		

	YES	NO
5. MOUTH		
Good Hygiene	✓	
Dentures - Partial		
Complete		
None	✓	
6. THROAT		
Tonsils - Absent		
Normal	✓	
Infected		
7. THYROID Normal	✓	
Enlarged		
Modular		
8. THORAX		
Chest - Symmetrical	✓	
Shape - Normal	✓	
Breath Sounds - Normal	✓	
Rales (Type and Location)		✓

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
9. BREASTS				
Normal	✓		✓	
Tumor		✓		✓
Other				

	SITTING RIGHT	SITTING LEFT
10. CIRCULATORY SYSTEM		
Blood Pressure		
Systolic	130	
Diastolic	90	

	YES	NO
Apex within M.C.L.	✓	
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal	✓	
Forceful		
Distant		
Murmurs		✓
Peripheral Vessels - Normal	✓	

	YES	NO
11. SPINE		
Cervical - Normal	✓	
Thoracic - Normal	✓	
Lumbar - Normal	✓	
Sacral - Normal	✓	
12. ABDOMEN		
Scars		
Masses		✓
Organmegly		
13. INGUINAL REGION		
Hernia		✓
Genitalia - Normal	✓	
Scrotal contents - Normal	✓	
14. RECTUM		
Ext. Hemorrhoids		✓
Int. Hemorrhoids		✓
Masses		✓
Prostate - Normal	✓	
15. EXTREMITIES - NORMAL		
Describe deformity or mal function including neuro	CMB	
16. MISCELLANEOUS		
Adenopathy -		
Locate		
Varicosities -		
Locate		
Etc.		
17. OTHER FINDINGS		
Pelvic - If Indicated		

LIST ALL POSITIVE FINDINGS OR ANY DIAGNOSIS INDICATED FROM ANSWERS TO THE ABOVE OR PREVIOUS PAGE.

NAME: C.L. Williams Age 44

SS NO.: 422-30-7090

CHEM. SCREEN: 5-12-93

LABSTIX

HEIGHT: 68 1/2

WEIGHT: 223 1/2

AUDIO SCREEN: OK

VISUAL SCREEN: OK

HISTORY COMPLETED

Hx CAT
Hx Hypertension
on Medication PRN

Ruth Boyd 6-3-93

C.L. Williams

SU-04 11/84

Physician's Examination and Consultation

1. GENERAL	YES	NO
Pallor		✓
Cyanosis		✓
Jaundice		✓
Skin Disease		✓

2. EYES	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Pupils - Regular	✓		✓	
Equal				
React to Light	✓		✓	
React to Acc.	✓		✓	
Exophthalmus		✓		✓
Nystagmus		✓		✓
Strabismus		✓		✓
Cataract	✓		✓	
Conjunctiva - Normal	✓		✓	
Congested				
Icteric				
Cornea - Normal	✓		✓	
Arcus senilis				
Ulcers				
Retina - Normal	✓		✓	
Hemorrhages				
Exudates				
Disc edema				
Nicking				

3. EARS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Discharge		✓		✓
Drum loose		✓		✓
Canals normal	✓		✓	

4. NOSE	YES	NO
Perforation of Septum		✓
Mucous Membranes Normal	✓	
Congested		
"Allergic"		
Polyps		

5. MOUTH	YES	NO
Good Hygiene	✓	
Dentures - Partial		
Complete		
None	✓	

6. THROAT		
Tonsils - Absent		
Normal	✓	✓
Inflamed		

7. THYROID - Normal	✓	
Enlarged		
Nodule		

8. THORAX		
Chest - Symmetrical	✓	
Shape - Normal	✓	
Breath Sounds - Normal	✓	
Rales (Type and Location)		✓

9. BREASTS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Normal	✓			✓
Tumor		✓		✓
Other				

10. CIRCULATORY SYSTEM	SITTING RIGHT	SITTING LEFT
Blood Pressure - Systolic	130	
Diastolic	90	

	YES	NO
Apex within M.C.L.	✓	
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal		
Forceful		
Thrills		
Murmurs		✓
Pheripheral Vessels - Normal	✓	

11. SPINE	YES	NO
Cervical - Normal	✓	
Thoracic - Normal		
Lumbar - Normal		
Sacral - Normal	✓	

12. ABDOMEN		
Scars		
Masses		✓
Organomegaly		

13. INGUINAL REGION		
Hernia		
Genitalia - Normal	✓	
Scrotal contents - Normal	✓	

14. RECTUM		
Ext. Hemorrhoids		✓
Int. Hemorrhoids		✓
Masses		✓
Prostate - Normal	✓	

15. EXTREMITIES - NORMAL	
Describe deformity or mal function including neuro.	WNL

16. MISCELLANEOUS	
Adenopathy - Locate	
Varicosities - Locate	
Etc.	

17. OTHER FINDINGS	
Pelvic - If indicated	

SL-04 11/84

Physician's Examination and Consultation

	YES	NO
1. GENERAL		
Pallor		
Cyanosis		
Jaundice		
Skin Disease		

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
2. EYES				
Pupils - Regular	✓		✓	
Equal	✓		✓	
React to Light	✓		✓	
React to Acc	✓		✓	
Exophthalmus		✓		✓
Nystagmus		✓		✓
Strabismus		✓		✓
Cataract		✓		✓
Conjunctiva - Normal	✓		✓	
Congested				
Icteric				
Cornea - Normal	✓		✓	
Arcus senilis				
Ulcers				
Retina - Normal	✓		✓	
Hemorrhages				
Exudates				
Disc edema				
Nicking				

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
3. EARS				
Discharge		✓		✓
Drum Intact	✓		✓	
Canals normal	✓		✓	

	YES	NO
4. NOSE		
Perforation of Septum		
Mucous Membranes Normal		✓
Congested		
"Allergic"		
Polyps		

	YES	NO
5. MOUTH		
Good Hygiene	✓	
Dentures - Partial		
Complete		
None	✓	

	YES	NO
6. THROAT		
Tonsils - Absent		
Normal	✓	
Infected		
7. THYROID - Normal	✓	
Enlarged		
Nodular		
8. THORAX		
Chest - Symmetrical	✓	
Shape - Normal	✓	
Breath Sounds - Normal	✓	
Rales (Type and Location)		✓

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
9. BREASTS				
Normal	✓		✓	
Tumor		✓		✓
Other				

	SITTING RIGHT	SITTING LEFT
10. CIRCULATORY SYSTEM		
Blood Pressure - Systolic	120	
Diastolic	90	

	YES	NO
Apex within M.C.L.	✓	
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal	✓	
Forceful		
Distant		
Murmurs		✓
Peripheral Vessels - Normal	✓	

	YES	NO
11. SPINE		
Cervical - Normal	✓	
Thoracic - Normal	✓	
Lumbar - Normal	✓	
Sacral - Normal	✓	
12. ABDOMEN		
Scars		✓
Masses		
Organmegly		✓
13. INGUINAL REGION		
Hernia		✓
Genitalia - Normal	✓	
Scrotal contents - Normal	✓	
14. RECTUM		
Ext. Hemorrhoids		✓
Int. Hemorrhoids		✓
Masses		✓
Prostate - Normal	✓	
15. EXTREMITIES - NORMAL	WNL	
Describe deformity or mal function including neuro		
16. MISCELLANEOUS		
Adenopathy - Locate		
Varicosities - Locate		
Etc.		
17. OTHER FINDINGS		
Pelvic - If indicated		

LIST ALL POSITIVE FINDINGS OR ANY DIAGNOSIS INDICATED FROM ANSWERS TO THE ABOVE OR PREVIOUS PAGE.

NAME: C L Williams Age 45

S.S. NO.:

BP 120/90

CHEM. SCREEN: 4-29-94 Hx. CAT

LABSTIX Hx Hypertension

Parents Stroke

HEIGHT: 69 1/2"

WEIGHT: 224 1/2 Medication? ER Dept 5-19-94

AUDIO SCREEN 4-29-94 OK

VISUAL SCREEN 4-29-94 20/33

HISTORY COMPLETED X C L Williams

SU-04 11/64

Physician's Examination and Consultation

1. GENERAL	YES	NO
Pallor		
Cyanosis		
Jaundice		
Skin Disease		

2. EYES	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Pupils - Regular				
Equal				
React to Light				
React to Acc				
Exophthalmos				
Nystagmus				
Strabismus				
Cataract				
Conjunctivae - Normal				
Congested				
Icteric				
Cornea - Normal				
Arcus senilis				
Ulcers				
Retina - Normal				
Hemorrhages				
Exudates				
Disc edema				
Nicking				

3. EARS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Discharge				
Drum intact				
Canals normal				

4. NOSE	YES	NO
Perforation of Septum		
Mucous Membranes		
Normal		
Congested		
"Allergic"		
Polyps		

5. MOUTH	YES	NO
Good Hygiene		
Dentures - Partial		
Complete		
None		

6. THROAT	YES	NO
Tonsils - Absent		
Normal		
Inflamed		

7. THYROID - Normal	YES	NO
Enlarged		
Modular		

8. THORAX	YES	NO
Chest - Symmetrical		
Shape - Normal		
Breath Sounds - Normal		
Rales (Type and Location)		

9. BREASTS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Normal				
Tumor				
Other				

10. CIRCULATORY SYSTEM	SITTING RIGHT	SITTING LEFT
Blood Pressure		
Systolic	120	
Diastolic	90	

	YES	NO
Apex within M.C.L.		
Rhythm - Normal Sinus		
Heart Sounds - Normal		
Forceful		
Distant		
Murmurs		
Peripheral Vessels - Normal		

11. SPINE	YES	NO
Cervical - Normal		
Thoracic - Normal		
Lumbar - Normal		
Sacral - Normal		

12. ABDOMEN	YES	NO
Scars		
Masses		
Organomegaly		

13. INGUINAL REGION	YES	NO
Hernia		
Genitalia - Normal		
Scrotal contents - Normal		

14. RECTUM	YES	NO
Ext. Hemorrhoids		
Int. Hemorrhoids		
Masses		
Prostate - Normal		

15. EXTREMITIES - NORMAL	YES	NO
Describe deformity or mal function including neuro		

16. MISCELLANEOUS	YES	NO
Adenopathy - Locate		
Varicosities - Locate		
Etc.		

17. OTHER FINDINGS	YES	NO
Pelvic - If indicated		

SU-04 11/84

Physician's Examination and Consultation

	YES	NO
1. GENERAL		
Pallor		✓
Cyanosis		✓
Jaundice		✓
Skin Disease		✓

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
2. EYES				
Pupils - Regular	✓		✓	
Equal	✓			✓
React to Light	✓		✓	
React to Acc	✓		✓	
Exophthalmos		✓		✓
Nystagmus		✓		✓
Strabismus		✓		✓
Cataract		✓		✓
Conjunctiva - Normal	✓		✓	
Congested				
Jaundice				
Cornea - Normal	✓		✓	
Arcus senilis				
Ulcers				
Retina - Normal	✓		✓	
Hemorrhages				
Exudates				
Disc edema				
Nicking				

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
3. EARS				
Discharge		✓		✓
Drum intact	✓		✓	
Canals normal	✓		✓	

	YES	NO
4. NOSE		
Perforation of Septum		✓
Mucous Membranes Normal	✓	
Congested		
"Allergic"		
Polyps		

	YES	NO
5. MOUTH		
Good Hygiene	✓	
Dentures - Partial		
Complete		
None	✓	
6. THROAT		
Tonsils - Absent		
Normal	✓	
Infected		
7. THYROID - Normal	✓	
Enlarged		
Nodular		
8. THORAX		
Chest - Symmetrical	✓	
Shape - Normal	✓	
Breath Sounds - Normal	✓	
Rales (Type and Location)		

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
9. BREASTS				
Normal	✓		✓	
Tumor		✓		✓
Other		✓		✓

	SITTING RIGHT	SITTING LEFT
10. CIRCULATORY SYSTEM		
Blood Pressure Systolic		140/84
Diastolic		

	YES	NO
Apex within M.C.L.	✓	
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal	✓	
Forceful		
Distant		
Murmurs		✓
Peripheral Vessels - Normal	✓	

	YES	NO
11. SPINE		
Cervical - Normal	✓	
Thoracic - Normal	✓	
Lumbar - Normal	✓	
Sacral - Normal	✓	
12. ABDOMEN		
Scars		✓
Masses	Hepatomegaly	
Organomegaly		✓
13. INGUINAL REGION		
Hernia		✓
Genitalia - Normal	✓	
Scrotal contents - Normal	✓	
14. RECTUM		
Ext. Hemorrhoids		—
Int. Hemorrhoids		—
Masses		—
Prostate - Normal	✓	
15. EXTREMITIES - NORMAL	✓	
Describe deformity or mal function including reason		
16. MISCELLANEOUS		
Adenopathy - Locate		
Varicosities - Locate		
Etc.		
17. OTHER FINDINGS		
Pelvic - If indicated		

LIST ALL POSITIVE FINDINGS OR ANY DIAGNOSIS INDICATED FROM ANSWERS TO THE ABOVE OR PREVIOUS PAGE.

NAME: C.L. Williams (/ 0 47)

S.S. NO.:

BP: 142/84

CHEM. SCREEN: 5-14-96

HX CA?. - heart pill, fluid pills

LABSTIX

HEIGHT: 69½

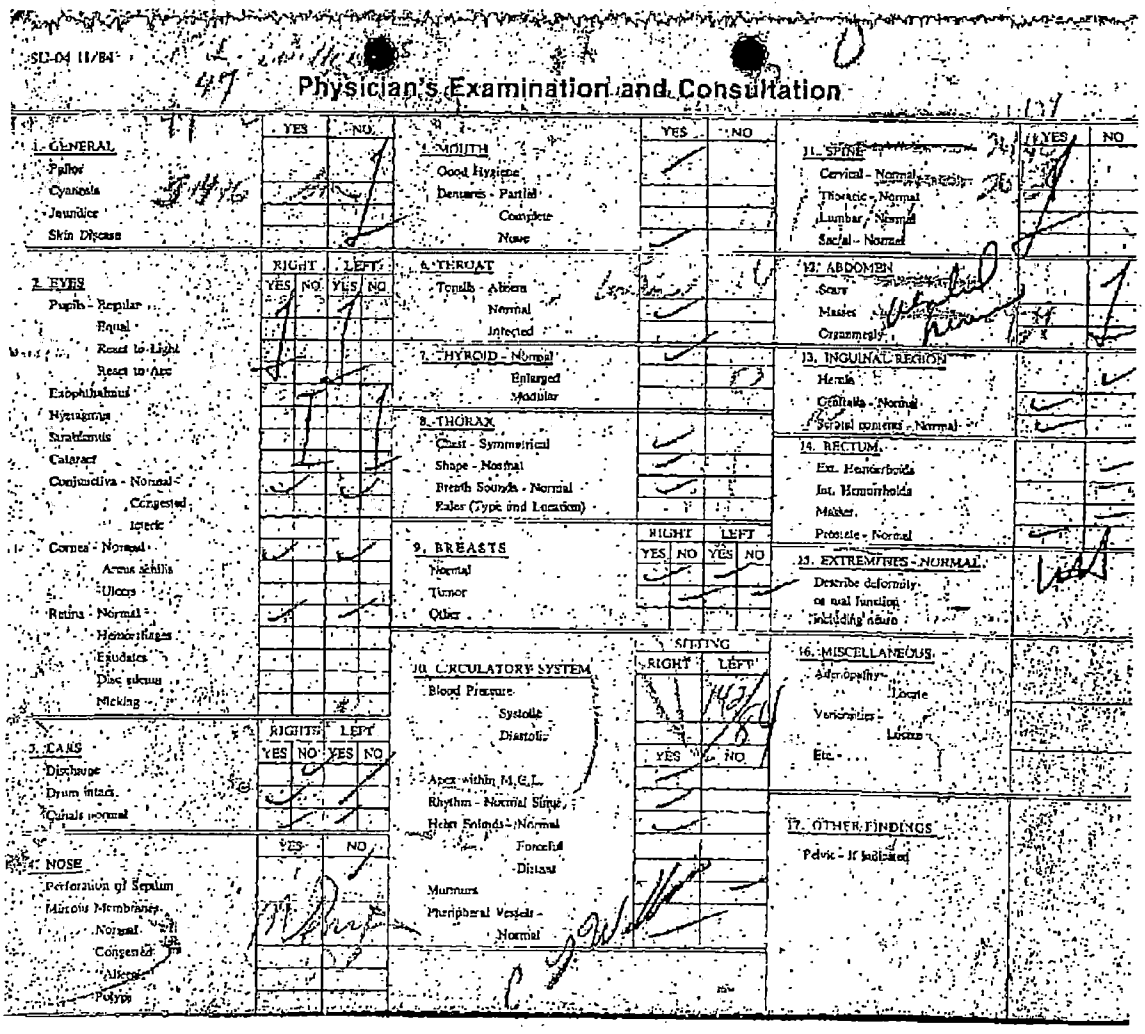
WEIGHT: 234½

Lucille Ford 5-21-96.

AUDIO SCREEN 5-14-96 Adj.

VISUAL SCREEN 5-14-96 20/29

HISTORY COMPLETED



SLI-04 11/84

Physician's Examination and Consultation

1. GENERAL — YES / NO
- Pallor
- Cyanosis
- Jaundice
- Skin Disease

2. EYES — RIGHT (YES / NO) / LEFT (YES / NO)
- Pupils - Regular
- Equal
- React to Light
- React to Acc
- Exophthalmus
- Hypertropia
- Strabismus
- Cataract
- Conjunctiva - Normal
- Congested
- Icteric
- Cornea - Normal
- Arcus senilis
- Ulcers
- Retina - Normal
- Hemorrhages
- Exudates
- Disc edema
- Micking

3. EARS — RIGHT (YES / NO) / LEFT (YES / NO)
- Discharge
- Drum intact
- Canals normal

4. NOSE — YES / NO
- Perforation of Septum
- Mucous Membranes - Normal
- Congested
- Atrophic
- Polyps

5. MOUTH — YES / NO
- Good Hygiene
- Dentures - Partial
- Complete
- None

6. THROAT
- Tonsils - Absent
- Normal
- Infected

7. THYROID - Normal
- Enlarged
- Nodular

8. THORAX
- Chest - Symmetrical
- Shape - Normal
- Breath Sounds - Normal
- Rales (Type and Location)

9. BREASTS — RIGHT (YES / NO) / LEFT (YES / NO)
- Normal
- Tumor
- Other

10. CIRCULATORY SYSTEM
- Blood Pressure — SITTING: RIGHT / LEFT
- Systolic
- Diastolic
- Apex within M.C.L. — YES / NO
- Rhythm - Normal Sinus
- Heart Sounds - Normal
- Forceful
- Distant
- Murmurs
- Peripheral Vessels - Normal

11. SPINE — YES / NO
- Cervical - Normal
- Thoracic - Normal
- Lumbar - Normal
- Sacral - Normal

12. ABDOMEN
- Scars
- Masses
- Organomegaly

13. INGUINAL REGION
- Hernia
- Genitalia - Normal
- Scrotal contents - Normal

14. RECTUM
- Ext. Hemorrhoids
- Int. Hemorrhoids
- Masses
- Prostate - Normal

15. EXTREMITIES - NORMAL
- Describe deformity or nail function including edema

16. MISCELLANEOUS
- Adenopathy
- Locate
- Varicosities
- Locate
- Etc.

17. OTHER FINDINGS
- Pelvic - If indicated

SU-04 11/84

Physician's Examination and Consultation

	YES	NO
1. GENERAL		
Pallor		✓
Cyanosis		✓
Jaundice		✓
Skin Disease		✓

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
2. EYES				
Pupils - Regular	✓		✓	
Equal	✓		✓	
React to Light	✓		✓	
React to Acc	✓		✓	
Exophthalmos		✓		✓
Nystagmus		✓		✓
Strabismus		✓		✓
Cataract		✓		✓
Conjunctiva - Normal	✓		✓	
Congested				
Icteric				
Cornea - Normal	✓		✓	
Arcus senilis				
Ulcers				
Retina - Normal	✓		✓	
Hemorrhages				
Exudates				
Disc edema				
Nicking				

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
3. EARS				
Discharge		✓		✓
Drum intact	✓		✓	
Canals normal	✓		✓	

	YES	NO
4. NOSE		
Perforation of Septum		✓
Mucous Membranes		
Normal	✓	
Congested		
"Allergic"		
Polyps		

	YES	NO
5. MOUTH		
Good Hygiene	✓	
Dentures - Partial		
Complete		
None	✓	
6. THROAT		
Tonsils - Absent		
Normal	✓	
Infected		
7. THYROID - Normal	✓	
Enlarged		
Modular		
8. THORAX		
Chest - Symmetrical	✓	
Shape - Normal	✓	
Breath Sounds - Normal	✓	
Rales (Type and Location)		

	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
9. BREASTS				
Normal	✓		✓	
Tumor		✓		✓
Other				

	SITTING RIGHT	SITTING LEFT
10. CIRCULATORY SYSTEM		
Blood Pressure		
Systolic		
Diastolic		

	YES	NO
Apex within M.C.L.	✓	
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal	✓	
Forceful		
Distant		
Murmurs		✓
Peripheral Vessels - Normal	✓	

	YES	NO
11. SPINE		
Cervical - Normal	✓	
Thoracic - Normal		
Lumbar - Normal	✓	
Sacral - Normal		
12. ABDOMEN		✓
Scars		
Masses		✓
Organomegly		
13. INGUINAL REGION	✓	
Hernia		✓
Genitalia - Normal	✓	
Sexual patients - Normal		✓
14. RECTUM		✓
Ext. Hemorrhoids		✓
Int. Hemorrhoids		✓
Masses		✓
Prostate - Normal		✓
15. EXTREMITIES - NORMAL	✓	
Describe deformity or past function including neuro		
16. MISCELLANEOUS		
Adenopathy - Locate		
Varicosities - Locate		
Etc.		
17. OTHER FINDINGS		
Pelvic - If indicated		

LIST ALL POSITIVE FINDINGS OR ANY DIAGNOSIS INDICATED FROM ANSWERS TO THE ABOVE OR PREVIOUS PAGE.

NAME: C.L. Williams Age 48

S.S. NO.

BP 112/84

CHEM. SCREEN: 4-17-97

LABSTIX

HEIGHT: 164½

WEIGHT: 239

AUDIO SCREEN: 4-17-97 Oh.

VISUAL SCREEN: 4-17-97 Oh.

HISTORY COMPLETED

1) Hx Hypertension
Rx - CAF.

Rec'd Appl 5-5-97

SU-04 11/84

Physician's Examination and Consultation

1. GENERAL	YES	NO
Pallor		✓
Cyanosis		✓
Jaundice		✓
Skin Disease		✓

2. MOUTH	YES	NO
Good Hygiene	✓	
Dentures - Partial		
Complete		
None	✓	

11. SPINE	YES	NO
Cervical - Normal	✓	
Thoracic - Normal		
Lumbar - Normal		
Sacral - Normal	✓	

7. EYES	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Pupils - Regular	✓		✓	
Equal	✓		✓	
React to Light	✓		✓	
React to Acc	✓		✓	
Exophthalmus		✓		✓
Nystagmus		✓		✓
Strabismus		✓		✓
Cataract		✓		✓
Conjunctiva - Normal	✓		✓	
Congested				
Icteric				
Cornea - Normal	✓		✓	
Arcus senilis				
Ulcers				
Retina - Normal	✓		✓	
Hemorrhages		✓		✓
Exudates		✓		✓
Disc edema		✓		✓
Nicking		✓		✓

6. THROAT	YES	NO
Tonsils - Absent		
Normal	✓	
Infected		

12. ABDOMEN	YES	NO
Scar		✓
Masses		✓
Organmegly		✓

7. THYROID - Normal	YES	NO
Enlarged		
Nodular		

13. INGUINAL REGION	YES	NO
Hernia		✓
Genitalia - Normal	✓	
Scrotal contents - Normal	✓	

8. THORAX	YES	NO
Chest - Symmetrical	✓	
Shape - Normal	✓	
Breath Sounds - Normal	✓	
Rales (Type and Location)		

14. RECTUM	YES	NO
Ext. Hemorrhoids		✓
Int. Hemorrhoids		✓
Masses		✓
Prostate - Normal		✓

9. BREASTS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Normal	✓		✓	
Tumor		✓		✓
Other				

15. EXTREMITIES - NORMAL	YES	NO
Describe deformity or mal function including scars		

10. CIRCULATORY SYSTEM	SITTING RIGHT	SITTING LEFT
Blood Pressure		
Systolic		
Diastolic		

	YES	NO
Apex within M.C.L.	✓	
Rhythm - Normal Sinus	✓	
Heart Sounds - Normal	✓	
Forceful		
Distant		
Murmurs		✓
Pheripheral Vessels - Normal	✓	

16. MISCELLANEOUS	YES	NO
Adenopathy -		
Locate		
Varicosities -		
Locate		
Eye:		

3. EARS	RIGHT YES	RIGHT NO	LEFT YES	LEFT NO
Discharge		✓		✓
Drum intact	✓		✓	
Canals normal	✓		✓	

4. NOSE	YES	NO
Perforation of Septum		✓
Mucous Membranes - Normal	✓	
Congested		
"Allergic"		
Polyps		

17. OTHER FINDINGS	YES	NO
Pelvic - If indicated		

The Precertification Center®

Health Benefits Management, Inc.

The Precertification Center®
P.O. Box 898125
Camp Hill, PA 17089-8125

DIS

DISABILITY CLAIM FORM

Name (Please print) C.L. Williams	Social Security Number	Employee Number 2150
Home Address 55 MAXWELL AVE SUMTER, SC 29150	Sex MALE ✓ FEMALE / Date of Birth	Group Number
Employer's Name and Address YUASA EXIDE, INC PO BOX 1197 SUMTER, SC 29150		
Physician's Name and Address DR MARION DAVIS PINEWOOD, SC 29153		If Accident, Date of Accident N/A
Physician's Phone Number (803) 452-5151		Was This Job Related YES / NO ✓
		Date of Surgery, If Applicable N/A
Type of Sickness or Injury Heart Trouble		If Pregnancy, Expected Date of Delivery N/A
If Hospitalized, Admission and Discharge Dates: Admission Date 2.10.98	Discharge Date 2.12.98	
Date First Seen By Any Doctor For This Condition 2/10/98	First Date Unable To Work Due To This Condition 2/10/98	Estimated Date Of Return To Work UNKNOWN

Employee Status

☐ Exempt ☐ Nonexempt ☑ Fulltime ☐ Parttime

Activities Involved In Your Job (Check all that apply)

- ___ Desk Job - If this applies, are you able to leave your desk as needed? Yes ___ No ___
- ✓ Walking (Most of the time)
- ✓ Lifting
- ✓ Bending
- ___ Typing or Operating a CRT
- ✓ Standing (Most of the time)
- ✓ Moving equipment, furniture, etc.
- ___ Driving a vehicle
- ✓ Operating Equipment
- ✓ Chemical Exposure
- ___ Other (Give a brief description) ___

I hereby authorize The Precertification Center® to obtain any medical information necessary to process this claim.

Signature C. L. Williams Date 2/16/98

1-800-441-9950 1-800-626-5050 FAX

2918 C 6/93

RECD 3/19/98

SUMTER MEDICAL CONSULTANTS, P.A.

250 W. WESMARK BLVD.
SUMTER, SOUTH CAROLINA 29150
Phone: 803/469-4011
Fax: 803/469-4192

Cardiovascular Disease
W. STRAT STAVROU, M.D., F.A.C.C., F.C.C.P.
J. DALE CANNON, JR., M.D., F.A.C.C.
C. WEST JACOCKS, IV, M.D., F.A.C.C.

Pulmonary Disease
CHARLES R. WHITE, JR., M.D., F.C.C.P.

Gastrointestinal Disease
T.D. WILLIAMS, III, M.D., F.A.C.G.
KENT N. CUNNINGHAM, M.D.
SCOTT R. McDUFFIE, M.D.

March 13, 1998

Ms. Heather Brown
Asst. Personnel Director
YUASA/EXIDE
Industrial Park
Sumter, S. C. 28150

Re: Mr. C. L. Williams
Our Record #: 17551

Dear Mr. Brown:

Mr. C. L. Williams is a patient of mine who has severe depressed left ventricular function and is unable to do heavy, repetitive labor. If other work could be found for him that does not involve repeated heavy lifting, I think he would be able to continue working.

Sincerely,

C. West Jacocks, IV, M.D., F.A.C.C.

CWJ/jkb

08/12/98 Yuasa-Exide, Inc. PAGE 1
M LP111 PERSONAL EMPLOYEE DATA ID: 28883

CLOCK# : 2150
NAME :C.L. Williams
HIRE DATE :03/12/76
TELEPHONE :773-9461

ADDRESS: 55 Maxwell Street
Sumter,SC 29150
SS# :
BADGE# :

CURRENT JOB STATUS

DEPT : 10
JOBTITLE :S&A
GRADE : 6
WAGE : 9.93
SHIFT :

PAYROLL PREMIUM:
* 0 -OOO/1ST
* 02-OOO/2ND
* 03-OOO/3RD

TRANSFERS- Permanent / Temporary

DEPT :
JOBTITLE :
GRADE :
WAGE :
SHIFT/PREM:

DIRECT / INDIRECT
INCENTIVE / NONINCENTIVE

Work Instruction Required:

___ BEREAVEMENT
___ VACATION
___ EXCUSED ABSENCE
___ REPLACEMENT
___ POLLING
✓ TERMINATED-ACCRUED VACATION HRS ___

___ MILITARY (Attach Pay Voucher)
___ JURY (Attach pay Voucher)
___ OTHER: _____
___ BIDDING

EFFECTIVE DATE(S): 8/12/98

COMMENTS: Employee terminated due to health related problems & is receiving DD & disability pension from the Co. is being processed (Kathy S. Brown)

SUP./MGR. _____ H.R. APPROVAL

DISTRIBUTION: ACCOUNTING ✓ SUPERVISOR ___ HUMAN RESOURCES ✓ MEDICAL ✓
QUALITY CONTROL ✓

P-11
11/96

PERIODIC HEALTH HISTORY

NOTE: This form is to be used only if employee has previously completed a Baseline Health History.

Please Print

Date: 5-24-93 Location of Examination: Sumter (City) SC (State)

Name: Williams (Last) CL (First) ____ (MI) Soc. Sec. #

Birthdate: ____ Sex: ☑ M ☐ F Marital Status: ☐ S ☑ M ☐ W ☐ D

Address: 55 Maxwell Ave Telephone: ____
Sumter SC

In case of emergency, notify:

Name: Katie G. Williams Telephone: 773-9461

Address: 55 Maxwell Ave
Sumter, SC

Family Doctor:

Name: ____ Telephone: ____

Address: ____

☑ I have no Family Doctor

FAMILY HISTORY:

YES	NO	UNKNOWN	
☐	☑	☐	Is your father living?
☐	☑	☐	Is your mother living?

MEDICAL HISTORY:

Since your last exam, have you:

YES	NO	
☑	☐	Been treated by a doctor, hospital or clinic,
☐	☑	Had surgery
☐	☑	Taken regular medicine or drugs

Is your health right now: ☐ Poor ☑ Good ☐ Excellent

In the past year, have you: ☐ Stayed the same weight ☑ Gained 5 or more pounds ☐ Lost 5 or more pounds

YES	NO	
☐	☑	Do you have any allergies?
☑	☐	Since your last exam, have you smoked any cigarettes?
☑	☐	Do you now smoke cigarettes? How many packs 2 per day?

How many drinks each week do you have? ____Liquor ____Beer ____Wine

YES	NO	
☐	☐	Since your last exam, has anyone ever considered you to be a heavy drinker?

Since your last exam, has your appetite: ☐ Increased ☐ Decreased ☐ Remained the same

YES	NO			
☐	☑	Do you drink coffee? If yes, cups per day:	____Decaf	____Caff
☐	☑	Do you drink tea? If yes, cups per day:	____Decaf	____Caff

5 or 6 How many hours sleep do you get each night?

YES	NO	
☐	☑	Do you have trouble sleeping?
☐	☑	Have your sleep habits changed in the past year?

Since your last exam, have you had or experienced:

Emotional

YES NO
- ☐ ☑ Frequent tension, can't relax
- ☐ ☑ Panic spells
- ☐ ☑ Dizziness or light headedness
- ☐ ☑ Frequent depression
- ☐ ☑ Frequent anger
- ☐ ☑ Other nervous problems

Eyes
- ☐ ☑ Not seeing properly
- ☐ ☑ Cataract removal
- ☐ ☑ Seeing double
- ☑ ☐ Red or runny eyes
- ☑ ☐ Pain in eyes
- ☐ ☑ Halo around lights
- ☐ ☑ Itchy eyelids
- ☐ ☑ Other eye trouble
- ☐ ☑ Contact lenses
- ☐ ☑ Eyeglasses
- ☐ ☑ Bifocals or trifocals
- ☐ ☑ Glaucoma

Nose, Ears and Hearing
- ☐ ☑ Difficulty in hearing
- ☐ ☑ Ringing or buzzing in ears
- ☐ ☑ More than one ear infection
- ☐ ☑ More than one earache
- ☐ ☑ Other ear trouble
- ☐ ☑ Sinus trouble
- ☐ ☑ Frequent colds or flu
- ☐ ☑ Frequent nosebleeds
- ☐ ☑ Loss of sense of smell
- ☐ ☑ Chronic drainage in nose or throat
- ☐ ☑ Other nose or sinus trouble

Mouth
- ☐ ☑ Sores on lips, tongue or mouth that won't heal
- ☐ ☑ Lumps on lips, tongue, mouth
- ☐ ☑ Frequent mouth sores
- ☐ ☑ Bleeding gums or mouth
- ☐ ☑ Other mouth trouble
- ☐ ☑ Wear full dentures
- ☐ ☑ Wear partial dentures
- ☐ ☑ Need teeth repair or dental work
- ☐ ☑ Need other attention to teeth or gums

Throat and Neck
- ☐ ☑ Frequent hoarseness
- ☐ ☑ Hard to swallow
- ☐ ☑ Frequent sore throat
- ☐ ☑ Frequent laryngitis
- ☐ ☑ Other throat trouble
- ☐ ☑ Lumps in neck
- ☐ ☑ Painful or tender spots in neck
- ☐ ☑ Frequent stiff neck
- ☐ ☑ Crunching or grinding noise in neck
- ☐ ☑ Other neck trouble
- ☐ ☑ Thyroid problems

YES NO
- ☐ ☑ Tuberculosis
- ☐ ☑ Asthma
- ☐ ☐ Emphysema
- ☑ ☐ Cough up phlegm
- ☐ ☑ Bronchitis
- ☑ ☐ Cough up phlegm in mornings at least once a week
- ☐ ☑ Tightness in chest
- ☐ ☑ Shortness of breath with heavy exertion
- ☐ ☐ Shortness of breath with mild exertion
- ☐ ☐ Any other trouble with lungs or breathing

Joints and Bones
- ☐ ☐ Torn cartilage
- ☐ ☑ Arthritis
- ☐ ☑ Gout
- ☐ ☑ Bursitis
- ☑ ☐ Pain in muscles
- ☑ ☐ Neck or shoulder pain
- ☐ ☑ Pain in upper back
- ☐ ☐ Pain in lower back
- ☐ ☑ Painful joints
- ☐ ☑ Broken bone(s)
- ☐ ☑ Other joint or bone trouble

Heart
- ☐ ☑ Heart murmur
- ☐ ☐ Shortness of breath
- ☐ ☑ Swelling of ankles
- ☐ ☐ Pain or pressure in chest
- ☐ ☑ Thumping, skipping or racing heart
- ☑ ☐ High blood pressure
- ☐ ☑ Heart attack
- ☐ ☑ Chest pain which goes into your arm or neck
- ☐ ☑ Other heart trouble

Digestive System (stomach)
- ☐ ☑ Ulcer
- ☐ ☑ Gall bladder trouble
- ☐ ☑ Frequent heartburn or indigestion
- ☐ ☑ Frequent diarrhea
- ☐ ☑ Frequent stomach pain
- ☑ ☐ Blood in your stool
- ☐ ☑ Vomit up blood
- ☐ ☑ Other digestive trouble

Neurological
- ☐ ☑ Stroke
- ☐ ☑ Skull fracture
- ☐ ☑ Concussion
- ☐ ☑ Loss of memory
- ☐ ☑ Loss of consciousness
- ☐ ☑ Head injury
- ☐ ☑ Numbness, tingling or weakness in part of your body
- ☐ ☑ Epilepsy, seizure or fits
- ☑ ☐ Frequent or persistent headaches
- ☐ ☐ Migraine headaches
- ☐ ☑ Passed out
- ☐ ☑ Other nervous or neurological trouble

Lungs

YES	NO	
☒	☐	Wheezing
☒	☐	Frequent or chronic cough
☐	☒	Cough up blood
☐	☒	Pneumonia
☐	☒	Pain in chest

Skin

YES	NO	
☐	☒	Warts or moles that change in size or color
☐	☒	Skin cancer
☐	☒	Eczema
☐	☒	Psoriasis
☐	☒	Hives
☐	☒	Sunburn easily
☐	☒	Persistent rash
☐	☒	Other skin trouble

Urinary System

YES	NO	
☐	☒	Kidney or bladder trouble
☐	☒	Bladder infection (cystitis)
☐	☒	Kidney stones
☐	☒	Difficult or painful urination
☐	☒	Blood in urine
☐	☒	Venereal disease
☒	☐	Awaken at night more than once to urinate
☐	☒	Other kidney or bladder trouble

Other Illnesses

YES	NO	
☐	☒	Diabetes
☐	☒	Cancer or Leukemia
☐	☒	Anemia

Since you last exam , have you had any other illness or surgery that we have not asked about?

☐ ☒

EXPOSURE HISTORY:

Do you work with any of the following in any outside activities (part-time or second job, hobby, gardening, farming, etc.)?

YES	NO	
☐	☒	Mist from spray paints
☐	☒	Spray or dust from insecticides
☐	☒	Fumes from solvents
☐	☒	Fumes from welding
☐	☒	Asbestos
☐	☒	Chromium
☐	☒	Arsenic
☐	☒	Lead
☐	☒	Mercury
☐	☒	Color dyes
☐	☒	Silica or sand dust
☐	☒	Benzene
☐	☒	Agricultural dust, mold or mildew
☐	☒	Oil mists
☐	☒	Carbon tetrachloride
☐	☒	Tar or pitch

YES	NO	
☐	☒	Lasers
☐	☒	Ultraviolet light
☐	☒	Beryllium
☐	☒	Benzidine
☐	☒	Cotton dusts
☐	☒	Sand blasting dust
☐	☒	Fumes from degreasing
☐	☒	Solvents
☐	☒	Furniture refinishing
☐	☒	Other chemicals or dusts
☐	☒	Other exposure

REPRODUCTIVE SYSTEMS

The Company encourages you to answer the following section regarding your reproductive system to ensure that the Medical Department has a full record of your health which is indispensable for a complete medical examination. As with all questions regarding medical history, your responses will be held in confidence. You have the right not to answer the questions in this section.

Men Only

Since your last exam, have you had or experienced:

YES	NO	
☐	☒	Prostate disease or trouble
☐	☒	Persistent sore on your testicles or penis
☐	☒	Pain, swelling or lumps in testicles

Since your last exam, have you had surgery for or on:

YES	NO	
☐	☒	Your testicles
☐	☒	Prostate
☐	☒	Vasectomy
☐	☒	Since your last examination has your spouse or mate had any pregnancies or borne any children?

Women Only

Since your last exam, have you had or experienced:

YES	NO	
☐	☐	Fibroids of uterus
☐	☐	Ovarian cyst
☐	☐	Lumps in breast
☐	☐	Irregular periods
☐	☐	Unexplained vaginal bleeding or spotting
☐	☐	Unusually painful menstrual periods
☐	☐	Persistent vaginal itch
☐	☐	Persistent white or yellow vaginal discharge
☐	☐	Menopause
☐	☐	Pap smear exam
☐	☐	Persistent wart or sores of the genitals
☐	☐	Vaginal bleeding or spotting after intercourse
☐	☐	Ectopic pregnancy

Since your last exam, have you had difficulty becoming pregnant?

YES	NO	
☐	☐	☐ Does not apply

Since your last exam, have you

YES	NO	
☐	☐	Taken birth control pills?
☐	☐	Used an IUD?

Are you now pregnant?

YES	NO	
☐	☐	☐ Do not know

Since your last exam, have you had surgery for:

YES	NO	
☐	☐	Removal of uterus (hysterectomy)
☐	☐	Removal of ovaries
☐	☐	Tubal ligation (tubes tied)
☐	☐	Any related surgery not otherwise mentioned

Since your last exam, have you:

YES	NO	UNKNOWN	
☐	☐	☐	Been pregnant? Number of times _____
☐	☐	☐	Had a miscarriage? Number of times _____
☐	☐	☐	Had a surgical abortion? Number _____
☐	☐	☐	Had a child with a birth abnormality? Number _____
☐	☐	☐	Had a stillborn infant? Number _____
☐	☐	☐	Had a normal child? Number _____
☐	☐	☐	Had an ectopic pregnancy?

Signature: C. L. Williams

Occupational Health History Questionnaire

IDENTIFICATION DATA Fill in the following information. PLEASE PRINT.

Name G Williams C L (Last, First, Initial)
Social Security No. _____ Employee No. 2150
Home address 55 Maxwell Ave
Sumter SC
Home telephone 803 773-5048 (area code)

Date Oct 13 19__ (Mo. Day Year) Plant Location _____
Date of birth __ Mo. __ Day __ Year — Sex (circle) M F
X Married — Separated — Divorced — Widowed — Single
Education: 6 years Elementary — years High School
— years College, Technical, Business, etc.
Job title _____

FAMILY HISTORY: Follow the lines across the page and mark an X in those boxes which indicate the present state of health (good, poor), or the death (indicate the cause) of your mother and father and any illnesses they or your blood relatives (grandparents, brothers, sisters, children) have ever had.

	Health		Cause of death						Allergies or asthma	Anemia	Bleeding tendencies	Cancer or tumor	Diabetes	Epilepsy	Glaucoma	Hearing trouble	Heart trouble	High blood pressure	Kidney or bladder trouble	Lung disease	Nervous breakdown	Rheumatism or arthritis	Stomach/duodenal ulcer
	Good	Poor	Dead	Age at Death	Cancer	Heart Trouble	Lung Disease	Other Causes															
Father	X		X					X	X									X					
Mother	X		X					X										X					
Blood relatives (mark an X for illnesses only)																							

YOUR HEALTH HISTORY: Mark an X in the box next to any of the following illnesses you now or ever have had.

- ☐ Anemia
- ☐ Asthma
- ☐ Bleeding tendencies
- ☐ Bronchitis
- ☐ Cancer or tumor
- ☐ Diabetes
- ☐ Diverticulosis
- ☐ Emphysema
- ☐ Epilepsy
- ☐ Eye problems
- ☐ Glaucoma
- ☐ Hay fever or allergies
- ☐ Hearing trouble
- ☐ Heart trouble
- ☐ Hemorrhoids
- ☐ Hernia
- ☒ High blood pressure
- ☐ Hives or rashes
- ☐ Kidney/bladder trouble
- ☐ Liver disease/hepatitis
- ☐ Lung disease
- ☐ Malaria
- ☐ Measles
- ☒ Mononucleosis
- ☐ Mumps
- ☐ Neuralgia/neuritis
- ☐ Nervous breakdown
- ☐ Pneumonia
- ☐ Polio
- ☐ Rheumatic fever
- ☐ Rheumatism/arthritis
- ☐ Skin disease
- ☐ Venereal disease
- ☐ Yellow jaundice
- ☐ Other _____

Have you ever been turned down for life insurance, military service, or employment because of health problems? (circle) ..... Yes No

Have you ever received a blood transfusion? (circle) ..... Yes No

Major Hospitalizations: If you have ever been hospitalized for any serious medical illness or operation, write in your most recent hospitalizations below. Check this box ☐ if you have had more than three such hospitalizations.

(Do not include normal pregnancies)	Year	Reason for Hospitalization	Name of Hospital	City and State
1st hospitalization	19			
2nd hospitalization	19			
3rd hospitalization	19			

Tests: Mark an X next to those which you have had. Enter the year when you last were given the test.

Mark an X after those tests which you know had abnormal results.

	Year		Abnormal
☐	19 80	chest X-ray	X
☐	19 80	kidney X-ray	X
☐	19__	GI series	☐
☐	19__	colon X-ray	☐
☐	19__	gallbladder X-ray	☐
☐	19__	electrocardiogram	☐
☐	19__	electrocardiogram with exercise	☐
☐	19 80	TB test	X
☐	19 80	Smallpox test	X
☐	19__	biopsy	☐
☐	19 80	Hearing test	X
☐	19__	other	☐

Medicines: Mark an X in the box next to any medicines that you are now taking or that you are allergic to.

taking	allergic to:		taking	allergic to:	
☐	☐	aspirin	☐	☐	high blood pressure medicine
☐	☐	penicillin	☐	☐	Dilantin/anticonvulsants
☐	☐	sulfa	☐	☐	birth control pills
☐	☐	codeine	☐	☐	diuretics/water pills
☐	☐	antibiotics	☐	☐	blood thinners (anticoagulants)
☐	☐	sedatives	☐	☐	cortisone-type drugs
☐	☐	stimulants	☐	☐	insulin/diabetic pills
☐	☐	Demerol	☐	☐	_____
☐	☐	laxatives	☐	☐	_____
☐	☐	cold tablets	☐	☐	_____
☐	☐	diet pills	☐	☐	_____
☐	☐	heart medicines	☐	☐	_____

Copyright © 1971, 1975 Hollister Incorporated. All rights reserved. Printed in U.S.A.

CONTINUE TO NEXT P

Name _____
Last First Initial

Date ___/___/___
Month Day Year

Soc. Sec. # _____

Employee No. _____

Special Problems _____

HEAD AND NECK
___ frequent headaches
___ neck pains
___ neck lumps or swelling

EYES
___ wears glasses/contact lenses
___ eyesight worsening
___ sees double
___ sees halo

EARS
___ others notice hearing problem
___ has hearing problem
___ decreased hearing after trauma
___ earaches
___ running ears
___ buzzing in ears
___ motion sickness

MOUTH
___ swellings on gums or jaws
___ sore tongue
___ taste changes

NOSE and THROAT
___ congested nose
___ running nose
___ sneezing spells
___ sore throat
___ hoarse voice
___ skin/neck X-ray

RESPIRATORY
___ wheezes or gasps
___ shortness of breath
___ coughing spells
___ coughs up phlegm
___ coughed up blood
___ chest colds
___ excessive sweating, night sweats

CARDIOVASCULAR
___ high blood pressure
___ racing heart
___ chest pains
___ dizzy spells
___ shortness of breath at night
___ more pillows to breathe
___ swollen feet or ankles
___ leg cramps
___ heart murmur
___ inflamed veins or blood clots in legs

NEUROLOGICAL
stutter or loss/speech ___
temp. loss of sight ___
numbness one side of body ___
weakness one side of body ___
faintness ___
numbness ___
convulsions ___
tremblers ___

DIGESTIVE
heartburn ___
bloated stomach ___
belching ___
stomach pains ___
nausea ___
vomited blood ___
difficulty swallowing ___
change in bowel habits ___
black stools ___
rectal surgery ___
rectal bleeding ___

URINARY
night frequency ___
day frequency ___
poor bladder control ___
burning on urination ___
brown, black, or bloody urine ___
difficulty starting urine ___
urgency ___

MALE GENITAL
weak urine stream ___
prostate trouble ___
burning or discharge ___
lumps on testicles ___

FEMALE GENITAL
menstrual trouble ___
breakthrough bleeding ___
heavy bleeding ___
bleeding after intercourse ___
premenstrual tension ___
hot flashes ___
birth control pill ___
lumps in breasts ___
vaginal discharge ___
PAP smear ___/___
last period ___/___

PREGNANCIES
gravida ___
miscarriages ___
stillbirths ___
premature births ___
para ___
cesarean ___
abortion ___

MUSCULOSKELETAL
___ aching muscles or joints
___ swollen joints
___ back or shoulder pains
___ back problem
___ painful feet
___ handicapped

SKIN
___ skin problems
___ bleeds easily
___ bruises easily

MOOD
___ nervous with strangers
___ difficulty in making decisions
___ lack of concentration or memory
___ lonely or depressed
___ hopeless outlook
___ difficulty relaxing
___ worries a lot
___ frightening dreams or thoughts
___ shy or sensitive
___ loses temper
___ work or family problems
___ sexual difficulties
___ considered suicide
___ desired psychiatric help

GENERAL
___ weight gain
___ weight loss
___ special diet
___ tends to be hot or cold
___ always hungry
___ more thirsty lately
___ sugar in system
___ loss of interest in eating
___ fatigue
___ sleeping difficulties
___ armpits or groin swelling
___ lack of exercise
___ smokes cigarettes
___ no. packs cig./day
___ no. of years smokes cig.
___ used to smoke cigs.
___ no. packs cig./day
___ no. years smoked cig.
19___ year stopped cigs.
___ smokes cigars/pipe a day
___ no. cigars/pipe a day
___ no. yrs. smokes cigars/pipe
___ used to smoke cigars/pipe
___ no. cigars/pipe a day
___ no. years smoked cigars/pipe
19___ year stopped cigars/pipes
___ 2 alcoholic drinks daily
___ 4 alcoholic drinks daily
___ heavy coffee or tea drinker
___ marijuana
___ heroin, LSD, similar drugs
___ doesn't use safety belts
___ visited in last 6 months

Signature

Copyright © 1971, 1975 Hollister Incorporated.
All rights reserved. Printed in U.S.A.

Occupational Health History Questionnaire

Occupational Health History Questionnaire

IDENTIFICATION DATA Fill in the following information. PLEASE PRINT.

D.O.H.

Date 12-31-86 Plant Location Sumter

Name Williams (Last) CL (First) (Initial) Sex (circle) M F

Social Security No. Employee No. 9650

Home address 55 Maxwell Ave

Sumter, SC

Home telephone 803 773-5048 (area code)

1 Married Separated Divorced Widowed Single

Education: 6 years Elementary years High School

years College, Technical, Business, etc.

Job title Machine Operator

FAMILY HISTORY: Follow the lines across the page and mark an X in those boxes which indicate the present state of health (good, poor), or the death (indicate the cause) of your mother and father and any illnesses they or your blood relatives (grandparents, brothers, sisters, children) have ever had.

	Health			Cause of death					Allergies or asthma	Anemia	Bleeding tendencies	Cancer or tumor	Diabetes	Epilepsy	Glaucoma	Hearing trouble	Heart trouble	High blood pressure	Kidney or bladder trouble	Lung disease	Nervous breakdown	Rheumatism or arthritis	Stomach/duodenal ulcer
	Good	Poor	Dead	Age at Death	Cancer	Heart Trouble	Lung Disease	Other Causes															
Father	Deceased					X																	
Mother	Deceased																						
Blood relatives (mark an X for illnesses only)																							

YOUR HEALTH HISTORY: Mark an X in the box next to any of the following illnesses you now or ever have had.

- ☐ Anemia
- ☐ Asthma
- ☐ Bleeding tendencies
- ☐ Bronchitis
- ☐ Cancer or tumor
- ☐ Diabetes
- ☐ Diverticulosis
- ☐ Emphysema
- ☐ Epilepsy
- ☐ Eye problems
- ☐ Glaucoma
- ☐ Hay fever or allergies
- ☐ Hearing trouble
- ☐ Heart trouble
- ☐ Hemorrhoids
- ☐ Hernia
- ☐ High blood pressure
- ☐ Hives or rashes
- ☐ Kidney/bladder trouble
- ☐ Liver disease/hepatitis
- ☐ Lung disease
- ☐ Malaria
- ☐ Measles
- ☐ Mononucleosis
- ☐ Mumps
- ☐ Neuralgia/neuritis
- ☐ Nervous breakdown
- ☐ Pneumonia
- ☐ Polio
- ☐ Rheumatic fever
- ☐ Rheumatism/arthritis
- ☐ Skin disease
- ☐ Venereal disease
- ☐ Yellow jaundice
- ☐ Other

Have you ever been turned down for life insurance, military service, or employment because of health problems? (circle) ..... Yes (No)

Have you ever received a blood transfusion? (circle) ..... Yes No

Major Hospitalizations: If you have ever been hospitalized for any serious medical illness or operation, write in your most recent hospitalizations below. Check this box ☐ if you have had more than three such hospitalizations.

(Do not include normal pregnancies)	Year	Reason for hospitalization	Name of hospital	City and State
1st hospitalization	19			
2nd hospitalization	19			
3rd hospitalization	19			

Tests: Mark an X next to those which you have had. Enter the year when you last were given the test.

Mark an X after those tests which you know had abnormal results.

	Year		Abnormal
☐	19__	chest X-ray	☐
☐	19__	kidney X-ray	☐
☐	19__	GI series	☐
☐	19__	colon X-ray	☐
☐	19__	gallbladder X-ray	☐
☐	19__	electrocardiogram	☐
☐	19__	electrocardiogram with exercise	☐
☐	19__	TB test	☐
☐	19__	breathing test	☐
☐	19__	biopsy	☐
☐	19__	hearing test	☐
☐	19__	other	☐

Medicines: Mark an X in the box next to any medicines that you are now taking or that you are allergic to.

taking	allergic to		taking	allergic to	
☐	☐	aspirin	☐	☐	high blood pressure medicine
☐	☐	penicillin	☐	☐	Dilantin/anticonvulsants
☐	☐	sulfa	☐	☐	birth control pills
☐	☐	codeine	☐	☐	diuretics/water pills
☐	☐	antibiotics	☐	☐	blood thinners (anticoagulants)
☐	☐	sedatives	☐	☐	cortisone-type drugs
☐	☐	stimulants	☐	☐	insulin/diabetic pills
☐	☐	Demerol	☐	☐	_____
☐	☐	laxatives	☐	☐	_____
☐	☐	cold tablets	☐	☐	_____
☐	☐	diet pills	☐	☐	_____
☐	☐	heart medicine	☐	☐	_____

Copyright © 1971, 1975 Miller Communications, Inc., Norwalk, CT 06856. All rights reserved. Printed in U.S.A.

CONTINUE TO NEXT PAGE

Name Williams C L NMN
Last First Initial

Date Aug 31 56
Month Day Year

Soc. Sec. # _____

Employee No. 8150

Special Problems: _____

HEAD AND NECK
- ___ frequent headaches
- ___ neck pains
- ___ neck lumps or swelling

EYES
- ___ wears glasses/contact lenses
- ___ eyesight worsening
- ___ sees double
- ___ sees halo

EARS
- ___ others notice hearing problem
- ___ has hearing problem
- ___ decreased hearing after trauma
- ___ earaches
- ___ running ears
- ___ buzzing in ears
- ___ motion sickness

MOUTH
- ___ swellings on gums or jaws
- ___ sore tongue
- ___ taste changes

NOSE and THROAT
- ___ congested nose
- ___ running nose
- ___ sneezing spells
- ___ sore throat
- ___ hoarse voice
- ___ skin/neck X-ray

RESPIRATORY
- ___ wheezes or gasps
- ___ shortness of breath
- ___ coughing spells
- ___ coughs up phlegm
- ___ coughed up blood
- ___ chest colds
- ___ excessive sweating, night sweats

CARDIOVASCULAR
- X high blood pressure
- ___ racing heart
- ___ chest pains
- ___ fluty spells
- ___ shortness of breath at night
- ___ more pillows to breathe
- ___ swollen feet or ankles
- ___ leg cramps
- ___ heart murmur
- ___ inflamed veins or blood clots in legs

NEUROLOGICAL
- ___ slurred or loss/speech
- ___ temp. loss of sight
- ___ numbness one side of body
- ___ weakness one side of body
- ___ faintness
- ___ numbness
- ___ convulsions
- ___ tremblers

DIGESTIVE
- ___ heartburn
- ___ broaked stomach
- ___ belching
- ___ stomach pains
- ___ nausea
- ___ vomited blood
- ___ difficulty swallowing
- ___ change in bowel habits
- ___ black stools
- ___ rectal surgery
- ___ rectal bleeding

URINARY
- ___ night frequency
- X day frequency
- ___ poor bladder control
- ___ burning on urination
- ___ brown, black, or bloody urine
- ___ difficulty starting urine
- ___ urgency

MALE GENITAL
- ___ weak urine stream
- ___ prostate trouble
- ___ burning or discharge
- ___ lumps on testicles

FEMALE GENITAL
- ___ menstrual trouble
- ___ breakthrough bleeding
- ___ heavy bleeding
- ___ bleeding after intercourse
- ___ premenstrual tension
- ___ hot flashes
- ___ birth control pill
- ___ lumps in breasts
- ___ vaginal discharge
- ___ PAP smear ___/___/___
- ___ last period ___/___/___

PREGNANCIES
- ___ gravida
- ___ miscarriages
- ___ stillbirths
- ___ premature births
- ___ para
- ___ cesareans
- ___ abortion

MUSCULOSKELETAL
- ___ aching muscles or joints
- ___ swollen joints
- ___ back or shoulder pains
- ___ back problem
- ___ painful feet
- ___ handicapped

SKIN
- X skin problems
- ___ bleeds easily
- ___ bruises easily

MOOD
- ___ nervous with strangers
- ___ difficulty in making decisions
- ___ lack of concentration or memory
- ___ lonely or depressed
- ___ hopeless outlook
- ___ difficulty relaxing
- ___ worries a lot
- ___ frightening dreams or thoughts
- ___ shy or sensitive
- ___ loses temper
- ___ work or family problems
- ___ sexual difficulties
- ___ considered suicide
- ___ desired psychiatric help

GENERAL
- ___ weight gain
- ___ weight loss
- ___ special diet
- ___ tends to be hot or cold
- ___ always hungry
- X more thirsty lately
- ___ sugar in system
- ___ loss of interest in eating
- ___ fatigue
- ___ sleeping difficulties
- ___ armpits or groin swelling
- X lack of exercise
- X smokes cigarettes
- 2 no. packs cig./day.
- 20 no. of years smokes cig.
- ___ used to smoke cigs.
- ___ no. packs cig./day
- ___ no. years smoked cig.
- 19___ year stopped cigs.
- ___ smokes cigars/pipe a day
- ___ no. cigars/pipe a day
- ___ no. yrs. smokes cigars/pipe
- ___ used to smoke cigars/pipe
- ___ no. cigars/pipe a day
- ___ no. years smoked cigars/pipe
- 19___ year stopped cigars/pipes
- X 2 alcoholic drinks daily
- ___ 4 alcoholic drinks daily
- ___ heavy coffee or tea drinker
- ___ marijuana
- ___ heroin, LSD, similar drugs
- X doesn't use safety belts

___ visited in last 6 months

Signature

Copyright © 1971, 1975 Miller Communications, Inc., Norwalk, CT 06850.
All rights reserved. Printed in U.S.A.

Occupational Health History Questionnaire

NAME WILLIAMS, C.L.

INDUSTRIAL HYGIENE LABORATORY STUDIES

DATE	Pb in Air UG/M³	ALA	URINE Sp. Gr.	URINE Pb UG/L	URINE Pb CORRECT TO 1.024 UG/L	BLOOD Pb UG/100 GMS	NOTES	JOB TITLE	CLOCK NO.	DEPT. NO.
3-15-76	.11	3-16-76	1.028	50		25	Hgb. 15 Hct 46		2150	
3-17-76			1.020	80			Urine spec.	21 Apr 76		
4-21-76			1.022	70						
5-21-76			1.025	130			Urine Spec.	11 June 76		
6-2-76	.29	6-2-76						6-5-76 Starting Oper.		
6-20-76			1.026	120			Urine Spec.	12 July 76		
7-14-76			1.029	150						
8-16-76			1.027	50			Blood & Urine Out (Lead in auto)	11 Oct 76 MHH		
9-16-76	.29	9-16-76	1.028	40		57				
10-17-76	.23	10-12-76	1.026	170						
11-16-76	.19	11-8-76	1.022	150						
12-14-76	.37	12-27-76	1.030	230						
1-11-77			1.025	250		65	Urine & Blood Pb — Health Conf.	7 Feb 77 and K		
2-15-77	.69	2-2-77	1.025	200		61	Urine Thru 77 Health Conference	9 Mar 77 MHH		
3-7-77	.46	2-7-77	1.020	170			All 3'77.			
3-15-77	.12	2-21-77	1.030	210		65	Health Conf.			
4-18-77	.24	5-11-77	1.025	240		63	Blood Pb Urine (Blood Thru 3/30/77 Health Conf	6 Apr 77 MHH		
5-17-77	.01	5-12-77	1.025	200		44	Health Conference			
6-14-77	.07	6-7-77	1.032	80						
7-12-77	.53	7-12	1.023	230		51	Health Conference			
8-23-77	.47	8-24-77	1.031	70			Blood Pb Urine Pb - Not for same	5 Oct 77 MHH		
9-20-77	.16	10-11-77	1.022	50						
10-19-77	.03	11-4-77	1.021	180		62	Health Conf.			
11-16-77	.08	12-12-77	1.027	240		57	Blood & Urine - Not Urine Health Conf	12 Dec 77 MHH		
12-14-77	.06	1-19-78	1.029	60						
1-16-78			1.023	180		44				
2-14-78			1.024	220			Health Conf.			
3-21-78	.34	3-28-78	1.027	170				Blood and Urine Analysis	2150	MHH
4-12-78						83	Health Conf.			
5-9-78						91	Health Conf.			
5-25-78						97				
6-16-78						79	Health Conf Blood Pb			
7-11-78						98	Health Conf Urine Pb			
8-1-78						44	Health Conf - Pb Urine			
8-14-78						77	Health Conf. Blood Pb			

1-0389 REV. 1/73

NAME Williams, C.L

INDUSTRIAL HYGIENE LABORATORY STUDIES

DATE	Pb in Air UG/M³	Pb Air Date	URINE Sp. Gr.	URINE Pb UG/L	UPINE Pb CORRECT TO 1.024 UG/L	BLOOD Pb UG/100 GMS	NOTES	JOB TITLE	CLOCK NO.	DEPT. NO.
8-24-78						37				
8-31-78						90	B/C Health Conf.			
9-26-78	.16	11-5-78				76	Health Conf.	cut Blade Op.		
10-26-78	/					65	Health Conf.			
11-21-78						60				
12-22-78						68				
1-10-79						65				
2-09-79						65				
3-21-79						70				
4-05-79						53				
4-11-79						89				
4-23-79						70				
5-03-79						64				
6-12-79						71				
6-25-79						71				
7-18-79						62				
8-17-79						53				
9-28-79						77				
10-31-79						69				
11-14-79						70				
1-30-80						51				
3-04-80	.21	4-17-80				62				
3-12-80	56	4-22-80				56				
3-28-80	150	4-28-80				64				
5-14-80	128	6-30-80				61				
7-09-80	100	7-22-80				65				
7-25-80	39	8-04-80				54				
9-22-80	136	9-28-80				61				
10-13-80	125	9-04-80				68				
11-14-80	50	10-14-80				62				
12-4-80	57	1-8-81				60				
12-17-80	.135	2/15/81				65	Equipment satisfied - Will chg monitor resp. wear			
12-30-80						68	Prompt dynamic work habits. 1-26-81			
1-21-81						60	Working fellow was hurt caught & welded - Admits			
1-27-81						62	to drinking moon shine liquor - Encouraged to			
2-26-81						57	drink bottled liquor only			

2-26-81 cont - Should be on 6 mon schedule for Pb & lead

1-5309 REV 1/79

NAME WILLIAMS, C.L.

INDUSTRIAL HYGIENE LABORATORY STUDIES

DATE	Pb in Air UG/M³	ALA	URINE Sp. Gr.	URINE Pb UG/L	~~URINE Pb CORRECT TO 1.024 UG/L~~	BLOOD Pb UG/100 GMS	NOTES	JOB TITLE	CLOCK NO.	DEPT. NO.
3-27-81	89	3/31/81				56		5/12/81 Temp chg for 3 days.		
4-27-81					157	60				
6-29-81	66	7/16/81			174	58				
8-24-81	31	7/14/81				57				
10-19-81	26	11/6/81			101	54				
12-21-81					123	54				
1-7-82					129	51	25 Jan 82 Asst Stanley K			
3-2-82					131	47				
4-28-82					200	56				
6/15/82					101	60		Stocker T/O 1st		
8/9/82					[illegible]	[illegible]		7-6-82 Stocker to Pre-A [illegible]		
10-05-82		1			114	56				
12-06-82					139	55				
1-31-83					226	69	2-07-83 Lic Not			
3/1/83					133	49	14 Feb 83 Asst			
4-4-83					152	47				
6-01-83					193	55				
8-04-83					145	55				
10-04-83					122	66				
10-27-83					132	57	Bld Pb Repeat - 3 Nov 83 - Blood Conc. >50 Ugm	Eligible for MRP		
12-06-83					162	58		Asst		
1-11-84					128	50	30 Jan 84 Asst			
3-1-84	1	1	1	1	1	11	13 Feb 84 - Removed Asst	4/30/84 Temp assigned to MRP	22	
3-05-84					117	55	23 Apr 84 - Restored Asst			
4-18-84					100	43				
5-22-84					119	48				
6-14-84					94	38				
6-26-84					118	41				
7-30-84					103	42				
8-15-84					128	48	Eligible for Asst			
9-06-84					103	40				
9-26-84					100	39				
10-09-84					96	44				
11-06-84					86	35				
11-19-84					79	36	Off MRP 11-28-84	12-03-84 To EV Stocking		
5-03-85					99	49	06 June 85 Asst			

1-6369 REV.1/73

NAME Williams, C. L.

INDUSTRIAL HYGIENE LABORATORY STUDIES

DATE	Pb in Air UG/M3	ALA	URINE Sp. Gr.	URINE Pb UG/L	URINE Pb CORRECT TO 1.024 UG/L	BLOOD Pb UG/100 GMS	NOTES	JOB TITLE	CLOCK NO.	DEPT. NO.
7-02-85						51				

1—8388 REV. 1/73

CHART #

FAMILY MEDICINE — OFFICE VISITS — 01 45 405

Formedic

NAME C. L. Williams — MARITAL STATUS S, M, W, D, SEP — DATE OF BIRTH

ADDRESS 55 Maxwell Street, Sumter, SC 29150 — PHONES (H) 773-9461 (O) 481-6345

OCCUPATION/EMPLOYER — INS. # — REFERRED BY

MEDICATIONS Lasix 40mgs. daily + K-Dur 20 daily — DRUG ALLERGIES NKMA

DATE / DIAGNOSIS / CPT CODE	HISTORY & PHYSICAL	HT	WT	BP	P	T

1-11-94

Ht: 5'9"
Wt: 218 lbs.
Temp: 96
BP 132/80

Problems c " fluid building up " 5 days.
" fever, cough + yellow x today

P/P ① Lasix CHF
② Lab KTS

Dr. Marion DAvis dictating: 45 year old black male new patient here. Complain is SOB which has been building up over about a week. He was seen in the ER earl this morning and we have those records. He was given LASIX 80 mgs. IV. Also had a CBC & SMA 7 done which were normal. Chest x-ray showed suspected mild congestive heart failure. He says that he was hosp. about 4 years ago for a similar problem and got some LASIX & POTASSIUM at that time. He has been to th ER I think he said 3 times over the last 3 or 4 months for a similar problem. Last night he was unable to lie down without getting short of breath and feelin like something was in his throat so he went to the ER and was diuresed. He says he is breathing a lot better now. He also relates questionable fever and he has been coughing up yellow sputum for about 2 weeks.
PMH: Borderline BP by his statement and what sounds like CHF.
ALLERGIES: none
TOBACCO HISTORY: 1 pack per day
ALCOHOL HISTORY: None for the last 2 months - Apparently he had quit for awhil and then drank some for maybe a year and now has quit for the last 2 months
O. Vital signs: Ht. 5'9", Wt. 218, Temp. 96.6. BP 132/80. PHYSICAL EXAM: MOUTH - mucus membranes are moist, ORAL PHARYNX is clear, NECK - no carotid bruits, HEART is regular without murmurs, rubs or gallops. LUNGS - coarse breath sounds bilaterally and decreased breath sounds in the right base. The X-ray report from early this morning does show vague horizontal opacities present in both bases. Findings consistent with discoid atelectasis. Also there is blunting of the right costo-phrenic angle, a change noted on previous studies and consistent with old pleural thickening.
A & P. 1. Mild CHF - He is given refills on FUROSEMIDE 40 mgs. 1 PO Q day #31, refills 2. Also KCL 20 MEQ, 1 PO Q day #31, refills 2. We are scheduling an echocardiogram to evaluate this heart failure.
2. Bronchitis - Plan is to prescribe TMP/SMX DS 1 PO BID times 14 days #28, refills 0. He is to return in 1 week for follow up and we will go over the echo results and check a POTASSIUM level. mp

1/18/94 wt 215 lbs.
T 97.6
Lungs Clear H.S. normal

p/p ① Rx
② Lab KTS

checkup for above re-

CONTINUED--

ONCE-A-DAY Zoloft (sertraline HCl) GETS DEPRESSED PATIENTS BACK INTO THE MAINSTREAM
Please see 20-page brief summary of prescribing information in the accompanying booklet.
Pfizer Roerig Pfizer Pharmaceuticals
© 1992, Pfizer Inc

DATE
DIAGNOSIS
CPT

113-976 HISTORY & PHYSICAL

C. L. Williams 481-6245(W)

Formedic

1-18-94 Dr. Marion Davis dictating: 45 year old black male. I had seen him on 1-11-94 after an ER visit for mild CHF. We started him on LASIX & POTASSIUM. We also got an echocardiogram which is in the chart. Conclusion reads 1. Suggests, decreased left ventricular and right ventricular function consistent with congestive cardiomyopathy, 2. Suggests 2-3 plus mitral insufficiency, 3. Suggest possible tricuspid and pulmonic insufficiency ty dopplar and that was read by Dr. Stavrou. Today he is here for follow up. WE had also treated him for bronchitis. He got half of his SEPTRA presc. filled and I have instructed him to go ahead and get the other half filled and finish that course. He has learned quite quickly that the FUROSEMIDE will cause him to urinate. This interfers somewhat with his sleeping if he takes it at night and somewhat with his work if he takes it in the morning. I have suggested that he try taking this as soon as he gets off work. That way he will have 6-7 hours before he goes to bed.
O. Vital signs: Wt. 215, Temp. 97.6, POTASSIUM 4.5, PHYSICAL EXAM NOT DONE.
A & P. (1.) CHF - Plan is starting LANOXIN 0.125 mgs. 1 PO Q day at 9-10 am #31, refill 2. Also start CAPTOPRIL 12.5 mgs. ½ tablet PO BID #16, refills 2. BP today is 104/80. They have brought a list of bp readings which are running 104-132/80-86. Probably the FUROSEMIDE has lowered his bp somewhat. He came to me with a history of borderline hypertension. He is to come back in 2 weeks for DIGOXIN LEVEL and then 2 days after that for a visit.
(2.) Bronchitis. He is to continue the SEPTRA.

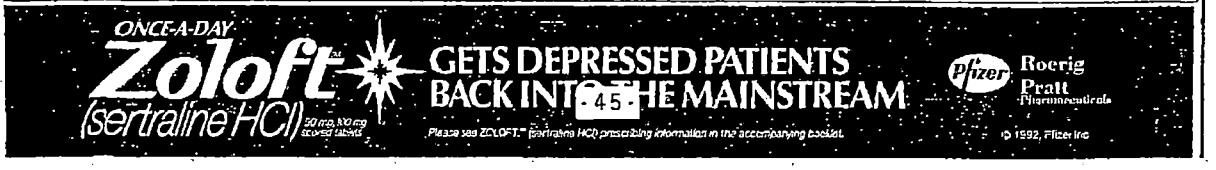
1-27-94 Blood drawn for digoxin level + sent to PSPA — MGibbs

1/31/94 wt. 221½ LB. T. 96.8 — Checkup for above — laboratory marked.

A/P. D CHF - Dig <0.5, ↑ Dig. to 0.125 mg ☞ QD, rev Dig level in wk. D 4/5 RN -

Dr. Marion Davis dictating: Mr. Williams is here for follow up on congestive heart failure. I saw him on 1-18-94, started him on LANOXIN & CAPTOPRIL, had him come back on the 27th for DIGOXIN LEVEL. That level is back and is subtheraputic at less than 0.5. His current digoxin dose is 0.125 mgs. Q day. He is having no problems and feels like he is doing fine.
O. Vital signs: Wt. 221½, Temp. 96.8. PHYSICAL EXAM - not done.
A & P. 1. CHF: Dig. level is subtheraputic. We plan to increase his dose to 0.125 mgs. 2 tablets Q day, have him come back for a dig. level in 1 week and then a visit after that level is drawn.
2. History of hypertension: The wife tells me that he was treated sever years ago by Dr. Abbott for hypertension and that after he got to feeling better he stopped the medicine on his own. He is currently not on anything for hypertension but we realize that the LASIX and CAPTOPRIL which he is on for heart failure will certainly affect his bp. MG

2/10/94 Digoxin level drawn + sent to PSPA (B. Samaa



DATE - TIME CPT CODE (DIAGNOSIS)	SUBJECTIVE / OBJECTIVE / ASSESSMENT / PLANS C. L. WILLIAMS	Formedic
3-7-96	CONTINUED. Prescriptions are given as follows: LANOXIN 0.25 mgs. 1 PO Q day, #31, refills 2; LASIX 40 mgs. 1 PO Q day, #31, refills 3; CAPTOPRIL 12.5 mgs. ½ PO BID, #16, refills 4; KCL 20 meqs. 1 PO Q day, #31, refills 5. Follow up with me in 2 weeks with DIGOXIN LEVEL. Chemzyme plus diarr i start to home - B Simon	
4/26/96 B/P	WS - 223 3/4 lbs (133 1/4 lbs) c/o "feeling like food build-up in Chest" SOB burning in Chest - belching T-96.5 138/88 Alot mostly after eating #'s pr - (1) Lanoxin DR. MARION DAVIS DICTATING: S: Complaints as above. Feeling like heartburn. comes on mostly after eating. O: Vital signs as above. BP 138/88. Physical exam: Heart is regular without murmurs, rubs or gallops. Lungs are clear. A & P: 1) DYSPEPSIA. PLAN - Schedule Upper GI to look for ulceration or Reflux. He can use OTC MAALOX or MYLANTA and start elevating the head of the bed. He is to avoid any aggravating foods. Return to me after the Upper GI. He is also to return at his convenience for the DIG LEVEL which he missed in March.	
12/16/96	TC from Rick - Super Rx (Liberty) - Lanoxin 0.25mg T po q day #7 NR - has appt next week	
12/23/96 B/P	WS - 231 3/4 lbs (↑8 lbs) T-97.3 140/86 #'s pr (1) Lanoxin (2) Lasix (3) KCL DR. MARION DAVIS DICTATING: S: See the note dated 4-26-96. Mr. Williams did not follow up on the Upper GI or the DIGOXIN Level at that time. Complaint today is coughing up yellow sputum. He is able to sleep on 2 pillows. He does have a history of CHF and he has run out of medications. O: VITAL SIGNS AS ABOVE. PHYSICAL EXAM: Heart is regular without murmurs, rubs or gallops. LUngs show rhonchi bilaterally.	Cheesy cough → yellow 2 pillows

IN CHRONIC ARTHRITIS EXPECT SUCCESS FROM NAPROSYN® (NAPROXEN) 500 mg tablets
© 1993 Syntex Puerto Rico, Inc. NP9127 SYNTEX
Please see full prescribing information in accompanying booklet
Also available in 375 and 250 mg tablets and in suspension 125 mg/5 mL

FAMILY MEDICINE — PROGRESS NOTES

NAME C. L. Williams — MARITAL STATUS M S W D SEP — DATE OF BIRTH

Formedic

DATE - TIME / CPT CODE / DIAGNOSIS	SUBJECTIVE / OBJECTIVE / ASSESSMENT / PLANS	HT	WT	T	ALLERGIES
		BP		P	Ø

2-14-94

W 222½ 165
T-97.8
B/P 126/84
A/P-① bronchitis - ② CHF

Check-up - results of blood work - Hx cough - congested Mon - yellowish clear sputum & lungs

Dr. Marion Davis dictating: Follow up on CHF & also complains of some cough productive of yellow sputum and congestion. Also some clear nasal drainage. DIGOXIN LEVEL was drawn on 2-10-94 and that is theraputic at 0.7. He denies any fever or chills and denies any nausea, vomiting or diarrhea. Denies any allergies.

O. Wt. 222½, Temp. 97.8. BP 126/84. PHYSICAL EXAM: EARS - TM's are clear bilaterally. SINUSES are non-tender. MOUTH - mucus membranes are moist. ORAL PHARYNX is clear. NECK - no adenopathy. HEART is regular without murmurs rubs or gallops. LUNGS are slightly coarse but clear with coughing.

A & P. 1. Bronchitis - Plan - prescribe TMP/SMX DS 1 PO BID times 14 days #28, refill 0.

2. CHF. DIG LEVEL is theraputic. He is to continue the current dosage which is 0.25 mgs. Q day. Return prn.

5-6-94

Dr. Marion Davis dictating/pb

Refill is given on LANOXIN 0.25 mgs. 1 PO Q day at 9 to 10 am. 1 month with refills prn. This is called in to Sur RX Pharmacy 775-6671.

3-7-96

W 220 165
T-98.8
B/P 124/82
A/P-① SOB

follow-up ER VS & 3/7/96 CXR x 2 views, 2 PA upright

DR. MARION DAVIS DICTATING:

S: Had a recent ER visit and he is following up on that. The complaint was shortness of breath. The diagnosis was CHF and Pleural effusion. The official x-ray report did not quite confirm that but Mr. Williams has been having to sleep on 3 pillows at night. The x-ray dated 3-6-96 showed no acute abnormality, chronic changes as described above which basically showed scarring in the right base unchanged from 1-11-94. He has not had any of his medicines for about the last 8 months. He does have a history of CHF. He has had a few fluid pills recently.

O: Vital signs as above. BP 124/82. Physical exam: Heart is regular without murmurs, rubs or gallops. Lungs are clear.

A & P: 1) SHORTNESS OF BREATH, PROBABLY RECURRENT CHF. PLAN - get him back on all of his medications. We will draw a Panel 7 on the way out today.

IN CHRONIC ARTHRITIS EXPECT SUCCESS FROM NAPROSYN (NAPROXEN) 500 mg tablets
© 1993 Syntex Puerto Rico, Inc. NP3812/ SYNTEX
Please see full prescribing information in accompanying booklet.
Also available in 375 and 250 mg tablets and in suspension 125 mg/5 mL

PROGRESS NOTES

NAME C. L. WILLIAMS DATE OF BIRTH PG# Formedic

DATE - TIME WT / BSA CPT CODE ALLERGIES

12-23-96 CONTINUED.

He does smoke 1 pack per week and drinks beer - he says 1 beer per weekend.

A & P: 1) BRONCHITIS. PLAN - Prescribe TMP/SMX DS 1 PO BID x 14 days, #28, refills 0.

2) HEALTH MAINTENANCE. Refill LANOXIN 0.25 mgs. 1 PO Q day, #31, refills 2; LASIX 40 mgs. 1 PO Q day, #31, refills 2; CAPTOPRIL 12.5 mgs. 1 PO BID, #62, refills 2; KCL 20 meqs. 1 PO Q day; #31, refills 2.

For the bronchitis and the abnormal lung sounds, he will go to the hospital and have a chest x-ray, PA & Lateral. he will also follow up with a timed DIGOXIN Level and also fasting Exec. Profile and TSH.

3) HYPERTENSION. BP today is 140/86 and we increased his CAPTORIL slightly The new dose is as prescribed above.

12/30/96 Digoxin Level drawn & sent to Work - B Inman

1-9-97 DR. PERRY DAVIS DICTATING: TC. Itching all over. Was using BENADRYL in the hospital. continued to do so since release and is not cutting it any longer. Wants something different called to SupRX on Liberty Street for PERIACTIN 4 mgs. TID or QID #28. If this does not relieve it then he should see Dr. Marion Davis.

1-20-97 wt - 231 lbs
T - 96.4
BP 126/84
Apical pulse 50

Check up re: hosp stay
Lungs fine, OF

DR. MARION DAVIS DICTATING:

S: Following up after hospitalization for Hemoptysis. He has been doing pretty well. His breathing is good. Denies any fever. Denies any more hemoptysis. Does feel like he is getting a fresh cold. He has a cough which is non-product He is smoking about 3 cigarettes per day.

O: VITAL SIGNS AS ABOVE.
BP 126/84.
PHYSICAL EXAM: Heart is regular without murmrus, rubs or gallops. Lungs show mild rhonchi bilateraly.

A & P: 1) HEMOPTYSIS. This seems to be pretty much resolved. We will send him for chest x-ray because of the lung sounds and for follow up. Prescribe TESSALON PERLES 100 mgs. 1 PO Q4H prn cough, #30, refills 0. Return in 2months or sooner if needed.

5-21-97 TC from Baker - Suplx - Lasix 40 mgs #7 1 daily
OK'd - appt 5-27-97

IMPALE® FORMEDIC® 1996 120 WORLDS FAIR DR. SOMERSET N.J. 08873 APPROVED BY FORMEDIC'S PHYSICIAN ADVISORY BOAR

Formedic Your Supplier of Patient Record Forms free
PARTNERS WITH PHYSICIANS SINCE 1982

DATE - TIME WT / BSA CPT CODE	C. L. WILLIAMS
5/27/97	No Show for 4:00 w/ Dr. Marion Davis – left message on answering machine to call re: [illegible]
5/28/97	Called pt about missed appt on 5/27/97 w/ Dr. Marion Davis – left message on answering machine @ 8:45am — LSSC
5/29/97	Sent pt a note re: missed appt. — LSSC
6-6-97	DR. PERRY DAVIS DICTATING: TC requesting POTASSIUM and fluid pills. I called in on 6-10-97 for KCL 20 meqs. 1 daily and LASIX 40 mgs. 1 daily, both for 1 month supply and he is to see Dr. Marion Davis within the next month.
6-11-97	WT - 241 lbs (↑ 10 lbs) T-97.2 BP 142/94 A/P – D/HTN BP Check – no meds new Rx's BP "good" @ plant
	DR. MARION DAVIS DICTATING:
	S: Follow up on BP and needing new refills. He says his BP has been "good" at the plant but he is not able to give me any numbers. He did not bring a list of readings or his medications.
	O: VITAL SIGNS AS ABOVE. WEIGHT 241. Note the 10 pound increase. BP 142/94. PHYSICAL EXAM: Heart is regular without murmurs, rubs or gallops. LUngsare clear.
	A & P: 1) HYPERTENSION. PLAN – Refill CAPTOPRIL 12.5 Mgs. 1 PO BID, #62, refills 5; LASIX 40 mgs. 1 PO Q day, #31, refills 3. Refill KCL 20 meqs. 1 PO Q day, #31, refills 2; LANOXIN 0.25 mgs. 1 PO Q day, #31, refills 4. Return in 1 month with a timed DIGOXIN Leve. Also, bring BP readings and all of his medication bottles.
7-9-97	no show for labs + Dr. visit w/ Dr. Marion Davis – called pt. – left message on ans. machine (RT)
7-11-97	note sent re: above appt. (RT)
11-6-97	DR. MARION DAVIS DICTATING: Note dated 11-5-97. TC needing refill on heart pill, fluid pill and POTASSIUM pill – enough to last until his appt. of Tuesday, 11-11-97, when he is to come in for timed DIGOXIN Level and office visit. I had staff call in to SupRX on Liberty for LANOXIN 0.25 mgs. 1 PO Q day, #14, refills 0; LASIX 40 mgs. 1 PO Q day, #14, refills 0; KCL 20 meqs. 1 PO Q day, #14, refills 0.

Formedic
Formedic Your Supplier of free Patient Record Forms
PARTNERS WITH PHYSICIANS SINCE 1932

DAVIS CLINIC
PROGRESS NOTES

NAME: Cl Williams DATE OF BIRTH: ? ACCT.: 597.3

DATE	
11/11/97	Wt - 240 1/2 lbs. Check up T. 97 Needs new Rx -
	BP 140/92 R Reg. Digoxin Level drawn & sent to A/P Dr. Hon. Hosp. /B Simon
	③ HM
	DR. MARION DAVIS DICTATING;
	S: Follow up on Hypertensio and also for DIGOXIN Level.
	O: VITAL SIGNS AS ABOVE. BP 140/92 in right arm with regular cuff. ~~PHYSICAL EXAM: Heart is regular without murmurs, rubs or gallops. Lungs are clear.~~
	A & P: 1) HYPERTENSION. PLAN - Refill LASIX 40 mgs. 1 PO Q day., #31, refills 4; ~~CAPTOPRIL 12.5 mgs. 1 PO BID, #62, refills 5.~~
	2) ~~HEALTH MAINTENANCE. PLAN - Refill LANOXIN 0.25 mgs. 1 PO Q day, #31,~~ refills 2. He requests refill on PERIACTIN 4 mgs. 1 PO TID prn itching, ~~#100, refills 3 and KCL 20 meqs. 1 PO Q day, #31, refills 6.~~ Return in2 months with a timed DIGOXIN leveland a POTASSIUM Level.
1-9-98	No Show for labs on 1-6-98 - called pt re: missed labs - left message on answering machine - TSC
1-13-98	pt sent note by mail re: missed appt. for labs - TSC
3/11/98	Wt - 235 1/2 lbs. Follow up & Hyp. T. 98 stay.
	BP - 104/64
	DR. MARION DAVIS DICTATING:
	S: Follow up from the hospitalization. He has a form to be filled out for his worksite and copy of that will be in the chart. He continues to smoke cigarettes Says he has decreased down to 2 cigarettes per day but that is only down from 1 cigarette back in June of 1997. Was recently admitted for Congestive Heart

CONTINUED.

DAVIS CLINIC
PROGRESS NOTES

NAME: C.L. WILLIAMS DATE OF BIRTH: ACCT.: 599.3

DATE	
3-11-98	CONTINUED.
	Failure. He has severe dilated congestive cardiomyopathy with an ejection fraction on Echocardiogram of about 15 to 20%. He will see Dr. Jacocks on April 24.
	O: VITAL SIGNS AS ABOVE.
	PHYSICAL EXAM: Heart is regular without murmurs, rubs or gallops. LUngs are clear.
	A & P: 1) CHF, RESOLVED. He will continue on LANOXIN & LASIX at the doses. The entire medicine list is reviewed today and updated, clarified for him.
	2) TOBACCO ABUSE. HE absolutely has to stop smoking.
	I have praised and commended him for trying to continue to work. However, with an Ejection Fraction of 15 to 20%, I am not sure how long he will be able to continue. I have suggested that he ask Dr. Jacocks about disability and that he might consider applying for disability at this point. Return to me in 2 months with DIGOXIN Level, Panel 7, Magnesium Level.
4/98	Digoxin Level, Panel 7 & Magnesium Level drawn & sent to Kmc. B. Griffin
5-1-98	wt. 238 lbs. follow-up on labs & B/P
	T. 97.6
	B/P 128/80
	R 18 - ① TBS
	② HTN
	③ Ⓡ Mobile pain

DAVIS CLINIC
PROGRESS NOTES

NAME: C. L. WILLIAMS DATE OF BIRTH: ACCT.: 599.3

DATE ALLERGIES:

5-7-98 CONTINUED.

DR. MARION DAVIS DICTATING:

S: Follow up from hospitalization. He also follows up on labs and BP. He went to see Dr. Jacocks yesterday. I don't have a note about that yet. Also, complains of some right shoulder pain.

O: VITAL SIGNS AS ABOVE. BP 128/80.
PHYSICAL EXAM: Heart is regular without murmurs, rubs or gallops. LUngs show some coarse breath sounds bilaterally, left greater than right and he continues to smoke 2 or 3 cigarettes per day.
LABORATORY: Panel 7 non-fasting showed a Glucose of 129, CO2 21, DIGOXIN level therapeutic at 1.14.

A & P: 1) ELEVATED BLOOD SUGAR. PLAN - Return in 2 months with fasting Panel 7 which will give us the fasting blood sugar.

2) HYPERTENSION. PLAN - No change there.

3) RIGHT SHOULDER PAIN. PLAN - Prescribe NAPROSYN 375 mgs. 1 PO TID with food prn pain, #30, refills 1.

Return as above with a DIGOXIN Level and a MAGNESIUM because of the Congestive Cardiomyopathy and the fact that he takes MAGNESIUM.

MV

-28-98 DR. MARION DAVIS DICTATING: Note requesting refill on his "heart pills". I have had staff call in to SupRX on Liberty Street for LANOXIN 0.25 mgs. 1½ PO Q a.m., #100, refills 2.

MV

6-Jones

6-30-98 Panel 7, Digoxin level, Magnesium drawn ⇒ TRAC Lab

7-2-98 Wt. 242½ lbs. follow-up on labs

T. 98.5 185

B/P [illegible] & chol., (3) [illegible]

2 wks - FBS, FLP, Glycohb

DAVIS CLINIC
PROGRESS NOTES

NAME: C. L. WILLIAMS DATE OF BIRTH: ACCT.: 599.3

ALLERGIES:

DATE	
7-2-98	CONTINUED.
	DR. MARION DAVIS DICTATING:
	S: Follow up on labwork. He also complains of an area on the right lower ribcage. Calls it a lump. Labwork dated 6-30-98 shows Glucose high at 127. See the note of 5-7-98 where there was a random non-fasting Glucose of 129.
	O: VITAL SIGNS AS ABOVE. PHYSICAL EXAM: Right ribcage area shows what appears to be a soft lipoma about 2 inches in diameter.
	A & P: 1) ELEVATED BLOOD SUGAR. PLAN - Return in 2 months with fasting Panel 7 and Glyco Hgb. to see whether we can confirm Diabetes. He is to work on diet and as much exercise as possible in the meantime, given the fact that he has a cardiomyopathy.
	2) ELEVATED CHOLESTEROL. PLAN - That was not checked at this visit but we will have him return in 2 months with a fasting Lipid Profile.
	3) LIPOMA, RIGHT RIBCAGE AREA. We are having him just keep an eye on that and if it changes or becomes painful or obstructive, we will see about having it removed. Return as above.
	MD
8-27-98	TC - would like to see him today -- coming in between pts (LO)
8-28-98	Panel 7, Lipid Profile, Hgb A1C drawn => TRRC lab
9-3-98	No show for appt. w/ Dr. Marion Davis today - called and left message on answering machine - JM
9-4-98	Pt. called to reschedule missed appt. - JM

DAVIS CLINIC
PROGRESS NOTES

NAME: C L Williams DATE OF BIRTH: ACCT.: 599.3

DATE ALLERGIES:

9-8-98 Ht. 348 Lb. Follow up on Labwork 8/98
T. 98°
B/P 140/80
8/8:00AM @ Night Tri. 196 FBT, Glyco Hgb, FLP

DR. MARION DAVIS DICTATING:

S: Follow up on labwork from 8-28-98., Glyco Hgb. is high at 7.1. Fasting Panel 7 shows Serum Glucose at 177, Chol. 156, HDL 26, LDL 91, Trig. 196. This is an improvement in Cholesterol but a worsening in Trig. His Glucose is high for at least the 2nd fasting reading. Therefore, we have said that he has Diabetes.

O: VITAL SIGNS AS ABOVE. BP 140/80.
PHYSICAL EXAM NOT DONE.

A & P: 1) DIABETES. PLAN - Begin GLUCOTROL XL 5 mgs. 1 PO Q a.m., #100, refills 1.

2) HYPERLIPIDEMIA. He does not recall being on a diet for this other than eating "diet foods". We have reviewed with him the Low Trig. and Low Chol. diet sheets today and he is to start on those. Also, to get as much walking or bicycle riding as he can tolerate, given his disability from Severe Dilated Congestive Cardiomyopathy.

Return in 2 months with fasting Panel 7 for Hypertension and Diabetes, Glyco Hgb. for Diabetes, fasting Lipid Profile for Hyperlipidemia.

AM

10-7-98 DR. MARION DAVIS DICTATING: Note from 10-6-98. Mr. Williams had requested refill on CAPOTEN. Review of the chart shows that on 5-6-98 Mr. Williams had seen Dr. Jacocks who had stopped the CAPOTEN and changed over to ALTACE. However, the patient has been taking the CAPTOPRIL for the last 5 months. My staff called the patient and, apparently, he is unclear or somehow did not get the idea that he was supposed to stop CAPTOPRIL and start ALTACE. At any rate, I have had staff call in to SupRX on Liberty for ALTACE 5 mgs. 1 PO Q day, #100, refills 1. That is based on the note from Dr. Jacocks which is in our chart and dated 5-6-98. Copy of this note to Dr. Jacocks.

1-3-99 [illegible] for Labs (FBT, FLP, & HbgA1C)

4/10/99 Panel 7, Lipid Profile & HgbA1C drawn & sent to [illegible]
B. Hufford LPN

DAVIS CLINIC
PROGRESS NOTES

NAME: C.L. Williams DATE OF BIRTH: ACCT.: 5473

DATE ALLERGIES:

11/19/98 Wt - 224.85 Needed Follow up.
T. 99.3
BP. 124/80

A/P/D DM 2 mos: Dg, Mg, Glyco Hb
HTN FP7
? diff, voiding
↑ lipids FLP, LFT's

DR. MARION DAVIS DICTATING:

S: Follow up on labwork. From 11-10-98 the Fasting Panel 7 shows Glucose of 368, Cholesterol 220 - that is up. LDL is up to 149. Glyco Hgb. 13.7. We have a note from Dr. Jacocks where he was seen on October 21, 1998. Dr. Jacocks DC'd the NITROGLYCERIN PATCH. Increased his ALTACE. He is no longer on CAPTOPRIL. The nurse's note mentioned head cold but the patient did not complain of anything.

O: VITAL SIGNS AS ABOVE. Temp. 99.3.
PHYSICAL EXAM: Not done.

A & P: 1) DIABETES. PLAN - Increase the GLUCOTROL XL to 10 mgs. 1 PO Q day, #100, refills 1. Return in 2 months with a Glyco Hgb.

2) HYPERTENSION. BP is well controlled. Return with a fasting Panel 7.

3) DIFFICULTY VOIDING. THE patient actually requests a circumcision. Feels that the foreskin may be interfering with his voiding. The genital area is not examined today. We will refer him to Urology for evaluation of that.

4) HYPERLIPIDEMIA. PLAN - Start LIPITOR 10 mgs. 1 PO Q day, #100, refills 2. Return with a fasting LIpid Profile and Liver Function tests.

Return with DIGOXIN & MAGNESIUM for non-ischemic cardiomyopathy. All of those labs will be in preparation for his visit 2 months from now.

M. Davis

1/4/99 Digoxin level drawn & sent to time-S. Butt

1-5-99 Magnesium, Lipid Profile, Liver function, Panel 7
Hgb A1c drawn → Time Lab

DAVIS CLINIC
PROGRESS NOTES

NAME: C. L. Williams DATE OF BIRTH: ACCT.: 5493

DATE ALLERGIES:

1/12/99 Ht. 5'3 1/8 S Follow up - labwork mailed
T. 98.3
BP 106/60 P. 64 Irregular
B.P. @ Dr. Davis

DR. MARION DAVIS DICTATING:

S: Follow up on labwork from 1-5-99. Fasting Panel 7 shows Glucose of 228. Liver Panel is normal. Lipid Profile is normal. From 1-4-99, Glyco Hgb. is high at 13.2. DIGOXIN, which appears to be drawn at the correct time, is less than 0.3. He says he has not missed any doses or run out of his LANOXIN.

O: VITAL SIGNS AS ABOVE.
PHYSICAL EXAM: Heart is mostly regular with an occasional irregular beat. Pulse is 64. Lungs are clear.

A & P: 1) DIABETES. PLAN - Increase the GLUCOTROL XL 10 mgs. to 1 PO BID, #100, refills 2. REturn in 6 weeks with a fasting blood sugar and Glyco Hgb.

2) HYPERTENSION. PLAN - BP is a little low at 106/60. We make no changes there.

DIGOXIN Level is slightly sub-therapeutic but we will defer adjustment of that to Dr. Jacocks.

1-18-99 Per Dr. M. Davis / called to Super X on Liberty St.
Lasix 80 mg. 1 PO q AM, #100, Refill x 1 S. Griffin
Periactin 4 mg. 1 PO Tid PRN itching, #100, Refill x 11 S. Griffin

1-26-99 Periactin not really working well, V.O. per Dr. M. Davis called to Super-X Liberty St. Atarax 25 mg 1 PO Q 4° PRN itching, #30, Refill x 1 S. Griffin

02-15-02 C. L. Williams #17551

Mr. Williams reports for wound check 10 days after ICD implant. From a cardiovascular standpoint, he is well compensated and has actually had a 3 lb weight loss. He notes no dyspnea at all. He has not had syncope or ICD discharge. The ICD incision was inspected and found to be healing appropriately with no evidence of infection. He will return to the clinic in 4 wks for an OV.

MITCHELL W. JACOCKS, MD
MWJ/kls

cc: Dr. Marion Davis

C. L. Williams
NAME

Medtronic
MANUFACTURER

7275
MODEL

2-5-02
INSERTION DATE

?	DATE	MODE	V LEAD	A LEAD	BATTERY VOLTS	BATTERY CHG TIME	BATTERY HV IMPED	EPISODES	THERAPIES DELIVERED	COMMENT	DR
	10-21-04	VVI ~~DDD~~	I-776 S-1.40 T-1.0/0.2	I-481 S-5 T-1.0/0.2	2.66	8.07	18	123-NST	⊘		mj
	6-22-05	VVI	I-776 S-10 T-	I-444 S-5 T-	2.59	8.74	17	619-NST 4-VT	4-VT therapy all RX I	NO CHANGES	mj
	10-20-05	VVI	I-716 S-9 T-1.0/.3	I-444 S-5 T-	2.59	9.79	16	[redacted] NST 98	⊘	no changes	mj
	3:58 2-06	VVI	1776 S 9.0 T 1.0/0.3	1 481 S 5.0 T	2.58	9.99	17	454 NST 1 VT	VT- RX I (AF-SVT)	no changes	mj
	5-25-06	VVI	1776 S 11 T 1.0/0.3	1444 S 5. T	2.58	10.14	17	1 SVT 467 NST	⊘	No changes	mj
	8-10-06	VVI	I-1776 S-10 T-1.0/0.3	I-481 S-7.5 T-	2.58	10.14	17	21 NST	⊘	No changes	mj
	2-8-07	VVI	I 716 S 9 T 1V/.2	I 481 S 5 T 1V/.2	2.58	10.25	17	280-NST		No changes	mj
	5-17-07	VVI	I 776 S 9.5 T 1.0V/0.2	I 444 S 6.5 T	2.57	10.4	17	179 NST VT-2	VT RX I (SUCCESSFUL)	NONE	mj
	8/9/07	VVI	I 644 S 9 T-1V0.2	I 444 S 5 T	2.57	10.43	16	1 VT- type 2 break w/ 1 burst ATP 48 NST		Pt returned for alert feature.	mj
	10/12/07	VVI	I 716 S 9.0 MV T 1.0V/0.2	I 481 S 5.5 MV T	2.57	10.39	17	39 NST	⊘	PATIENT ALERT FOR B. VOLTAGE	mj
	1:1N.M										

C. J. Williams
NAME

Medtronic
MANUFACTURER

Maximo DR 7278
MODEL

11-15-07
INSERTION DATE

DATE	MODE	RV LEAD	LV LEAD	A LEAD	BATTERY VOLTS	BATTERY CHG TIME	BATTERY HV IMPED	EPISODES	THERAPIES DELIVERED	COMMENT	D
4-17-08	VVI	1.7Ω S 13.2 T 1.0V | .3		1 448 S 3.8 T 1.0V | 0.2	3.19	7.33	47	VF-aborted FVT-2 VT-2	FVT-ATP VT-ATP		

03-15-02 C. L. Williams #17551

VITALS: BP-94/58 P-120 WT-228.6 HT-5'10"

CHIEF COMPLAINT:

FU after ED visit on 3/9/02 with chief complaint of "I feel OK."

HISTORY OF PRESENT ILLNESS:

Pt is a 53 YO man with hypertension, Type II diabetes mellitus, COPD, and severe nonischemic cardiomyopathy, EF of 15-20% with symptomatic ventricular arrhythmia s/p Medtronic 7275 ICD implant in 2/00. He was recently seen in the ED with a complaint of ICD discharge. He was found to have atrial fibrillation with a rapid ventricular response, which reduced ICD discharge. Of note, the pt was hypokalemic. At that time, he had not taken his meds. Potassium was supplemented and home medications were resumed. Coumadin therapy was initiated, and he was discharged to home in satisfactory condition from the ED. He reports today for FU. Since hospital D/C, he has had no syncope, presyncope, or sustained palpitations. He denies any chest pressure, heaviness, or other anginal symptoms. He sleeps on two pillows chronically and has not noted any need to sleep at a higher elevation. He has not had LE edema, PND. He remains dyspneic with mild activity. He has had no syncope, presyncope, or ICD discharge.

CURRENT MEDICATIONS:

Toprol 50 mg qd, potassium 40 mEq qd, Lasix 80 mg bid, Theophylline ER 200 mg bid, Spironolactone 25 mg qd, Precose 25 mg tid, Pepcid 20 mg bid, Digitek 0.125 mg qd, Accupril 40 mg qd, Coreg 12.5 mg bid, Flomax 0.4 mg qd, Glucophage XR 1 gm bid, Captopril 50 mg q8h, Slo Mag 2 qd, Albuterol and Atrovent inhalers, Coumadin as directed, PTU 50 mg qd.

EXAMINATION:

NAD. A&O x 3. HEENT no JVD or carotid bruit. Chest CTAP with decreased breath sounds. No wheezing or rales. CV PMI is downwardly and laterally displaced, diffuse with LV heave. RRR with normal S1, S2, S3, S4. II/VI SEM at the apex radiating laterally. Abdomen soft, nontender. No organomegaly. No masses. Extremities with no clubbing, cyanosis, or edema.
The ICD site is appropriately healed.

EKG:

Revealed normal sinus rhythm, 100 beats/minute, left atrial enlargement, LVH with secondary ST-T abnormality.

(Continued)

IMPRESSION:

1. Nonischemic cardiomyopathy, overall fairly well compensated. However, his medical regimen is somewhat redundant, and pt is confused about his meds. I have gone over these meds extensively with him and provided written instructions. We will change the Captopril to tid until gone, then take 40 mg of Accupril qd. Toprol XL will be taken 50 mg bid until gone and then Coreg 12.5 mg bid will be resumed.
2. Paroxysmal atrial fibrillation, currently in normal sinus rhythm on Coumadin therapy.
3. With clinical hyperthyroidism, on PTU.
4. Hypertension, controlled.
5. Type II diabetes mellitus.
6. Chronic obstructive pulmonary disease.
7. Probable obstructive sleep apnea. Pt evaluation is in progression.
7. Medical noncompliance.

PLAN:

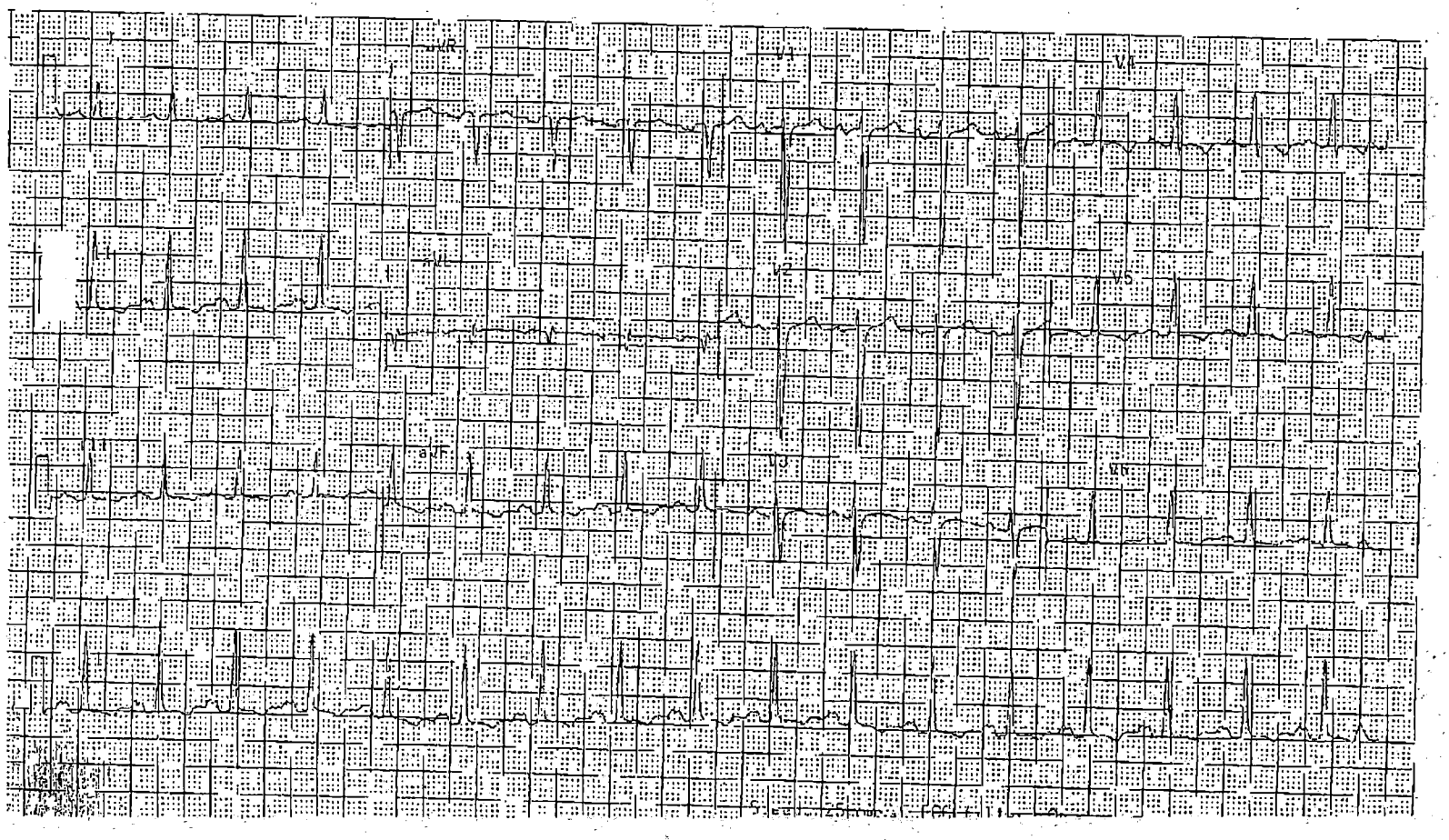
1. Medication changes as above.
2. Potassium, BUN, creatinine.
3. PT today.
4. Return to clinic in 4 wks.

MITCHELL W. JACOCKS, MD
MWJ/kls

cc: Dr. Marion Davis

Name: C L WILLIAMS
ID :
Date: 03/15/02 Time: 17:33
Age : 53 Sex : Male
Hgt : Wgt :
Med1:
Med2:
Race:
Cmnt:

BP: Rate: PR:
Qrs: Qt: Axis:
P Waves: T Waves: St Segment:
Interpretation:



04-12-02 C.J. Williams #17551

VITALS: BP-92/68 P-102 WT-228.8 HT-5'10"

CHIEF COMPLAINT:

FU congestive heart failure

HISTORY OF PRESENT ILLNESS:

Pt is a 53 YO man with hypertension, Type II diabetes mellitus, COPD, and severe nonischemic cardiomyopathy with EF 15-20% with symptomatic VT s/p implantation of a Medtronic 7275 ICD. He was recently seen in the ED with ICD D/C secondary to atrial fibrillation. He has been treated with Coumadin therapy and continued rate control. Since last seen, he has not had syncope, near syncope, and he has noted no palpitations. He has not had ICD D/C. He denies any chest pressure, heaviness, or other anginal symptoms. He is ambulating and not limited by dyspnea. He sleeps on two pillows chronically and has not had increased orthopnea, PND, or LE edema.

CURRENT MEDICATIONS:

Potassium 40 mEq qd, Lasix 80 mg bid, Theophylline ER 200 mg bid, Aldactone 25 mg qd, Precose 25 mg tid, Pepcid 20 mg bid, Digitek 0.125 mg qd, Accupril 40 mg qd, Coreg 12.5 mg bid, Flomax 0.4 mg qd, Glucophage XR 1 gm bid, Slow-Mag 2 qd, Coumadin as directed, PTU 50 mg qd, Albuterol and Atrovent inhalers.

EXAMINATION:

NAD. A&O x 3. HEENT no JVD or bruit. Chest CTAP. No wheezing or rales. CV PMI is downwardly displaced with LV heave. RRR with normal S1, S2, S3, and S4. II/VI SEM at the apex radiating laterally. Abdomen soft, nontender. No organomegaly. No masses. No palpable AAA. Extremities with no clubbing, cyanosis, or edema. ICD incision is well healed.

DATA REVIEWED:

P7 on 3/18/02 remarkable for a sodium of 134, BUN 22, creatinine 1.4.

IMPRESSION:

1. Nonischemic cardiomyopathy, currently New York Heart Class I-II.
2. Paroxysmal atrial fibrillation - currently normal sinus rhythm on Coumadin therapy.
3. Subclinical hyperthyroidism on PTU.
4. Hypertension, controlled.

(Continued)

5. Type II diabetes mellitus.
6. COPD.
7. Obstructive sleep apnea, work up in progress.
8. History of noncompliance.

PLAN:

1. Continue current meds.
2. Return to clinic in 2 months for ICD check.

MITCHELL W. JACOCKS, MD
MWJ/kls

cc: Dr. Marion Davis

06/07/02 C. L. Williams #17551

Ht- 5' 10" Wt- 225.2 Bp- 92/68 P- 76

CHIEF COMPLAINT: ICD FU

HISTORY OF PRESENT ILLNESS:

This 53 yo man w/HTN, DM-II, COPD, nonischemic cardiomyopathy, EF 15-20% with ventricular tachycardia, now s/p implantation of Medtronik 7275 ICD. He reports today for regular FU. Since last seen he has had no syncope or ICD discharge. He notes no chest symptoms suggestive of angina. He sleeps on 2 pillows chronically. He has not had increased orthopnea, PND, or LE edema.

CURRENT MEDICATIONS:

K Cl 40 mEq qd; Lasix 80 mgs bid; Theophylline 200 mgs bid; Aldactone 25 mgs qd; Precose 25 mgs tid; Pepcid 20 mgs bid; Digitek 0.125 mgs qd; Accupril 40 mgs qd; Coreg 12.5 mgs bid; Flomax 0.4 mgs qd; Glucophage XR 1 gm bid; Albuterol and Atrovent inhalers; coumadin as directed; PTU 50 mgs qd.

PHYSICAL EXAMINATION: NAD, AXOX3.

HEENT: No JVD; no bruits.
Chest: CTAP.
CV: PMI downwardly displaced with LV heave, RRR, normal S1 & S2; S3 no S4; II/VI SEM at apex radiating laterally.
Abdomen: Soft, nontender, no palpable AAA, no appreciable organomegaly or mass.
Extremities: No clubbing, cyanosis or edema.

ICD INTERROGATION:

Battery 3.16 v; atrial imp. 481 Ohms; ventricular imp. 716 Ohms; P-waves 5 mv; R-wave 12 mv; atrial threshold 3 v @ .06 msecs; ventricular threshold 4v @</= .03 msecs. Ventricular tachycardia was treated twice with ATP therapy with success.

ASSESSMENT:

1. Nonischemic cardiomyopathy, New York Heart Class II.
2. Paroxysmal atrial fib. currently in NSR, on coumadin therapy.
3. Subclinical hypothyroidism on PTU.
4. HTN, controlled.
5. Symptomatic ventricular arrhythmia with normal ICD function.
6. COPD.
7. DM.
8. Sleep apnea: he is undergoing evaluation.
9. Medical noncompliance.

PLAN:

1. Cont. current meds.
2. RTC 3 months for ICD check.

MITCHELL W. JACOCKS, MD
MWJ/jkh

xc: Dr. Marion Davis

08-13-02 C. L. Williams #17551

VITALS: Bp-102/70 P-88 Wt.-224.0

CHIEF COMPLAINT: ICD FU.

HISTORY OF PRESENT ILLNESS:

53 YO man with HTN, DM-II, COPD, nonischemic cardiomyopathy, EF 15-20%, w/ventricular tachycardia, s/p Medtronik 7275 ICD implant 02/02. Since last seen Mr. Williams has had persistent dry cough without fever or hemoptysis. He has not had syncope or near-syncope, no chest pressure, heaviness, or other anginal symptoms. He has not had ICD discharge. He sleeps on 2 pillows chronically with no change. He has not had LE edema or PND. He has dyspnea with mild activity.

MEDICATIONS:

K Cl 20 mEq bid; Lasix 80 mgs bid; Theophylline ER 200 mgs bid; Aldactone 25 mgs qd; Precose 25 mgs tid; Pepcid 20 mgs bid; Digitek 0.125 mgs qd; Accupril 40 mgs qd; Coreg 12.5 mgs bid; Flomax 0.4 mgs qd; Glucophage XR 1.0 gm bid; Slo-Mag 2 qd; PTU 50 mgs qd.

EXAMINATION:

NAD. A&OX3. HEENT-no JVD or bruit. Chest CTAP. CV: PMI downwardly and laterally displaced w/LV heave, RRR, normal S1, S2; no audible S3 or S4, II/VI SEM at apex radiating laterally. Abd. no palpable AAA, organomegaly, or mass; active bowel sounds. Ext. no clubbing, cyanosis or edema. Neurologic exam is nonfocal.

DATA:

ICD Interrogation demonstrates 3 episodes of ventricular tachycardia 07/10, 07/22, & 08/01, terminated by ATV pacing or low energy cardioversion. Battery 3.14 v, atrial impedance 444 Ohms, ventricular imp. 661 Ohms, R-waves 12 mv, P-waves 7 mv, atrial threshold 2 v at .06 msecs, ventricular threshold 2 v at .06 msecs. Counters were then cleared.

ASSESSMENT:

1) Nonischemic cardiomyopathy, New York Heart Class III.
2) Peristernal a-fib, currently in NSR on coumadin therapy.
3) Subclinical hypothyroidism on PTU.
4) HTN, controlled.
5) VT with normal ICD function.
6) COPD.
7) DM.
8) Sleep apnea: to undergo sleep study later this month.

PLAN:

1) Change Accupril to Avapro X 2 weeks to see if this improves his cough. Cont. other medications.
2) RTC 3 months for ICD check.

MITCHELL W. JACOCKS, MD
MWJ/jkb

11/19/02 Williams, C. L. 1755

VITALS: BP-98/62 P-74 WT-231 lbs. HT-6' 0"

CHIEF COMPLAINT: ICD follow-up

HISTORY OF PRESENT ILLNESS:

53-year-old man with hypertension, diabetes mellitus, COPD, nonischemic cardiomyopathy with LVEF 15-20% and ventricular tachycardia status post implantation of a Medtronic 7275 ICD 2/02. Since last seen he has not had chest pain, pressure, heaviness, or other systems typical angina. He sleeps on two pillows chronically and has noted no need to sleep more upright, no edema, PND. He has chronic dyspnea with mild activity which is unchanged from his baseline. He has not had syncope, palpitations or ICD discharge.

MEDICATIONS:

KCL 20 mEq b.i.d., Lasix 80 mg b.i.d., theophylline ER 200 mg b.i.d., Aldactone 25 mg q.d., Coumadin as directed, PTU 50 mg q.d., Precose 25 mg t.i.d., Pepcid 20 mg q.d., digitalis.125 mg q.d., Coreg 12.5 mg b.i.d., Glucophage XR 1 gram b.i.d., slow Mag two q.d., Flomax .4 mg q.d.

EXAMINATION:

NAD, alert., oriented. HEENT: No JVD or brult. Chest CTAP. No wheezing or rales. CV: PMI laterally displaced with LV heave, regular rhythm with normal S1, S2, no S3 or 4, 2/6 SEM audible at the apex radiating laterally, peripheral pulses are palpable without bruit abdomen: Soft, nontender, no palpable AAA. Extremities no clubbing, cyanosis, or edema.

DATA:

ICD interrogation: Battery 3.11V, atrial impedance 444 ohms, ventricular impedance 661 ohms, atrial threshold 2 V at .06 milliseconds, ventricular threshold 2 V at .06 milliseconds, P-wave = 8 mV, R-wave = 12 mV, ATP pacing was delivered for what appears to be atrial fibrillation no other therapy was delivered, counts were cleared.

IMPRESSION:

1. Nonischemic cardiomyopathy. Accupril was discontinued secondary to cough; his cough has significantly improved off ACE inhibitor, unfortunately he did not fill his prescription for Avapro.
2. Ventricular arrhythmia with normal ICD function
3. Transient A fib on Coumadin anticoagulation.
4. Hypertension, controlled

5. Subclinical hypothyroidism on PTU
6. COPD
7. Diabetes mellitus
8. Sleep apnea

PLAN:

1. Continue current medications with RTC 3 months for ICD check
2. Obtain TFTs and CBC on PTU.
3. Start Diovan at the time of next appointment

Mitchell W. Jacocks, M. D.

cc: Dr. Marion Davis

12/3/02 Williams, C.L. 17551

VITALS: BP-92/60 P-90 WT-232 lbs. HT-5' 10"

CHIEF COMPLAINT: "I got shock"

HISTORY OF PRESENT ILLNESS:

53 remained hypertension, diabetes mellitus, COPD, OSA and a nonischemic cardiomyopathy with LVEF 15-20% status post implantation of a Medtronic 7275 ICD 2/02. He reports today for an unscheduled visit with complaints of device discharge not decision was syncope or palpitations. He denies medical noncompliance. He continues to sleep on two pillows chronically without change and has not had PND, edema or worsening dyspnea over his baseline. He has not had chest pain, pressure, heaviness or other systems of angina.

MEDICATIONS:

KCL 20 mEq b.i.d., Lasix 80 mg b.i.d., theophylline ER 200 mg b.i.d., Aldactone 25 mg q.d., Precose 25 mg t.i.d., Pepcid 20 mg q.d., Lanoxin .25 mg q.d., Coreg 12.5 mg b.i.d., Flomax .4 mg q.d., Glucotrol is XR 1 gram b.i.d., slow Mag -2 q.d., Coumadin as directed, PTU 50 mg q.d.

EXAMINATION:

NAD, alert, oriented. HEENT: No JVD or bruit. Chest: CTAP. No rales. CV: PMI laterally displaced LV heave, regular rhythm with normal S1, S2, no S3, soft S4. 2/6 SEM at the apex radiating laterally. Peripheral pulses are palpable without bruit Abdomen: Soft, nontender, no palpable AAA. Extremities: No clubbing, cyanosis, or edema.

DATA:

ICD interrogation: Battery 3.10 volts, atrial impedance 444 ohms, ventricular impedance 661 ohms, atrial threshold 3 volt at < .03 msec, ventricular threshold to volt at < .03 msec, R-wave = 12 mV, P-wave = 5 mV, two episodes of ventricular tachycardia occurred which were treated by ATP therapy which failed, both episodes were then terminated with a 5 joule cardioversion. Counters were subsequently cleared.

IMPRESSION:

1. Nonischemic cardiomyopathy overall at his baseline. He has not refilled his Accupril as requested
2. Ventricular tachycardia with normal ICD function. Of note he was seen in ED after one discharge and was discharged home.
3. Transient atrial arrhythmia on chronic Coumadin anticoagulation

4. Hypertension, controlled
5. COPD
6. OSA
7. Subclinical hyperthyroidism on PTU. He has not obtained the requested labs
8. Diabetes mellitus.

PLAN:

1. Discontinue Coreg, add Toprol XL 100 mg q.d.
2. RTC three months, continue regular follow-up with Dr. Davis

Mitchell W. Jacocks, M. D.

cc: Dr. Davis

3/21/03 Williams, C.L. 17551

VITALS: BP-120/70 P-92 WT-232 lbs. HT-5' 10"

CHIEF COMPLAINT: " I'm feeling OK"

HISTORY OF PRESENT ILLNESS:

53 year old male with hypertension, diabetes mellitus, COPD, OSA and a nonischemic cardiomyopathy with LVEF 15-20% status post implantation of a Medtronic 7275 ICD 2/02. Since last seen he's had no chest pain, pressure, heaviness or any other symptoms of angina. He has dyspnea with mild to moderate activity but has not had PND, orthopnea or edema. No syncope or ICD discharge. No TIA like symptoms. He denies medical noncompliance.

MEDICATIONS:

KCL 20 mEq b.i.d., Lasix 80 mg b.i.d., theophylline ER 200 mg b.i.d., Aldactone 25 mg q.d., Precose 25 mg t.i.d., Pepcid 20 mg q.d., Lanoxin .25 mg q.d., Flomax .4 mg q.d., Glucotrol is XR 1 gram b.i.d., slow Mag -2 q.d., Coumadin as directed, PTU 50 mg q.d., Toprol XL 100 mg q.d.

EXAMINATION:

NAD, alert, oriented. HEENT: No JVD or bruit. Chest: CTAP. No rales. CV: PMI laterally displaced LV heave, regular rhythm with normal S1, S2, no S3, soft S4. 2/6 SEM at the apex radiating laterally. Peripheral pulses are palpable without bruit Abdomen: Soft, nontender, no palpable AAA. Extremities: No clubbing, cyanosis, or edema.

DATA:

ICD interrogation: Battery 3.10 volts, atrial impedance 444 ohms, ventricular impedance 716 ohms, atrial threshold 3 volt at < .03 msec, ventricular threshold 2 volt at < .06 msec, R-wave = 12 mV, P-wave = 7 mV. No therapy delivered counters were cleared.

IMPRESSION:

1. Nonischemic cardiomyopathy overall at his baseline.
2. Ventricular tachycardia with normal ICD function.
3. Transient atrial arrhythmia on chronic Coumadin anticoagulation
4. Hypertension, controlled
5. COPD
6. OSA
7. Subclinical hyperthyroidism on PTU.
8. Diabetes mellitus.

PLAN:

1. Increase Toprol XL to 200 mg q.d. over the next two weeks. Restart ACE inhibitor at the time of next office visit. RTC 3 months

Mitchell W. Jacocks, M. D.

cc: Dr. Davis

6/18/03 Williams, C.L. 17551

VITALS: BP-106/68 P-82 WT-226 lbs. HT-5' 10"
CHIEF COMPLAINT: " I'm about the same "
HISTORY OF PRESENT ILLNESS:
53 year old male with hypertension, diabetes mellitus, COPD, OSA and a nonischemic cardiomyopathy with LVEF 15-20% status post implantation of a Medtronic 7275 ICD 2/02. He's had no chest tightness, heaviness, pressure or other symptoms of angina. He continues to have dyspnea with mild activity which is unchanged from his chronic baseline, he has limited his activity due to dyspnea but has not had PND, orthopnea or edema. He's had no syncope, presyncope, palpitations, slurred speech or any other TIA like symptoms. No bleeding complication on Coumadin.

MEDICATIONS:
KCL 20 mEq b.i.d., Lasix 80 mg b.i.d., theophylline ER 200 mg b.i.d., Aldactone 25 mg q.d., Precose 25 mg t.i.d., Pepcid 20 mg q.d., Lanoxin .25 mg q.d., Flomax .4 mg q.d., Glucotrol is XR 1 gram b.i.d., slow Mag -2 q.d., Coumadin as directed, PTU 50 mg q.d., Toprol XL 100 mg q.d.

EXAMINATION:
NAD, alert, oriented. HEENT: No JVD or bruit. Chest: CTAP. No rhonchi or rales. CV: PMI laterally displaced LV heave, regular rhythm with normal S1, S2, no S3, soft S4. 2/6 SEM at the apex radiating laterally. Peripheral pulses are palpable without bruit Abdomen: Soft, nontender, no palpable AAA. Extremities: No cyanosis or edema, warm.

DATA:
ICD interrogation: Battery 3.03 volts, atrial impedance 481 ohms, ventricular impedance 776 ohms, atrial threshold 2 volt at .06 msec, ventricular threshold 2 volt at .06 msec, R-wave = 12 mV, P-wave = 7 mV. No therapy delivered counters were cleared.

IMPRESSION:
1. Nonischemic cardiomyopathy , New York heart class 3
2. Ventricular tachycardia , normal ICD function.
3. Transient atrial arrhythmia on chronic Coumadin anticoagulation, asymptomatic
4. Hypertension, controlled
5. COPD
6. OSA, sleep study was not done
7. Subclinical hyperthyroidism on PTU.
8. Diabetes mellitus.

PLAN:

1. Continue current medications RTC in 3 months for ICD check. He would benefit from Natrecor infusion and will be enrolled in the Natrecor Clinic.

Mitchell W. Jacocks, M. D.

cc: Dr. Davis

2/26/04 Williams, C.L. 17551

VITALS: BP-126/76 P-94 WT-223 lbs. HT-5' 10"
CHIEF COMPLAINT: " I'm feeling okay "
HISTORY OF PRESENT ILLNESS:
54 year old male with hypertension, diabetes mellitus, COPD, OSA and a nonischemic cardiomyopathy with LVEF 15-20% status post implantation of a Medtronic 7275 ICD 2/02. He reports continued dyspnea with mild activity without change in his baseline; he has not had edema, orthopnea or PND. He denies chest fullness, pressure, heaviness or other angina symptoms. He's not had palpitations, syncope or near syncope. He denies ICD discharge. He has undergone sleep study but does not comply with CPAP.

MEDICATIONS:
KCL 20 mEq b.i.d., Lasix 80 mg b.i.d., theophylline ER 200 mg b.i.d., Aldactone 25 mg q.d., Precose 25 mg t.i.d., Pepcid 20 mg q.d., Lanoxin .25 mg q.d., Flomax .4 mg q.d., Glucotrol is XR 1 gram b.i.d., slow Mag -2 q.d., Coumadin as directed, PTU 50 mg q.d., Toprol XL 200 mg q.d.

EXAMINATION:
NAD, alert, oriented. HEENT:, No JVD at 45°, no carotid bruit. Chest: CTAP: No or rhonchi rales. CV: PMI laterally displaced LV heave, regular rhythm with normal S1, S2, no S3, soft S4. 2/6 SEM at the apex radiating laterally. Peripheral pulses are palpable without bruit Abdomen: Soft, nontender, active bowel sounds, nondistended, no palpable AAA. Extremities: No edema, no rash or ulceration.
DATA:
ICD interrogation: Battery 2.93 volts, atrial impedance 444 ohms, ventricular impedance 776 ohms, atrial threshold 2 volt at .06 msec, ventricular threshold 2 volt at .06 msec, R-wave = 6 mV, P-wave = 4 mV. One episode of ventricular tachycardia was terminated by ATP pacing.
IMPRESSION:
1. Nonischemic cardiomyopathy , New York heart class 2-3
2. Ventricular tachycardia , normal ICD function.
3. Transient atrial arrhythmia on chronic Coumadin anticoagulation, asymptomatic
4. Hypertension, controlled
5. COPD
6. OSA, noncompliant
7. Subclinical hyperthyroidism on PTU.
8. Diabetes mellitus.
PLAN:
1. Continue current medications with RTC three months for ICD check.

Mitchell W. Jacocks, M. D.
cc: Dr. Davis

11/24/04 Williams, C.L. 17551

VITALS: BP-136/88 P-115 WT-228.0 lbs. HT-5' 10"
CHIEF COMPLAINT: " I'm doing just fine "
HISTORY OF PRESENT ILLNESS:
54 year old male with hypertension, diabetes mellitus, COPD, OSA, paroxysmal atrial fibrillation and a nonischemic cardiomyopathy with LVEF 15-20% status post implantation of a Medtronic 7275 ICD 2/02. He presented to the office today with complaints of ICD discharge. He states that after a meal and while at rest, he noticed ICD discharge which was not associated with palpitations or syncope. He has been noncompliant with medications secondary to cost. He denies any chest pain, tightness or pressure. He's had no increased dyspnea of her baseline and no PND, orthopnea or edema.

MEDICATIONS:
KCL 20 mEq b.i.d., Lasix 80 mg b.i.d., theophylline ER 200 mg b.i.d., Aldactone 25 mg q.d., Glycet 25 mg t.i.d., Pepcid 20 mg q.d., Lanoxin .25 mg q.d., Flomax .4 mg q.d., slow Mag -2 q.d., Coumadin as directed, PTU 50 mg q.d., Toprol XL 200 mg q.d., Glucophage XR 850 mg t.i.d., Avandamet 4/1000 b.i.d.

EXAMINATION:
NAD, alert, oriented. HEENT: No JVD at 45°, no bruit. Chest: CTAP. No rhonchi or rales. Nontender. CV: PMI laterally displaced with LV heave, regular rhythm with normal S1, S2, no S3, soft S4. 2/6 SEM at the apex radiating laterally. Abdomen: Soft, nontender, active bowel sounds, nondistended, no guarding or rebound. Extremities: No edema, no rash

DATA:
ICD interrogation: Battery 2.64 volts, atrial impedance 444 ohms, ventricular impedance 716 ohms, atrial threshold 1 volt at .2 msec, ventricular threshold 1 volt at .2 msec, R-wave = 12 mV, P-wave = 5 mV. He has received three episodes of therapy; one episode was for atrial fibrillation which fell within the VT zone and two episodes of ventricular tachycardia terminated with ATP pacing.

IMPRESSION:
1. Nonischemic cardiomyopathy , New York heart class 2-3
2. Ventricular tachycardia , normal ICD function. He has received therapy for ventricular tachycardia as well as atrial arrhythmia. Of note he is not taken Toprol XL in four days.
3. Transient atrial arrhythmia on chronic Coumadin anticoagulation
4. Hypertension, controlled
5. COPD
6. OSA, noncompliant
7. Subclinical hyperthyroidism on PTU.
8. Diabetes mellitus.
9. Polypharmacy

PLAN:
1. Compliance with medication was emphasized and Toprol samples were provided. Pharmacy assistance forms have been filled out. Check potassium, magnesium, creatinine. He will continue regular follow-up as previously scheduled.

Mitchell W. Jacocks, M. D.
cc: Dr. Davis

7/16/04 Williams, C.L. 17551

VITALS: BP-124/74 P-88 WT-224.6 lbs. HT-5' 10"
CHIEF COMPLAINT: " I'm doing just fine "
HISTORY OF PRESENT ILLNESS:
54 year old male with hypertension, diabetes mellitus, COPD, OSA and a nonischemic cardiomyopathy with LVEF 15-20% status post implantation of a Medtronic 7275 ICD 2/02. He has not been very active and denies any chest tightness, heaviness, fullness or other anginal quality symptoms. He has dyspnea with moderate activity but no PND, orthopnea or edema. He has not had palpitations, syncope or near syncope and he denies ICD discharge. He has had no TIA like symptoms and denies any bleeding complication on Coumadin.

MEDICATIONS:
KCL 20 mEq b.i.d., Lasix 80 mg b.i.d., theophylline ER 200 mg b.i.d., Aldactone 25 mg q.d., Glycet 25 mg t.i.d., Pepcid 20 mg q.d., Lanoxin .25 mg q.d., Flomax .4 mg q.d., slow Mag -2 q.d., Coumadin as directed, PTU 50 mg q.d., Toprol XL 200 mg q.d., Glucophage XR 850 mg t.i.d., Avandamet 4/1000 b.i.d.

EXAMINATION:
NAD, alert, oriented. HEENT: No JVD at 30°, no carotid bruit. Chest: CTAP. No wheezes or rales. Nontender. CV: PMI laterally displaced with LV heave, regular rhythm with normal S1, S2, no S3, soft S4. 2/6 SEM at the apex radiating laterally. Abdomen: Soft, nontender, active bowel sounds, nondistended, no guarding or rebound. Extremities: No edema, warm, dry

DATA:
ICD interrogation: Battery 2.79 volts, atrial impedance 444 ohms, ventricular impedance 716 ohms, atrial threshold 2 volt at .06 msec, ventricular threshold 2 volt at .06 msec, R-wave = 7 mV, P-wave = 4 mV. Counters cleared. No therapy delivered.

IMPRESSION:
1. Nonischemic cardiomyopathy , New York heart class 2-3
2. Ventricular tachycardia , normal ICD function.
3. Transient atrial arrhythmia on chronic Coumadin anticoagulation
4. Hypertension, controlled
5. COPD
6. OSA, noncompliant
7. Subclinical hyperthyroidism on PTU.
8. Diabetes mellitus.
9. Polypharmacy

PLAN:

1. Continue present cardiac medications; I have asked him to see Dr. Davis regarding clarification of his diabetic drugs as he seems to be on a very high dose of metformin.
2. Check LFTs, TFTs, lipid panel, hemoglobin, creatinine, potassium
3. *ICD interrogation three months*
4. RTC 6 months for office visit

Mitchell W. Jacocks, M.D.

cc: Dr. Davis

TUOMEY HEALTHCARE SYSTEM
129 N. WASHINGTON STREET
SUMTER, SC 29150

CONSULTATION NOTE

17501

Name: WILLIAMS,C L
Att. Dr: MEDICAL SERVICES - TMP
Adm. Dr: MEDICAL SERVICES - TMP
Admit Date: 01/01/05
Location: TELE

MR#: 271254
RM#: 325-1
DOB:
Disharge Date:
Service: Medicine

NAME: WILLIAMS, C L

U#: 271254

ACCOUNT NUMBER: 12341489

DATE OF BIRTH:

DATE OF CONSULTATION: 01/01/2005

REQUESTING PHYSICIAN: WILMOT S MCCOLLOUGH III, MD

REASON FOR CONSULTATION: Congestive heart failure.

HISTORY: This is a 56-year-old black male who has a history of known nonischemic cardiomyopathy with severely depressed LV function. His EF is approximately 15% to 20%. He is status post implantation of a Medtronic 7275 ICD in February 2002. He has had fairly stable class 2 to 3 heart failure. He says over the last several weeks he has noted increased shortness of breath. His medications are somewhat unclear. He listed Coreg and Toprol as medications, although by Dr. Mitchell Jacocks' note from February 2004, he was only supposed to be on Toprol. He had been on Coreg in the past. The patient also admits to some dietary indiscretion over the holidays and has probably had several high salt meals. He was seen in the emergency room on December 28, 2004, and given IV Lasix. This improved his symptoms and he was discharged home. He had recurrent symptoms of shortness of breath, however, and returned to the emergency room on the 31st of December and was admitted for heart failure exacerbation. He has been started on IV Natrecor and given IV Lasix. He has improved, but is still not quite back to baseline.

PAST MEDICAL HISTORY: Remarkable for the nonischemic cardiomyopathy with an EF of 15% to 20%. He had cardiac catheterization in 1998 that showed no significant coronary disease. He had an ICD implanted in February 2002 after having long episodes of nonsustained ventricular tachycardia. He had discharge of his device around Thanksgiving, but has had no other discharges. He has a history of hypertension and COPD. His FEV1 is 69% of predicted. He has type 2 diabetes. He has a history of heavy alcohol use in the past, but none over the last 6 years. He also has obstructive sleep apnea, but does not use his CPAP as he should. He has a history of hyperthyroidism. HE HAS NO KNOWN DRUG ALLERGIES.

MEDICATIONS: As per Dr. Mitchell Jacocks' note from February 2004 was potassium chloride 20 mEq b.i.d., Lasix 80 mg b.i.d., theophylline SR 200 mg

b.i.d., Aldactone 25 mg per day, Precose 25 mg t.i.d., Pepcid 20 mg per day, Lanoxin 0.25 mg per day, Flomax 0.4 mg per day, Glucotrol XR 1 g b.i.d., Slow-Mag 2 per day, Coumadin as directed, PTU 50 mg per day, and Toprol XL 200 mg per day. It is not clear exactly what medications the patient is taking. On the ER note, the following medications were listed: Albuterol inhaler, theophylline, K-Dur, Glucophage, Lasix, amitriptyline, spironolactone, Flomax, Accupril, Lanoxin, Toprol, Pepcid, Coreg, Precose, Flovent, Humalog, and Lescol. The patient says that he ran out of his Toprol around Thanksgiving.

FAMILY HISTORY: His mother died in her 30s from sudden death. His father died, also in his 30s, from cerebral hemorrhage. Another brother apparently died of sudden death at approximately age 30.

SOCIAL HISTORY: The patient is married and lives with his wife. He has a 4th grade education and cannot read or write. He is on disability. He has a history of alcohol and tobacco use in the past, but none over the last 6 to 8 years.

REVIEW OF SYSTEMS: He denies any fevers or chills. He has not had any auditory or visual problems. He has not had any significant chest pain. He has had the shortness of breath as noted above. He denies any nausea, vomiting, diarrhea, hematuria, dysuria or discharge, trauma, rash, extremity weakness, depression, excessive thirst, or abnormal bleeding.

PHYSICAL EXAMINATION: On physical exam, his blood pressure is 108/65 with a pulse of 81. He is afebrile. In general, he is a well-developed, well-nourished, obese black male in no acute distress, although he is mildly short of breath when he talks. He is able to lie fairly flat without difficulty. HEENT Exam: Normocephalic and atraumatic. Pupils equal, round, and reactive to light. His dentition is poor. His neck is supple. No adenopathy or thyromegaly. He has JVD halfway up at 45 degrees. His lungs have mild diffuse wheezes throughout with crackles in both bases. Cardiac exam is regular rate and rhythm with occasional early beat; 2/6 systolic murmur. Normal S1 and S2. No S3 or S4 could be appreciated. His PMI could not be appreciated, but his chest is very thick. His abdomen is obese, soft, and nontender with no masses or hepatosplenomegaly. His extremities show no significant clubbing, cyanosis, and no significant edema. His pulses are all 2+ and symmetric throughout with no bruits. Neurologically, he is alert and oriented X3 with no focal deficits. His skin is warm and dry with no rashes.

His EKG shows a sinus tachycardia with frequent PACs and diffuse nonspecific

TUOMEY HEALTHCARE SYSTEM
129 N. WASHINGTON STREET
SUMTER, SC 29150

CONSULTATION NOTE

Name: WILLIAMS,C L
Att. Dr: MEDICAL SERVICES - TMP
Adm. Dr: MEDICAL SERVICES - TMP
Admit Date: 01/01/05
Location: TELE

MR#: 271254
RM#: 325-1
DOB:
Discharge Date:
Service: Medicine

ST-T wave abnormalities.

LABORATORY DATA: His sodium is 139, potassium 3.2, chloride 102, CO2 26, serum glucose 95, BUN 22, creatinine is 1.1, calcium of 9.2. Protein 6.9, albumin 3.3, total bilirubin of 0.7. His SGPT and SGOT are both elevated at 361 and 222 with an alkaline phosphatase of 162. His CK total is 36. His BNP at time of admission was 1180. His white count is 8700, hemoglobin 14.5, hematocrit 43.8, with 291,000 platelets. His PT is 29.9 with an INR of 3.13. His troponin I is less than 0.04. His digoxin level is less than 0.1 and his theophylline level is less than 2. His TSH is 0.45.

ASSESSMENT: Patient with a severe nonischemic cardiomegaly who presents with CHF exacerbation. I suspect that there is a combination of dietary and medical noncompliance contributing to this exacerbation.

RECOMMENDATIONS: Would recommend discontinuing Coreg. He has been continued on Toprol. His wife needs to bring in all of his medications so that they can be reviewed. He has already been started on Natrecor and I agree with the Natrecor for 48 hours. The patient may be a candidate for the FUSION-II trial, although it is unclear how reliable he would be. Medical therapy is all that can be offered.

CHARLES JACOCKS, MD

Dict. Date: 01/01/2005 Trans. Date: 01/01/2005
578905 / 33905 /
Job: 1990

TUOMEY HEALTHCARE SYSTEM
129 N. WASHINGTON STREET
SUMTER, SC 29150

CONSULTATION NOTE

Name:	WILLIAMS,C L	MR#:	271254
Att. Dr:	MEDICAL SERVICES - TMP	RM#:	325-1
Adm. Dr:	MEDICAL SERVICES - TMP	DOB:	
Admit Date:	01/01/05	Discharge Date:	
Location:	TELE	Service:	Medicine

CC: MARION DAVIS, MD
WILMOT MCCOLLOUGH, III, MD

<SIGNED ELECTRONICALLY>

Electronically Signed by: JACOCKS,CHARLES WEST

6/13/06 Williams, C.L. 17551

VITALS: BP-120/68 P-74 WT-225.0 lbs. HT-5' 10"
CHIEF COMPLAINT: " I'm doing just fine "
HISTORY OF PRESENT ILLNESS:
56 year old male with hypertension (> 10 year duration), diabetes mellitus (> 10 year duration), COPD, OSA, paroxysmal atrial fibrillation and a nonischemic cardiomyopathy with LVEF 15-20% status post implantation of a Medtronic 7275 ICD 2/02. He has not had syncope, palpitations or ICD discharge. He denies any chest heaviness, pressure, tightness or symptoms of angina. He has chronic dyspnea with more activity but no change from baseline. He denies orthopnea or edema. He has not had PND. He denies any focal weakness, slurred speech, facial droop or symptoms of TIA and he's had no bleeding problems on Coumadin. He denies claudication. Otherwise, he's without complaint but has been sporadically compliant with medications secondary to cost.

MEDICATIONS:
KCL 20 mEq b.i.d., Lasix 80 mg b.i.d., Lanoxin .25 mg q.d., Flomax .4 mg q.d., magnesium oxide 400 mg q.d., Coumadin as directed, PTU 50 mg q.d., Toprol XL 200 mg q.d., Accupril 40 mg q.d., Atrovent, Avandia 8 mg q.d.

EXAMINATION:
NAD, alert, oriented. HEENT: No JVD at 45°, no bruit. Chest: CTA, no wheezing, no rhonchi. No CVA or spinal tenderness. Symmetric chest expansion. CV: PMI laterally displaced with LV heave, regular rhythm with normal S1, S2, no S3, S4. 2/6 SEM apex. Abdomen: soft, nontender, nondistended, no mass or organomegaly palpable. Extremities: No edema, warm, dry; no rash, normal skin turgor.

DATA:
ICD interrogation: Normal 5/25/06

IMPRESSION:
1. Nonischemic cardiomyopathy, functional class 2-3 symptoms
2. Ventricular tachycardia with normal ICD function
3. Atrial arrhythmia on chronic Coumadin anticoagulation
4. Hypertension, controlled
5. COPD
6. OSA, noncompliant with therapy
7. Subclinical hyperthyroidism on PTU.
8. Diabetes mellitus.

PLAN:
1. Suggest a thyroid profile, lipid panel, BMP. Labs have been obtained at Colonial Family Practice and will be reviewed. Otherwise, continue regular ICD follow-up and present medications.

Mitchell W. Jacocks, M. D.

cc: Dr. Grunsky

17551

TUOMEY
HEALTHCARE SYSTEM
Bringing more to life.

PATIENT NAME:	WILLIAMS, C L
MRN:	QUORTU271254
ACCOUNT # (Opt):	12843062
DATE OF BIRTH:	
REQUESTING DR:	JACOCKS, MITCHELL WELLS MD
ATTENDING DR:	
CC DR:	
ORDER LOCATION:	OP
ROOM NUMBER:	HTCATH^^
SERVICE DATE:	11-14-2007 09:28
DICTATED DATE:	11-14-2007 10:01
REPORT DATE:	11-14-2007 11:12

PROCEDURE PERFORMED ACCORDING TO DEPARTMENTAL PROTOCOLS

STUDY: RAD.CH XR CHEST (PA OR AP 1 VIEW)0608
REASON: ARRYTHMIA,V58.61,MEDTRONIC DEFIB GENARTOR CHANGE

CHEST

REASON: Arrhythmia. Medtronic defib generator change.

REPORT: PA chest film shows an AICD over the left midchest with leads in the right atrium and right ventricle. The lungs show no acute infiltrates and the pulmonary vascular pattern is normal. The heart is normal in size. Old pleural thickening about the right costophrenic angle is unchanged. The left costophrenic angle is sharply outlined.

IMPRESSION:
1. Old pleural thickening, right base.
2. AICD implant as noted.

TRANSCRIPTIONIST: WHEELER, SUE
READING RADIOLOGIST: ROSEFIELD JR, M K MD
This report has been reviewed and released by:

ROSEFIELD JR, M K MD

TUOMEY HEALTHCARE SYSTEM
129 N. WASHINGTON STREET
SUMTER, SC 29150

OPERATIVE REPORT

Name: WILLIAMS, C L
Att. Dr: JACOCKS, MITCHELL WELLS
Adm. Dr:
Admit date:
Location: HTCATH
Account#: 12843062
ID: JACM

MR#: 271254
RM#:
DOB:
Service:

17551

NAME: WILLIAMS, C L

U#: 271254

ACCOUNT NUMBER: 12843062

DATE OF BIRTH:

DATE OF PROCEDURE: 11/15/2007

PREOPERATIVE DIAGNOSIS: ERI Medtronics 7275 ICD.

POSTOPERATIVE DIAGNOSIS: ERI Medtronics 7275 ICD.

OPERATOR: Mitchell Wells Jacocks, MD

PROCEDURE:
1. Explantation of Medtronics 7275 GEM3DR (serial number PJM229158H) ICD.
2. Implantation of Medtronics 7278 (serial number PRM129602H) ICD, utilizing chronic Medtronic atrial lead 5076-52 (serial number PJN134298V), and Medtronic RV lead system 6944-65 (serial 2DC015995V), originally implanted 2/5/02.

METHODS: Informed consent was obtained. The patient was taken to the catheterization lab in a fasting state. Valium 5 mg given as preoperative sedation. Ancef 1 g IV given as preoperative antibiotic. Please see the cath lab flow sheet for details of medications administered. The patient was prepped and draped in the usual sterile manner, 0.5% Sensorcaine was given for local anesthesia. A 4-cm incision was made over the chronic generator and by blunt dissection, the chronic generator was exposed and then explanted. Via a PSA, the ventricular lead was tested, found to have a paced threshold of 0.6 volts at 0.5 ms at a current of 1.0 mA with R waves of 30 mV, slew rate of 4.0 volts/second, lead impedance of 888 ohms. Atrial threshold was 0.6 volts at 0.5 ms at a current of 1.0 mA with P waves of 7 mV, slew rate of 2.5 volts/second, lead impedance of 555 ohms. The pocket was irrigated with antibiotic solution. Excellent hemostasis was noted at all times. The Medtronics 7278 ICD was then attached to the lead system in an appropriate manner. The device and leads were then placed back into the pocket. The pocket was then closed with 2 layers of Vicryl, 3-0 and 4-0 subcuticular, Steri-Strips, and a pressure dressing were applied. The patient tolerated the procedure well without complication. He tolerated the procedure well without complication. He was then transported to day surgery recovery area in stable condition. The device was programmed in an active mode. Of note, the device was tested and found to have P waves of 5.7 mV, atrial impedance of 472 ohms, atrial threshold of 1 volt at 0.3 seconds. R

OPERATIVE REPORT

Name: WILLIAMS,C L
Account#: 12843062

---

waves were 16.4 mV through the device with an impedance of 768 ohms, threshold of 1 volt at 0.3 ms. HVB impedance 49 ohms, HVX impedance 61 ohms, thus confirming electrical integrity of the system. The device was programmed in the active mode, bradycardic pacing VBI 40, two volts at 0.6 ms at 0.3 millivolt sensitivity. A VF zone was established at 300 ms with max. output therapy, VT zone at 380 ms with a burst pacing, followed by 5 joule cardioversion, followed by max. output therapy. Fast VT zone at 240 with 88% burst, followed by max. output therapy.

CONCLUSION: Successful ICD changeout of Medtronic 7275 ICD for Medtronic 7278 ICD with utilization of the chronic 5076 atrial lead and 6944 RV lead system.

MITCHELL JACOCKS, MD

Dict. Date: 11/15/2007 10:57 Trans. Date: 11/15/2007 12:47
240239 / 33462 /
Job: 20110

CC: MITCHELL GRUNSKY, MD

JACM

The South Carolina Second Injury Fund

WILLIAM E. GUNN
INTERIM DIRECTOR

100 EXECUTIVE CENTER DRIVE, SUITE 101
SANTEE BUILDING
COLUMBIA, S.C. 29210
www.scsif.sc.gov

(803) 798-2722
FAX: (803) 798-5290

September 20, 2011

Vernon F. Dunbar, Esquire
Turner Padget
P.O. Box 1509
Greenville, SC 29602

RE: C.L. Williams vs Yuasa Exide
SIF# 141031 WCC File # 9930399

Dear Vernon:

I have now completed my review of the above-mentioned claim. It appears that the claimant worked for the employer for 25 years. His blood lead levels ranged from 21-91. He was removed from his department on several occasions due to high levels of lead. He had a pre-existing history of uncontrolled hypertension, which lead to congestive heart failure, and a diagnosis of diabetes. At this time, we are in a position to offer [redacted] weeks, which is an amount certain of $[redacted] Please take this offer to your client.

Sincerely,

Maria I. Renborn
Claims Analyst

Patti
RECEIVED
SEP 21 2011
3180.167

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION
W.C.C. FILE NO: 9930399

C.L. WILLIAMS, Employee, Claimant, vs. YUASA-EXIDE, INC., Employer, AND LIBERTY MUTUAL INSURANCE COMPANY, Carrier, Defendants.	**EMPLOYEE/CLAIMANT KNOWLEDGE AFFIDAVIT**

PERSONALLY APPEARED before me, C.L. Williams, who, after being duly sworn, deposes and says that:

I am the Claimant in this worker's compensation matter. Prior to sustaining a work related accident or contracting an occupational disease, I was aware of the following preexisting conditions as indicated below:

(1) Arthritis ____

(2) Epilepsy ____

(3) Diabetes ____

(4) Cardiac Disease ✓

(5) Loss of sight of one or both eyes or partial loss of uncorrected vision of more than 75% bilateral ____

Mcott
Received
12/10/NE
0380.00167

(6) Cerebral Palsy

(7) Multiple sclerosis

(8) Parkinson's disease

(9) Cerebral vascular accident

(10) Tuberculosis

(11) Silicosis

(12) Depression or psychological injury (psycho/neurotic disability)

(13) Disability from poliomyelitis

(14) Chronic osteomyelitis and ankylosis of joints

(15) Hyperinsulinism

(16) Arterial sclerosis

(17) Muscular dystrophy

(18) Thrombophlebitis

(19) Varicose veins

(20) Heavy metal poisoning

(21) Compressed air sequelae

(22) Ruptured intervertebral disc

(23) Hodgkin's disease

(24) Brain damage

(25) Deafness

(26) Cancer

(27) Pulmonary disease

2

(28) Sickle Cell anemia ____

(29) Mental retardation ____

(30) Other (please specify) ____

FURTHER AFFIANT SAYETH NOT.

C.L. Williams

August 23, 2010

Witnessed by:

(Signature)

(Printed name)

SOUTH CAROLINA WORKERS' COMPENSTION COMMISSION

WCC FILE NO.: 9930399

LTD Services & Accident Insurance Company, Plaintiffs, v. South Carolina Second Injury Fund, Defendant.	NOTICE OF WITNESSES AND WRITTEN REPORT(S) PHYSICIAN OR OTHER EVIDENCE TO BE INTRODUCED ON BEHALF OF THE S.C. SECOND INJURY FUND
IN RE: C.L. Williams , Employee, v. Yuasa Exide, Inc., Employer, and Arrowpoint Capital Corporation, Carrier.	

TO: SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION, VERNON F. DUNBAR, ESQUIRE, ATTORNEY FOR EMPLOYER/CARRIER.

YOU ARE HEREBY NOTIFIED THAT THE South Carolina Second Injury Fund (hereinafter "the Fund"), pursuant to the provisions of the South Carolina Workers' Compensation Act and South Carolina Code Section 1-23-330 (1976, as amended), herewith submit the following reports/physicians or other evidence on behalf of the Fund, to wit:

NAME OF PHYSICIAN/OTHER	DATE	PAGE NOS.
Letter from S.C. Second Injury Fund	November 28, 2011	1

YOU ARE FURTHER HEREBY NOTIFIED that you have the right to cross-examination; and, should you desire to exercise said right, you are to forthwith schedule the deposition(s) of any of the physicians or other person(s), whose reports are submitted, for the purposes of cross-examination.

YOU ARE FURTHER HEREBY NOTIFIED that the originals of the documents referred herein, or photocopies received from physicians/others, are being hereby forwarded to the South Carolina Workers' Compensation Commission, for insertion in the file of the South Carolina Workers' Compensation Commission and inclusion into evidence on behalf of the Fund.

YOU ARE FURTHER NOTIFIED that the following witnesses may be called on behalf of the Fund: N/A

The Defendant reserves the right to cross-examine any expert submitted by the Plaintiffs pursuant to Section 1-23-330(3).

Latonya Dilligard Edwards, Esquire
Dilligard Edwards, LLC
3790 Fernandina Road, Suite 103
Columbia, South Carolina 29210
(803) 750-2214 – Phone
(803) 750-2377 – Fax

Columbia, South Carolina

August 3, 2012

The South Carolina Second Injury Fund

100 EXECUTIVE CENTER DRIVE, SUITE 101
SANTEE BUILDING
COLUMBIA, S.C. 29210
www.scsif.sc.gov

STEPHEN L ELLIOTT
INTERIM DIRECTOR

(803) 798-2722
FAX: (803) 798-5260

November 28, 2011

Vernon F. Dunbar, Esquire
Turner Padget
P.O. Box 1509
Greenville, SC 29602

RE: C.L. Williams vs Exide
SIF# 141031 WCC File # 9930399

Dear Vernon:

On September 20, 2011 we issued a compromise on this claim of an amount certain of $38,000.00. I sent you an email on November 9th again offering the above amount. As of today, I have failed to hear from you regarding the above offer and at this time have no choice but to withdraw our offer and issue a denial on the above claim. Please consider this a general denial at this time.

The Second Injury Fund reserves the right to utilize any additional defenses available should you decide to pursue this claim.

Sincerely,

Marla I. Rehborn
Claims Analyst

Patti — 65 Pkt

NOV 29 2011

3180.167

SCANNED

Yuasa-Exide, Inc.
v.
South Carolina Second Injury Fund

In re:

James W. Huggins – SIF No.: 141445
C.L. Williams - SIF No.: 141031
Joe Mathis, Jr. - SIF No.: 146812
Mary C. McConico - SIF No.: 147569

August 13, 2012

OVERVIEW OF LEAD CASES

Vernon F. Dunbar, Esquire
Turner Padget Graham & Laney P.A.
Telephone: 864-552-4601

INDEX

1. Article regarding lead

   a) Health Effects of Chronic Lead Exposure among Employees of Exide Battery Production Plant – Sumter SC

   b) Lead-related nephrotoxicity: A review of the epidemiologic evidence

   c) Literature Review – Lead and Health Effects

   d) Statistical Data Concerning Hypertension, Diabetes, Renal Kidney Failure, and Memory Loss in Society

   e) Case Studies in Environmental Medicine – Lead Toxicity

2. OSHA and Yuasa-Exide Regulations

   a) Electronic Code of Federal Regulations

   b) Lead Control Program - Yuasa-Exide Regulations

3. Commission and Court cases involving *Lee Ernest Franklin*

   a) Employee's Notice of Claim and/or Request for Hearing dated July 3, 2001

   b) Decision and Order of Commissioner George N. Funderburk dated June 10, 2005

   c) Appellate Panel Decision and Order of the South Carolina Workers' Compensation Commission dated May 3, 2006

   d) Decision and Order of Commissioner George N. Funderburk dated January 31, 2008

   e) Decision of the Supreme Court of South Carolina, Opinion No.: 26880 dated September 13, 2012

Health Effects of Chronic Lead Exposure among Employees of Exide Battery Production Plant- Sumter SC

Report prepared at the Request of The Steinberg Law Firm, Charleston, SC

July, 2009

Report authored by Edward L. Baker MD, MPH, Howard Hu MD, DrPH, and J. Routt Reigart MD

I. Executive Summary

Based upon our extensive experience as experts in the field of occupational and environmental medicine, toxicology, epidemiology, pediatrics, and internal medicine, and upon an extensive review of scientific literature, battery manufacturing processes, and health records of Exide workers, we conclude that workers at the Exide plant in Sumter SC experienced lead exposure which caused or contributed to the occurrence of a range of significant health problems. These health problems include hypertension, heart disease, kidney failure, nervous system damage, gout and kidney cancer. These conditions are or will be associated with significant morbidity and mortality resulting in decreased quality of life and premature death.

As experts in the fields noted above, we have received specialized training and education in occupational and environmental medicine, toxicology, and clinical medicine. We have practiced medicine and have personally examined and treated individuals with lead poisoning and diseases caused by toxic chemicals. We have conducted research at major US medical centers and at the Centers for Disease Control and Prevention (CDC) on the health effects of lead exposure and have published this research in peer-reviewed publications. We have been invited to write chapters in medical textbooks on the subject of lead poisoning and have been invited to speak at medical conferences and workshops around the world on our research. We have served as professors at Harvard University, the Medical University of South Carolina, the University of North Carolina and the University of Michigan, where we have taught medical doctors and other health professionals in the areas of toxicology, epidemiology and occupational and environmental medicine. We have been invited to consult with major government organizations including the World Health Organization, the US Environmental Protection Agency, the Centers for Disease Control and Prevention and the Occupational Health and Safety Administration. One of us held the position of Deputy Director of the US National Institute for Occupational Safety and Health (NIOSH). We all remain active in our fields and are currently involved in research and academic publication in the field of occupational and environmental medicine.

Our health hazard analysis of the Exide lead battery plant consisted of a review of the battery plant processes in general and the Exide processes in particular. We utilized detailed information provided by the US Occupational Health and Safety Administration on health hazards of the battery manufacturing to identify specific plant processes associated with exposure to lead dust and fumes. We reviewed research on other battery plants, including a study conducted by one of us, to determine the nature and extent of lead exposure in other battery plants. We met with former workers at the plant to review plant processes in detail and to develop an understanding of the nature of work activities in specific areas of the Exide plant. We reviewed and summarized the health literature on lead poisoning to identify those conditions which have been shown in scientific research to be caused by or significantly contributed to by lead exposure in adults.

We then reviewed health records of former Exide workers including extensive records of the results of personal blood lead testing performed by the Exide plant. We reviewed medical records documenting the health status and treatment histories of these workers. We used the results of blood lead testing to calculate a cumulative blood lead index (CBLI) measurement for each worker and then compared the CBLI results with those of the normal US population. From the review of the health records, we identified a set of health conditions which are known to be caused by lead exposure.

Our extensive reviews of the records of the Exide workers have revealed that these individuals had very elevated blood lead concentrations. The elevated blood lead concentrations were caused by exposure to lead dust and fumes during their performance of their normal job duties at the Exide plant in Sumter SC; we did not identify any other plausible source of significant lead exposure to these individuals. We conclude that exposure to lead dust and fumes in the Exide plant resulted in inhalation or ingestion of lead into the body of Exide workers.

We also conclude that lead, once absorbed into the body, was distributed to various parts of the body, including the brain, the kidneys and bone, and caused damage to the body of Exide workers. The damage to the body of Exide workers was manifested by the development or aggravation of a number of diseases or health conditions. These include hypertension, heart disease, kidney failure, nervous system damage, gout, and kidney cancer. Exide workers who developed these conditions experienced significant morbidity and premature mortality and experienced impairment of

the normal functions of daily life and work. These workers required medical care including hospitalization, medications, and ongoing medical treatment. These diseases and health conditions that they experienced are likely to be permanent and will persist indefinitely; these former Exide workers will require long term medical care for these conditions. Furthermore, some former Exide workers are likely to develop disease or health conditions in the future caused by or aggravated by their prior exposure to lead at the Exide plant as a result of the latent toxicity of lead.

We hold these opinions to a reasonable degree of medical certainty and base our opinions on our extensive professional experience and expertise, our review of battery manufacturing processes in general and the Exide plant in particular, our review of the scientific literature on adult lead poisoning (some of which we have written), and our review of the health records of Exide workers. We may supplement or modify this report as additional information becomes available to us.

II. Qualifications

A. Edward L. Baker MD, MPH

Dr. Baker is a medical doctor, having received his medical degree from Baylor College of Medicine in Houston, Texas (1972). Dr. Baker is licensed to practice medicine in the state of Texas. Dr. Baker completed his internship and residency in internal medicine at Boston City Hospital (1972-73) and Framingham Union Hospital (1973-74, 1977-78). He is board certified in Internal Medicine (1980) and Preventive Medicine (Occupational Medicine) (1978). He has also completed a General Preventive Medicine Residency at the Centers for Disease Control (1975-77) and is board eligible in General Preventive Medicine. He received a Master of Public Health degree (1989) and a Master of Science degree in Epidemiology and Occupational Health (1980) from Harvard School of Public Health. He completed a Fellowship in Occupational Neurology in the Department of Neurology at Boston University Medical Center (1978-79).

Dr. Baker was trained in epidemiology at the Centers for Disease Control and Prevention through the Epidemic Intelligence Service, an internationally-recognized training program for epidemiologists from 1974 to 1976. At CDC from 1974 to 1977, Dr. Baker directed studies of children exposed to lead near lead smelters, studies of children exposed to lead as a result of parents carrying home lead-containing dust on work clothing, and studies of pesticide poisoning in Pakistan. Findings of his research were used to create portions of the OSHA lead standard and to develop standards for prevention of pesticide poisoning among workers throughout the world.

Following the completion of his residency training and graduate study, Dr. Baker served as a professor at Harvard University School of Public Health from 1980-1985, where he conducted research related to the toxic effects of workplace chemicals on the nervous system. His research focused primarily on the study of nervous system effects of occupational exposure to lead and organic solvents. While at Harvard, he directed the Occupational Medicine Residency Program, an accredited training program for occupational physicians. He also taught courses in industrial toxicology and occupational epidemiology to physicians, nurses, industrial hygienists and other Harvard graduate students.

While on the Harvard faculty from 1980-1985, he led a research effort in occupational neuroepidemiology, conducting epidemiological studies of the health effects of occupational exposure to lead and organic solvents, with particular emphasis on the central nervous system. He has been invited to present the results of his research at conferences in the United States, Europe and Australia, particularly in the area of occupational neuroepidemiology. His research has received a number of awards, including recognition by the American College of Occupational and Environmental Medicine for research on solvent neurotoxicity.

From 1985-1990, Dr. Baker served as Assistant Director and later Deputy Director of the National Institute for Occupational Safety and Health (NIOSH), the federal agency charged with supporting and conducting research in occupational safety and health and providing recommendations and guidelines to the medical and scientific communities and to OSHA on prevention of occupational disease and injury. His duties included providing oversight to the Institute's research and educational activities and representing NIOSH in national and international settings.

Dr. Baker served as Assistant Surgeon General in the U.S. Public Health Service from 1995-2003 and as Director of the Centers for Disease Control and Prevention's Public Health Practice Program Office, from 1990 until 2003. In this position, he led national programs to strengthen our nation's public health infrastructure, representing CDC throughout the nation. He was intimately involved in the public health

response to the terrorist attacks of Sept. 11, 2001 and in the leadership of a multibillion dollar initiative to rebuild the Nation's public health system as a defense against bioterrorism. He retired from active duty in the U.S. Public Health Service in 2003.

Dr. Baker currently serves as professor of health policy and epidemiology in the University of North Carolina Gillings School of Global Public Health and as Director of the North Carolina Institute for Public Health. He also teaches and advises graduate students in the Departments of Health Policy and Management and Epidemiology.

Dr. Baker has received numerous awards and honors from a range of organizations in the United States and abroad including the American College of Occupational Medicine, the Royal Society of Medicine (London), the International Commission on Occupational Health, the National Association of County and City Heath Officials and other groups. He has been asked to serve on expert committees on subjects related to occupational health and public health under the auspices of the World Health Organization, and the Centers for Disease Control and Prevention.

Dr. Baker has been invited to author a number of textbook chapters and review articles on the subject of occupational neurological disease, including articles on the health effects of toxic chemicals on the nervous system. He was invited to participate in expert workshops convened by the World Health Organization and the U.S. Environmental Protection Agency on the areas of solvent neurotoxicity and neuroepidemiology. He has served as a peer reviewer and on the editorial boards of a number of medical journals, including the American Journal of Industrial Medicine, the Journal of the American Medical Association, Health Affairs, and the Annual Review of Public Health. Dr. Baker has published over 100 articles in peer-review journals on the subjects of occupational health, environmental health and public health. Dr. Baker has been a member of the American Public Health Association, the International Commission on Occupational Health, the American College of Occupational and Environmental Medicine, the American College of Epidemiology and other professional organizations. Dr. Baker has also been a consultant to various governmental organizations in the United States and abroad, including the World Health Organization, the Occupational Safety and Health Administration, and the US Environmental Health Administration. He has also served as an occupational medicine consultant to private industry and labor unions in the United States and Great Britain.

Dr. Baker has practiced occupational medicine at Brigham and Women's Hospital, Massachusetts General Hospital, and Braintree Hospital while he was on the faculty at Harvard and in Atlanta at Emory University Clinic. He directed the Occupational Medicine Clinic at Braintree Hospital. Dr. Baker was actively engaged in patient diagnosis and care at these clinics, where he managed cases of individuals with occupational disease.

He has also led the conduct of numerous research studies of the exposure of workers to toxic substances encountered in the workplace and has received various awards and recognition in the United States and abroad for that work. Dr. Baker has also served as a reviewer of the work of other researchers in the context of peer review for journal publication, in the context of writing review articles and book chapters, and in the context of teaching epidemiology at Harvard, the Centers for Disease Control and Prevention and at the University of North Carolina.

Dr. Baker has been asked, along with other experts noted below, to provide an evaluative report describing the extent and nature of toxic exposures at the Exide facility in Sumter SC with respect to potential health impacts to employees of the facility. Dr. Baker has agreed to serve as an expert in the legal proceedings being filed on behalf of former employees of this facility. He will serve in this capacity as a private individual and not as a representative of the University of North Carolina. All of Dr. Baker's opinions will be given to a reasonable degree of scientific and medical certainty. Dr. Baker has served as a medical expert in legal proceedings in the past.

B. Howard Hu MD, MPH, ScD

Dr. Hu is the NSF International Endowed Chair of the Department of Environmental Health Sciences and Professor of Environmental Health, Epidemiology and Internal Medicine at the University of Michigan Schools of Public Health and Medicine. He is physician board-certified in internal medicine and occupational/environmental medicine who also holds a doctoral degree in epidemiology. He came to the University of Michigan in 2006 from the Harvard School of Public Health and the Brigham and Women's Hospital, where he directed the Harvard Residency Program in Occupational and Environmental Medicine, the Harvard Metals Epidemiology Research Group, and the Center for Children's Environmental Health and

Disease Prevention Research; he worked with the Harvard NIEHS Center and the Channing Laboratory of the Brigham and Women's Hospital. He taught the main introductory courses in environmental health and medicine at the Harvard SPH for 16 years. Dr. Hu continues his research on metals toxicity by co-directing, with Dr. Robert Wright, what is now the Michigan-Harvard Metals Epidemiology Research Group, which is engaged in multiple NIH- and EPA-funded epidemiologic investigations of the contribution of metals exposure and genetics to the causation of chronic diseases in adults and impaired development in children.

Dr. Hu's research interests also encompass clinical syndromes such as idiopathic environmental intolerances (chemical sensitivities) and emerging children's environmental health issues such as neonatal exposure to phthalates. He served on 3 fact-finding missions and on the board of directors for Physicians for Human Rights and in 1992-1995 was the Chair of the Commission for Research on the Health and Environmental Effects of Nuclear Weapons Production and Testing for the International Physicians for the Prevention of Nuclear War (IPPNW; Nobel Peace Prize, 1985). He has authored or co-authored over 200 scientific papers and book chapters, of which over 120 have focused on the subject of lead exposure and/or toxicity, and co-edited or co-authored 7 books.

Dr. Hu was the founding medical editor (and continues as the Associate Medical Editor) of *Environmental Health Perspectives*, the journal of the National Institute of Environmental Health Sciences. Among the awards and honors Dr. Hu has received have been the 1994 Will Solimene Award of Excellence, American Medical Writers Association, the 1997 Alice Hamilton Lectureship at the University of California at San Francisco, the 1998 First Prize for Best Infant Nutrition Research from the Instituto Danone of Mexico, the 1999 NIEHS Scientific Advance of the Year, the 2000 Hoopes prize for mentorship of environmental research, a Senior United States Faculty Fulbright Award to work as a scholar in India 2000-2001, the 2005 Adolph Kammer award for authorship by the American College of Occupational and Environmental Medicine, and the 2006 Harriett Hardy award from the New England College of Occupational and Environmental Medicine.

Dr. Hu has been asked, along with other experts, to provide an evaluative report describing the extent and nature of toxic exposures at the Exide facility in Sumter SC with respect to potential health impacts to employees of the facility. Dr. Hu has agreed to serve as an expert in the legal proceedings being filed on behalf of former employees of this facility. He will serve in this capacity as a private individual and not as a representative of the University of Michigan. All of Dr. Hu's opinions will be given to a reasonable degree of scientific and medical certainty. Dr. Hu has served as a medical expert in legal proceedings in the past.

C. John Routt Reigart, II, MD

Dr. Reigart is a medical doctor licensed in the state of South Carolina. He is a graduate of Dartmouth College, Dartmouth Medical School, and Harvard Medical School and he completed residency training at Children's Hospital Medical Center in Boston, MA. He is certified by the American Board of Pediatrics, the National Board of Medical Examiners and the South Carolina Board of Medical Examiners.

He has held faculty appointments at Harvard Medical School and at the Medical University of South Carolina where he is currently Professor of Pediatrics. He has held numerous hospital appointments including Director of the Division of General Pediatrics and Director of the Poison Control Center at the Medical University Hospital in Charleston SC. He has received numerous honors and awards including special recognition for his efforts to end childhood lead poisoning. He is a member of numerous professional organizations and has served as an expert consultant to the US Environmental Protection Agency, the President's Council on Environmental Quality, and the Centers for Disease Control and Prevention in areas related to clinical toxicology and lead poisoning. Dr. Reigart has chaired the Committee on Environmental Health of the American Academy of Pediatrics, the CDC Advisory Committee on Childhood Lead Poisoning, and the USEPA's Children's Health Protection Advisory Committee. He served for several years as a contract consultant to the Director, National Institute of Environmental Health Sciences, NIH, DHHS for metals toxicity. He has participated on multiple review panels for grant submissions to NIEHS for environmental health research. He is an active member of the South Carolina Medical Association and the Charleston County Medical Society.

He has served on the editorial boards or as editor of medical journals and has published extensively in the peer reviewed literature on the subject of lead poisoning and other toxicological issues. He has been an invited speaker at medical conferences and workshops around the world on the subject of lead poisoning.

Dr. Reigart has been asked, along with other experts, to provide an evaluative report describing the extent and nature of toxic exposures at the Exide facility in Sumter SC with respect to potential health impacts to employees of the facility. Dr. Reigart has agreed to serve as an expert in the legal proceedings being filed on behalf of former employees of this facility. He will serve in this capacity as a private individual and not as a representative of the Medical University of South Carolina. All of Dr. Reigart's opinions will be given to a reasonable degree of scientific and medical certainty. Dr. Reigart has served as a medical expert in legal proceedings in the past.

III. The plant

A. Plant description and location

The Yuasa/Exide manufacturing facility in Sumter, South Carolina is located on S-43/458 off US Highway 15-S near Sumter, South Carolina. Yuasa formed a joint/venture company with General Battery Corporation in 1976. Yuasa was sold by General Battery Corporation to Exide Corporation in 1987 to form Yuasa/Exide Battery Corporation. Yuasa/Exide purchased Exide's industrial division to become Yuasa, Inc., in 1991. In 2000, Yuasa, Inc., sold off its industrial division to Enersys.

The Yuasa/Exide facility in Sumter, South Carolina totals 299,762 sq. ft. Of the total square feet, 21,029 sq. ft. are devoted to office use. The facility sits on a 41 acre site and was constructed in 1965 with additions in 1991 and 1998. The floor of the facility is concrete and brick and the walls are constructed of brick on concrete block. Ceiling heights within the facility vary from 16-17 feet. Lighting is both fluorescent and vapor. 100% of the manufacturing space within the plant is heated. Some limited cooling is provided by air handlers.. At present, the facility is for sale, last having been used by UPS Battery Manufacturing.

During its operation and ownership by Yuasa/Exide and Enersys, the plant produced various industrial batteries from large commercial batteries used in industrial applications to heavy equipment batteries and motorcycle/ATV batteries. From 1965 to around 1974 the plant manufactured nickel/iron batteries. Beginning around 1974 the plant was retooled to manufacture lead acid batteries. The major component to the lead acid batteries produced at the facility was inorganic lead. Lead used in the production process came to the plant either in ingot form or in a powdered form. Depending upon the product being manufactured, lead was mixed with various other chemicals and eventually cast into plates which were then packaged in a casing and filled with an acid base. The final product, through the chemical reaction between the lead plates and the acid base, produced electricity. A floor plan (see figure below) of the facility demonstrates a range of production areas related to the primary battery production process.

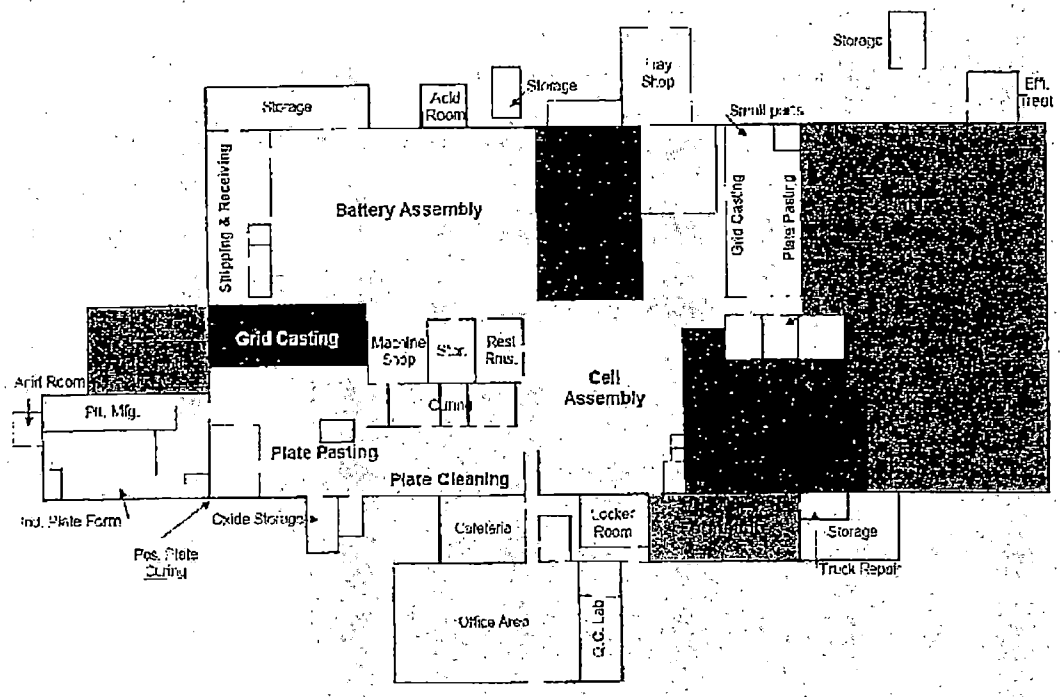


Figure : Floor plan of Exide facility- Sumter SC

B. Plant processes and related workplace lead exposures

1. <u>Manufacturing processes and potential sources of lead exposure at Exide facility in Sumter SC</u>

(Source: OSHA website, including photos, and former Exide employee interviews)

Battery Manufacturing is the process of producing lead-acid batteries, commonly used in automobiles, fork trucks, material handling, and standby power applications. Employees working in battery manufacturing plants can be exposed to significant lead concentrations. There are a number of operations workers perform in battery manufacturing plants. These processes were performed by workers at the Exide facility. Workers were exposed to lead dust and fumes as a result of performing their normal job duties, as described below. Lead was absorbed into the body of these workers by inhalation or ingestion of lead-containing dust and fumes.

a. <u>Oxide and Grid Production</u>

Lead oxide is made from refined lead. The oxide is then made into a paste by mixing it with water, acid, and other chemicals. In a process parallel to the production of paste, grids are cast from lead alloy. The paste and grids are later combined in the pasting process.

1. Oxide Production: Lead oxide, which is used to create the paste used on the battery plates, is either produced by a production process in the plant (figure below) or purchased from an oxide producer. The major sources of lead exposure in the oxide production process come from leaking equipment and system upsets. Sources of exposure in oxide production include:

- Lead dust becomes airborne due to improper air flow or exhaust ventilation, which results in "puffing" of lead oxide at the mill inlet.
- Exposure occurs from handling lead pigs while loading the mill pots or the ball mill.
- Lead oxide dust spills or leaks from trunnion seals, bearing seals, conveying systems, and transfer points.
- Exposure to lead occurs when drossing the lead pots during the Barton process (see Figure) and during maintenance operations that require entry into the pot for cleanout.
- Lead oxide leaks or spills during drive fitting and transfer operations.
- Lead exposure occurs while collecting and testing oxide samples.
- Operators are exposed to lead oxide while cleaning the oxide production line.
- Exposure occurs due to vehicular traffic in adjacent roadways stirring up oxide.

Oxide production furnace

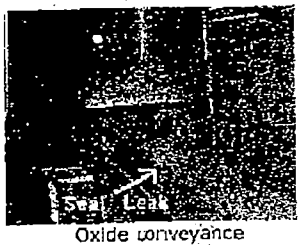
Drossing out the lead pot

2. Oxide Receiving: Oxide is delivered to plants in "pod" cars, which loads the material into storage by pneumatic conveying (blowing) it into storage tanks. All lead oxide storage systems have the potential for leaking, particularly around seals and sampling points. Sources of exposure in this area include:

- Leaking conveying equipment, particularly around flanges, seals, and transfer points.

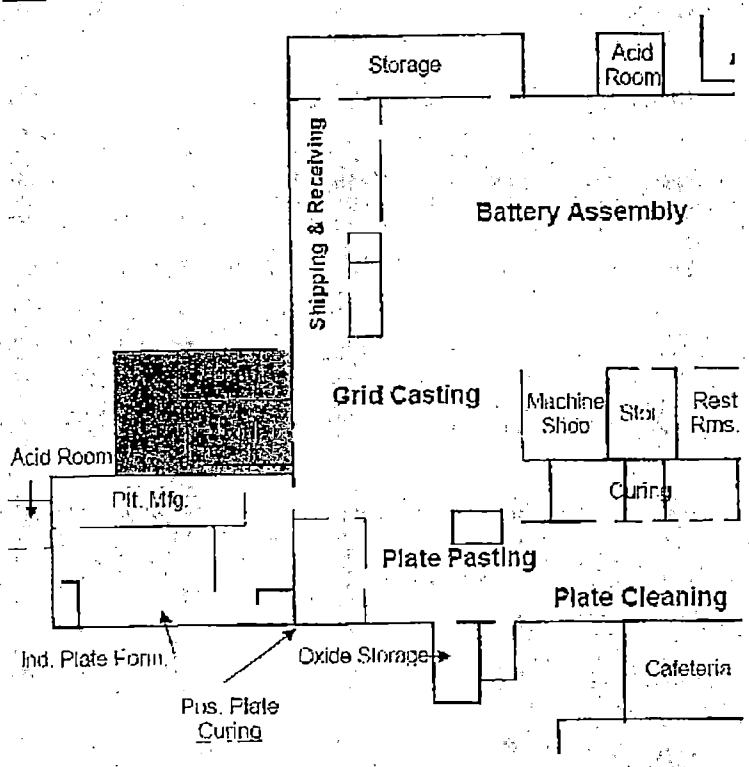
3. Oxide Conveyance and Classification: Certain material transporting operations call for mechanical conveyance. Classification separates the oxide particles by size, allowing only particles within the desired size range to the paste mixing operation. Pneumatic conveying systems use air to transfer lead oxide from one point to another via pipes. Sources of exposure include:

- Leaking equipment particularly in the pneumatic system.
- Damaged bags in bin vent or baghouse.

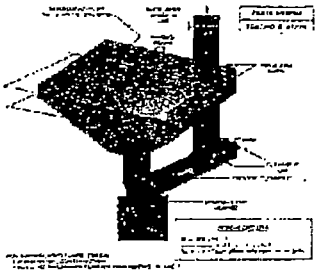


Oxide conveyance

MAP: OXIDE MANUFACTURING AREA



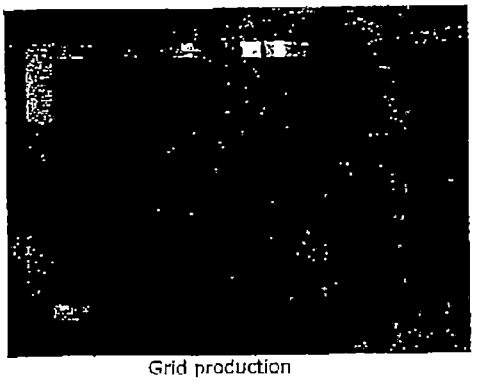
4. Paste mixing: During the paste mixing process, lead oxide, water, acid, and other chemicals are blended in a mixer to form a thick paste. The major source of lead exposure in this process is from lead oxide that escapes from the paste mixing machine, dried, and becomes airborne.



Sources of exposure in paste mixing include:

- Leaking equipment:
  - oxide conveyance,
  - weigh hoppers,
  - mixer door seals.
- Limited ventilation due to moist material plugging the system allowing lead dust to become airborne, or allowing rising air currents to carry suspended lead to the employee's breathing zone.
- Sampling and testing oxide or paste increase the employee's exposure to lead.
- Lead-containing expander becomes airborne when added to the paste mixer.
- Manually scraping and cleaning the mixer during cleanup or while changing pastes caused lead particles to become airborne.
- Lead exposure increases when there is re-entrainment of dried and settled lead on buckets, the cone feeder, or other equipment.
- Lead dust becomes airborne when workers fold lead-containing expander bags for disposal outside of a ventilated enclosure.
- Lead oxide becomes airborne when dried oxide flakes off of gloves and hand tools.
- When a cone feeder is empty, heat rising from the pasting machine causes a chimney affect. This dries out the oxide and suspends it in the rising air currents.
- Lead dust and particles become airborne due to drafts and air currents caused by moving equipment or motor drives.

b. Grid production and parts casting: Grid production and parts casting involve book casting, continuous casting, and strip casting. In all of these processes, lead pigs are melted down and the molten lead is poured into molds or continuously cast into grids, strips, or parts. The major source of lead exposure in this process is from lead fumes and lead oxide which become easily airborne.

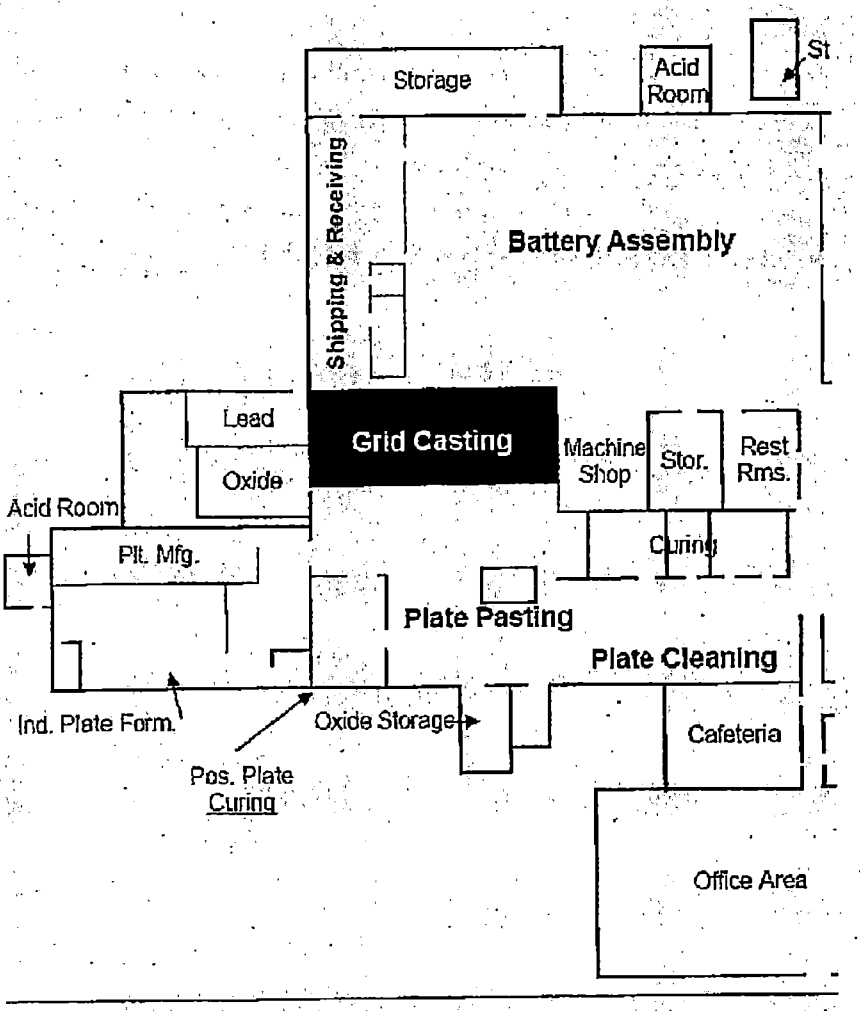


Grid production

Sources of exposure in this area include:

- Drossing the lead pot increases lead exposure.
- High levels of lead fumes are generated when a flame is put in contact with lead, such as when cutting out frozen pots.
- Settled lead dust on surfaces and equipment become airborne due to exhaust from fuel-powered fork-lifts or vehicular traffic.
- High exposure to lead fumes occurs when the pig is loaded into the lead pot.
- Airborne lead dust migrates from other areas of the plant.
- Separator boards become contaminated by using lead contaminated pallets that have not been cleaned after being returned from the pasting line.
- Dust becomes airborne as a result of using a rotary sander for cleaning molds.
- Oxide collected on the dross ladle becomes airborne while cleaning or storing it in an area outside the hood.

MAP: GRID CASTING AREA



b. Plate Processing

Plate processing combines the oxide paste and the grids to prepare plates for assembly into batteries.

1. Grid pasting: During the pasting process, lead oxide paste is applied to the grid panels in a pasting machine to fill the spaces of the grid. The major source of lead exposure in the pasting process comes from lead oxide in the paste which becomes airborne once it dries. Workers are exposed to lead dust during pasting and take-off operations. Contaminated gloves, clothing, tools, and equipment are also a source of lead exposure. Dried paste on equipment and other areas, such as the mixer, cone feeder, paste transfer equipment, paste return belt, pasting machine, floors, and adjacent areas, become airborne due to equipment vibration or by being disturbed.

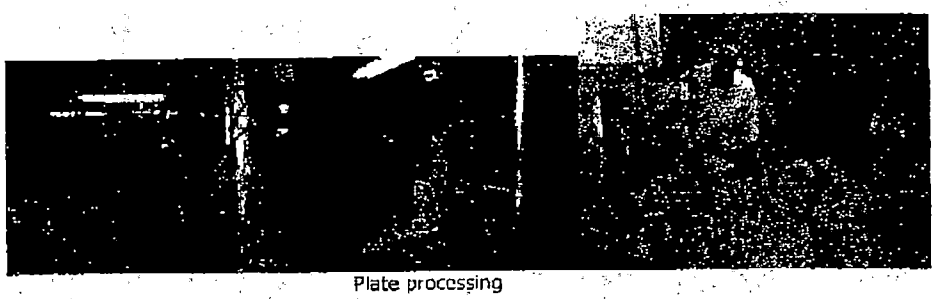
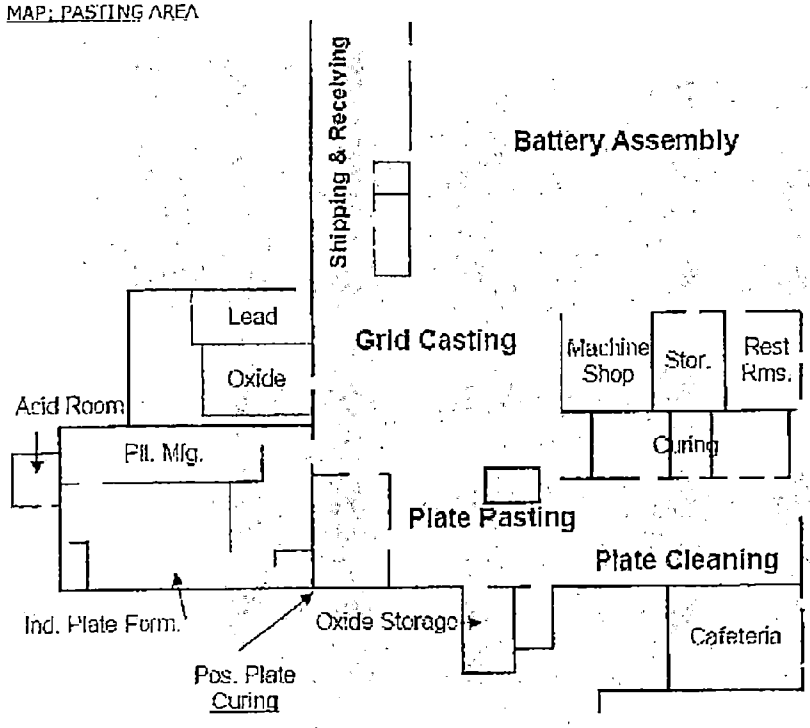


Plate processing

Sources of exposure to lead in the pasting process include:

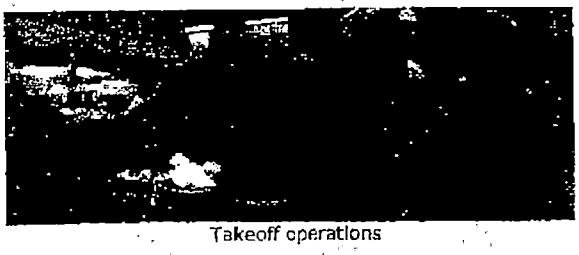
- Scraping lead oxide off the hopper and exterior of the cone feeder during cleanup and upsets.
- Inadequate exhaust ventilation on the drying oven.
- Settled oxide on the housings of rotating drives and motor cooling fans.
- Improper handling and disposal of paste accumulated on the floor or other surfaces.
- Dried dust on equipment, controls, and gloves during operation of equipment located near the breathing zone.
- Dried oxide on the floor or other surfaces being disturbed by employees, vehicle traffic, or cross-drafts.

MAP: PASTING AREA



Lead oxide dust also becomes airborne in the take-off/offbearer operations:

- From handling dry plates or moving plates in and out of the workstation.
- From the buildup of dust on equipment, racks, and floor.
- When workers brush plate lugs during parting.
- From inadequate capture velocity for tamping plates.
- From upset conditions or while clearing jam-ups in equipment.
- From dried oxide on contaminated gloves, clothing, shoes, and tools.

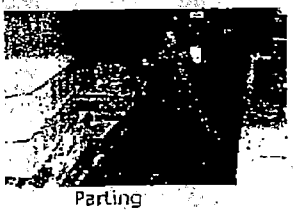


Takeoff operations

2. Hydrosetting: The major source of lead exposure in the hydrosetting process comes from lead oxide when the grids are handled incorrectly. Sources of exposure in this area include:

- Lead becomes airborne from handling or moving dry plates.
- Lead particulate becomes airborne from positioning pallets of pasted plates near drafts or thermals.
- Handling burlap, used to cover grids that have been contaminated with lead oxide improperly.

3. Parting: Grids are produced and pasted as pairs called pasted plates or panels. They are separated before the battery is built. The major source of lead exposure in the parting process come as a result of lead particles becoming airborne when plates are cut or broken.



Parting

4. Enveloping and Wrapping: Enveloping involves placing a plate within porous membranes. Sources of exposure in this area include:
- Lead oxide becomes airborne when workers handle plates at the feed and discharge ends of enveloping machines as well as from the bellows effect when handling unsealed envelopes.
- Exposure to lead occurs due to the lead oxide that accumulates on equipment, racks, and floors become airborne.
- Increased exposure to lead oxide occurs when tamping plates in unventilated areas, such as the rack, created puffs of lead dust.

5. Handling and Transport: Handling and transporting of materials is an essential part of the battery manufacturing process. Sources of exposure in this area include:
- Lead dust from feed materials become airborne during handling and transport.
- Lead dust that settles on surfaces and equipment becomes airborne due to vehicle traffic and cross-drafts.

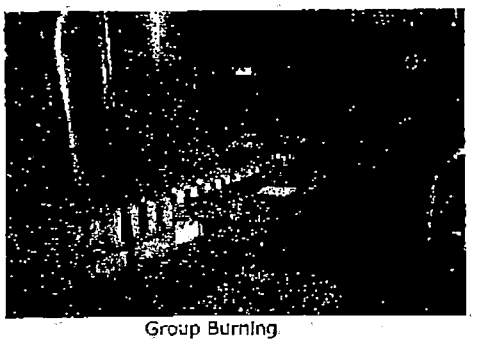
c. ***Battery Assembly***

Battery assembly combines the plates container, and other parts into a functional battery. Battery charging is also an important part of assembly.

1. Stacking: After curing, the plates are stacked so that positive and negative plates alternate with an insulating separator in between. The major source of lead exposure in the stacking process is from oxide from the plates which become easily airborne. Sources of exposure include:

- Tamping plates in unventilated areas.
- Lead oxide on equipment, racks, and floors become airborne.
- Clearing stacking equipment jam-ups.

2. Group Burning : After the plates have been stacked, they are joined with small connecting parts and burned together to form cell elements or groups.



Group Burning

Sources of exposure include:

- Handling dry plates or moving plates in and out of the workstation.
- When lead oxide that accumulated on racks, equipment, and floors becomes airborne.
- When groups are dropped into the cases.
- During cleaning and roughing with a wire brush.
- During drossing or charging the lead pot.
- Burning with torches.
- Moving groups in and out of the workstation and in and out of the burning box.
- Adjusting and repairing equipment, or during changeovers.
- Cleaning molds using compressed air or torches.

3. Intercell welding and post burning: After workers place all the groups in the battery case, the straps are fused together using a torch or high electrical power source. The connections are then welded. The units are then tested and the posts attached. The major source of lead exposure in the intercell welding process comes from lead fumes.

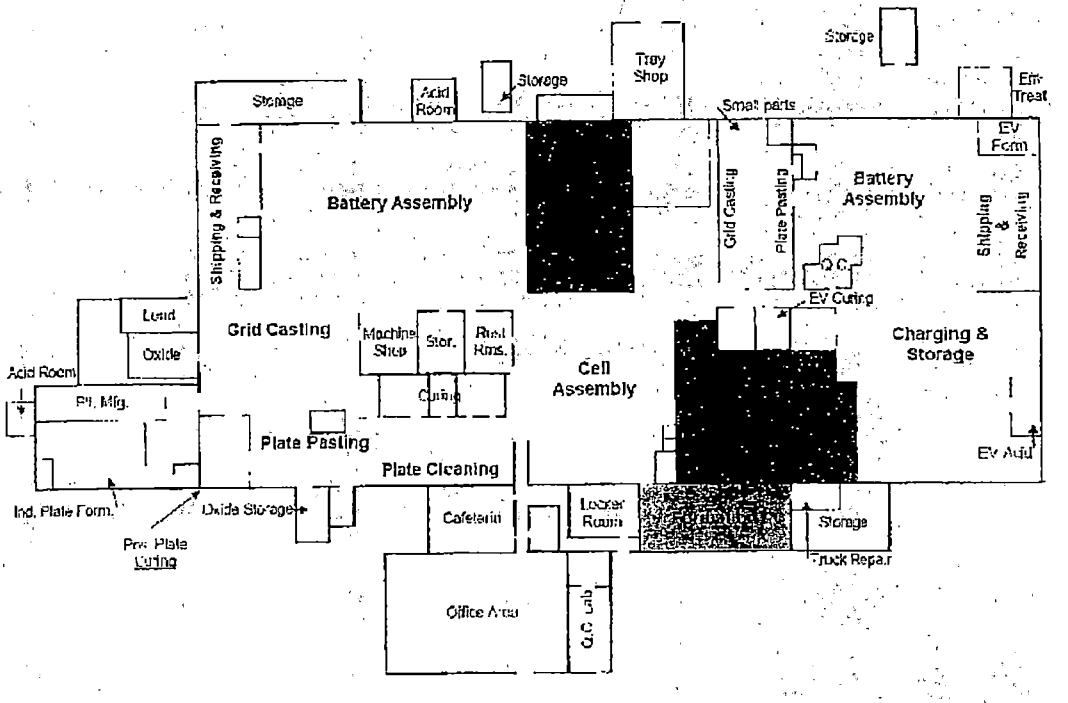
Sources of Exposure Include:

- Lead fumes are emitted during the manual burn welding process.
- Manual handling of burning jigs or contaminated dams.
- Repairing batteries.

4. Formation: In the dry charge process, the first step, called tacking consists of plates being placed in a tank and lead bars being welded, or "tacked" on. Battery plates are then formed (or charged) in tanks. The major source of lead exposure in the formation process comes from lead fumes. Sources of exposure include:

- Lead fumes emitted from using a torch to weld lead.
- Moving plates in and out of acid tanks, drying ovens, and racks.
- Cleaning of panel lugs using brushes.

MAPS: FORMATION AREAS



d. Battery Repair and Reclaim

Battery repair operations repair and salvage defective batteries. Reclaim operations involve recycling scrap grids, plates, and straps.

Battery repair: Defective batteries are repaired and salvaged. The plate groups are tested and reused when possible. Groups are removed by sawing or cutting the covers off the battery, breaking or burning the intercell connectors, and removing the groups.

The major source of lead exposure in the battery repair process comes from lead oxide which become airborne. Sources of exposure include:

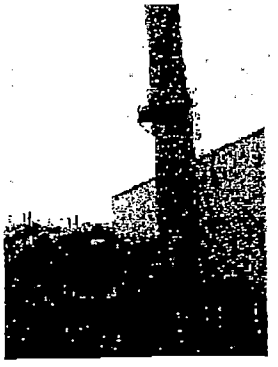
- While removing plates, oxide spills out of the battery case.
- High levels of lead fumes are generated when flames are put in contact with lead.

Reclaim: Reclaim operations involve recycling scrap grids, plates, and straps. The major source of lead exposure in the reclaim process comes from lead oxide which becomes airborne. Sources of exposure include:

- When barrels are dumped, or spilled, leaded material becomes airborne.
- Handling leaded materials contaminate clothing or results in airborne exposure.
- Lead pots emit high levels of lead fume and operators are exposed during cumping and drossing.
- The handling of material or dross cause lead fume emissions and expose the operator.

e. ***Environmental Controls***

Employees are exposed to lead while servicing and maintaining environmental controls.



1. Waste Water Treatment: while handling lead-bearing wastes, sludge, and filters employees come in contact with lead-containing wastes.

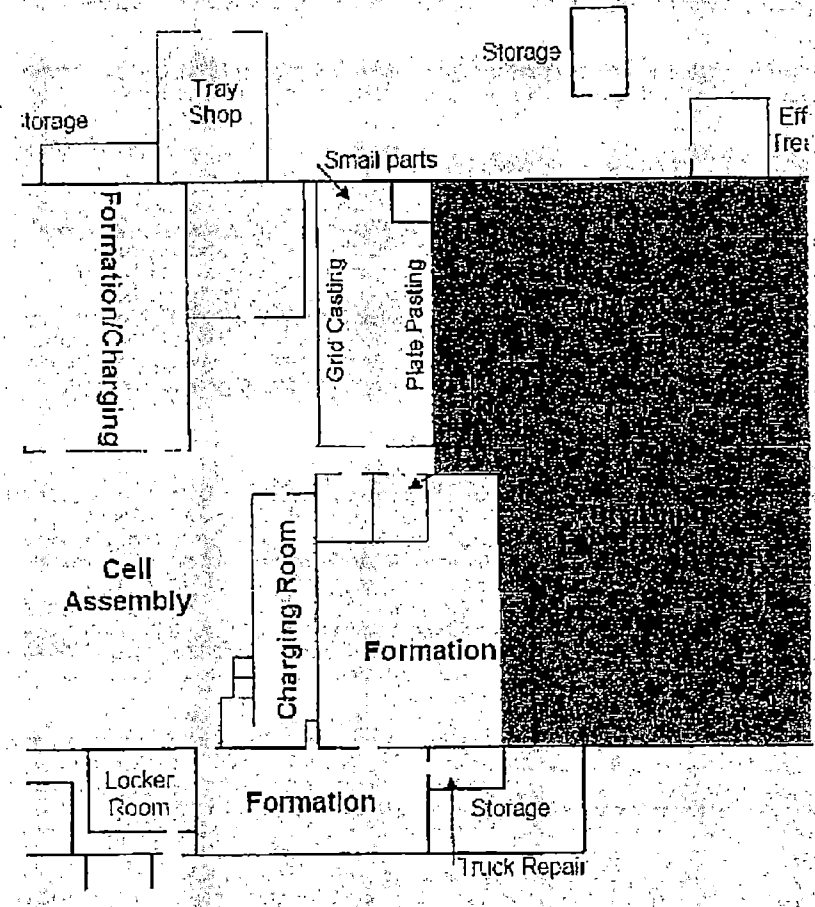
2. Baghouse: Dust collection systems are a source of lead exposure. Sources of exposure include:

    - While cleaning and maintaining dust collection systems (cyclones and baghouses).

- While handling and transporting materials that are removed from dust collection systems (cyclones and baghouses).
- While working near dust collection systems.

E. Maintenance

Maintenance personnel are at risk of exposure to lead fumes and dust during maintenance operations involving equipment that is contaminated with lead. Maintenance shop personnel are exposed to lead dust when servicing and handling equipment that is contaminated with lead.



MAP: DISTRIBUTION AREA

IV. Lead Toxicology

A. General

Lead poisoning is a multisystem disease characterized by a wide spectrum of injury, most strikingly observed by neurologic, renal and cardiovascular injury. There appears to be no threshold for this effect with demonstrated effects down to blood

lead levels which represent population level exposure. In adults, multiple effects seen with occupational exposures are characterized by hypertension and other cardiovascular dysfunctions (including coronary artery disease, stroke mortality, and peripheral artery disease), renal toxicity, cognitive dysfunction, peripheral neuropathy and anemia. Both acute and chronic lead exposure is a cause of cognitive dysfunction and hypertension in adults as they age.

It is important to recognize that lead is a cumulative toxicant in terms of health effects as well as storage in the body with a mean life in bone of approximately thirty years. Therefore chronic effects that are seen in adults can be related to exposures throughout life and that processes later in life, such as pregnancy, immobilization (such as with bed rest or treatment of fractures), renal disease, osteoporosis of aging, and other aging and disease processes mobilize lead from bone increasing the risk and severity of the chronic lead effects. It has become clear from many studies that lifetime lead exposure can be expressed later in life and particularly in the aged, where it results, among other effects, in increased rates of decline in cognition.

Most persons with chronic lead poisoning have no acute symptoms, even with marked elevations of blood lead. In persons with hypertension and other cardiovascular disorders and lead exposure, lead toxicity should be considered as causative or as an aggravating factor.

B. Exposure

Lead exposure occurs through inhalation and ingestion only. Airborne lead is characterized by point-source and non point-source exposure. In the USA removal of lead from gasoline beginning in 1975 dramatically decreased the population wide exposure to lead from this source and closure of lead smelters in USA has removed the major point-source exposure. Decreases in airborne lead in the USA, as well as the removal of lead from solder used in food cans, paint, and other products, resulted in a decline in blood lead levels for individuals 1-74 years of age from 12.8 ugm/dl in 1976-80 to 2.8 ugm/dl in 1991. For adults, the primary remaining source of lead exposure is occupational (such as scraping off old lead paint, smelters, battery manufacturing, radiator repair, etc.). Occupational exposures have declined significantly in recent years. With the multiple real and potential sources of lead exposure throughout life, since lead is a cumulative toxicant with very prolonged residence in bone, evaluation of sources of exposure requires evaluation of prior occupational exposure, particularly in older adults.

C. Toxic effects

Lead toxicity results almost exclusively from inhalation or ingestion of lead from environmental sources, food, or water. Small lead particulates are well absorbed from the lungs. Ingested particles are absorbed less well, with absorption efficiency partially determined by particle size as small particles promote increased absorption. Once absorbed, most circulating lead is bound to red blood cells, at least below blood lead levels less than 60 ugm/dl. The small fraction in plasma is distributed to tissues at rather slow rates with the majority excreted in urine. The proportion distributed to target organs, including particularly the nervous system, bone marrow, vascular system, and kidneys is responsible for the observed toxicity of lead. Due to this relatively slow transport of lead to target organs, clinical toxicity generally requires long term exposure, measured in months to years. Bone is a major repository of lead, containing the vast majority of body lead burden (>90%) and the residency of lead in bone is measured in decades, thereby providing a continual source of lead to target tissues, even after cessation of exposure and/or chelation therapy to remove the lead.

Lead competes with other minerals particularly calcium in cellular and sub-cellular systems. For instance, it has been shown to directly affect mitochondrial function *in vitro* by interfering with calcium uptake. Lead damages the production of heme, the protein responsible for carrying oxygen throughout the body. Its effect on heme synthesis has been studied for many years and it has been demonstrated that at very low levels it inhibits aminolevulinic acid dehydratase and at somewhat higher levels inhibits heme sythetase. It is felt that this effect may have important diffuse effects on a variety of heme-dependent processes. Similarly, it is felt that lead is a potent inhibitor of protein kinase C, a calcium dependent enzyme critical to brain function. It also appears to interfere with calcium dependent control of neurotransmitter function at pre-synaptic nerve terminals. Lead has likewise been known for many years to be a potent toxin to the proximal renal tubules in the kidneys, with accumulation of intracellular inclusions at high levels of exposure. This dysfunction results in a renal fanconi syndrome, with phosphaturia, glucosuria, and aminoaciduria. It is less clear what is the etiology of the association with hypertension and other cardiovascular manifestations of lead exposure seen in adult subjects. Some studies suggest renal causes due to decreased glomerular filtration rates or stimulation of the renin-angiotensin system. Other proposed hypotheses suggest a more direct effect on vascular tone in the body. It is also possible that lead exerts some of its effects through epigenetic mechanisms with resulting impacts on gene programming and gene expression.

Lead poisoning is a chronic illness. It has long been recognized that much of the toxicity of lead poisoning is not reversible by medical therapy. Prevention of exposure is the main aim in lead poisoning management as treatment has little effect on

reversing toxicity or preventing toxicity later in life related to lead mobilization from bone. Since lead remains in bone lead stores for many decades, it is considered a chronic illness requiring long term management and observation

D. Diagnosis

The measurement of whole blood lead is the hallmark of diagnosis. Regular blood lead testing at the Exide plant provides very extensive data on the amount of lead absorbed into the bodies of the Exide workers and is therefore a very useful diagnostic tool. Another approach to estimating the cumulative amount of lead in the body is the direct measurement of lead in bone. A third approach to measuring the amount of lead that has been absorbed into the body involves the calculation of a cumulative blood lead index (CBLI) by averaging the results of blood lead test results over time. The CBLI has recently been shown to be a useful approach to assessing the level of cumulative lead exposure, particularly in situations where repeated blood lead determinations have been made over time (e.g. in the workplace). The CBLI provides an index of an individual's cumulative lead exposure which is correlated with the risk of development of conditions caused by lead exposure. An approach has been developed and published for converting between CBLI and bone lead levels.

E. Conditions caused by lead exposure in the workplace

1. **Acute lead poisoning**- Workers with lead exposure develop acute lead poisoning with symptoms of abdominal colic, fatigue, anemia and peripheral neuropathy. Such manifestations are due to high lead exposure levels which, at times, is also associated with alteration of central nervous system function and, in rare cases, acute toxic encephalopathy. More often, lead exposure is associated with the manifestations of chronic lead poisoning.

2. **Chronic lead poisoning**- Exposed workers also experience a range of health problems caused by chronic exposure to lead. Research has shown a range of health problems due to lead exposure at increasingly lower levels of exposure, leading the medical community to lower estimates of the "safe" level of exposure in the workplace. These health problems typically develop insidiously and are often not initially attributed to lead exposure. Further, lead exposure aggravates a range of medical conditions, thereby enhancing the development of morbidity and mortality due to these conditions. It is clear that lead toxicity occurs at levels down to the lowest observed lead levels and that occupational standards for lead exposure do not prevent lead toxicity with resulting medical impairment. Chronic lead poisoning is manifested by a range of damage to various systems of the body.

   a) **Hematologic Toxicity**- Lead damages the body's ability to produce heme resulting in **anemia.** The higher the blood lead concentration the greater the prevalence of anemia in lead-exposed individuals. Typically, once exposure ceases and the blood lead level falls, the anemia is reversible. A blood test measuring the concentration of zinc protoporphyrin is used to quantify the inhibitory effect of lead on the red blood cell producing system.

   b) **Neurologic Toxicity**- Lead causes damage to the peripheral and central nervous systems which persists after exposure ceases. Damage to peripheral nerves causes a **peripheral neuropathy** which is manifest as motor weakness or, at times, as sensory impairment. Nerve conduction testing is used to diagnose lead-related peripheral neuropathy. Also excessive lead exposure has been shown to cause **cognitive impairment,** characterized by memory impairment, mood changes, reduced visual-motor function, and other abnormalities of neurobehavioral function. The onset of cognitive impairment is often insidious and often the association with lead exposure is not immediately recognized. Formal neurobehavioral testing is used to diagnose **cognitive impairment** in lead exposed individuals.

   Cognitive Reserve Capacity- Recent research has shown that individuals exposed to toxic agents do not demonstrate evidence of cognitive impairment until some time after the exposure has ceased as a result of the existence of cognitive reserve capacity. As noted in the figures below, cognitive function improves through early childhood and commonly declines in later years without reaching a threshold for the manifestation of dementia.

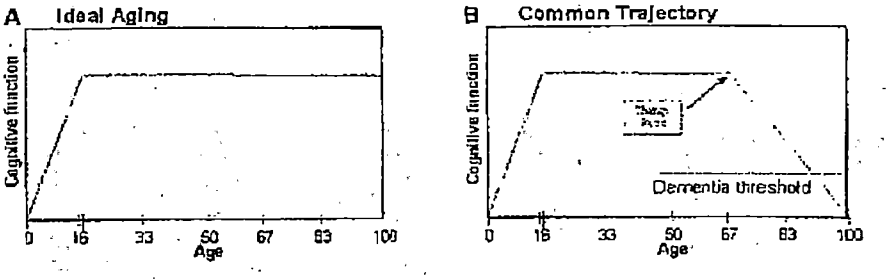


Figure: Ideal and normal changes in cognitive function over a life time.

Toxic exposures cause delayed cognitive impairment- Exposure to a neurotoxic agent such as lead causes brain damage which is manifest only later in life during the time in which normal aging occurs, thereby **accelerating the aging process** (as noted in the figure below). As a result, symptoms of lead-related cognitive impairment develop but are not apparent until later in life.

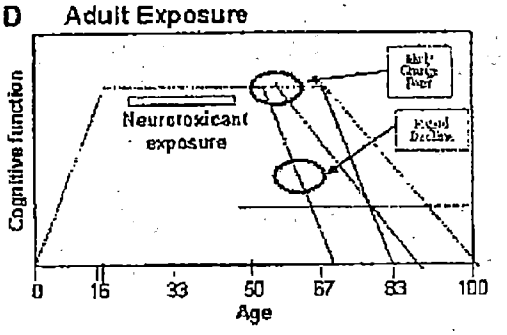


Figure: Impact of adult lead exposure on decline in cognitive function

c) **Renal Toxicity-** Lead is known to cause damage to the kidney which results in **chronic renal failure** and causes or contributes to the development of **hypertension.** Lead is known to damage the kidney tubules leading to Fanconi syndrome, a condition in which glucose, amino acids and phosphates are spilled into the urine. Lead poisoning also causes **gout** due to the disturbance of clearance of uric acid by the kidney. Tests of kidney function are used to diagnose renal toxicity caused by lead exposure.

d) **Hypertension and heart disease-** Human and animal research has clearly demonstrated an association between elevated lead exposure and the occurrence of **hypertension.** Further, a clear association exists between the development of hypertension and the occurrence of **heart disease** and **stroke.** Therefore, lead exposure is one cause of **heart disease** and **stroke;** specific cardiovascular conditions caused by lead exposure include **myocardial infarction** (i.e. "heart attack") and **left ventricular hypertrophy** (i.e. "enlargement of the heart").

Hypertension is defined by the American Heart Association as follows: '

- Normal blood pressure: In the range 130/85
- High normal: In the ranges 130–140/85–90
- Mild hypertension: In the ranges 140–160/90–100
- Moderate hypertension: In the ranges 160–180/100–110
- Severe hypertension: In the ranges 180–210/110–120
- Very severe hypertension: Higher than 210/120

e) **Reproductive Toxicity**– Lead also causes impairment of reproductive function in both men and women exposed to lead. Women with lead exposure have been found to have increased rates of spontaneous abortion. Men with lead exposure have been found to have low sperm counts and reduced androgen concentrations, leading to decreased fertility. Lead passes through the placental barrier and low concentrations of lead in the blood of a pregnant woman causes damage to the developing brain of the fetus. Further, since pregnancy can be associated with the mobilization of lead from bone stores, a woman who had developed lead toxicity in the past can be at risk for lead-related damage to her fetus.

f) **Carcinogenicity**– Lead has been recognized by the International Agency for Research on Cancer (IARC) as being a "probable human carcinogen (group 2A)". Kidney cancer has been associated with lead exposure in a number of studies.

V. Analyses of Exide worker health records

A. **Health Record Review**– Preliminary review of medical records of a subset of Exide workers revealed that a significant number of them were diagnosed with conditions that are known to be caused by or aggravated by lead exposure. These include:

- Hypertension
- Heart disease
- Kidney failure
- Neuropathy
- Memory complaints
- Gout
- Kidney cancer

B. **Blood Lead Levels** – A review of the records of blood lead testing of Exide workers revealed that workers had blood lead tests results as high as 102 ugm/dl, with many whose blood lead concentrations remained above 50 ug/dl for extended periods of time. Blood lead concentrations in the Exide workers were many times the normal blood lead levels of the general population. Currently, the average blood lead level of the general population is approximately 3 ugm/dl.

VI. Overview

- Battery manufacturing is known to be associated with significant worker exposure to lead
- Lead is known to cause damage to the body if significant exposures occur
- Health effects known to be associated with significant lead exposure include:
  1. Hypertension
  2. Anemia
  3. Brain damage
  4. Peripheral nerve damage
  5. Kidney failure
  6. Gout
  7. Kidney cancer
  8. Heart disease

- Workers employed at the Exide battery manufacturing plant in Sumter SC experienced significant, prolonged exposure to lead as a result of inhalation and ingestion of lead-containing dust emanating from production processes in the plant
- As a result of prolonged lead exposure, workers had elevated blood lead levels, indicating increased lead absorption into the body
- Workers experienced a range of adverse health effects which are known to be caused or aggravated by lead exposure, These conditions include:
    1. Hypertension
    2. Heart disease
    3. Kidney failure
    4. Neuropathy
    5. Memory and mood complaints
    6. Gout
    7. Kidney cancer
- The conditions which they experienced are associated with significant morbidity and mortality
- The conditions which they experienced become worse with age as lead is mobilized from bone, where the mean life of lead in bone is 30 years.

VII. Summary and Conclusions

Based upon our extensive experience as experts in the field of occupational and environmental medicine, toxicology, epidemiology, pediatrics, and internal medicine, with over 80 years of combined research and clinical experience, and upon an extensive review of scientific literature, battery manufacturing processes, and health records of Exide workers, we conclude that workers at the Exide plant in Sumter SC experienced lead exposure which caused or contributed to the occurrence of a range of significant health problems. These health problems include hypertension, heart disease, kidney failure, nervous system damage, gout and kidney cancer. These conditions are or will be associated with significant morbidity and mortality resulting in decreased quality of life and premature death.

As experts in the fields noted above, we have received specialized training and education in occupational and environmental medicine, toxicology, and clinical medicine. We have practiced medicine and have personally examined and treated individuals with lead poisoning and diseases caused by toxic chemicals. We have conducted research at major US medical centers and at the Centers for Disease Control and Prevention (CDC) on the health effects of lead exposure and have published this research in peer-reviewed publications. We have been invited to write chapters in medical textbooks on the subject of lead poisoning and have been invited to speak at medical conferences and workshops around the world on our research. We have served as professors at Harvard University, the Medical University of South Carolina, the University of North Carolina and the University of Michigan, where we have taught medical doctors and other health professionals in the areas of toxicology, epidemiology and occupational and environmental medicine. We have been invited to consult with major government organizations including the World Health Organization, the US Environmental Protection Agency, the Centers for Disease Control and Prevention and the Occupational Health and Safety Administration. One of us held the position of Deputy Director of the US National Institute for Occupational Safety and Health (NIOSH). We all remain active in our fields and are currently involved in research and academic publication in the field of occupational and environmental medicine.

Our health hazard analysis of the Exide lead battery plant consisted of a review of the battery plant processes in general and the Exide processes in particular. We utilized detailed information provided by the US Occupational Health and Safety Administration on health hazards of the battery manufacturing to identify specific plant processes associated with exposure to lead dust and fumes. We reviewed research on other battery plants, including a study conducted by one of us, to determine the nature and extent of lead exposure in other battery plants. We met with former workers at the plant to review plant processes in detail and to develop an understanding of the nature of work activities in specific areas of the Exide plant. We reviewed and summarized the health literature on lead poisoning to identify those conditions which have been shown in scientific research to be caused by or significantly contributed to by chronic lead exposure in adults.We then reviewed health records of former Exide workers including extensive records of the results of personal blood lead testing performed by the Exide plant. We reviewed medical records documenting the health status and treatment histories of these workers. We used the results of blood lead testing to calculate a cumulative blood lead index (CBLI) measurement for each worker and then compared the CBLI results with those of the normal US population. From the review of the health records, we identified a set of health conditions which are known to be caused by lead exposure.

Our extensive reviews of the records of the Exide workers have revealed that these individuals had very elevated blood lead concentrations. The elevated blood lead concentrations were caused by exposure to lead dust and fumes during their performance of their normal job duties at the Exide plant in Sumter SC; we did not identify any other plausible source of significant lead exposure to these individuals. We conclude that exposure to lead dust and fumes in the Exide plant resulted in inhalation or ingestion of lead into the body of Exide workers.

We also conclude that lead, once absorbed into the body, was distributed to various parts of the body, including the brain, the kidneys and bone, and caused damage to the body of Exide workers. The damage to the body of Exide workers was manifested by the development or aggravation of a number of diseases or health conditions. These include hypertension, heart disease, kidney failure, nervous system damage, gout, and kidney cancer. Exide workers who developed these conditions experienced significant morbidity and premature mortality and experienced impairment of the normal functions of daily life and work. These workers required medical care including hospitalization, medications, and ongoing medical treatment. These diseases and health conditions that they experienced are likely to be permanent and will persist indefinitely; these former Exide workers will require long term medical care for these conditions. Furthermore, some former Exide workers are likely to develop disease or health conditions in the future caused by or aggravated by their prior exposure to lead at the Exide plant as a result of the latent toxicity of lead.

We hold these opinions to a reasonable degree of medical certainty and base our opinions on our extensive professional experience and expertise, our review of battery manufacturing processes in general and the Exide plant in particular, our review of the scientific literature on adult lead poisoning (some of which we have written), and our review of the health records of Exide workers. We may supplement or modify this report as additional information becomes available to us.

VIII. References

1. Association of Occupational and Environmental Clinics: Medical Management Guidelines for Lead-Exposed Adults April 2006. www.aoec.org/documents.

2. Hu H, Rabinowitz M, Smith D. Bone lead as a biological marker in epidemiologic studies of chronic toxicity: conceptual paradigms. Environ Health Perspect. 1998 Jan;106(1):1-8

3. Dolcourt JL, Hamrick HJ, O'Tuama LA, Wooten J, Baker EL Jr Increased lead burden in children of battery workers: asymptomatic exposure resulting from contaminated work clothing. Pediatrics. 1978 Oct;62(4):563-6.

4. Schwartz BS, Hu H Adult Lead Exposure: Time for Change. Environ Health Perspect 115: 451-454 (2007).

5. Hu H, Shih R , Rothenberg S, et al The Epidemiology of Lead Toxicity in Adults: Measuring Dose and Consideration of Other Methodological Issues. Environ Health Perspect 115: 455-462, (2007).

6. Baker EL and Fiedler N, Neurobehavioral disorders, in Levy B, Wegman D, Baron S and Sokas R, Occupational and Environmental Health, 6th Edition, in press.

7. Reigart, JR, Lead Toxicity. BMJ Point-of-Care 2009; www.pointofcare.bmj.com (in press)

8. Baker EL, Landrigan PJ, Barbour AG, et al. Occupational Lead Poisoning in the United States. Brit J Ind Med 36: 314-22, 1979.

9. Stewart WF, Schwartz BS. Effects of Lead on the Adult Brain; A 15-year Exploration. Amer J Ind Med 50: 729-739, 2007.

10. Occupational Safety and Health Administration, Battery Manufacturing, OHSA website, www.osha.gov, accessed May 2009.

11. Pilsner JR, Hu H, Ettinger A, Sanchez BN, Wright RO, Cantonwine D, Lazarus A, Lamadrid -Figueroa H, Mercado-Garcia A, Tellez-Rojo MM, Hernandez-Avila M. Influence of Prenatal Lead Exposure on Genomic DNA Methylation of Umbilical Cord Blood. Environ Health Perspec (in press) on-line 3/25/09.

12. Weisskopf MG, Jain N, Nie H, Sparrow D, Vokonas P, Schwartz J, Hu H. A prospective study of bone lead concentration and death from all causes, cardiovascular diseases, and cancer in the VA Normative Aging Study. Circulation (in press).

IX. Other Sources of Information

IX. Other Sources of Information

1. Former worker interviews
2. Medical records
3. Plant map and material safety data sheets

X. Report authored by:

Edward L. Baker MD, MPH

9/11/09

Date

Howard Hu MD, MPH, ScD

9/2/09

Date

John Routt Reigart, II, MD

9/17/09

Date

http://www.kidney-international.org
© 2006 International Society of Nephrology

Lead-related nephrotoxicity: A review of the epidemiologic evidence

EB Ekong[1], BG Jaar[2,3] and VM Weaver[1,3]

[1]Division of Occupational and Environmental Health, Department of Environmental Health Sciences, Johns Hopkins University Bloomberg School of Public Health, Baltimore, Maryland, USA; [2]Department of Epidemiology, Johns Hopkins University Bloomberg School of Public Health, Baltimore, Maryland, USA and [3]Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland, USA

Chronic kidney disease (CKD) represents a major global public health concern. Efforts to prevent and/or slow progression of CKD are essential. Lead nephropathy, characterized by chronic tubulointerstitial nephritis, is a well-known risk of chronic, high-level lead exposure. However, in recent years, lead exposure has declined sharply, particularly in developed countries. We reviewed epidemiologic research in general, occupational, and patient populations to assess whether lead, at current exposure levels, still contributes to nephrotoxicity. Other pertinent topics, such as risk in children, genetic susceptibility, and coexposure to cadmium, are also considered. The data reviewed indicate that lead contributes to nephrotoxicity, even at blood lead levels below 5 μg/dl. This is particularly true in susceptible populations, such as those with hypertension (HTN), diabetes, and/or CKD. Low socioeconomic status is a risk factor for both lead exposure and diseases that increase susceptibility. Future public health risk for lead-related nephrotoxicity may be most significant in those rapidly developing countries where risk factors for CKD, including obesity and secondary HTN and diabetes mellitus, are increasing more rapidly than lead exposure is declining. Global efforts to reduce lead exposure remain important. Research is also needed to determine whether specific therapies, such as chelation, are beneficial in susceptible populations.

Kidney International (2006) 70, 2074–2084. doi:10.1038/sj.ki.5001809; published online 25 October 2006

KEYWORDS: environmental lead exposure; kidney function; occupational lead exposure; renal biomarkers

Correspondence: *VM Weaver, Division of Occupational and Environmental Health, Johns Hopkins University Bloomberg School of Public Health, 615 N. Wolfe St., Rm. 7041, Baltimore, Maryland 21205, USA.*
E-mail: vweaver@jhsph.edu

Received 13 February 2006; revised 11 July 2006; accepted 18 July 2006; published online 25 October 2006

Chronic kidney disease (CKD) represents a major public health concern. A recent analysis of US National Health and Nutrition Examination Survey data estimated that 19 million Americans have CKD, which includes end-stage renal disease and the four stages of renal dysfunction that precede it.[1] Worldwide, the estimated number of end-stage renal disease patients increased from 886 023 in 1999 to 1 131 594 in 2003.[2] Prevalence rates vary and are much higher in certain groups. In the US, for example, African-Americans have rates of end-stage renal disease that are four times higher than those in Caucasians.[2] Despite improvements in therapy, mortality remains substantial. Fewer than 40% of patients survive more than 5 years after onset of dialysis.[2] Recent data indicate that obesity is also increasing globally,[3,4] resulting in greater prevalence of diabetes mellitus and hypertension (HTN),[5–8] which are the leading causes of CKD. Obesity may increase CKD risk by other pathways as well.[9]

In this setting, strategies to prevent CKD and/or slow progression at earlier stages are imperative and will require a global effort. Exposure to environmental and occupational nephrotoxicants is not commonly considered in this regard. However, chronic, high-level lead exposure, that is, blood lead levels persistently above 70–80 μg/dl, is an established risk factor for CKD. This has been documented in long-term follow-up of children in the Queensland, Australia lead poisoning epidemic,[10] mortality studies of lead exposed workers,[11] historical occupational experience,[12] and animal models.[13] At this level of exposure, lead is generally the primary cause of renal pathology, which is characterized by chronic tubulointerstitial nephritis and diagnosed as lead nephropathy.[14] Fortunately, exposure at these levels is increasingly rare, particularly in developed countries. The geometric mean blood lead level in adults declined from 13.1 μg/dl in the National Health and Nutrition Examination Survey II, conducted between 1976–1980,[15] to 1.56 μg/dl in the most recent survey conducted between 2001 and 2002.[16] Similarly, occupational lead exposure in industrialized countries, although much higher than environmental exposure, is well below historic levels associated with lead nephropathy.

In this review, we consider whether current lower levels of lead exposure continue to contribute to nephrotoxicity. There are a number of reasons for concern in this regard. Globally, environmental exposure to lead is ubiquitous. Lead is stored in bone resulting in ongoing endogenous exposure. Body burdens of such cumulative toxicants tend to increase with age as does risk for renal disease from other factors. In addition, despite the overall decline in exposure, certain populations, even in developed countries, continue to experience higher lead exposure.[17,18] These include inner city children and adults of lower socioeconomic status, particularly African-Americans who, as noted above, also have a higher prevalence of end-stage renal disease. Occupational settings of particular concern include small and/or mobile workplaces, such as radiator repair shops and construction sites. Children continue to be exposed from lead paint. Many other sources of lead exposure have been identified, such as children's jewelry,[19] folk remedies, glazed pottery, and even candy.[20] Recently, attention has focused on urban water as a continued source of lead exposure.[21,22] Internationally, blood lead levels are higher in developing countries owing to continued use or later phase-out of leaded gasoline and paint.[23,24] Occupational exposure in these countries is higher as well.

In order to review recent research of relevance for the question of lead-related nephrotoxicity at current exposure levels, we have categorized the literature by study population as strengths, weaknesses, and conclusions that can be made are different in each setting. Several lead dose measures are used in this body of literature. Blood lead is a short-term measure (half-life of 30 days[25]) that reflects exposure from current exogenous sources and the release of endogenous lead from bone. Bone lead is a cumulative dose measure that also provides information on potential for endogenous exposure.[25,26] Lead in trabecular bone (commonly measured in the patella or calcaneus) is more bioavailable than lead in cortical bone (measured in the mid-tibia) and has a shorter half-life (estimated at 1–16 years compared to 10–30 years for cortical bone.[25,27] Chelatable lead is thought to represent a bioavailable pool of lead from blood, soft tissue, and bone. Calcium disodium ethylenediaminetetraacetic acid (EDTA) has traditionally been used for chelation; dimercaptosuccinic acid (Succimer) is a more recent option.

LITERATURE SEARCH STRATEGY

We searched MEDLINE for studies involving the effect of lead on the kidney in humans using the following terms: lead and (occupational, environmental, or exposure) and (kidney, renal or nephrotoxicity). Limits included human and English language. The time period covered 1 January 1985–31 December 2005 to include the initial general population studies on the topic. An additional search for general population studies was conducted using the same search terms and time period in an Embase search, which was limited to humans but had no language limits. Pertinent earlier papers from the investigators' files were discussed when relevant. We manually reviewed cited references from identified articles. Owing to the heterogeneous populations studied, small number of publications in general populations, and the range of renal outcomes analyzed, no attempt to pool data quantitatively was made. Therefore, studies were not excluded *per se*. However, studies that incorporated stronger designs and analyzed renal outcomes of known prognostic value were emphasized.

GENERAL POPULATION STUDIES

We identified 17 publications within the past two decades that have evaluated the effect of environmental lead exposure on renal function in adult general populations.[17,28–43] In six publications, early biological effect markers were the only renal outcome measures analyzed.[38–43] Determining the clinical relevance of these results is difficult owing to the limited number of prospective studies documenting the prognostic value of these markers in lead exposed populations. Therefore, the 11 studies that analyzed clinical renal outcomes (blood urea nitrogen, serum creatinine, measured and/or estimated creatinine clearance, and/or estimated glomerular filtration rate (GFR)) were summarized (Table 1). These publications included eight populations, primarily in the US and Europe. Four publications addressed different time points or lead dose measures in the same longitudinal study (Normative Aging Study). Statistically significant associations between higher lead dose and worse renal function were observed in nine publications (representing six different populations). Associations between blood lead and creatinine-based renal outcomes were the most commonly reported.[17,29–31,33,35,36] Results in longitudinal data were consistent with those observed cross-sectionally, although significant associations were limited to susceptible populations (diabetics and hypertensives) in one study.[33] Hypertensives also emerged as a susceptible group in National Health and Nutrition Examination Survey data.[35] Both studies that measured bone lead reported associations not observed with blood lead, suggesting that assessment of cumulative lead dose is important in understanding lead-related nephrotoxicity.[32,33] A review of the two studies in which no significant associations were observed is notable for the fact that one is limited by small sample size and minimal statistical analysis.[37]

Overall, these studies have a number of strengths including assessment of a range of lead dose measures and renal outcomes; statistical analysis that adjusted for numerous renal risk factors and, in two, evaluated longitudinal data;[31,33] and generally large sample sizes. The analyses of National Health and Nutrition Examination Survey data have the advantage of being representative of the US non-institutionalized, civilian population.[17,35] The general consistency of the results provides important evidence that lead-related nephrotoxicity remains a public health concern, particularly in susceptible populations.

Table 1 | Research on the renal effects of lead exposure in general populations[a]

Reference and study location	Study population; date; sample size; % male	Mean (s.d. or range) Age (years) Blood lead (μg/dl)	Study design	Statistical modeling; covariates	Adjusted β coefficients (95% CI) for observed associations
Longitudinal studies					
Kim *et al.*[31] Boston, MA, USA	Normative Aging Study; 1979–1994; 459; 100%	At baseline: 56.9 (37.7–87.5) years 9.9 (6.1) μg/dl	Longitudinal and cross-sectional analysis of data from examinations conducted every 3–5 years; Exclusionary criteria not specified	Random effects Baseline age, time since initial visit, body mass index, smoking status, alcohol ingestion, education level, hypertension, and in longitudinal analysis, serum creatinine at the beginning of the follow-up interval and time between evaluations	*Longitudinal analysis* Log-transformed blood lead and change in serum creatinine over subsequent follow-up period in: All participants (P=0.07) 0.02 (−0.004, 0.044) 428 whose peak blood lead was ⩽25 μg/dl 0.027 (0.0, 0.054) *Cross-sectional analysis* Log-transformed blood lead and concurrent serum creatinine in: All participants 0.033 (0.009, 0.057) 741 whose peak blood lead was ⩽10 μg/dl 0.06 (0.023, 0.097) 10-fold higher blood lead level associated with 0.08 (95% CI: 0.02, 0.13) mg/dl higher concurrent serum creatinine and a 0.05 (95% CI: −0.01, 0.10) mg/dl greater change in serum creatinine over the subsequent follow-up interval in all participants after adjustment
Tsaih *et al.*[33] Boston, MA, USA	Normative Aging Study; 1991 to ~2001; 448; 100%	At baseline: 66.0 (6.5) years; 6.5 (4.2) μg/dl	Longitudinal and cross-sectional analysis of data from two evaluations over a mean 6-year period Six (n=26) and 26% of subjects had diabetes and hypertension, at baseline, respectively Tibia and patella lead also measured Exclusionary criteria=no follow-up in 259; serum creatinine, lead dose, and key covariates not statistically different at baseline between those with and without follow-up	Multiple linear regression, interaction models Age, age squared, body mass index, diabetes, hypertension, smoking status, alcohol consumption, analgesic, and diuretic use, and in longitudinal analyses, baseline serum creatinine and its square	*Longitudinal analysis* Baseline lead dose not associated with change in creatinine in all 448. 0.009 (−0.001, 0.019) (for natural ln-transformed blood lead) Significant interaction of ln baseline blood and tibia lead with diabetes in models of annual change in serum creatinine. For ln blood lead: 0.076 (0.031, 0.121) in diabetics 0.006 (−0.004, 0.016) in non-diabetics Interaction also noted in hypertensive participants with tibia lead, although the diabetics likely present in this group may have been influential there as well. *Cross-sectional analysis* No significant associations between lead dose and concurrent serum creatinine at baseline in all 448; ln-transformed tibia lead associated with serum creatinine in diabetics. One of three associations at follow-up significant: natural ln-transformed blood lead with concurrent serum creatinine 0.149 (0.041, 0.257)
Cross-sectional studies; Renal outcomes include estimated/measured creatinine clearance and/or estimated GFR					
Akerson *et al.*[36] Sweden	Women's Health in the Lund Area Study; 1999–2003; 820; 0%	Medians: 58 (53–64) years 2.2 (1.1–4.6 (5th and 95th %)) μg/dl	Exclusionary criteria=renal cancer (n=1) and lithium treatment (n=3)	Multiple linear regression Age, body mass index, diabetes, hypertension, and regular use of nephrotoxic drug, blood, and urinary cadmium (in separate models), smoking status (by stratification)	Blood lead (ug/dl) with GFR (ml/min) estimated from cystatin C[34] −2.0 (−3.2, −0.9) Blood lead (μg/dl) with estimated creatinine clearance[b] (ml/min): −1.8 (−3.0, −0.7)
Muntner *et al.*[35] US	NHANES III; 1988–1994; Study population representative of US non-institutionalized,	Age reported categorically 4.21 (0.14) μg/dl (hypertensives) 3.30 (0.10) μg/dl (normotensives)	Renal outcomes=elevated serum creatinine (defined as ⩾99th percentile of each race-gender-specific distribution for participants aged 20–39 years without	Multiple logistic regression Age, race, gender, diabetes, systolic blood pressure, smoking	Odds ratios for both renal outcomes increased by quartile of blood lead among hypertensives but not normotensives. Odds ratios for CKD in hypertensives after adjustment

Table 1 continued on following page

Table 1 | Continued

Reference and study location	Study population; date; sample size; % male	Mean (s.d. or range) Age (years) Blood lead (μg/dl)	Study design	Statistical modeling; covariates	Adjusted β coefficients (95% CI) for observed associations
	civilian population; 15 211 (4813 hypertensives); 48%		hypertension or diabetes) and CKD (defined as estimated GFR[a] <60 ml/min/1.73 m^2) Exclusionary criteria not specified; data from 13 141 of 15 825 adults used in final models	status, history of cardiovascular disease, body mass index, alcohol consumption, household income, education level, marital status, and health insurance	*Blood lead* — %: *Odds ratio (95% CI)* Quantile 1 (0.7–2.4): 6.1: 1.00 Quantile 2 (2.5–3.8): 10.4: 1.44 (1.00, 2.09) Quantile 3 (3.9–5.9): 10.8: 1.85 (1.32, 2.59) Quantile 4 (6.0–56.0): 14.1: 2.60 (1.52, 4.45) $P<0.001$ for χ^2 test for trend Twofold higher blood lead associated with odds ratio for CKD of 1.38 (95% CI: 1.15, 1.66) in hypertensives after adjustment In normotensives, higher blood lead was associated with a higher prevalence of CKD in diabetics
Muntner *et al.*[17] US	NHANES 1999–2002; 9561; gender not reported	Age not reported Geometric mean (95% CI) 1.64 (1.59, 1.68) μg/dl	CKD (defined as estimated GFR[a] <60 ml/min/1.73 m^2) Exclusionary criteria not specified	Multiple logistic regression Age, race/ethnicity, gender, diabetes, smoking status, alcohol, body mass index, education, and health insurance	Odds ratios for CKD in all increased by quartile of blood lead, significant χ^2 test for trend
Payton *et al.*[20] Boston, MA, USA	Normative Aging Study; 1988–1991; 744; 100%	64.0 (7.4) years; 8.1 (3.9) μg/dl	Exclusionary criteria=missing data present in 251	Multiple linear regression Age, body mass index, analgesic & diuretic use, alcohol consumption, smoking status, systolic/diastolic blood pressure	Log transformed (ln) blood lead with ln 24-h measured creatinine clearance −0.04 (−0.079, −0.001) After adjustment, a 10 μg/dl higher ln blood lead was associated with a 10.4 ml/min lower ln creatinine clearance. A 10-fold increase in blood lead associated with 9% decrease in creatinine clearance (as noted by Kim *et al.*[11]) Borderline significant ($P<0.1$) associations between ln blood lead and both serum creatinine (β=0.027) and ln estimated creatinine clearance[b] (β=0.022)
Staessen *et al.*[29] Belgium	Cadmibel study; 1985–1989; general Belgian population in four cadmium polluted and unpolluted areas; 1981; 48.7%	48 (16) years Geometric mean Males: 11.4 (2.3–72.5) μg/dl Females: 7.5 (1.7–60.3) μg/dl	Exclusionary criteria=missing data present in 302; potentially inaccurate 24-h urine collections in 44	Multiple linear regression Age, age squared, gender (by stratifying), body mass index, blood pressure, ferritin level, smoking status, alcohol ingestion, rural vs urban residence, analgesic, and diuretic use, blood, and urinary cadmium, diabetes, occupational exposure to heavy metals, and gamma glutamyl transpeptidase	Log transformed blood lead with 24 h measured creatinine clearance −9.5 (−18.1, −0.9) in males −12.6 (−20.3, −5.0) in females 10-fold higher blood lead associated with 10 and 13 ml/min lower measured creatinine clearance in men and women, respectively Log-transformed blood lead also negatively associated with estimated creatinine clearance[b] but not significantly associated with serum creatinine
Wu *et al.*[22] Boston, MA, USA	Normative Aging Study, 1991–1995 709; 100%	67.0 (7.4) years 6.2 (4.1) μg/dl	Tibia and patella lead also measured Exclusionary criteria not specified	Multiple linear regression Age, body mass index, hypertension, smoking status, alcohol ingestion, analgesic medication use	Significant negative association between patella lead and estimated creatinine clearance[b]: β=−0.069; P=0.03 (neither s.e. nor CI provided) Borderline significant (P=0.06) negative association between tibia lead and creatinine clearance. No lead measure was significantly associated with serum creatinine; no blood lead associations were significant
Cross-sectional studies: Other clinical renal outcomes					
De Burbure *et al.*[24] France	399 adults who lived ≥8 years near two nonferrous smelters; 50% males	Means ranged from 34.6 (8.9) years (exposed males) to 35.9 (9.6) years (exposed females)	Exclusionary criteria included pregnancy, cancer, diabetes, kidney disease, occupational smelter	Multiple linear regression Age, sex, body mass	No significant difference in mean serum creatinine between exposed and unexposed groups. After adjustment for covariates, log-transformed blood lead not significantly

Table 1 continued on following page

Table 1 | Continued

Reference and study location	Study population; date; sample size; % male	Mean (s.d. or range) Age (years) Blood lead (μg/dl)	Study design	Statistical modeling; covariates	Adjusted β coefficients (95% CI) for observed associations
	201 age- and gender-matched controls Date not provided (Note – only results in adults presented herein)	Geometric means ranged from 4.2 (0.2) μg/dl (control females) to 7.1 (0.18) μg/dl (control males)	exposure, recent dental work, and missing data. Numbers excluded not stated, however, serum creatinine data reported in 479 of original 600	index, area of residence, smoking, alcohol ingestion, log urine mercury, log blood cadmium and urinary creatinine	associated with serum creatinine (data not presented in publication).
Mortada *et al.*[39] Egypt	Not applicable – details of population recruitment not provided; date not provided 68; 100%	30 (25–35) years (smokers) 31.8 (25–38) years (non-smokers) 14.4 (3.4) μg/dl (smokers) 10.2 (3.1) μg/dl (non-smokers)	Also measured cadmium, and mercury in blood and all three metals in urine, hair, and nails. Exclusionary criteria=occupational exposure to any of the three metals, dental amalgams, drug intake, diabetes, hypertension, and hepatic, renal or urological disease. Smokers and non-smokers matched on socioeconomic status.	t-test for independent samples; Spearman correlation	No significant difference in serum creatinine or BUN by smoking status (blood lead levels significantly higher in smokers as compared to non-smokers). No significant correlations of any of the four lead measures with renal outcomes (only assessed in 35 smokers; data not presented in publication)
Pocock *et al.*[28] Britain	British Regional Heart Study; year not provided; 7364; 100%	Mean not reported, range 40–59 years Mean blood lead not reported	Exclusionary criteria not specified	Correlation; analysis of covariance adjusting for alcohol	Correlations of blood lead with log transformed serum creatinine, urate, and log urea=0.00, +0.1, −0.08 (unadjusted data) ($P<0.001$ for urate and urea) Partial correlations for urate and urea after adjustment for alcohol=+0.06 and −0.05 (P-values not reported)

BUN, blood urea nitrogen; CI, confidence interval; CKD, chronic kidney disease; GFR, glomerular filtration rate; NHANES, National Health and Nutrition Examination Survey.
[a]Studies in adult general populations published in the last 25 years in which relations between lead dose and clinical renal outcomes (BUN, serum creatinine, measured or estimated creatinine clearance, or estimated GFR) were analyzed.
[b]Creatinine clearance estimated using the Cockcroft-Gault equation.[43]
[c]GFR estimated by the abbreviated Modification of Diet in Renal Disease (MDRD) equation.[46]

OCCUPATIONAL POPULATION STUDIES

Research on renal outcomes from occupational lead exposure is categorized by reported results (Table 2). Compared to the work described above in general populations, this body of literature is larger, however, the results are less consistent. This is puzzling as most dose–response relations are linear. Biologically, lead dose consistent with occupational exposure (i.e., blood lead levels between 20 and 50 μg/dl) should be nephrotoxic if lower (<10 μg/dl) and higher (>80 μg/dl) doses are. A number of factors may be involved in this seeming paradox. Some are unique to the occupational literature. These studies generally have small sample sizes, resulting in less power to detect significant differences. Most are cross-sectional studies of currently employed workers, a group that is well known to be healthier than the general population (the healthy worker effect). Lead workers who are followed in a medical surveillance program (a common practice) are often removed from exposure if renal function decline is observed. This may result in removal from the workplace, yet few studies have included former workers. In many studies, exclusionary criteria for a range of diseases, such as HTN and diabetes, were applied. The number of workers subsequently excluded was not always reported, making it difficult to determine if the healthy worker effect was substantially increased. Statistical analyses were more limited than in general population studies. Analyses limited to comparisons of crude mean outcomes between exposed and control workers are problematic, when lead levels in the latter group are in the range associated with adverse renal outcomes in the general population. Limited lead exposure assessment may also be a factor, as few studies have included cumulative measures of lead dose and blood lead varies more owing to external exposure in the occupational setting. Other limitations that are pertinent for research on the adverse renal effects of lead exposure in any population include insensitivity of the clinical renal outcomes and the lack of uniformly accepted early markers of renal damage in lead exposure. As discussed below, coexposure to environmental cadmium may also account for differences in reported associations between studies, particularly for *N*-acetyl-β-D-glucosaminidase (NAG). In general, these limitations result in bias towards the null meaning that actual associations are obscured.

Finally, one factor that has received relatively little attention to date involves the paradoxical inverse associations observed in some studies (Table 2; Category 4). These unexpected associations have been reported with blood, tibia, and dimercaptosuccinic acid-chelatable lead in the

Table 2 | Research on the renal effects of occupational lead exposure[a]

Significant results	Details and examples	References
Mean difference in one or more measures of clinical renal function[b] between lead workers and control group and/or positive association(s) between higher lead dose and worse clinical renal function	Traditional pattern of lead-related nephrotoxicity. Mean serum creatinine higher in exposed compared to control workers. Higher blood or bone lead associated with higher serum creatinine in all workers.	47–57
Mean difference or association for renal biomarkers[c]; clinical outcomes not assessed	Relatively recent approach to lead-related nephrotoxicity to address issue of insensitivity of clinical renal measures. Higher blood lead associated with higher RBP	58–66
Significant results for renal biomarkers but not clinical outcomes	Higher blood lead associated with higher RBP but not serum creatinine	67–78
Paradoxical associations with higher mean creatinine clearance in the lead exposed group and/or inverse associations between higher lead dose and worse clinical renal function	May indicate lead-related hyperfiltration. Higher lead dose associated with lower BUN and serum creatinine and/or higher creatinine clearance	47,52,79,80,84
None[d]		82–86

BUN, blood urea nitrogen; RBP, retinol-binding protein.
[a]Published between 1985–2005, adult populations.
[b]Clinical renal function defined as BUN, serum creatinine, estimated and/or measured creatinine clearances, and/or estimated GFR.
[c]Renal biomarkers examples include *N*-acetyl-β-D-glucosaminidase, RBP, β_2 microglobulin, and α_1-microglobulin.
[d]Interestingly, one of these studies[85] included two workers with high lead dose and lower creatinine clearance who had biopsy evidence of chronic interstitial nephritis, thus a pattern consistent with lead nephropathy. In another, the association was no longer significant after the removal of two participants with abnormal renal function that appear to be outliers.[86]

occupational setting and with blood lead in a recent study of environmentally exposed children.[87] Higher mean creatinine clearance compared to controls was also observed in one study of adults who were previously lead poisoned as children.[88] Although observed renal function was not consistently in a supranormal range, these associations may indicate a lead-related hyperfiltration process. Data from lead workers in South Korea suggest a temporal pattern that is consistent with hyperfiltration.[47] Inverse associations (i.e., higher lead dose associated with lower serum creatinine) were noted in younger workers; however, the opposite was observed in older workers (i.e., higher lead dose associated with higher serum creatinine). Two longitudinal studies in rats are critical to our understanding of this process in lead exposure. High exposure animals had mean blood lead levels of ~50, 90 and 125 μg/dl at 1, 3, and 6 months, respectively, at which point lead exposure was reduced and levels declined to ~60 μg/dl at 12 months.[13] Lower exposure resulted in mean blood lead levels between ~20 and 30 μg/dl throughout.[89] Compared to controls, mean GFR (measured with ^{125}I-iothalamate clearance) was significantly higher at 1 month in the lower exposure animals and in both groups at 3 months. In the high exposure rodents, a positive association between blood lead and GFR was observed in the first 6 months; however, tubulointerstitial fibrosis became apparent at 6 months and GFR was decreased compared to controls at 12 months. Interestingly, despite a similar degree of hyperfiltration initially (and an earlier onset), subsequent renal damage was much less severe in the lower exposed animals.

Whether this process contributes to pathology in human lead exposure remains unclear and will require longitudinal studies. Regardless, significant associations could be obscured if opposite direction associations are present in different segments of the study population and interaction models to address this are not performed. This is a valid concern as the factors involved in these inverse associations in lead exposed populations are not well defined at present.

PATIENT POPULATION STUDIES

Studies in various patient populations have also contributed to the body of knowledge concerning the adverse renal impact of lead. Patients with CKD, gout, and/or HTN have been the focus of this work as risk for these diseases is increased with high-level lead exposure, particularly when two or more coexist in the same patient. Early research focused on lead nephropathy and body burdens were high (>600 to 650 μg lead excreted in 72 h following chelation with EDTA).[90,91] More recent work has involved patients with earlier stages of CKD and much lower lead body burdens, thus addressing the issue of low-level lead as a cofactor with other renal risk factors in susceptible populations. This work has been published by Lin and co-workers in Taiwan; a discussion of two recent studies serves to illustrate the current state of this research and the different approaches they use to study this issue. The first approach involves prospective study of susceptible patient populations to determine if renal function decline is greater in participants with higher baseline chelatable lead body burdens. Yu *et al.*[92] followed 121 CKD patients over a 4-year period. Eligibility criteria included stable renal function (serum creatinine from 1.5 to 3.9 mg/dl), well-controlled blood pressure, cholesterol and daily protein intake, and EDTA-chelatable lead <600 μg/72 h. Sixty-three patients had 'high-normal' EDTA-chelatable lead levels (≥80 but <600 μg/72 h);

58 patients had 'low-normal' EDTA-chelatable lead levels (<80 μg lead/72 h). Mean blood lead was higher in the former group (4.9 vs 3.4 μg/dl). The groups were similar in most other baseline risk factors. However, borderline statistically significant ($P<0.1$) differences were present (older age and chronic glomerulonephritis more prevalent in the 'high-normal' lead group chronic interstitial nephritis and hypertensive nephropathy less prevalent). Fifteen patients in the 'high-normal' chelatable lead group reached the primary end point (doubling of serum creatinine or need for hemodialysis) compared to only two in the 'low-normal' group ($P=0.001$).

Associations between baseline chelatable or blood lead levels and change in GFR were modeled separately using generalized estimating equations. Based on these models, 10 μg/72 h higher chelatable lead and 1 μg/dl higher blood lead were associated with a reduction in GFR of 1.3 and 4.0 ml/min/1.73 m^2, respectively, during the 4-year study period. Given the lead dose ranges in this study, this is a clinically relevant association.

The second approach used by Lin *et al.*, involves randomized EDTA chelation trials to determine if this therapy changes the rate of renal function decline. Chelation in lead exposure is controversial owing to the potential for it to be used in lieu of exposure reduction. In addition, cases of acute tubular necrosis were reported following early clinical use of EDTA that involved very large doses.[93] However, adverse renal effects have not been observed in subsequent work using lower doses.[91,93] In their largest chelation trial to date, Lin *et al.*[94] randomized 64 patients whose EDTA-chelatable lead levels were 80–600 μg/72 h; half received weekly chelation with 1 g EDTA intravenously for up to 3 months (mean 5 weeks) and half received a weekly placebo infusion for 5 weeks. Renal risk factors were similar in the two groups. Mean blood lead levels were 6.1 and 5.9 μg/dl in treated and control groups, respectively. In the subsequent 24 months, chelation was repeated in 19 (59%) participants owing to increases in serum creatinine in association with rebound increases in chelatable lead levels. Each received one additional chelation series (mean of four weekly infusions), a mean of 13.7 months after the first chelation period. At the end of the 2-year study period, mean estimated GFR increased by 2.1 ml/min/1.73 m^2 in the chelated group compared to a decline of 5.0 ml/min/1.73 m^2 in the controls ($P<0.01$). Benefits from chelation have also been observed in rodent models of lead-related nephrotoxicity.[95–97] This did not appear to occur via reversal of structural damage;[97] improved hemodynamics from reduction of reactive oxidant species may be a mechanism.[98] It is also important to note that chelation may have a direct beneficial effect on kidney function, irregardless of lead exposure. Dimercaptosuccinic acid has been reported to prevent renal damage when coadministered during induction of nephrosclerosis in a non-lead-exposed rat model.[98]

The unique body of work by Lin *et al.*, (which also includes work in patient populations with gout and HTN not discussed herein) has numerous strengths including prospective study design, randomization, lead dose assessment that includes bioavailable body burden, longitudinal statistical analysis, and control for multiple renal risk factors. However, to date, this work has been performed in small groups at one clinical center and thus the generalizability of the results to broader populations is unknown. In addition, the observed effect of lead on decline in GFR has been variable. In an earlier study of 202 patients, an increase of 10 μg/72 h in baseline EDTA-chelatable lead was associated with a decline in GFR of 0.03 ml/min/1.73 m^2 over a 2-year observation period.[94] When adjusted for the shorter follow-up period, this effect, although statistically significant ($P<0.001$), is 20-fold lower than in the most recent work discussed above.[92] Small sample sizes and differences in renal diagnoses between groups may be factors in this variability.

Although preliminary, this line of research could yield important public health benefits if confirmed in large populations (and shown not to worsen cognition). This would indicate that lead body burden contributes to worsening renal function in populations with CKD from a range of causes at much lower levels than previously recognized. Therapeutic options would be available for high-risk patients, who, despite reductions in lead exposure, still experience lead-related nephrotoxicity.

ADDITIONAL CONSIDERATIONS

A number of additional issues may be relevant in assessing the adverse renal impact of lead exposure. However, data are currently too limited to merit in depth discussion. Such areas include the potential for lead-related nephrotoxicity in children, genetic susceptibility, and coexposures, of which cadmium is the most important. Lead-poisoned children who are not chelated are at increased risk for nephropathy as young adults.[10] Recent work in children exposed at lower levels has generally relied on renal biomarkers for outcome assessment. As discussed above, few prospective studies documenting the prognostic value of these markers in lead exposure have been published. Further, some data suggest that renal biomarker levels may decrease post-puberty,[99] so prospective biomarker studies specifically in lead exposed children will be needed to interpret the existing literature. However, two recent publications deserve comment. A positive association between blood lead and serum cystatin C was observed in 200 Belgian adolescents.[100] In 600 European children, higher blood lead was associated with lower serum creatinine and cystatin C,[87] in models that adjusted for age, gender, body mass index, and either blood or urine cadmium (A. Bernard, e-mail communication). Prospective studies of renal function in lead-exposed children are needed to understand the clinical significance of these findings.

Research in the past two decades suggests that certain genetic polymorphisms affect lead toxicokinetics (i.e., modify the relation between lead exposure and internal dose).

The gene that encodes for δ-aminolevulinic acid dehydratase has received most attention in this regard to date. Overall, current data suggest that tighter binding of lead by the isoenzymes of the variant δ-aminolevulinic acid dehydratase[2] allele leads to higher blood lead levels and decreased lead sequestration in bone.[101,79,102] Data to determine whether the δ-aminolevulinic acid dehydratase polymorphism confers additional toxicodynamic risk for the kidney are still quite limited, but are suggestive of an increased risk in lead-exposed populations with the variant allele.[32,79,102–104]

Finally, exposure to other environmental nephrotoxicants may affect risk. Cadmium is likely to be the most important in this regard as this metal has many similarities to lead. It is also a cumulative toxicant that is stored long term in the body. Environmental cadmium exposure in the US occurs primarily through food and smoking.[105] The existing data indicate that cadmium exposure, at levels common in the US, confounds associations between lead exposure and at least one renal outcome, NAG. Studies have reported higher mean NAG in lead workers compared to controls; however, NAG was correlated with urinary cadmium (CdU; a cumulative measure of exposure) rather than lead dose.[80,67] CdU was associated with the NAG-B isoenzyme (released with breakdown of proximal tubular cells) even at CdU levels <2 μg/g creatinine.[106] In Korean lead workers, both CdU and tibia lead were positively associated with NAG[47]. However, a 0.5 μg/g creatinine increase in cadmium had the same effect on NAG as a 66.9 μg lead/g bone mineral. The fact that mean CdU was 1.1 μg/g creatinine, indicating environmental rather than occupational exposure, again illustrates the impact of cadmium on NAG.

Lower level cadmium exposure may also confound or modify relations between lead exposure and clinical renal outcomes, although the data are too limited to draw firm conclusions. Occupational cadmium exposure increases the risk for clinical renal dysfunction,[107,108] as does high-level environmental exposure.[109,110] However, most recent studies of lower level cadmium exposure are cross-sectional and have assessed renal biomarkers, rather than clinical renal outcomes.[43,111–113] Two recent exceptions include a report of increased renal dialysis and transplantation rates in residents of cadmium-polluted areas in Sweden[114] and associations of higher blood lead and blood and CdU with lower estimated creatinine clearance and GFR in Swedish women.[30] However, as noted above, higher blood lead was associated with lower creatinine clearance in the Cadmibel study whereas urinary and blood cadmium were not.[29] Additional studies assessing both lead and cadmium are needed.

CONCLUSIONS

The research reviewed herein utilized a variety of study approaches in different populations. Overall, these diverse lines of evidence indicate that lead exposure, at much lower levels than those causing lead nephropathy, acts as a cofactor with more established renal risk factors to increase the risk for CKD and the rate of progression. Adverse renal effects have been reported at mean blood lead levels <5 μg/dl. Cumulative lead dose was also associated with worse renal function. The data available to date are not sufficient to determine whether current blood lead level or cumulative exposure with higher past blood lead levels is the more important determinant of nephrotoxicity. However, Kim *et al.*[31] noted associations in participants whose peak blood lead levels, dating back to 1979, were ≤10 μg/dl. Populations with diabetes, HTN, and/or CKD appear to be at greater risk for adverse renal effects from lead. Moreover, recent research suggests that the adverse impact of lead on renal function decline in CKD from a range of causes may be reduced with chelation, even at lead body burdens previously considered normal. Although preliminary, this line of research deserves further study as it could yield important public health benefits if confirmed in large populations.

Residual confounding is unlikely to explain associations between lead exposure and renal function. Muntner *et al.*[35] observed that the odds ratios for both renal outcomes assessed in hypertensives, initially adjusted for age, race, and gender, actually increased slightly following additional adjustment for a range of covariates including diabetes, blood pressure, smoking, cardiovascular disease, body mass index, alcohol, and socioeconomic status indicators. Furthermore, most studies adjusted for blood pressure, which is likely to be in the causal pathway and thus may result in an underestimate of effect. Reverse causality, which attributes increased lead dose in general population studies to reduced lead excretion as a consequence of renal insufficiency, is also not likely to be a major explanatory factor. Longitudinal data indicate that lead dose at baseline is associated with subsequent decline in renal function. Furthermore, associations in the Normative Aging Study population occurred over the entire serum creatinine range,[31] and persisted when data from participants with serum creatinine >1.5 mg/dl were removed.[35] The impact of publication bias is always difficult to assess. A type of publication bias is present in the reviewed studies that reported no significant associations but did not show the data. In addition, at least one cadmium study appears to include unpublished longitudinal lead data.[115] However, no other evidence of unpublished data on associations between lead dose and renal function in general populations was identified through the MEDLINE and Embase search strategies used or in population descriptions and references of reviewed papers.

Inverse associations between higher lead dose and worse renal function may be mediated through a hyperfiltration mechanism. Although potentially of mechanistic importance, a more immediate concern is the fact that, if interaction is not specifically explored in data analysis, risk may be underestimated. The potential for such underestimation in lead workers, along with new knowledge regarding susceptible populations, indicates that lead exposure in workers must be controlled not simply to reduce risk for lead nephropathy but also to minimize steeper renal function decline with aging as other cofactors develop. Monitoring of

cumulative lead dose may be important in this effort. Given these limitations, current permissible exposure levels[116,117] may not be as protective for lead workers as previously thought. Finally, there are a number of data gaps, in terms of effects in children, genetic susceptibility factors, and co-exposures, which require further study.

Globally, despite substantial reductions in lead exposure overall, two risk groups for lead-related nephrotoxicity remain: (1) those with higher exposure levels still common in developing countries and in minority populations in developed countries and (2) those with other renal risk factors. From a public health perspective, certain groups, such as those of lower socioeconomic status, are at highest risk as they have higher lead exposure and prevalence of diseases that increase susceptibility. Ultimately, the global public health impact may be most significant in developing countries where obesity, and secondary HTN and diabetes mellitus, are increasing more rapidly than lead exposure is declining. Continued global efforts to reduce lead exposure are obviously important; technology transfer is critical to reduce associated costs. Given the increasing prevalence of renal disease, research to better delineate the contribution made by lead exposure and to determine whether chelation is beneficial is also needed.

ACKNOWLEDGMENTS

This work was supported by NIEHS Grant No. 2 R01 ES007198.
We thank Dr B Schwartz for valuable comments on the manuscript.

REFERENCES

1. Coresh J, Astor BC, Greene T *et al.* Prevalence of chronic kidney disease and decreased kidney function in the adult US population: Third National Health and Nutrition Examination Survey. *Am J Kidney Dis* 2003; 41: 1–12.
2. US Renal Data System. *USRDS 2005 Annual Data Report: Atlas of End-Stage Renal disease in the United States, National Institutes of Health.* National Institute of Diabetes and Digestive and Kidney Diseases: Bethesda, MD, 2005.
3. Mendez MA, Monteiro CA, Popkin BM. Overweight exceeds underweight among women in most developing countries. *Am J Clin Nutr* 2005; 81: 714–721.
4. Monteiro CA, Moura EC, Conde WL, Popkin BM. Socioeconomic status and obesity in adult populations of developing countries: a review. *Bull World Health Organ* 2004; 82: 940–946.
5. Kim S, Popkin BM. Commentary: Understanding the epidemiology of overweight and obesity – a real global public health concern. *Int J Epidemiol* 2006; 35: 60–67.
6. Wild S, Roglic G, Green A *et al.* Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care* 2004; 27: 1047–1053.
7. Yach D, Stuckler D, Brownell KD. Epidemiologic and economic consequences of the global epidemics of obesity and diabetes. *Nat Med* 2006; 12: 62–66.
8. Poirier P, Giles TD, Bray GA *et al.* Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss: an update of the 1997 American Heart Association Scientific Statement on Obesity And Heart Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Circulation* 2006; 113: 898–918.
9. Hsu CY, McCulloch CE, Iribarren C *et al.* Body mass index and risk for end-stage renal disease. *Ann Intern Med* 2006; 144: 21–28.
10. Inglis JA, Henderson DA, Emmerson BT. The pathology and pathogenesis of chronic lead nephropathy occurring in Queensland. *J Pathol* 1978; 124: 65–76.
11. Steenland K, Selevan S, Landrigan P. The mortality of lead smelter workers: an update. *Am J Pub Health* 1992; 82: 1641–1644.
12. Wedeen RP, Mallik DK, Batuman V. Detection and treatment of occupational lead nephropathy. *Arch Intern Med* 1979; 139: 53–57.
13. Khalil-Manesh F, Gonick HC, Cohen AH *et al.* Experimental model of lead nephropathy. I. Continuous high-dose lead administration. *Kidney Int* 1992; 41: 1192–1203.
14. Bernard AM. Clinical renal toxicology. In: Sullivan JB, Krieger GR (eds) *Clinical Environmental Health and Toxic Exposures.* 2nd edn, Lippincott Williams & Wilkins: Philadelphia, 2001 pp 281–289.
15. Pirkle JL, Brody DJ, Gunter EW *et al.* The decline in blood lead levels in the United States. *JAMA* 1994; 272: 284–291.
16. National Center for Environmental Health. *Third National Report on Human Exposure to Environmental Chemicals, Centers for Disease Control and Prevention,* NCEH Pub. No. 05-0570, July, Department of Health and Human Services: Atlanta, Georgia 2005, pp 38–46.
17. Muntner P, Menke A, DeSalvo KB *et al.* Continued decline in blood lead levels among adults in the United States: The National Health and Nutrition Examination Surveys. *Arch Intern Med* 2005; 165: 2155–2161.
18. National Center for Environmental Health, Centers for Disease Control and Prevention. Blood lead levels – United States, 1999–2002. *MMWR* 2005; 54: 513–516.
19. Dooley EE. Tragic trinkets. *Environ Health Perspect* 2004; 112: A803.
20. Medlin J. Sweet candy, bitter poison. *Environ Health Perspect* 2004; 112: A803.
21. Maas RP, Patch SC, Morgan DM, Pandolfo TJ. Reducing lead exposure from drinking water: recent history and current status. *Public Health Rep* 2005; 120: 316–321.
22. Bryant SD. Lead-contaminated drinking waters in the public schools of Philadelphia. *J Toxicol Clin Toxicol* 2004; 42: 287–294.
23. Kaiser R, Henderson AK, Daley WR *et al.* Blood lead levels of primary school children in Dhaka, Bangladesh. *Environ Health Perspect* 2001; 109: 563–566.
24. Clark CS, Thuppil V, Clark R *et al.* Lead in paint and soil in Karnataka and Gujarat, India. *J Occup Environ Hyg* 2005; 2: 38–44.
25. Hu H, Rabinowitz M, Smith D. Bone lead as a biological marker in epidemiological studies of chronic toxicity: conceptual paradigms. *Environ Health Perspect* 1998; 106: 1–8.
26. Barbosa Jr F, Tanus-Santos JE, Gerlach RF, Parsons PJ. A critical review of biomarkers used for monitoring human exposure to lead: advantages, limitations, and future needs. *Environ Health Perspect* 2005; 113: 1669–1674.
27. Chettle DR. *In vivo* X-ray fluorescence of lead and other toxic trace elements. *Adv X-ray Anal* 1995; 38: 563–572.
28. Pocock SJ, Shaper AG, Ashby D *et al.* Blood lead concentration, blood pressure, and renal function. *BMJ* 1984; 289: 872–874.
29. Staessen JA, Lauwerys RR, Buchet JP *et al.* Impairment of renal function with increasing blood lead concentrations in the general population. *N Engl J Med* 1992; 327: 151–156.
30. Payton M, Hu H, Sparrow D, Weiss ST. Low-level lead exposure and renal function in the Normative Aging Study. *Am J Epidemiol* 1994; 140: 821–829.
31. Kim R, Rotnitzky A, Sparrow D *et al.* A longitudinal study of low-level lead exposure and impairment of renal function. *J Am Med Assoc* 1996; 275: 1177–1181.
32. Wu M-T, Kelsey K, Schwartz J *et al.* A δ-aminolevulinic acid dehydratase (ALAD) polymorphism may modify the relationship of low-level lead exposure to uricemia and renal function: The Normative Aging Study. *Environ Health Perspect* 2003; 111: 335–340.
33. Tsaih S-W, Korrick S, Schwartz J *et al.* Lead diabetes, hypertension, and renal function: The Normative Aging Study. *Environ Health Perspect* 2004; 112: 1178–1182.
34. de Burbure C, Buchet JP, Bernard A *et al.* Biomarkers of renal effects in children and adults with low environmental exposure to heavy metals. *J Toxicol Environ Health A* 2003; 66: 783–798.
35. Muntner P, He J, Vupputuri S *et al.* Blood lead and chronic kidney disease in the general United States population: results from NHANES III. *Kidney Int* 2003; 63: 1044–1050.
36. Åkesson A, Lundh T, Vahter M *et al.* Tubular and glomerular kidney effects in Swedish women with low environmental cadmium exposure. *Environ Health Perspect* 2005; 113: 1627–1631.
37. Mortada WI, Sobh MA, El-Defrawy MM. The exposure to cadmium, lead and mercury from smoking and its impact on renal integrity. *Med Sci Monit* 2004; 10: CR112–116.
38. Factor-Litvak P, Stein Z, Graziano J. Increased risk of proteinuria among a cohort of lead-exposed pregnant women. *Environ Health Perspect* 1993; 101: 418–421.

39. Lin J-L, Yeh K-H, Tseng H-C *et al.* Urinary *N*-acetyl-glucosaminidase excretion and environmental lead exposure. *Am J Nephrol* 1993; 13: 442–447.
40. Satarug S, Nishijo M, Ujjin P *et al.* Evidence for concurrent effects of exposure to environmental cadmium and lead on hepatic CYP2A6 phenotype and renal function biomarkers in nonsmokers. *Environ Health Perspect* 2004; 112: 1512–1518.
41. Satarug S, Ujjin P, Vanavanitkun Y *et al.* Effects of cigarette smoking and exposure to cadmium and lead on phenotypic variability of hepatic CYP2A6 and renal function biomarkers in men. *Toxicology* 2004; 204: 161–173.
42. Cui Y, Zhu Y-G, Zhai R *et al.* Exposure to metal mixtures and human health impacts in a contaminated area in Nanning, China. *Environ Int* 2005; 31: 784–790.
43. Alfven T, Jarup L, Elinder C-G. Cadmium and lead in blood in relation to low bone mineral density and tubular proteinuria. *Environ Health Perspect* 2002; 110: 699–702.
44. Larsson A, Malm J, Grubb A, Hansson LO. Calculation of glomerular filtration rate expressed in ml/min from plasma cystatin C values in mg/L. *Scand J Clin Lab Invest* 2004; 64: 25–30.
45. Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. *Nephron* 1976; 16: 31–41.
46. Levey AS, Bosch JP, Lewis JB *et al.* A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. *Ann Intern Med* 1999; 130: 461–470.
47. Weaver VM, Lee B-K, Ahn K-D *et al.* Associations of lead biomarkers with renal function in Korean lead workers. *Occup Environ Med* 2003; 60: 551–562.
48. Ehrlich R, Robins T, Jordaan E *et al.* Lead absorption and renal dysfunction in a South African battery factory. *Occup Environ Med* 1998; 55: 453–460.
49. Smith CM, Wang X, Hu H, Kelsey KT. A polymorphism in the δ-aminolevulinic acid dehydratase gene may modify the pharmacokinetics and toxicity of lead. *Environ Health Perspect* 1995; 103: 248–253.
50. Pinto de Almeida AR, Carvalho FM, Spinola AG, Rocha H. Renal dysfunction in Brazilian lead workers. *Am J Nephrol* 1987; 7: 455–458.
51. Lim YC, Chia KS, Ong HY *et al.* Renal dysfunction in workers exposed to inorganic lead. *Ann Acad Med Singapore* 2001; 30: 112–117.
52. Wang VS, Lee MT, Chiou JY *et al.* Relationship between blood lead levels and renal function in lead battery workers. *Int Arch Occup Environ Health* 2002; 75: 569–575.
53. Jung KY, Lee SJ, Kim JY *et al.* Renal dysfunction indicators in lead exposed workers. *J Occup Health* 1998; 40: 103–109.
54. Chia KS, Jeyaratnam J, Tan C *et al.* Glomerular function of lead-exposed workers. *Toxicol Lett* 1995; 77: 319–328.
55. Endo G, Horiguchi S, Kiyota I. Urinary *N*-acetyl-beta-d-glucosaminidase activity in lead-exposed workers. *J Appl Toxicol* 1990; 10: 235–238.
56. Ong CN, Endo G, Chia KS *et al.* Evaluation of renal function in workers with low blood lead levels. In: Foa V (ed). *Occupational and Environmental Chemical Hazards*. Halstead Press: New York, 1987 pp 327–333.
57. Weaver VM, Lee B-K, Todd AC *et al.* Associations of patella lead and other lead biomarkers with renal function in lead workers. *J Occup Environ Med* 2005; 47: 235–243.
58. Coratelli P, Giannattasio M, Lomonte C *et al.* Enzymuria to detect tubular injury in workers exposed to lead: a 12-month follow-up. *Contrib Nephrol* 1988; 68: 207–211.
59. Garcon G, Leleu B, Zerimech F *et al.* Biologic markers of oxidative stress and nephrotoxicity as studied in biomonitoring of adverse effects of occupational exposure to lead and cadmium. *J Occup Environ Med* 2004; 46: 1180–1186.
60. Chia KS, Mutti A, Tan C *et al.* Urinary *N*-acetyl-beta-d-glucosaminidase activity in workers exposed to inorganic lead. *Occup Environ Med* 1994; 51: 125–129.
61. Chia KS, Mutti A, Alinovi R *et al.* Urinary excretion of tubular brush-border antigens among lead exposed workers. *Ann Acad Med Singapore* 1994; 23: 655–659.
62. Chia KS, Jeyaratnam J, Lee J *et al.* Lead-induced nephropathy: relationship between various biological exposure indices and early markers of nephrotoxicity. *Am J Ind Med* 1995; 27: 883–895.
63. Huang JX, He FS, Wu YG, Zhang SC. Observations on renal function in workers exposed to lead. *Sci Total Environ* 1988; 71: 535–537.
64. El-Safty IA, Afifi AM, Shauman AE *et al.* Effects of smoking and lead exposure on proximal tubular integrity among Egyptian industrial workers. *Arch Med Res* 2004; 35: 59–65.
65. Vyskocil A, Fiala Z, Salandova J *et al.* The urinary excretion of specific proteins in workers exposed to lead. *Arch Toxicol Suppl* 1991; 14: 218–221.
66. Kumar BD, Krishnaswamy K. Detection of sub-clinical lead toxicity in monocasters. *Bull Environ Contam Toxicol* 1995; 54: 863–869.
67. Cardenas A, Roels H, Bernard AM *et al.* Markers of early renal changes induced by industrial pollutants. II Application to workers exposed to lead. *Br J Ind Med* 1993; 50: 28–36.
68. Kumar BD, Krishnaswamy K. Detection of occupational lead nephropathy using early renal markers. *Clin Toxicol* 1995; 33: 331–335.
69. Fels LM, Herbort C, Pergande M *et al.* Nephron target sites in chronic exposure to lead. *Nephrol Dial Transplant* 1994; 9: 1740–1746.
70. Gerhardsson L, Chettle DR, Englyst V *et al.* Kidney effects in long term exposed lead smelter workers. *Br J Ind Med* 1992; 49: 186–192.
71. Pergande M, Jung K, Precht S *et al.* Changed excretion of urinary proteins and enzymes by chronic exposure to lead. *Nephrol Dial Transplant* 1994; 9: 613–618.
72. Verschoor M, Wibowo A, Herber R *et al.* Influence of occupational low-level lead exposure on renal parameters. *Am J Ind Med* 1987; 12: 341–351.
73. Cardozo dos Santos A, Colacciopo S, Dal Bo CMR, Guimaim dos Santos NA. Occupational exposure to lead, kidney function tests, and blood pressure. *Am J Ind Med* 1994; 26: 635–643.
74. Mortada WI, Sobh MA, El-Defrawy MM, Farahat SE. Study of lead exposure from automobile exhaust as a risk for nephrotoxicity among traffic policemen. *Am J Nephrol* 2001; 21: 274–279.
75. Endo G, Konishi Y, Kiyota A, Horiguchi S. Urinary alpha 1 microglobulin in lead workers. *Bull Environ Contam Toxicol* 1993; 50: 744–749.
76. Konishi Y, Endo G, Kiyota A, Horiguchi S. Fractional clearances of low molecular weight proteins in lead workers. *Ind Health* 1994; 32: 119–128.
77. Ankrah N-A, Kamiya Y, Appiah-Opong R *et al.* Lead levels and related biochemical findings occurring in Ghanian subjects occupationally exposed to lead. *East African Med J* 1996; 73: 375–379.
78. Oktem F, Arslan MK, Dundar B *et al.* Renal effects and erythrocyte oxidative stress in long-term low-level lead-exposed adolescent workers in auto repair workshops. *Arch Toxicol* 2004; 78: 681–687.
79. Weaver VM, Schwartz BS, Ahn K-D *et al.* Associations of renal function with polymorphisms in the δ-aminolevulinic acid dehydratase, vitamin D receptor, and nitric oxide synthase genes in Korean lead workers. *Environ Health Perspect* 2003; 111: 1613–1619.
80. Roels H, Lauwerys R, Konings J *et al.* Renal function and hyperfiltration capacity in lead smelter workers with high bone lead. *Occup Environ Med* 1994; 51: 505–512.
81. Hsiao C-Y, Wu H-DI, Lai J-S, Kuo H-W. A longitudinal study of the effects of long-term exposure to lead among lead battery factory workers in Taiwan (1989–1999). *Sci Total Environ* 2001; 279: 151–158.
82. Al-Neamy FR, Almehdi AM, Alwash R *et al.* Occupational lead exposure and amino acid profiles and liver function tests in industrial workers. *Int J Environ Health Res* 2001; 11: 181–188.
83. Gennart JP, Bernard A, Lauwerys R. Assessment of thyroid, testes, kidney and autonomic nervous system function in lead-exposed workers. *Int Arch Occup Environ Health* 1992; 64: 49–57.
84. Omae K, Sakurai H, Higashi T *et al.* No adverse effects of lead on renal function in lead-exposed workers. *Ind Health* 1990; 28: 77–83.
85. Pollock CA, Ibels LS. Lead intoxication in Sydney Harbour bridge workers. *Aust NZ J Med* 1988; 18: 46–52.
86. Staessen J, Yeoman WB, Fletcher AE *et al.* Blood lead concentration, renal function, and blood pressure in London civil servants. *Br J Ind Med* 1990; 47: 442–447.
87. de Burbure C, Buchet JP, Leroyer A *et al.* Renal and neurological effects of cadmium, lead, mercury and arsenic in children: evidence of early effects and multiple interactions at environmental exposure levels. *Environ Health Perspect* 2006; 114: 584–590.
88. Hu H. A 50-year follow-up of childhood plumbism. *AJDC* 1991; 145: 681–687.
89. Khalil-Manesh F, Gonick HC, Cohen AH. Experimental model of lead nephropathy. III. Continuous low-level lead administration. *Arch Environ Health* 1993; 48: 271–278.
90. Batuman V, Landy E, Maesaka JK, Wedeen RP. Contribution of lead to hypertension with renal impairment. *N Engl J Med* 1983; 309: 17–21.
91. Sanchez-Fructuoso AI, Torralbo A, Arroyo M *et al.* Occult lead intoxication as a cause of hypertension and renal failure. *Nephrol Dial Transplant* 1996; 11: 1775–1780.
92. Yu CC, Lin JL, Lin-Tan DT. Environmental exposure to lead and progression of chronic renal diseases: a four-year prospective longitudinal study. *J Am Soc Nephrol* 2004; 15: 1016–1022.

93. Wedeen RP, Batuman V, Landy E. The safety of the EDTA lead-mobilization test. *Environ Res* 1983; 30: 58–62.
94. Lin JL, Lin-Tan DT, Hsu KH, Yu C-C. Environmental lead exposure and progression of chronic renal diseases in patients without diabetes. *N Engl J Med* 2003; 348: 277–286.
95. Sanchez-Fructuoso AI, Blanco J, Cano M *et al.* Experimental lead nephropathy: treatment with calcium disodium ethylenediaminetetraacetate. *Am J Kidney Dis* 2002; 40: 59–67.
96. Sanchez-Fructuoso AI, Cano M, Arroyo M *et al.* Lead mobilization during calcium disodium ethylenediaminetetraacetate chelation therapy in treatment of chronic lead poisoning. *Am J Kidney Dis* 2002; 40: 51–58.
97. Khalil-Manesh F, Gonick HC, Cohen A *et al.* Experimental model of lead nephropathy. II. Effect of removal from lead exposure and chelation treatment with dimercaptosuccinic acid (DMSA). *Environ Res* 1992; 58: 35–54.
98. Gonick HC, Cohen AH, Ren Q *et al.* Effect of 2,3-dimercaptosuccinic acid on nephrosclerosis in the Dahl rat. I. Role of reactive oxygen species. *Kidney Int* 1995; 50: 1572–1581.
99. Sarasua SM, Mueller P, Kathman S *et al.* Confirming the utility of four kidney biomarker tests in a longitudinal follow-up study. *Renal Failure* 2003; 25: 797–817.
100. Staessen JA, Nawrot T, Den Hond E *et al.* Renal function, cytogenetic measurements, and sexual development in adolescents in relation to environmental pollutants: a feasibility study of biomarkers. *Lancet* 2001; 357: 1660–1669.
101. Kelada SN, Shelton E, Kaufmann RB, Khoury MJ. aminolevulinic acid dehydratase genotype and lead toxicity: a HuGE review. *Am J Epidemiol* 2001; 154: 1–13.
102. Weaver VM, Schwartz BS, Jaar BG *et al.* Associations of uric acid with polymorphisms in the δ-aminolevulinic acid dehydratase, vitamin D receptor, and nitric oxide synthase genes in Korean lead workers. *Environ Health Perspect* 2005; 113: 1509–1515.
103. Ye XB, Wu CE, Hua F *et al.* Associations of blood lead levels, kidney function, and blood pressure with δ-aminolevulinic acid dehydratase and vitamin D receptor gene polymorphisms. *Toxicol Mech Methods* 2003; 13: 139–146.
104. Weaver VM, Lee B-K, Todd AC *et al.* Effect modification by δ-aminolevulinic acid dehydratase vitamin D receptor and nitric oxide synthase gene polymorphisms on associations between patella lead and renal function in lead workers. *Environ Res* 2006; 102: 61–69.
105. National Center for Environmental Health. *Third National Report on Human Exposure to Environmental Chemicals. Centers for Disease Control and Prevention*, NCEH Pub. No. 05-0570, July, Department of Health and Human Services: Atlanta, Georgia 2005, pp 26–30.
106. Bernard A, Thielmans N, Roels H, Lauwerys R. Association between NAG-B and cadmium in urine with no evidence of a threshold. *Occup Environ Med* 1995; 52: 177–180.
107. Roels HA, Lauwerys RR, Buchet JP *et al.* Health significance of cadmium induced renal dysfunction: a five year follow up. *Br J Ind Med* 1989; 46: 755–764.
108. Roels HA, Van Assche FJ, Oversteyns M *et al.* Reversibility of microproteinuria in cadmium workers with incipient tubular dysfunction after reduction of exposure. *Am J Ind Med* 1997; 31: 645–652.
109. Nogawa K, Kobayashi E, Okubo Y, Suwazono Y. Environmental cadmium exposure, adverse effects, and preventive measures in Japan. *Biometals* 2004; 17: 581–587.
110. Kido T, Nogawa K, Ishizaki M *et al.* Long-term observation of serum creatinine and arterial blood pH in persons with cadmium-induced renal dysfunction. *Arch Environ Health* 1990; 45: 35–41.
111. Noonan CW, Sarasua SM, Campagna D *et al.* Effects of exposure to low levels of environmental cadmium on renal biomarkers. *Environ Health Perspect* 2002; 110: 151–155.
112. Olsson IM, Bensryd I, Lundh T *et al.* Cadmium in blood and urine – impact of sex, age, dietary intake, iron status, and former smoking – association of renal effects. *Environ Health Perspect* 2002; 110: 1185–1190.
113. Jarup L, Hellstrom L, Alfven T *et al.* Low level exposure to cadmium and early kidney damage: the OSCAR study. *Occup Environ Med* 2000; 57: 668–672.
114. Hellstrom L, Elinder CG, Dahlberg B *et al.* Cadmium exposure and end-stage renal disease. *Am J Kidney Dis* 2001; 38: 1001–1008.
115. Hotz P, Buchet J P, Bernard A *et al.* Renal effects of low-level environmental cadmium exposure: 5-year follow-up of a subcohort from the Cadmibel study. *Lancet* 1999; 354: 1508–1513.
116. Occupational Safety & Health Administration, US Department of Labor. Safety and Health Regulations for Construction, Occupational Health and Environmental Controls: Lead, 29 Code of Federal Regulations 1926.62.
117. Occupational Safety & Health Administration, US Department of Labor. Occupational Safety and Health Standards, Toxic and Hazardous Substances: Lead, 29 Code of Federal Regulations 1910.1025.

INTERNATIONAL CENTER
FOR TOXICOLOGY AND MEDICINE

Literature Review

Lead and Health Effects

David Lukcso MD, MPH
November 27, 2009

E-MAILED

2301 Research Blvd. • Suite 210 • Rockville, Maryland 20850-3204 • 301-519-0300 • fax 301-

INTERNATIONAL CENTER
FOR TOXICOLOGY AND MEDICINE

Lead is one of the most useful elements in industry;[1] it is resistant to corrosion, easily molded and shaped. "Lead and lead alloys are commonly found in pipes, storage batteries, weights, shots and ammunition, cable covers and sheets used to shield us from radiation. The largest use for lead is in storage batteries in cars and other vehicles."[2] The diversity of uses of lead has resulted in increased levels of lead in the environment. Human exposure to lead is common and results from the many uses of this metal due to its exceptional properties.[2] Prior to 1987 lead was used as a pigment in paints and was used as an additive in gasoline until 1995. In the general US population surveys, National Health and Nutrition Examination Surveys (NHANES) the mean blood lead level in 1976 was 15.8 µg/dL for US citizens.[3]

An enormous amount of information is available on the health effects of lead, many effects of lead have been known for centuries.[2] "Lead can induce a wide range of adverse effects in humans depending on the dose and duration of exposure."[4] "Under conditions of extremely high respiratory exposure to lead, an acute encephalopathy can develop, accompanied by renal failure and severe gastrointestinal symptoms."[5] In most cases, lead is absorbed more slowly and the clinical course is subacute or chronic. The adverse effects of chronically elevated blood lead levels range from the inhibition of enzymes associated in the synthesis of hemoglobin to severe pathology such as cerebral edema, usually associated with blood lead levels more than 150 µg/dL.[5] The following table, from the toxicology textbook, *Clinical Environmental Health and Toxic Exposures*, highlights the range of adverse effects on adults based on blood lead levels.

TABLE 73-6. Summary of lowest observed effect levels for key lead-induced health effects in adults

Lowest observed effect level (blood lead µg/dl)	Heme synthesis and hematologic effects	Neurologic effects	Renal effects	Reproductive effects	Cardiovascular effects
100–120	—	Encephalopathic signs and symptoms	—	—	—
60	Anemia	Encephalopathy symptoms	Chronic nephropathy	—	—
60	—	—	—	Reproductive effects in women	—
50	Reduced hemoglobin production	Overt subencephalopathic neurologic symptoms	—	—	—
40	Increased urinary ALA and elevated coproporphyrins	Peripheral nerve dysfunction (slowed nerve conduction)	—	—	—
30	—	—	—	Altered testicular function	Elevated blood pressure (white men, aged 40–59)
25–30	Erythrocyte protoporphyrin elevation in men	—	—	—	—
15–20	Erythrocyte protoporphyrin in women	—	—	—	—
<10	ALA-D inhibition	—	—	—	—

ALA, aminolevulinic acid; ALA-D, aminolevulinic acid dehydrase.

Table 73-6 Clinical Environmental Health and Toxic Exposure[5]

2301 Research Blvd. • Suite 210 • Rockville, Maryland 20850-3204 • 301-519-0300 • fax 301-519-1307

Encephalopathy, Dementia & Neurobehavioral Effects in Adults

Confusion, is a behavioral state of reduced mental clarity, coherence and capacity to reason. Inattention and disorientation are the main early signs, however, as the confusional state worsens there is deterioration in memory, perception, comprehension, problem solving, language and various aspects of emotional behavior.[6] "When there is in addition to confusion an element of drowsiness, the patient is said to have an encephalopathy."[7]

"Dementia is a syndrome with many causes [and] affects more than 4 million Americans; 10% of persons over 70 and 20-40% of individuals over 85 have clinically identifiable memory loss."[6] A simple definition of dementia is a deterioration in cognitive abilities that impairs successful performance of activities of daily living. Memory is the most common cognitive ability lost with dementia. "The most common causes of dementia are: Alzheimer's disease, Multi-Infarct dementia, Alcoholism, Parkinson's disease and medication intoxication."[6]

The literature on an association between blood lead levels and neurobehavioral effects are inconsistent.[8,9] The ATSDR's *Toxicological Profile for Lead* acknowledges that "the literature contains numerous case reports and small cohort studies that describe a higher incidence of these [non-specific] symptoms, including malaise, forgetfulness, irritability, lethargy, headache, fatigue, impotence, decreased libido, dizziness, weakness and paresthesia at blood leads that range from approximately 40 to 120 µg/dl following acute-, intermediate-, and chronic duration occupational exposure."[2]

In a meta-analysis in 2000, Meyer-Baron and Seeber found a statistically significant effect for 3 of 13 tests, Block Design, Logical Memory, and Santa Ana (dominant hand) tests.[2] A follow up meta-analysis in 2002 by Goodman et al found only 2 of 22 tests to have a statistical difference between exposed and unexposed workers, the Digit Symbol and D2 tests and no difference in the three tests found to be statistically different by Meyer-Baron and Seeber. Goodman et al concluded that "The data available to date are inconsistent and are unable to provide adequate information on the neurobehavioral effects of exposure to moderate blood concentrations of lead. A lack of consistency should caution against far reaching conclusions about the true biological effects of low to moderate lead concentrations."[8]

Despite the inconsistency in the literature, the ATSDR noted, "in studies where adults were exposed occupationally to lead, a number of neurobehavioral parameters were reportedly affected. Although as Goodman et al. (2002) pointed out, the lack of true measures of pre-morbid state, observer bias, and publication bias affect the overall assessment, the preponderance of the evidence indicates that lead is associated with neurobehavioral impairment in adult workers at blood leads below 70 µg/dl."[2] The abnormal neuropsychological test finding, even if truly associated with elevated blood lead levels, may be reversible and of no long-term significance.[10] Lindgren et al found

"reversibility of function may occur when proximate blood lead level is maintained below 40 µg/dl."[11]

Renal Effects

The kidney is a very complex organ, "One of the most highly differentiated organs in the body. Nearly 30 different cell types can be found. This panoply of cells modulates a variety of complex physiologic processes."[8] The glomeruli are complex filters with fenestrated epithelia, the renal tubules are composed of highly differentiated cells that vary dramatically in function along the nephron allowing for selective transport of ions, molecules and fluids. Each of these specialized and unique cell types provide very unique functions. The complexity of the kidney with its many different cell types provides for a multitude of injuries each with different clinical effects and each with a multitude of different causes. "Well-described risk factors for the progressive loss of renal function include systemic hypertension, diabetes and activation of the renin-angiotensin-aldosterone system. Cigarettes smoking has been shown to either predispose or accelerate the progression of nephron loss. Lipid oxidation associated with obesity can accelerate progressive renal damage."[6]

Chronic kidney disease encompasses a spectrum of abnormal kidney function, and a progressive decline in glomerular filtration rate. Chronic renal failure typically corresponds to chronic kidney disease stages 3-5. End-stage renal disease represents a stage of chronic kidney disease where the accumulation of toxins, fluids and electrolytes that require removal by renal replacement therapy using dialysis or kidney transplant.[6]

Based on population survey data, "at least 6% of the adult population in the Unites states has chronic kidney disease at stages 1 or 2. An additional 4.5% of the U.S. population is estimated to have stage 3 and 4 chronic kidney disease."[6] Diabetes mellitus and hypertension are the leading causes of chronic renal failure.

Acute exposure to high concentrations of lead can result in proximal tubular dysfunction, manifest by glycosuria and aminoaciduria. The renal manifestations of acute lead nephrotoxicity are usually reversible after treatment and removal from lead exposure.[5,12]

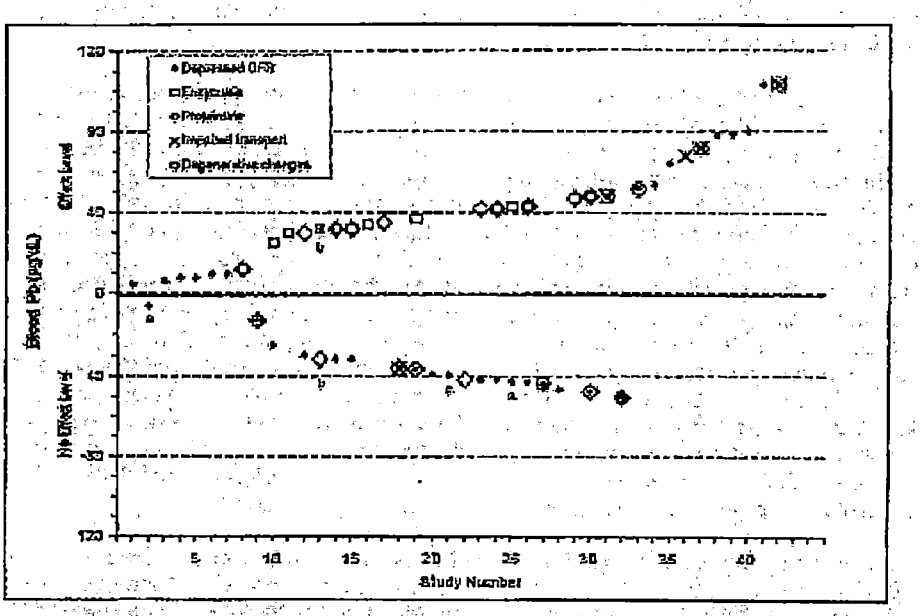
Chronic exposure to high blood lead levels may cause chronic interstitial nephritis which may then progress to progressive nephron loss and renal failure, hypertension, hyperuricemia and gout.[1,4,5] The dose and duration of lead exposure in relationship to renal dysfunction has been extensively studied. "Studies suggest that blood lead level of 60 µg/dl is the threshold for proximal tubular cell injury from lead. This finding is consistent with the observation of Buchet et al that workers who do not have a blood lead level over 62 or 63 µg/dl for up to 12 years do not have lead nephropathy."[13] In a group of 53 workers with an average blood lead of 47 µg/dl, with 12 of the workers blood lead greater than 60 µg/dl and one with a blood lead over 70 µg/dl, the authors found "no adverse effects on kidney function were detected in the exposed

population."[14] In his review, *Renal Effects of Environmental and Occupational Lead Exposure*, Loghman-Adham concluded, "Chronic lead poisoning may result in what has been termed lead nephropathy. The latter seems to develop when blood lead levels exceed a threshold of 60 µg/dl."[1]

The duration for significant exposure required to induce renal effects has been investigated in many studies. Chronic lead nephropathy may develop after "years of excessive exposure."[15] In a study of smelter workers with an average of 18 years exposure (minimum 5 years) to continuously elevated blood lead levels (range 26 to 68 µg/dl) the authors found "that in adult male workers with long term moderate occupational exposure to lead, no significant change in the urinary excretion of low or high molecular weight proteins is detected and that serum concentrations of creatinine were not affected."[16] They concluded that "adverse renal changes are unlikely to occur in most adult male lead workers when their blood lead concentration is regularly kept below 70 µg/dl."[16] Another study of active (14.3 years of exposure) and retired (32.6 years of exposure) lead smelter workers, the authors concluded that "despite many years of moderate to heavy exposure to lead no signs of adverse effects on the kidney such as early tubular or glomerular malfunction were found."[17]

The Agency for Toxic Substances and Disease Registry's (ATSDR) *Toxicological Profile for Lead* describes the findings of a number of studies regarding the relationship between blood lead and qualitative aspects of kidney response. The studies reveal "inconsistencies in the reported outcomes [including proteinuria and glomerular filtration rate (GFR)] across studies may derive from several causes. Epidemiological studies of general populations have shown a significant effect of age on the relationship between glomerular filtration rate and blood lead levels. Furthermore, hypertension can be both a confounder in studies of associations between lead exposure and creatinine clearance and a covariable."[2]

The ATSDR's review included the attached chart; "a cluster of observations of decrements in glomerular filtration rate appear at the low end of the blood lead range (<20 µg/dl range."[2] The review contained seven studies that evaluated either serum creatine or creatine clearance in populations with blood lead levels below 10 µg/dl. The ATSDR indicates that the study authored by Dr. Hu[18] found an "increase in creatinine clearance" associated with exposure to lead.[2] The remaining six studies had problems with results that conflicted with other studies of the same populations, made invalid conclusions or suffered from the lack of inclusion of covariants known to be strongly associated with the study outcome.



*Refer to Table 3-3 for study details (indexed by study number)

a = Increase in creatinine clearance; b = >45 years of age; c = Decrease in serum creatinine; GFR = glomerular filtration rate

The first study cited in the above chart represents an analysis of the NHANES III data, the authors discuss finding of an association between higher blood lead levels and a higher prevalence of elevated serum creatinine; they do not indicate if this association was statistically significant. They compared persons with and without hypertension and found a significant difference, "mean blood lead and serum creatinine were higher and estimated GFR was lower for persons with hypertension. In contrast, no association was present between blood lead and either elevated serum creatine or chronic kidney disease among persons without hypertension"[19] The association found in analysis of the NHANES III data was not found in a previous NHANES review, "No interaction between blood lead, blood pressure and kidney function was found from NHANES II."[18]

The third study in the low exposure group was undertaken "to determine whether long-term low-level environmental lead exposure plays a role in the progression of renal insufficiency in patients with chronic renal insufficiency."[20] The authors make an invalid conclusion; "long-term low-level lead exposure may subtly affect progressive renal insufficiency in the general population."[20] They attempt to generalize findings from persons with chronic renal insufficiency at the onset of their study to the general

population; any such attempt to generalize to non-similar populations is incorrect and such conclusions are invalid.

The next two[21,22] studies involved evaluation of data from the Normative Aging Study, both analyses "considered the potential covariates known to be associated with blood lead and/or serum creatinine levels."[22] While both of these studies considered age, body mass index, smoking status, alcohol intake, and hypertension; neither of these studies included diabetes which is the most frequent cause of chronic kidney disease.[6] Payton et al noted the limitations of their study including the absence of sources of confounding could result in erroneous conclusions; "Renal function is determined by many factors, as in any epidemiologic study, there may be unknown sources of confounding, such as an unmeasured variable that could explain the relation of lead exposure to creatinine clearance."[21] Kim et al "investigated the relationship between low-level lead exposure and renal function using longitudinal data from the Normative Aging Study."[22] Without the inclusion of diabetes as a covariate both studies found that low level lead exposure correlates with impairment in renal function. However, in another evaluation of the same data from the Normative Aging Study, Wu et al "did not find any significant association between blood lead and impaired renal function as measured by serum creatinine or estimated creatinine clearance."[23]

"Outcomes for the various renal toxicity end points are mixed over the blood lead range 20-50 µg/dl."[2] In contrast to the very low level studies, depressed glomerular filtration rate was, "with only one exception, not observed over this blood lead range."[2] The studies that produced the one exception in this range were analyses of South Korean lead workers in which the authors "found that lead has an adverse effect on renal function in the moderate dose range, particularly in older workers."[24,25] The authors compared six renal outcomes with three hematological outcomes and four lead exposure markers, therefore they generated 42 model analyses. "When influential outliers were removed, higher lead measures remained associated with worse renal function in nine models. An additional five associations were in the opposite direction."[24] Therefore, 33 models found no association (including the measured creatine clearance) or actual improvement in outcomes with elevated blood lead. The findings were inconsistent across the age groups studied, "in several of the clinical outcome models, associations in the youngest age tertile were in the opposite direction of associations in the oldest age tertile. Higher lead measures were associated with worse renal function in older workers, but with lower BUN and serum creatinine in younger workers."[24]

The authors of the South Korean studies noted the significance of covariates and confounding; "because non-lead related factors contribute to both renal dysfunction and elevated blood pressure, lack of adjustment for these variables in such models likely results in residual confounding."[25] Analysis of the covariates and confounders included in each of these studies revealed the likelihood of serious under estimation of "diabetes and hypertension the leading underlying etiology of chronic renal disease and end stage renal disease."[26] The authors state that covariates assessed "included diabetes and

hypertension (both based on participant report of physician diagnosis)."[24,25] Using self reported diagnoses revealed the prevalence of diabetes in the study population to be 0.8% and the prevalence of hypertension in the study population to be 7.2%. These prevalences are significantly less than the reported prevalences of the general South Korean population. In a population survey in 1999, the prevalence of diabetes was found to be 15.8%, although only 5.8% of the population had previously been diagnosed.[27] This was confirmed in the Korean Health and Genome Study which found the prevalence of diabetes to be 12.5% and hypertension to be 25.1% in Korean men.[28] The significant difference in the prevalence of diabetes and hypertension in these study populations and the general Korean population make any conclusions suspect as the "variables in such models likely result in residual confounding."[25] Support for a lack of association between blood lead and renal dysfunction when significant covariables are included in the analysis was evidenced by Dr. Hu et al in 2004, "in the absence of diabetes or hypertension, we did not see statistically significant associations of bone lead levels with either cross-sectional or longitudinal measures of renal function."[29]

The ATSDR review concluded that "the overall dose-effect pattern suggests an increasing severity of nephrotoxicity associated with increasing blood lead with deficits in function and pathological changes occurring in association with blood leads exceeding 50 µg/dl."[2] This conclusion is supported by the consensus that long term exposure to blood lead levels in excess of 60 µg/dl may cause progressive lead induced renal disease.[1,13-17,30]

Gout

Gout is a metabolic disease most often affecting middle-aged to elderly men. Acute arthritis is the most frequently early clinical manifestation of gout. Usually, only one joint is affected initially, but polyarticular acute gout can occur in subsequent episodes. The metatarsophalangeal joint of the first toe is often involved, but tarsal joints, ankles, and knees are commonly affected.

Gout is the result of an increased body pool of urate with hyperuricemia. Hyperuricemia may be related to the overproduction of uric acid, or the under excretion. Excretion of >800 mg of uric acid per 24 hours on a regular diet suggests that causes of overproduction of purine should be considered.[5] A 24-hour urine collection for uric acid can, in some cases be useful in assessing the risk of stones, in elucidating overproduction or under excretion of uric acid. Serum uric acid levels can be normal or low at the time of the acute attack. This limits the value of serum uric acid determinations for the diagnosis of gout. Nevertheless, serum urate levels are almost always elevated at some time.

The relationship of gout and lead is due to the accumulation of lead in the proximal tubular cells which affect urate excretion.[5] The basic toxicologic principle regarding dose and duration of exposure hold for lead and gout. Dr. Hu et al noted that chronically elevated "levels of lead (e.g., blood lead levels >60 µg/dL) are clearly

associated with gouty arthritis."[31] Others have concluded that "Chronic high-dose lead exposure has been implicated in the development of gout."[1]

Diabetes Mellitus

Diabetes mellitus (DM) is a common metabolic disorder resulting in hyperglycemia (high blood sugar). "The metabolic dysregulation associated with DM causes secondary changes in multiple organ systems that impose a tremendous burden on the individual with diabetes. In the United States, DM is the leading cause of end-state renal disease (ESRD). It also predisposed to cardiovascular disease."[6]

The prevalence of DM has risen dramatically over last 30 years. The rise in type 2 DM has been much more rapid because of increasing obesity and reduced activity levels. DM increases with age, in the US the prevalence of DM is about 10% in individuals aged 20-60, and 21% in persons over the age of 60. "DM prevalence also varies among different ethnic populations. In 2005, the Centers for Disease Control and Prevention (CDC) estimated that the prevalence of DM in the United States, (age > 20 years) was 13.3% in African Americans, 9.5% in Latinos and 8.7% in non-Hispanic whites."[6]

Type 2 DM has a strong genetic component, "individuals with a parent with type 2 DM have an increased risk of diabetes; if both parents have type 2 DM, the risk approaches 40%. The disease is polygenic and multifactorial, since in addition to genetic susceptibility, environmental factors (such as obesity, nutrition, and lack of physical activity) modulate the phenotype."[6]

The chronic complications of DM affect many organ systems and are responsible for the majority of morbidity and mortality associated with the disease. The chronic complications include, renal disease, retinopathy, neuropathy, coronary artery disease, peripheral arterial disease, and cerebrovascular disease. The risk of coronary heart disease events and mortality are two to four times greater in patients with type 2 DM.[6]

The ATSDR's Toxicological Profile for Lead[2] section on endocrine effects of lead does not consider lead to be associated with diabetes mellitus. There is no support to consider lead as a causal factor for the development of diabetes mellitus or any of its many chronic complications.

Coronary Heart Disease, Cardiomyopathy and Stroke

Atherosclerosis is a major cause of death and disability in developed societies. Atherosclerosis of the coronary arteries leads to Coronary Heart Disease (CHD) causing heart attacks and angina. Atherosclerosis of the arteries supplying the central nervous system frequently provokes strokes. Atherosclerosis can affect the renal vessels causing renal disease. The clinical expression of atherosclerosis may be chronic or a dramatic acute events such as a heart attack, stroke or sudden cardiac death.[6]

CHD is "the most common, serious, chronic, life-threatening illness in the United States."[6] Obesity, type 2 diabetes mellitus, elevated cholesterol, hypertension and smoking are powerful risk factors for CHD. Other risk factors such as age and male gender are strong non-modifiable risk factors for disease.

The ATSDR's Toxicological Profile for Lead section on cardiovascular effects does not consider CHD or stroke to be potential effect of lead exposure.[2] Pocock et al found that after controlling for cigarette smoking there was no association between blood lead and heart attack or stroke. They concluded, "we see no convincing epidemiological evidence at present to support the claim that moderate elevations in body lead burden are of relevance to the risk of cardiovascular disease."[32] There is no support to consider lead as a causal factor for the development of coronary heart disease, cardiomyopathy or stroke.

Peripheral Neuropathy

Peripheral neuropathy is a general term indicating peripheral nerve disorders of any cause. Peripheral neuropathies affect up to 8% of adults; the incidence increases with age.[5] The multitude of causes and manifestations of such disorders make diagnosis bewildering.[7] Evaluation begins with a history focusing on the time course of the illness, symptoms, medical conditions that predispose to neuropathy (e.g. diabetes mellitus, hypothyroidism, connective tissue disease, infection such as Lyme and HIV, and nutritional deficiency) toxic exposures (e.g., alcohol, arsenic, and lead) and family history. The distribution of sensory, motor and reflex changes determines whether the neuropathy is asymmetric or symmetric and whether the neuropathy is a polyneuropathy or a mononeuropathy. Electrodiagnostic studies help to classify the neuropathy into one of the three major categories, axonal, demyelinating, or neuronal.[6]

The ATSDR's *Toxicological Profile for Lead* section on neurologic effects of lead exposure reports that there are numerous studies available on peripheral nerve function.[2] Given the multitude of disease entities that are included under such a broad term, it is not surprising to find that the results of the studies are inconsistent. Ishida et al found no significant association between blood lead and median nerve conduction velocity.[33] Ehle reviewed many of the nerve conduction studies and "concluded that a

mild slowing of certain motor and sensory NCVs may occur at blood leads below 60 µg/dl, but that the majority of studies did not find correlations between blood lead and nerve conduction velocities below 70 µg/dl."[34] Several studies found improvement in NCV of the median (motor) NCV after decrease of blood lead levels. The conclusion of the ATSDR's review, "The results of these studies indicate that NCV effects occur in adults at blood leads <70 µg/dl, and possibly as low as 30 µg/dl."[2]

Anemia

"The anemia induced by lead is microcytic and hypochromic and results primarily from both the inhibition of heme synthesis and shortening of the erythrocyte lifespan. Lead interferes with heme synthesis by altering the activities of δ-aminolevulinic acid dehydratase (ALAD) and ferrochelatase. Correlations between blood lead and log erythrocyte protoporphyrin (EP) or zinc protoporphyrin (ZPP) indicate an apparent threshold for EP elevation in male workers at 25-35 µg/dl. In summary, of all the parameters examined, ALAD activity appears to be the most sensitive indicator of lead exposure. The threshold for blood lead increases in urinary ALA in adults is approximately 40 µg/dl; for increases in blood EP or ZPP, the threshold in adults is around 30 µg/dl."[2]

Erectile Dysfunction

Erectile dysfunction is associated with physiologic aging. In the Massachusetts Male Aging Study, a survey of men 40 to 70 years of age, 52% of respondents reported some degree of erectile dysfunction, 35% was of moderate or complete. The incidence of erectile dysfunction is also higher among men with certain medical disorders such as diabetes mellitus, obesity, lower urinary tract symptoms secondary to benign prostatic hyperplasia, heart disease, hypertension, and decreased HDL levels. Smoking is a significant risk factor in the development of erectile dysfunction. Medications used to treat diabetes or cardiovascular disease are additional risk factors.[6]

The ATSDR's Toxicological Profile for Lead section on reproductive effects does not even mention an erectile dysfunction as a potential reproductive effect of lead exposure.[2] The toxicologic profile does indicate that the literature supports lead induced changes in the quantity and quality of sperm in males with elevated blood lead levels. "Although the evidence for reduced fertility is not conclusive, it appears that a threshold for fertility effects in men could be in the blood lead range of 30-40 µg/dl."[2]

Hypertension

Hypertension is an extremely common chronic illness present in all populations. In the United States, based on results of National Health and Nutrition Examination Survey (NHANES), 33.5% of non-Hispanic blacks and 28.9% of non-Hispanic whites have hypertension. "Hypertension doubles the risk of cardiovascular diseases, including CHD, congestive heart failure, ischemic and hemorrhagic stroke, renal failure and peripheral arterial disease."[6]

There are many well recognized factors that contribute to blood pressure and idiopathic hypertension including age, obesity (elevated Body Mass Index – BMI), dietary salt intake, alcohol consumption, psychological stress, low levels of physical activity, and many medications. Hypertension increases with age; 65.4% of individuals 60 years of age or older have hypertension. In these hypertensive individuals, approximately 80-95% have primary or idiopathic hypertension. The prevalence of hypertension also varies with geographic region; "The prevalence of hypertension and stroke mortality rates are higher in the southeastern Unites States than in other regions."[6] Family studies document a significant inheritable component to blood pressure and hypertension, in fact high blood pressure before age 55 occurs 3.8 times more frequently among persons with a family history of hypertension. "In African Americans, hypertension appears earlier, is generally mores severe, and results in higher rates of morbidity and mortality from stroke, left ventricular hypertrophy, congestive heart failure and end-stage renal disease."[6]

In contrast to the many well known and accepted causal risk factors for the development of hypertension, "On balance across all human studies the relationship between blood pressure and blood lead concentration is inconsistent."[35] At first glance, meta-analysis of the literature may show a weak association between blood lead levels and blood pressure, however, further analysis of the literature provides no support for a causal relationship and raises significant questions regarding whether even an association exists.

The early controversy regarding a possible association relates to initial analysis of the NHANES II data.[36] Despite the inconsistent findings of the study, Harlan et al concluded that "analyses of the NHANES II data provide a link between blood lead levels and blood pressure." Their findings included "blood lead levels were significantly higher in younger men and women (aged 21 to 55 years) with high diastolic pressure than in those with normal blood pressure. However, in older persons (aged 56-74), blood lead levels were not significantly different for any of the blood pressure categories and there were no important trends."[37] They also found that a greater body mass index (weight/height^2) was significantly associated with high diastolic blood pressure in men and women in both age groups. When the authors looked specifically at gender, they found that "for men, blood lead had a significant relationship to systolic and diastolic pressure, [but] for women, blood lead was not independently related to blood

pressure."[37] The authors did warn: "No causal inferences should be drawn from this cross-sectional survey about the blood pressure effects of lead, calcium, and zinc."[37]

The NHANES II data has been subsequently analyzed many times with conflicting results. In 1988 Harlan re-analyzed the data and found a slightly different finding. In 1985 Harlan et al found that blood lead was only associated with elevated diastolic pressure, re-analysis in 1988 found an association with both diastolic and systolic pressure in men. He confirmed his earlier findings that "blood lead did not enter the final regression model for blood pressure in women." [38] In 1988 Gartside's analysis using a "more reliable forward stepwise regression"[39] found that "these results do not provide evidence of any reliability or of any consistency for the hypothesized association. The results obtained by this careful process lacked consistency, lacked reliability, and were weak or negligible. It is concluded, therefore, that there is no convincing evidence of an association between blood lead and blood pressure in NHANES II."[39] De Hond et al analyzed more recent NHANES data and found "there is no consistent relationship between blood pressure and blood lead in NHANES III dataset."[40]

The contradictory results from many of the early studies investigating a possible association between blood lead levels and elevated blood pressures "are due to variation in criteria for including participants in the analysis (age groups, gender, race), weighting factors, covariates selected as confounders and statistical methods."[40] Harlan et al found thirty-four variables having significant correlations with blood pressure and that "age and body mass index were significant and independent predictors of blood pressure."[37] "Human population studies have not always been able to adequately address the impact of potentially confounding factors."[41]

In the most recent meta-analysis by Nawrot et al,[35] several of the early studies included in the analysis found very large changes in blood pressure associated with blood lead concentrations. Each of these early studies were significantly flawed in that they did not include many of the most relevant confounders. In the Kromhout[42] study, with one of the largest reported associations, the only confounders included were smoking and Quetelet Index (weight/height^2 equivalent to BMI). In the discussion Kromhout noted that "additional analyses showed that this relation was influenced by one person with a high blood lead who also had severe hypertension. After exclusion of this person, the regression coefficient was reduced. The relation between blood lead and systolic blood pressure was no longer statistically significant after adjusting for age and Quetelet Index."[42]

Orssaud et al[43] considered only age, BMI, and alcohol as confounders. They found two findings that argue against a dose-response that one would expect in a causally related interaction. Their results "indicate that an increase in blood lead value might result in an increase in systolic blood pressure up to a limiting value, while higher lead concentrations do not relate to blood pressure. In our study blood lead values are significantly related to blood pressure; this correlation, highly significant in young

subjects (24-34 years), decreases with age (not significant in 45-55 year olds)."[43] The statistically significant finding would be attenuated with inclusion of the many additional significant influences on blood pressure, such as smoking and family history.

The study by Weiss et al[44] which found the highest association between blood lead levels and hypertension also had the largest variability in their results. The study began on 89 policemen; however, the final analysis was conducted on only 70 of the subjects. The authors indicate that they examined the relationship of blood lead to blood pressure adjusting for "other independent variables known to affect blood pressure." They specifically address age, body mass index and smoking as variables. They discuss alcohol, but note that they "could find no independent effect of alcohol consumption on blood pressure." This is in distinct contrast to other findings, "Alcohol intake was significantly associated with systolic and diastolic blood pressure in NHANES I. Alcohol intake is an independent cause of increased blood pressure."[45] Grandjean et al[45] found that "lead is related to increased blood pressure. However, the statistical significance of this association disappears when alcohol intake and hemoglobin are included in a multiple regression analysis." In the Weiss study, there is no discussion on how alcohol consumption was quantified in the study subjects; the lack of association may be related to recall bias on the part of the policemen. The initial results, without inclusion of many know confounders, was a statistically significant association between a high level of blood lead and elevation in systolic pressure. No significant association was found between blood lead and diastolic pressure. The authors then investigated "whether the observations were the product of a few influential points. For systolic pressure three individuals needed to be excluded." With the removal of these individuals, the association was only deemed "borderline significant"[44] as the authors chose to use a lower level of significance, not the usual 95% scientific confidence level. The Weiss et al analyses was significantly altered by the exclusion of three study participant outliers as can often be seen in small data sets. They did not find an association with alcohol consumption, a known confounder, and did not consider many other well known and important confounders. Inclusion of these confounders would have decreased the size of the finding and may have diminished the statical significance even further.

The analysis by Kort and Zwennis[46] which contributed the fifth largest relationship between blood lead and blood pressure in Nawrot's[35] meta-analysis, was a very small study that did not include the most basic confounders. The authors stated, "From the study, which had its limitations (small groups, insufficient data on body mass index), it was concluded that a positive relationship may exist between blood lead level and blood pressure (notably systolic blood pressure)."[46] However, when the authors did analyze the data controlling for age, heart rate, and urinary cadmium they found "a nonsignificant result."[46] This small study provides evidence that inclusion of even minor confounders can alter the results of an analysis.

Neri et al[36] reported on two studies; an evaluation of Canadian population data and another study of a cohort of 500 foundry workers. The authors analysis of the Canadian population data of 2193 subjects including only 4 confounders; age, BMI, serum zinc

and hemoglobin level. In multiple regressions they found that "none of those employing control for zinc and hemoglobin attained significance."[35] They also noted that "body build other than slim entails a fourfold risk of antihypertensive medications and, in untreated subjects, a doubling of the prevalence of elevated blood pressure (over 90 mm Hg)."[35] Their analysis of the 500 foundry workers contributed the 3rd highest association between blood lead and blood pressure in the Nawrot et al[35] meta-analysis. The only confounders analyzed were age, weight (not BMI), and urinary cadmium levels. The authors found an association between diastolic blood pressure and blood lead, but the association with systolic blood pressure falls "short of conventional significance".[36] When the authors included urinary cadmium into the analysis they excluded weight "because of the limited number of data points per subject." With the inclusion of age, and urinary cadmium, no significant association was found between blood lead and blood pressure.

Moller and Kristensen's analysis of 1,052 subjects provided further evidence that consideration of confounders and influential variables alters the findings of a study. They concluded that there is a "weak positive association between blood lead and increased blood pressure, this association becomes weaker and statistically nonsignificant when changes in other risk factors are controlled."[47] They did make a very important clinical statement, "This association has little significance for the individual."[47]

Current studies continue to exclude analyses of important confounders such as family history. The inclusion of confounders is highlighted by Hu et al[48], "the most significant factors related to the occurrence of hypertension were body mass index, family history of hypertension, and tibial lead level." A study participant was 2.329 times more likely to be hypertensive if he had a family history of hypertension. A study participant's odds of being hypertensive was only increased by 0.019 if he had an elevated tibial lead level.

Glenn et al[49] noted that "the few longitudinal studies that have examined the association of blood lead levels on changes in blood pressure have not consistently found lead to be a risk factor." While they concluded that blood lead was associated with systolic blood pressure, they "did not have data for family history of hypertension and therefore were not able to use this variable to analyze the genetic contribution to blood pressure change or its effect as a confounder." The failure to include factors related to the occurrence of hypertension, such as family history will cause overestimation of the relationship of evaluated factors.

In addition to the lack of inclusion of the multiple factors that significantly influence blood pressure, the results of the multitude of studies are inconsistent. For example, Harlan's analysis of the NHANES II data found that "blood lead levels were significantly related to systolic and diastolic pressure in males but not females."[37] Schwartz's[50] analysis on the NHANES II data was different; "this study confirms the association in males aged 20 to 74 and documents a significant, although weaker, association in females as well." A third analysis of the same NHANES II data reported still different findings. "A

statistically significant increase in blood pressure occurs in only 24 out of 156 analyses. The increase in blood pressure for the older age groups in white males is missing in white females and is contradicted in older blacks, where instead it decreases."[39] Sharp et al published exactly the opposite findings; "blood lead concentration plays a strong role as a determinant of blood pressure level among black males. An inverse relationship between systolic blood pressure and blood lead concentration was noted in nonblack males."[41]

Even some authors report findings in direct conflict with their earlier findings. In 1991 Joel Schwartz found in, "Lead has been shown to be associated with elevated blood pressure, this study confirms the association in males aged 20-74 and documents a significant, although weaker, association in females as well."[50] In 2001 Schwartz and colleagues assessed the association of hypertension and lead; "No association was found with blood lead."[51]

Parkinson et al evaluated the association of blood pressure and occupational lead exposure in workers in three battery plants. The author's analysis included several of the "known risk factors: age, alcohol use, a quantitative measure of current cigarette use, level of exercise and body mass index."[52] They found that "blood pressure measurements were significantly correlated with age, body mass index and alcohol consumption. After controlling for other known risk factors, the associations between exposure and blood pressure were small and non-significant."[52]

In another occupational study Maheswaran et al studied the association of blood pressure and occupational lead exposure in a lead battery manufacturer in the United Kingdom. In their article they discussed the weaknesses of several of the earlier occupational studies. "Three of these studies found a significant association between lead and blood pressure. In all three studies, however, alcohol consumption was either not taken into account or did not appear to influence blood pressure. De Kort et al did not measure body mass index in their study."[53] Maheswaran et al examined blood lead levels, zinc protoporphyrin and years of industrial exposure to lead. "Following adjustment for the confounding effects of age, body mass index and alcohol consumption, the effect of blood lead on systolic pressure was not significant. There was no association between diastolic blood pressure and blood lead. Zinc protoporphyrin levels and years of industrial lead exposure did not raise adjusted systolic or diastolic pressure."[53]

In another occupational study, Glenn et al found that "change in systolic blood pressure during the study was associated with lead dose." A major weakness in this analysis of lead exposed workers was the lack of inclusion of two influential factors on blood pressure, alcohol and family history. "Very high alcohol consumption was associated with systolic blood pressure. We did not keep alcohol consumption in the final model because we did not have data on this variable for all subjects. We did not have data for family history of hypertension and therefore were not able to use this variable to analyze the genetic contribution to blood pressure change or its effect as a confounder."[49]

The investigation of a possible association between blood lead and blood pressure has been ongoing for many years. There have been a multitude of studies including population and occupationally based; the results have been inconsistent and even contradictory with the majority of the studies failing to reach statistical significance.[35] Many of these studies have been included in the three meta-analyses discussed in the Agency for Toxic Substances and Disease Registry's (ATSDR) *Toxicologic Profile for Lead* which noted that "meta-analyses of the epidemiological findings have found a persistent trend in the data that supports a relatively weak, but significant association."[2] Even these three meta-analyses are inconsistent. Schwartz, in 1995 found "a highly significant and moderately consistent association was found, i.e., decrease of blood lead from 10 mg/dL to 5 mg/dL associated with a decrease of 1.25 mmHg."[54] Staessen et al (1994) found that "there was no relationship across studies between the strength of the blood pressure-blood lead relationship and the mean blood lead concentration. The published evidence suggests that there can only be a weak positive association between blood pressure and lead exposure. Any such relationship may not be causal."[55] Nawrot et al[35], authors of the most recent meta-analysis caution; "These overall results must be cautiously interpreted. In the majority of the population studies, the association between blood pressure and blood lead concentration did not reach statistical significance. The present meta-analysis shows an overall statistically significant positive relationship. On the other hand epidemiological studies can only show associations but not prove causation."[35]

The overall evidence from a long search for a possible relationship between blood lead and blood pressure is inconsistent and contradictory. Many studies have found an association between blood lead and blood pressure, but the majority have not reached statistical significance. Statistical significance implies that the results are unlikely to have occurred by chance; therefore the results in the many studies that did not reach statistical significance may have occurred by chance alone. Associations that are found to be statistically significant are not necessarily important finding as was discussed by Moller et al,[47] "this association has little significance for the individual." Most importantly a statistically significant association does not prove causation. The literature raises significant questions regarding whether an association exists and clearly does not support a causal relationship between blood lead and blood pressure elevations.

David Lukcso MD, MPH

Reference List

(1) Loghman-Adham M. Renal effects of environmental and occupational lead exposure. *Environ Health Perspect.* 1997;105:928-938.

(2) Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for lead. August 2007. 1-582. 2007. Atlanta, GA, Agency for Toxic Substances and Disease Registry. Toxicological Profiles.

(3) CDC. Current Trends Blood-Lead Levels in US Population. 3-19-1982. 10-4-2009.
Ref Type: Internet Communication

(4) Klaassen CD. *Casarett and Doull's Toxicology. The basic science of poisons.* 7th ed. New York: McGraw Hill Medical; 2008.

(5) Sullivan JB, Krieger GR. *Clinical environmental health and toxic exposures, 2nd Ed.* Baltimore: Williams & Wilkins; 2001.

(6) Fauci AS. *Harrison's Principles of Internal Medicine, 17th Edition.* New York: McGraw-Hill; 2008.

(7) *Harrison's Principles of Internal Medicine, 14th Edition.* New York: McGraw-Hill.; 1998.

(8) Goodman M, LaVerda N, Clarke C, Foster ED, Iannuzzi J, Mandel J. Neurobehavioural testing in workers occupationally exposed to lead: systematic review and meta-analysis of publications. *Occup Environ Med.* 2002;59:217-223.

(9) Lindgren KN, Masten VL, Ford DP, Bleecker ML. Relation of cumulative exposure to inorganic lead and neuropsychological test performance. *Occup Environ Med.* 1996;53:472-477.

(10) Parkinson DK, Ryan C, Bromet EJ, Connell MM. A psychiatric epidemiologic study of occupational lead exposure. *Am J Epidemiol.* 1986;123:261-269.

(11) Lindgren KN, Ford DP, Bleecker ML. Pattern of blood lead levels over working lifetime and neuropsychological performance. *Arch Environ Health.* 2003;58:373-379.

(12) Goyer RA. Lead toxicity: a problem in environmental pathology. *Am J Pathol.* 1971;64:167-182.

(13) Goyer RA. Lead toxicity: current concerns. *Environ Health Perspect.* 1993;100:177-187.

(14) de Kort WL, Verschoor MA, Wibowo AA, van Hemmen JJ. Occupational exposure to lead and blood pressure: a study in 105 workers. *Am J Ind Med.* 1987;11:145-156.

(15) Cardenas A, Roels H, Bernard AM et al. Markers of early renal changes induced by industrial pollutants. II. Application to workers exposed to lead. *Br J Ind Med.* 1993;50:28-36.

(16) Roels H, Lauwerys R, Konings J et al. Renal function and hyperfiltration capacity in lead smelter workers with high bone lead. *Occup Environ Med.* 1994;51:505-512.

(17) Gerhardsson L, Chettle DR, Englyst V et al. Kidney effects in long term exposed lead smelter workers. *Br J Ind Med.* 1992;49:186-192.

(18) Hu H. Knowledge of diagnosis and reproductive history among survivors of childhood plumbism. *Am J Public Health.* 1991;81:1070-1072.

(19) Muntner P, He J, Vupputuri S, Coresh J, Batuman V. Blood lead and chronic kidney disease in the general United States population: results from NHANES III. *Kidney Int.* 2003;63:1044-1050.

(20) Lin JL, Tan DT, Hsu KH, Yu CC. Environmental lead exposure and progressive renal insufficiency. *Arch Intern Med.* 2001;161:264-271.

(21) Payton M, Hu H, Sparrow D, Weiss ST. Low-level lead exposure and renal function in the Normative Aging Study. *Am J Epidemiol.* 1994;140:821-829.

(22) Kim R, Rotnitsky A, Sparrow D, Weiss S, Wager C, Hu H. A longitudinal study of low-level lead exposure and impairment of renal function. The Normative Aging Study. *JAMA.* 1996;275:1177-1181.

(23) Wu MT, Kelsey K, Schwartz J, Sparrow D, Weiss S, Hu H. A delta-aminolevulinic acid dehydratase (ALAD) polymorphism may modify the relationship of low-level lead exposure to uricemia and renal function: the normative aging study. *Environ Health Perspect.* 2003;111:335-341.

(24) Weaver VM, Lee BK, Todd AC et al. Associations of patella lead and other lead biomarkers with renal function in lead workers. *J Occup Environ Med.* 2005;47:235-243.

(25) Weaver VM, Jaar BG, Schwartz BS et al. Associations among lead dose biomarkers, uric acid, and renal function in Korean lead workers. *Environ Health Perspect.* 2005;113:36-42.

(26) *Harrison's Principles of Internal Medicine, 16th Edition.* I and II ed. New York, NY: McGraw-Hill; 2005.

(27) Kim SG, Yang SW, Jang AS et al. Prevalence of diabetes mellitus in the elderly of Namwon County, South Korea. *Korean J Intern Med.* 2002;17:180-190.

(28) Shin C, Abbott RD, Lee H, Kim J, Kimm K. Prevalence and correlates of orthostatic hypotension in middle-aged men and women in Korea: the Korean Health and Genome Study. *J Hum Hypertens.* 2004;18:717-723.

(29) Tsaih SW, Korrick S, Schwartz J et al. Lead, diabetes, hypertension, and renal function: the normative aging study. *Environ Health Perspect.* 2004;112:1178-1182.

(30) Buchet JP, Roels H, Bernard A, Lauwerys R. Assessment of renal function of workers exposed to inorganic lead, cadmium or mercury vapor. *J Occup Environ Med.* 1980;22:741-750.

(31) Wu MT, Kelsey K, Schwartz J, Sparrow D, Weiss S, Hu H. A delta-aminolevulinic acid dehydratase (ALAD) polymorphism may modify the relationship of low-level

lead exposure to uricemia and renal function: the Normative Aging Study. *Environ Health Perspect.* 2003;111:335-341.

(32) Pocock SJ, Shaper AG, Ashby D, Delves HT, Clayton BE. The relationship between blood lead, blood pressure, stroke, and heart attacks in middle-aged British men. *Environ Health Perspect.* 1988;78:23-30.

(33) Ishida M, Ishizaki M, Yamada Y. Decreases in postural change in finger blood flow in ceramic painters chronically exposed to low level lead. *Am J Ind Med.* 1996;29:547-553.

(34) Ehle AL. Lead neuropathy and electrophysiological studies in low level lead exposure: a critical review. *Neurotoxicology.* 1986;7:203-216.

(35) Nawrot TS, Thijs L, Den Hond EM, Roels HA, Staessen JA. An epidemiological re-appraisal of the association between blood pressure and blood lead: a meta-analysis. *J Hum Hypertens.* 2002;16:123-131.

(36) Neri LC, Hewitt D, Orser B. Blood lead and blood pressure: analysis of cross-sectional and longitudinal data from Canada. *Environ Health Perspect.* 1988;78:123-126.

(37) Harlan WR, Landis JR, Schmouder RL, Goldstein NG, Harlan LC. Blood lead and blood pressure. Relationship in the adolescent and adult US population. *JAMA.* 1985;253:530-534.

(38) Harlan WR. The relationship of blood lead levels to blood pressure in the U.S. population. *Environ Health Perspect.* 1988;78:9-13.

(39) Gartside PS. The relationship of blood lead levels and blood pressure in NHANES II: additional calculations. *Environ Health Perspect.* 1988;78:31-34.

(40) Den HE, Nawrot T, Staessen JA. The relationship between blood pressure and blood lead in NHANES III. National Health and Nutritional Examination Survey. *J Hum Hypertens.* 2002;16:563-568.

(41) Sharp DS, Benowitz NL, Osterloh JD, Becker CE, Smith AH, Syme SL. Influence of race, tobacco use, and caffeine use on the relation between blood pressure and blood lead concentration. *Am J Epidemiol.* 1990;131:845-854.

(42) Kromhout D. Blood lead and coronary heart disease risk among elderly men in Zutphen, The Netherlands. *Environ Health Perspect.* 1988;78:43-46.

(43) Orssaud G, Claude JR, Moreau T, Lellouch J, Juguet B, Festy B. Blood lead concentration and blood pressure. *Br Med J (Clin Res Ed).* 1985;290:244.

(44) Weiss ST, Munoz A, Stein A, Sparrow D, Speizer FE. The relationship of blood lead to blood pressure in a longitudinal study of working men. *Am J Epidemiol.* 1986;123:800-808.

(45) Grandjean P, Hollnagel H, Hedegaard L, Christensen JM, Larsen S. Blood lead-blood pressure relations: alcohol intake and hemoglobin as confounders. *Am J Epidemiol.* 1989;129:732-739.

(46) de Kort WL, Zwennis WC. Blood lead and blood pressure: some implications for the situation in The Netherlands. *Environ Health Perspect.* 1988;78:67-70.

(47) Moller L, Kristensen TS. Blood lead as a cardiovascular risk factor. *Am J Epidemiol.* 1992;136:1091-1100.

(48) Hu H, Aro A, Payton M et al. The relationship of bone and blood lead to hypertension. The Normative Aging Study. *JAMA.* 1996;275:1171-1176.

(49) Glenn BS, Stewart WF, Links JM, Todd AC, Schwartz BS. The longitudinal association of lead with blood pressure. *Epidemiology.* 2003;14:30-36.

(50) Schwartz J. Lead, blood pressure, and cardiovascular disease in men and women. *Environ Health Perspect.* 1991;91:71-75.

(51) Cheng Y, Schwartz J, Sparrow D, Aro A, Weiss ST, Hu H. Bone lead and blood lead levels in relation to baseline blood pressure and the prospective development of hypertension: the Normative Aging Study. *Am J Epidemiol.* 2001;153:164-171.

(52) Parkinson DK, Hodgson MJ, Bromet EJ, Dew MA, Connell MM. Occupational lead exposure and blood pressure. *Br J Ind Med.* 1987;44:744-748.

(53) Maheswaran R, Gill JS, Beevers DG. Blood pressure and industrial lead exposure. *Am J Epidemiol.* 1993;137:645-653.

(54) Schwartz J. Lead, blood pressure, and cardiovascular disease in men. *Arch Environ Health.* 1995;50:31-37.

(55) Staessen JA, Bulpitt CJ, Fagard R et al. Hypertension caused by low-level lead exposure: myth or fact? *J Cardiovasc Risk.* 1994;1:87-97.

Statistical Data Concerning Hypertension, Diabetes, Renal Kidney Failure, and Memory Loss in Society

I. Hypertension.....3

II. Diabetes.....4

III. Renal Kidney Failure.....6

IV. Memory Loss.....8

V. Attachments.....10
- A. Memory Loss Chart
- B. Facts on High Blood Pressure
- C. Hypertension Table

I. Hypertension

- **Source: National Kidney Foundation, http://www.kidney.org/news/newsroom/fs_new/10factsabtaframerkd.cfm (last visited Oct. 30, 2009).**
- **Internal Citation for FN1:** National Center for Health Statistics. Health, United States, 2008, with Chartbook on the Health of Americans. Hyattsville, MD: 2008. Available at http://www.cdc.gov/nchs/data/hus/hus08.pdf
  - It is estimated that about 90% of middle-aged adults will develop high blood pressure in the remainder of their lifetime.
  - In the United States, the prevalence of hypertension is higher among blacks than whites.[1]
  - The prevalence of high blood pressure is particularly high among black women at 44%.[1]
  - **Age-adjusted Percent of Adults Aged 20 Years and Older With Hypertension and/or Taking Blood Pressure-Lowering Medication, 2003–2006**

Overall:	31.3%
Women:	
All Women	30.3%
White	28.3%
African American	44.1%
Mexican American	28.6%
Men:	
All Men	31.8%
White	31.2%
African American	42.2%
Mexican American	24.8%
Poverty Status:	
Poor	35.0%
Near Poor	34.1%
Non-Poor	30.3%

Internal Source: *Health*, United S tates, 2008[1]

- **Source: National Kidney Foundation, http://www.kidney.org/site/kidneyDisease/index.cfm?ch=209 (last visited Oct. 30, 2009).**
  - More than 40 percent of African Americans are believed to have high blood pressure, however.[2] African-Americans also have higher death rates from causes related to high blood pressure. In 2004, for example, the death rates per 100,000 population from high blood pressure were 15.6 for white men, 49.9 for black men, 14.3 for white women and 40.6 for black women.[3]
  - South Carolina is second in the nation in the prevalence of diabetes, and a leader in the prevalence of high blood pressure.

II. Diabetes

- **Source: American Diabetes Ass'n, http://www.diabetes.org/diabetes-statistics.jsp (last visited Oct. 30, 2009).**
  - 23.6 million people in the United States, or 8% of the population, have diabetes.
  - **Total Prevalence of Diabetes**
    - **Age 20 years or older:** 23.5 million, or 10.7% of all people in this age group have diabetes.
    - **Non-Hispanic Whites:** 14.9 million, or 9.8% of all non-Hispanic whites aged 20 years or older have diabetes.
    - **Non-Hispanic Blacks:** 3.7 million, or 14.7% of all non-Hispanic blacks aged 20 years or older have diabetes.
    - The prevalence of diabetes is at least 2 to 4 times higher among non-Hispanic Black, Hispanic/Latino American, American Indian, and Asian/Pacific Islander women than among non-Hispanic white women.
  - **Race and Ethnic Differences in Prevalence of Diagnosed Diabetes:** After adjusting for population age differences, 2004-2006 national survey data for people diagnosed with diabetes, **aged 20 years or older** include the following prevalence by race/ethnicity:
    - 6.6% of non-Hispanic whites
    - 7.5% of Asian Americans
    - 10.4% of Hispanics
    - 11.8% of non-Hispanic blacks

- **Source: National Kidney Foundation, http://www.kidney.org/news/newsroom/fs_new/10factsabtaframerkd.cfm (last visited Oct. 30, 2009).**
  - Diabetes is the leading cause of kidney failure in African Americans.
  - The prevalence of diabetes in African Americans is much higher than in white Americans. Among those aged 40 to 74 years, the rate is 11.2 percent for white Americans, compared with 18.2 percent for African Americans.[2]
  - Approximately 13.3 percent of all African Americans over 20 years of age (3.2 million) have diabetes.
  - On average, African Americans are twice as likely to have diabetes as white Americans of similar age.
  - The most common type of diabetes in African Americans is type 2 diabetes. The risk factors for this type of diabetes include: family history, impaired glucose tolerance, diabetes during pregnancy, high blood insulin levels and insulin resistance, obesity and physical inactivity.[2]
  - African Americans with diabetes are more likely to develop complications of diabetes and to have greater disability from these complications than white Americans.
  - African-Americans experience kidney failure about four times more often than white Americans with diabetes.

- African-Americans are also more likely to develop other serious complications such as heart disease and strokes and to experience greater disability than white Americans with diabetes.
- Death rates associated with diabetes are 20 percent higher for African American men and 40 percent higher for African American women, compared with their white counterparts.[2]

- **Source: Kaiser Family, http://www.statehealthfacts.org/profileind.jsp?ind=70&cat=2&rgn=42 (last visited, Oct. 30, 2009).**
  - **South Carolina: Percent of Adults Who Have Ever Been Told by a Doctor that They Have Diabetes, 2008**

Percent of Adults Who Have Ever Been Told by a Doctor that They Have Diabetes, 2008 Compare

	13.1%	8.2%
	0.7%	0.8%
	87.4%	90.0%
	1.6%	1.0%

- **Source: South Carolina Department of Health and Environmental Control, http://www.scdhec.gov/health/chcdp/diabetes/statistics.htm (last visited, Oct. 30, 2009).**
  - Diabetes is a major public health problem across the nation and the seventh leading cause of death in South Carolina (2005).
  - According to the 2006 Behavior Risk Factor Surveillance Survey (BRFSS), South Carolina ranks seventh in diabetes prevalence, where an estimated 414,840 people in South Carolina are aware that they have diabetes.
  - The prevalence of diabetes among adults in South Carolina was 9.6% in 2006 (BRFSS, SC DHEC).
  - South Carolina is 8th in the nation for diabetes prevalence among African Americans (BFRSS, SC DHEC 2006).
  - In South Carolina, African Americans of either gender are twice as likely to have diabetes as their white counterparts. About 13.8 % of African-Americans said that they had diabetes, while only 8.2% of whites reported having the disease.

III. Renal Kidney Failure

- **Source: National Institute of Diabetes and Digestive and Kidney Diseases, NIH, http://kidney.niddk.nih.gov/kudiseases/pubs/kustats/index.htm (last visited Oct. 30, 2009).**
  - **Prevalence for Kidney Disease:** (1999–2004): An estimated 11.5 percent of adults ages 20 or older (23 million adults) have physiological evidence of chronic kidney disease determined from data collected through the National Health and Nutrition Examination Survey. [1]
  - **End-stage Renal Disease (ESRD)**
    - **Prevalence (2006):** 506,256 U.S. residents were under treatment as of the end of the calendar year.[2]
    - **Resulting from these primary diseases:**
      - **Diabetes:** 188,381
      - **Hypertension:** 122,339
      - **Glomerulonephritis:** 80,164
      - **Cystic kidney:** 23,685
      - **Urologic disease:** 13,371
      - **All other:** 78,316

- **Source: National Kidney Foundation, http://www.kidney.org/news/newsroom/fs_new/10factsabtaframerkd.cfm (last visited Oct. 30, 2009).**
- **Internal Citations:**

1. US Renal Data System 2006 Annual Data Report (www.usrds.org)
2. "Diabetes in African Americans," from the National Kidney and Urologic Diseases Information Clearinghouse (www.niddk.nih.gov)
3. American Heart Association High Blood Pressure Statistics (www.americanheart.org)
4. "Early Identification of Renal Disease Among African Americans: A Continuing Problem," M Thornhill-Joynes and M. Moore, *Nephrology News & Issues*, November 1995, p.16-18.

  - Due to high rates of diabetes, high blood pressure and heart disease, African Americans have an increased risk of developing kidney failure, and requiring dialysis treatments or a kidney transplant to sustain life.
  - In 2004, the incidence of kidney failure per million population was 968 in African Americans, compared with 263 in white Americans.[1] African Americans constitute about 32 percent of all patients treated for kidney failure in the U.S., but only about 12 percent of the overall U.S. population.[1]
  - African-Americans also develop kidney failure at an earlier age than white Americans. In 2006, the mean age for African-Americans at the start of treatment for kidney failure was 56.4 years, compared with 59.6 in white Americans.[1]
  - Diabetes is the leading cause of kidney failure in African Americans. The prevalence of diabetes in African Americans is much higher than in white Americans. Among those aged 40 to 74 years, the rate is 11.2 percent for white Americans, compared with 18.2 percent for African Americans.[2]
  - High blood pressure is the second leading cause of kidney failure among African Americans, accounting for 34 percent of the new cases each year.1 However, high

blood pressure remains the leading cause of death overall in African Americans because of its link with heart attacks and strokes.[2]

- Source: Office of Chronic Disease Epidemiology, http://www.scdhec.gov/health/epidata/docs/FACTckd09.pdf (last visited Oct. 30, 2009).
  - WHO IS AT RISK FOR KIDNEY FAILURE?
    - Diabetes is the most common cause of kidney failure.
    - High blood pressure is the second leading cause of kidney failure.
    - African Americans are four times more likely to develop kidney failure than Whites.
    - Diabetes and high blood pressure account for more than 70 percent of kidney failure in African Americans.
  - KIDNEY DISEASE IN SC
    - 1 in 8 South Carolinians have Chronic Kidney Disease (CKD).
    - Approximately 460,000 people have CKD in South Carolina.
    - South Carolina is, per capita, third in the nation with the number of patients on dialysis.
    - 75% of kidney patients in South Carolina are African American.
    - Three out of four of those waiting for a kidney are African American, and South Carolina ranks second in the nation in the need for organs among African Americans.

- Source: National Kidney Foundation, http://www.kidney.org/site/kidneyDisease/index.cfm?ch=209 (last visited Oct. 30, 2009).
  - 1 in 8 South Carolinians has CKD.
  - Approximately 546,000 people have CKD in South Carolina.
  - South Carolina is, per capita, third in the nation with the number of patients on dialysis.
  - Diabetes and high blood pressure are the leading causes of kidney disease
  - South Carolina is second in the nation in the prevalence of diabetes, and a leader in the prevalence of high blood pressure.
  - Kidney disease disproportionately affects African-Americans. 30% of South Carolinians are African-American, and nearly 75% of kidney patients in South Carolina are African-American.
  - More than 600 South Carolinians are waiting for a kidney transplant.
  - Three out of four of those waiting for a kidney are African-American, and South Carolina ranks second in the nation in the need for organs among African-Americans.

IV. Memory Loss & Aging

- **Source: Melissa Healy, Memory Loss: What's Normal What's Not, Los Angeles Times, Nov. 17, 2008.**
  - By about age 25, however, a human's memory has typically peaked and a long period of decline begins.
  - Around about the half-century mark, cognitive performance starts to slide noticeably. Keys and glasses are forgotten. Names, places and words linger on the tip of the tongue.

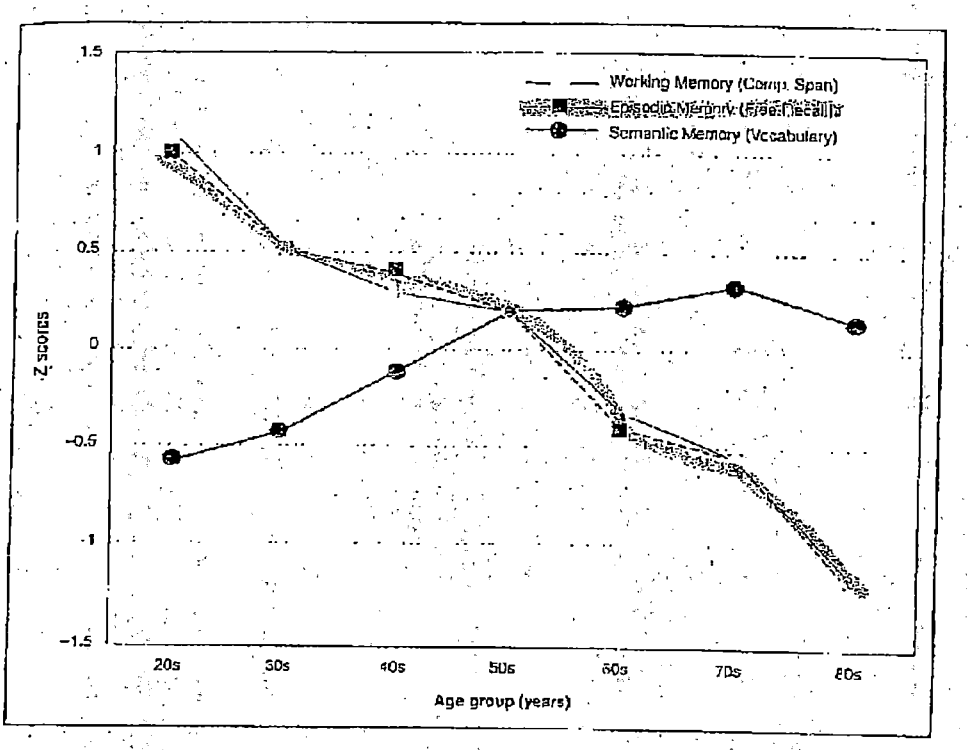
- **Source: 3 Encyclopedia of Aging, 902, 905, & 914 (2002, David J. Ekerdt).**
  - Most people recognize that their memories are changing as they grow older. They have a hard time coming up with names; they have a harder time finding things they need; they have to rely more on external memory cues such as notes or calendars. P. 902
  - Memory declines are experienced throughout the adult life span and not just in the older ages. P. 902
  - Working memory and episodic memory decline significantly across the life span, while semantic memory increases significantly. P. 902
  - **Episodic memories:** recollections that are actively retrieved as previous personal experiences. P. 905
    - The memory is a reconstruction of an earlier experience very much like looking something up in an internal cognitive diary.
    - Ex: "What did I have for dinner last night?" "Where did I park my car?" "Did I take my medicine this morning?" "Did you see Joan at the party last night?"
    - To answer each question, one attempts to reconstruct the original event.
    - Older adults do worse on most episodic memory tasks than do younger adults. (see chart saved in file 01464.04709)
    - Episodic memory requires one to put what one is trying to remember in a specific context.
  - **Everyday memory-** refers to memory operations that routinely occur in one's daily environment. P. 914
    - Ex: remembering names, remembering plans for the day, recalling items that one needs to purchase at the store, remembering to take medications, remembering telephone numbers, directions, or recent newsworthy events.
    - Hallmark of everyday memory is that it involves the performance of tasks that occur naturalistically in the real world.
    - Laboratory studies generally provide evidence for a decline with age in everyday memory process. **See the West et al study (on order from library). These studies found evidence for age-related declines on a wide range of every-day memory lab tasks (remembering names, locations of objects, grocery lists, faces, telephone numbers, and news events)

every-day memory lab tasks (remembering names, locations of objects, grocery lists, faces, telephone numbers, and news events)

- **Source: R.L.C. West et al, Everyday Memory Performance Across the Life Span: Effects of Age and Noncognitive Individual Differences, Psychology & Aging, 72–82 (1992).**
  - Source is on order from Greenville County Library System

Figure 1

Age group differences in working memory (as measured by computation span), episodic memory (as measured by free recall), and semantic memory (as measured by vocabulary).



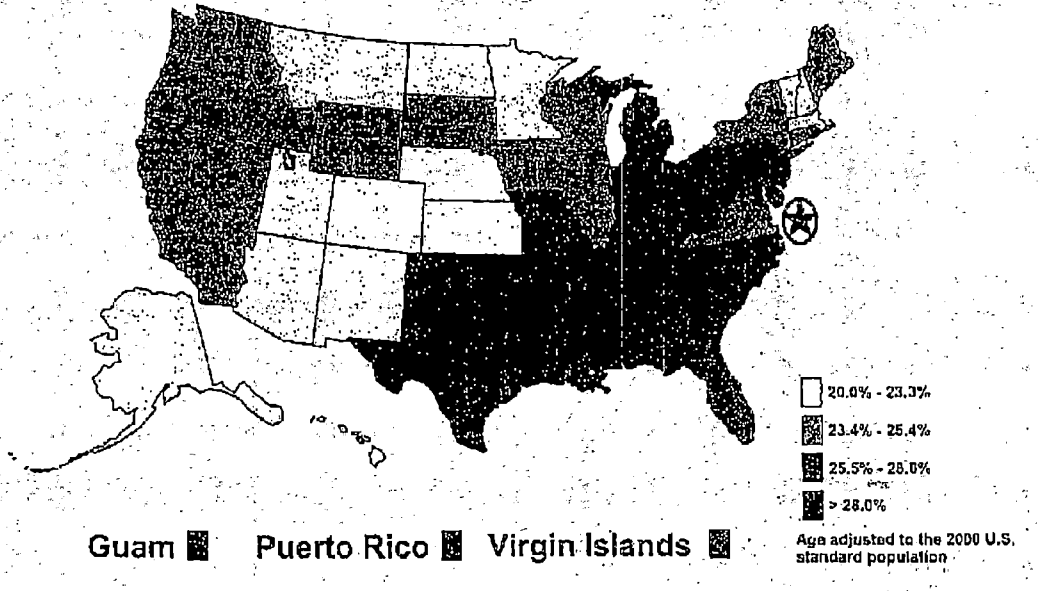
SOURCE: Adapted from Park, Lautenschlager, et al. in press. Copyright © 2002 by the American Psychological Association. Adapted with permission.

difficult to accomplish in aging research, however, because adults of different ages may process information differently at one of the earlier stages, thus violating the requirement that all be held constant until retrieval (Smith).

The major reason, however, for the loss of interest in identifying the stage at which aging had its effects is clear evidence that age has effects on all stages of memory: encoding, storage, and retrieval. For reasons not directly related to memory stage theory, however, memory research in the 1970s and early 1980s focused heavily on the encoding stage of memory. This focus on encoding was due to the development of a conceptual view of memory, the "levels of processing" framework, which proposed that the ability to remember was determined by the extent of semantic processing during the encoding of the to-be-remembered information (Craik and Lockhart). For this reason, much research on memory and aging during this period focused on the nature of encoding processes in different age groups. Even with the magnitude of the research effort, however, the relationship between aging and levels of processing is still unclear. The research did, however, suggest that encoding is especially affected by adult aging. For example, the memory performance of older adults, relative to younger adults, is more detrimentally affected by

Facts on High Blood Pressure

Percent of persons who were ever told they had high blood pressure, Adults aged 20 years and older, 2003



20.0% - 23.3%: Alaska, Arizona, Colorado, Hawaii, Kansas, Massachusetts, Minnesota, Montana, Nebraska, New Hampshire, New Mexico, North Dakota, Utah, Vermont	**25.5% - 28.0%:** District of Columbia, Florida, Guam, Indiana, Maryland, Michigan, Missouri, New Jersey, Ohio, Oklahoma, Pennsylvania, Texas, Virgin Islands
23.4% - 25.4%: California, Connecticut, Idaho, Illinois, Iowa, Maine, Nevada, New York, Oregon, South Dakota, Virginia, Washington, Wisconsin, Wyoming	**>28.0%:** Alabama, Arkansas, Delaware, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, Puerto Rico, South Carolina, Rhode Island, Tennessee, West Virginia

Source: CDC, Behavioral Risk Factor Surveillance System

June 2005

Table 71 (page 1 of 2). Hypertension and elevated blood pressure among persons 20 years of age and over, by selected characteristics: United States, 1988–1994, 1999–2002, and 2003–2006

[Data are based on interviews and physical examinations of a sample of the civilian noninstitutionalized population]

Click here for spreadsheet version

Sex, age, race and Hispanic origin[1], and percent of poverty level	Hypertension[2,3] (elevated blood pressure and/or taking antihypertensive medication)			Elevated blood pressure[2]		
	1988–1994	1999–2002	2003–2006	1988–1994	1999–2002	2003–2006
	Percent of population					
20 years and over, age-adjusted[4]						
Both sexes[5]	25.5	30.0	31.3	18.5	19.9	17.9
Male	26.4	28.8	31.8	20.6	19.1	18.2
Female	24.4	30.6	30.3	16.4	20.2	17.3
Not Hispanic or Latino:						
White only, male	25.6	27.6	31.2	18.7	17.6	17.4
White only, female	23.0	28.5	28.3	15.1	18.5	15.9
Black or African American only, male	37.5	40.6	42.2	30.3	28.2	26.5
Black or African American only, female	38.3	43.5	44.1	26.4	28.8	23.9
Mexican male	26.9	26.8	24.8	22.2	21.6	15.3
Mexican female	25.0	27.9	28.6	20.4	21.2	19.2
Percent of poverty level:[6]						
Below 100%	31.7	33.9	35.0	22.5	23.3	22.6
100%–less than 200%	26.6	33.5	34.1	19.3	23.0	21.1
200% or more	23.9	28.2	30.3	17.5	18.2	16.6
20 years and over, crude						
Both sexes[5]	24.1	30.2	32.1	17.6	19.9	18.2
Male	23.8	27.6	31.3	18.7	18.2	17.9
Female	24.4	32.7	32.9	16.5	21.6	18.6
Not Hispanic or Latino:						
White only, male	24.3	28.3	32.4	18.7	17.8	17.9
White only, female	24.6	32.8	33.4	16.4	21.6	18.8
Black or African American only, male	31.1	35.9	38.8	25.5	25.2	24.8
Black or African American only, female	32.5	41.9	42.8	22.2	27.2	22.4
Mexican male	16.4	16.5	16.5	13.9	14.1	10.9
Mexican female	15.9	18.8	20.0	12.7	13.8	13.0
Percent of poverty level:[6]						
Below 100%	25.7	30.3	28.8	18.7	21.1	16.3
100%–less than 200%	28.7	34.8	36.8	19.8	24.1	22.5
200% or more	22.2	28.2	31.1	16.2	17.8	16.8
Male						
20–34 years	7.1	*8.1	9.2	6.6	*7.3	7.6
35–44 years	17.1	17.1	21.1	15.2	12.1	13.2
45–54 years	29.2	31.0	38.2	21.9	20.4	21.0
55–64 years	40.6	45.0	50.2	28.4	24.8	26.4
65–74 years	54.4	59.6	64.1	39.9	34.9	29.2
75 years and over	60.4	69.0	65.0	49.7	50.6	38.2
Female						
20–34 years	2.9	*2.7	*2.2	*2.4	*1.4	*
35–44 years	11.2	15.1	12.6	5.4	8.5	5.8
45–54 years	23.8	31.8	36.2	13.7	19.1	20.0
55–64 years	42.6	53.9	54.4	27.0	31.9	28.6
65–74 years	56.2	72.7	70.8	38.2	53.0	40.8
75 years and over	73.6	83.1	80.2	59.9	54.4	55.4

See footnotes at end of table.

Section Contents
En español
Learning Objectives
Introduction
Neurological Effects
Children
Adults
Renal Effects
Hematological Effects
Endocrine Effects
Gastrointestinal Effects
Cardiovascular Effects
Reproductive Effects
Developmental Effects
Other Potential Effects
Key Points
Progress Check

Case Contents
Table of Contents
Cover Page
How to Use the Course
Initial Check
What is Lead
Where Found
Exposure Routes
Who Is at Risk
Safety Standards
Biological Fate
Physiological Effects
Patient Evaluation
Diagnostic Tests
Patient Treatment
Patient Education
More Information
Posttest

Case Studies in Environmental Medicine (CSEM) Lead Toxicity

- Literature Cited
- Acronyms/Abbreviations
- Patient Education Sheet

Environmental Medicine
- CSEM
- GREM
- PEHT
- Continuing Education
- Online Registration
- Patient Education
- Community Education

Related Documents
- CABS: Lead
- Lead Screening
- PHS: Lead
- Pb/As/Cd/Cr Interaction
- Pb/Mn/Zn/Cu Interaction
- Pb/Chlorpyrifos/Hg/Me-Hg
- ToxFAQs™: Lead
- Toxguide: Lead
- Tox Profile: Lead

ATSDR Resources
- ATSDR en Español
- Case Studies (CSEM)
- Exposure Pathways
- Health Assessments
- Health Statements
- Interaction Profiles
- Interactive Learning
- Managing Incidents
- Medical Guidelines
- Minimal Risk Levels
- Priority List
- ToxFAQs™
- ToxFAQs™ CABS
- Toxicological Profiles
- Toxicology Curriculum

External Resources

What Are the Physiologic Effects of Lead Exposure?

CDC
Cancer
eLCOSH
EPA
Healthfinder®
Medline Plus
NCEH
NIEHS
NIOSH
OSHA

Learning Objectives	Upon completion of this section, you will be able to • describe how lead affects adults and children • describe the major physiologic effects of chronic low- level lead exposure • describe the major physiologic effects of acute high-level lead exposure.

Introduction	Lead serves no useful purpose in the human body, but its presence in the body can lead to toxic effects, regardless of exposure pathway. • Lead toxicity can affect every organ system. • On a molecular level, proposed mechanisms for toxicity involve fundamental biochemical processes. These include lead's ability to inhibit or mimic the actions of calcium (which can affect calcium-dependent or related processes) and to interact with proteins (including those with sulfhydryl, amine, phosphate and carboxyl groups) (ATSDR, 2005). It must be emphasized that **there may be no threshold** for developmental effects on children. • The practicing health care provider can distinguish overt clinical symptoms and health effects that come with high exposure levels on an individual basis. • However, lack of overt symptoms does not mean "no lead poisoning." • Lower levels of exposure have been shown to have many subtle health effects. • Some researchers have suggested that lead continues to contribute significantly to socio-behavioral problems such as juvenile delinquency and violent crime (Needleman 2002, Nevin 2000). • It is important to prevent all lead exposures. While the immediate health effect of concern in children is typically

Neurological Effects	The nervous system is the most sensitive target of lead exposure. • There may be no lower threshold for some of the adverse neurological effects of lead in children. • Neurological effects of lead in children have been documented at exposure levels once thought to cause no harmful effects (<10 µg/dL) (Canfield 2003; CDC 1997a). • Because otherwise asymptomatic individuals may experience neurological effects from lead exposure, clinicians should have a high index of suspicion for lead exposure, especially in the case of children.

Children	In children, acute exposure to very high levels of lead may produce encephalopathy and other accompanying signs of • ataxia • coma • convulsions • death • hyperirritability • stupor The BLLs associated with encephalopathy in children vary from study to study, but BLLs of 70-80 µg/dL or greater appear to indicate a serious risk (ATSDR 2005). • Even without encephalopathy symptoms, these levels are associated with increased incidences of lasting neurological and behavioral damage (ATSDR 2005). Children suffer neurological effects at much lower exposure levels. • Neurological effects may begin at low (and, relatively speaking, more widespread) BLLs, at or below 10 µg/dL in some cases, and it may not be possible to detect them on clinical examination. • Some studies have found, for example, that for every 10 µg/dL increase in BLL, children's IQ was found to be lower by four to seven points (Yule *et al.*, 1981; Schroeder *et al.*, 1985; Fulton *et al.*, 1987; Landsdown *et al.* 1986; Hawk *et al.* 1986; Winneke *et al.* 1990 as cited in AAP 1993). • There is a large body of evidence that associates decrement in IQ performance and other

Adults	There can be a difference in neurological effects between an adult exposed to lead as an adult, and an adult exposed as a child when the brain was developing. • Childhood neurological effects, including ADHD, may persist into adulthood. Lead-exposed adults may also experience many of the neurological symptoms experienced by children, although the thresholds for adults tend to be higher. Lead encephalopathy may occur at extremely high BLLs, *e.g.*, 460 µg/dL. (Kehoe 1961 as cited in ATSDR 2005) • Precursors of encephalopathy, such as dullness, irritability, poor attention span, muscular tremor, and loss of memory may occur at lower BLLs. Less severe *neurological and behavioral effects* have been documented in lead-exposed workers with BLLs ranging from 40 to 120 µg/dL. (ATSDR 2005) These effects include: • decreased libido • depression/mood changes, headache • diminished cognitive performance • diminished hand dexterity • diminished reaction time • diminished visual motor performance • dizziness • fatigue • forgetfulness • impaired concentration • impotence • increased nervousness • irritability

Renal Effects

Many studies show a strong association between lead exposure and renal effects. (ATSDR 1999)

- Acute high dose lead-induced impairment of proximal tubular function manifests in aminoaciduria, glycosuria, and hyperphosphaturia (a Fanconi-like syndrome). These effects appear to be reversible (ATSDR 1999).
- However, continued or repetitive exposures can cause a toxic stress on the kidney, if unrelieved, may develop into chronic and often irreversible lead nephropathy (*i.e.*, chronic interstitial nephritis).

The lowest level at which lead has an adverse effect on the kidney remains unknown.

- Most documented renal effects for occupational workers have been observed in acute high-dose exposures and high-to-moderate chronic exposures (BLL > 60 µg/dL).
- Currently, there are no early and sensitive indicators (*e.g.*, biomarkers) considered predictive or indicative of renal damage from lead. (ATSDR 2000) Serum creatinine and creatinine clearance are used as later indicators.
- However, certain urinary biomarkers of the proximal tubule (*e.g.*, NAG) show elevations with current exposures, even at BLLs less than 60 µg/dL; and some population-based studies show accelerated increases in serum creatinine or decrements in creatinine clearance at BLLs below 60 µg/dL. (Staessen *et al.* 1992;

Hematological Effects

Lead inhibits the body's ability to make hemoglobin by interfering with several enzymatic steps in the heme pathway.

- Specifically, lead decreases heme biosynthesis by inhibiting *d*-aminolevulinic acid dehydratase (ALAD) and ferrochelatase activity.
- Ferrochelatase, which catalyzes the insertion of iron into protoporphyrin IX, is quite sensitive to lead.
- A decrease in the activity of this enzyme results in an increase of the substrate, erythrocyte protoporphyrin (EP), in the red blood cells (also found in the form of ZPP—bound to zinc rather than to iron).
- Also associated with lead exposure is an increase in blood and plasma d-aminolevulinic acid (ALA) and free erythrocyte protoporphyrins (FEP) (EPA 1986a as cited in ATSDR 1999).

EPA estimated the threshold BLL for a decrease in hemoglobin to be 50 µg/dL for occupationally exposed adults and approximately 40 µg/dL for children, although other studies have indicated a lower threshold (*e.g.*, 25 µg/dL) for children. (EPA 1986b as cited in ATSDR 1999; ATSDR 1999)

- Recent data indicate that the EP level, which has been used in the past to screen for lead toxicity, is not sufficiently sensitive at lower levels of blood lead and is therefore not as useful a screening test as previously thought (see the *"Laboratory Evaluation"* section for further discussion of EP testing.).

Lead can induce two types of anemia

Endocrine Effects	Studies of children with high lead exposure have found that a strong inverse correlation exists between BLLs and vitamin D levels. ▪ Lead impedes vitamin D conversion into its hormonal form, 1, 25-dihydroxyvitamin D, which is largely responsible for the maintenance of extra- and intra-cellular calcium homeostasis. • Diminished 1, 25-dihydroxyvitamin D, in turn, may impair cell growth, maturation, and tooth and bone development. • In general, these adverse effects seem to be restricted to children with chronically high BLLs (most striking in children with BLLs > 62 µg/dL) and chronic nutritional deficiency, especially with regard to calcium, phosphorous, and vitamin D (Koo *et al.* 1991 as cited in ATSDR 1999). • However, Rosen *et al.* (1980) noted that in lead-exposed children with blood lead levels of 33-55 µg/dL, 1, 25-dihydroxyvitamin D levels were reduced to levels comparable to those observed in children with severe renal insufficiency. • Lead appears to have a minimal, if any, effect on thyroid function.
Gastrointestinal Effects	In severe cases of lead poisoning, children or adults may present with severe cramping abdominal pain, which may be mistaken for an acute abdomen or appendicitis.

Cardiovascular (Hypertension) Effects	Hypertension is a complex condition with many different causes and risk factors, including age, weight, diet, and exercise habits. • Lead exposure is one factor of many that may contribute to the onset and development of hypertension. • Although low to moderate lead level exposures (BLL<30 µg/dL) show only a low degree of association with hypertension, higher exposures (primarily occupational) increase the risk for hypertensive heart disease and cerebrovascular disease as latent effects. • One study found that adults who experienced lead poisoning as children had a significantly higher risk of hypertension 50 years later (relative to control adults without childhood lead exposure). (Hu, 1991, as cited in ATSDR 2000) The association has been shown in population-based studies with BLLs below 10 µg/dL. Data supports an association between lead exposure and elevations in blood pressure. (Victery *et al.* 1988; Schwartz 1995 as cited in ATSDR 2000; Korrick *et al.* 1999; Hu *et al.* 1996) • It is estimated that, on a population basis, blood lead can account for a 1% to 2% variance in blood pressure. (ATSDR 2000) This could increase the incidence of hypertension a substantial amount, due to the high prevalence of hypertension of all causes in general populations.

Reproductive Effects	Reproductive effects examined in the literature include sperm count, fertility, and pregnancy outcomes. While several studies have implicated lead as contributing to reproductive and developmental effects, these effects have not been well-established at low exposure levels. ***Male Reproductive Effects*** Recent reproductive function studies in humans suggest that current occupational exposures decrease sperm count totals and increase abnormal sperm frequencies (Alexander *et al.* 1996; Gennart *et al.* 1992; Lerda 1992; and Lin *et al.* 1996 as cited in ATSDR 2000; Telisman *et al.* 2000). ▪ Effects may begin at BLLs of 40 µg/dL. (ATSDR 2005) ▪ Long-term lead exposure (independent of current lead exposure levels) also may diminish sperm concentrations, total sperm counts, and total sperm motility (Alexander *et al.* 1996 as cited in ATSDR 2000). ▪ It is unclear how long these effects may last in humans after lead exposure ceases. ***Fertility*** It is not currently possible to predict fertility outcomes based on current BLLs or past lead exposure levels. (ATSDR 2000) ***Pregnancy Outcomes*** The effect of low-level lead exposures on pregnancy outcomes is not clear. Thus it appears that at higher (*e.g.*, occupational) exposure levels, the evidence is clearer for an association

Developmental Effects	Developmental effects examined in the literature include pregnancy outcomes (*e.g.*, premature births and low birth weights), congenital abnormalities, and post birth effects on growth or neurological development. • Increasing evidence indicates that lead, which readily crosses the placenta, adversely affects fetus viability as well as fetal and early childhood development. • Prenatal exposure to low lead levels (*e.g.*, maternal BLLs of 14 µg/dL) may increase the risk of reduced birth weight and premature birth (ATSDR 1999). • Although lead is an animal teratogen, most human studies have not shown a relationship between lead levels and congenital malformations. • A study by Needleman *et al.* (1984) correlated increased prenatal lead exposure with increased risk for minor congenital abnormalities (*e.g.*, minor skin abnormalities and undescended testicles). • No association between prenatal lead exposure and major congenital abnormalities has been found (Ernhart *et al.* 1985, 1986; McMichael *et al.* 1986). • In a retrospective study, a higher proportion of learning disabilities were found among school-aged children with biological parents who were lead poisoned as children 50 years previously (Hu 1991).

Other Potential Effects	Lead has been linked to problems with the development and health of bones. At high levels, lead can result in slowed growth in children. • Studies have shown increased likelihood of osteoporosis (weakened bones later in life) in animals exposed to lead. A review of this issue can be found in Puzas (1992). Although this link has not been established in humans, it is likely that upon closer examination of lead-exposed individuals, lead will be shown to be a new risk factor for the disease. • Research currently underway may provide more information about potential impacts of lead on osteoporosis (bone health) in the future. Current available data are not sufficient to determine the carcinogenicity of lead in humans. • EPA has classified elemental lead and inorganic lead compounds as Group 2B: probable human carcinogens. (ATSDR 1999) This classification is based in part on animal studies, which have been criticized because the doses of lead administered were extremely high (ATSDR 1999). • The National Toxicology Program classifies lead and lead compounds as "reasonably anticipated to be a carcinogen" (NTP 2004). • Information regarding the association of occupational exposure to lead with increased cancer risk is generally limited. This is because these occupational exposure studies, which primarily

Key Points	• Effects in children generally occur at lower BLLs than in adults. • The developing nervous system of a child can be affected adversely at BLLs of less than 10 µg/dL. It is often impossible to determine these effects upon clinical examination. • There is a wide range of neurological effects associated with lead exposure, some of which may likely be irreversible. • Lead exposure can lead to renal effects such as Fanconi-like syndromes, chronic nephropathy, and gout. • Most lead-associated renal effects or disease are a result of ongoing chronic or present high acute exposure or can be a latent effect of chronic past lead exposure. • Lead inhibits several enzymes critical to the synthesis of heme, causing a decrease in blood hemoglobin. • Today, lead exposure in children only rarely results in frank anemia. • Lead's impairment of heme synthesis can affect other heme-dependent processes in the body outside of the hematopoietic system. • Lead interferes with a hormonal form of vitamin D, which affects multiple processes in the body, including cell maturation and skeletal growth. • Lead exposure may lead to increased risk for hypertension and its sequelae. • Evidence suggests an association between lead exposure and certain reproductive and developmental

Progress Check

7. How do lead's effects **differ** in children and adults?

- A. Effects in children are more likely to be reversible.
- B. Adults suffer more neurological damage.
- C. Children are less likely to become anemic.
- D. Effects in adults tend to begin at higher exposure levels than in children.

Answer:

Clear

To review relevant content, see Adults in this section.

8. Lead toxicity can affect
- A. the kidneys and brain
- B. IQ and neurological development in children
- C. sperm count
- D. All of the above.

Answer:

Clear

To review relevant content, see Neurological Effects, Children, Renal Effects, and Reproductive Effects in this section.

Previous Section **Next Section**

Revised 2007-08-20.

Agency for Toxic Substances and Disease Registry
4770 Buford Hwy NE, Atlanta, GA 30341-3717
Contact CDC: 800-232-4636 / TTY: 888-232-6348
cdcinfo@cdc.gov

USA.gov Government Made Easy

UNITED STATES DEPARTMENT OF LABOR
SEARCH
A to Z Index | En Español | Contact Us | FAQs | About OSHA
OSHA
OSHA QuickTakes Newsletter RSS Feeds Print This Page Text Size
Occupational Safety & Health Administration We Can Help
What's New | Offices
Home | Workers | Regulations | Enforcement | Data & Statistics | Training | Publications | Newsroom | Small Business
OSHA

Regulations (Standards - 29 CFR) - Table of Contents

- **Part Number:** 1910
- **Part Title:** Occupational Safety and Health Standards
- **Subpart:** Z
- **Subpart Title:** Toxic and Hazardous Substances
- **Standard Number:** 1910.1025
- **Title:** Lead.
- **Appendix:** A, B, C, D

1910.1025(a)

Scope and application.

1910.1025(a)(1)

This section applies to all occupational exposure to lead, except as provided in paragraph (a)(2).

1910.1025(a)(2)

This section does not apply to the construction industry or to agricultural operations covered by 29 CFR Part 1928.

1910.1025(b)

Definitions.

Action level means employee exposure, without regard to the use of respirators, to an airborne concentration of lead of 30 micrograms per cubic meter of air (30 ug/m^3) averaged over an 8-hour period.

Assistant Secretary means the Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, or designee.

Director means the Director, National Institute for Occupational Safety and Health (NIOSH), U.S. Department of Health, Education, and Welfare, or designee.

Lead means metallic lead, all inorganic lead compounds, and organic lead soaps. Excluded from this definition are all other organic lead compounds.

1910.1025(c)

Permissible exposure limit (PEL).

1910.1025(c)(1)

The employer shall assure that no employee is exposed to lead at concentrations greater than fifty micrograms per cubic meter of air (50 ug/m^3) averaged over an 8-hour period.

1910.1025(c)(2)

If an employee is exposed to lead for more than 8 hours in any work day, the permissible exposure limit, as a time weighted average (TWA) for that day, shall be reduced according to the following formula:

Maximum permissible limit (in ug/m^3)=400 divided by hours worked in the day.

1910.1025(c)(3)

When respirators are used to supplement engineering and work practice controls to comply with the PEL and all the requirements of paragraph (f) have been met, employee exposure, for the purpose of determining whether the employer has complied with the PEL, may be considered to be at the level provided by the protection factor of the respirator for those periods the respirator is worn. These periods may be averaged with exposure levels during periods when respirators are not worn to determine the employee's daily TWA exposure.

1910.1025(d)

Exposure monitoring -

1910.1025(d)(1)

General.

1910.1025(d)(3)(i)

For the purposes of paragraph (d), employee exposure is that exposure which would occur if the employee were not using a respirator.

1910.1025(d)(3)(ii)

With the exception of monitoring under paragraph (d)(3), the employer shall collect full shift (for at least 7 continuous hours) personal samples including at least one sample for each shift for each job classification in each work area.

1910.1025(d)(3)(iii)

Full shift personal samples shall be representative of the monitored employee's regular, daily exposure to lead.

1910.1025(d)(2)

Initial determination. Each employer who has a workplace or work operation covered by this standard shall determine if any employee may be exposed to lead at or above the action level.

1910.1025(d)(3)

Basis of initial determination.

1910.1025(d)(3)(i)

The employer shall monitor employee exposures and shall base initial determinations on the employee exposure monitoring results and any of the following, relevant considerations:

1910.1025(d)(3)(i)(A)

Any information, observations, or calculations which would indicate employee exposure to lead;

1910.1025(d)(3)(i)(B)

Any previous measurements of airborne lead; and

1910.1025(d)(3)(i)(C)

Any employee complaints of symptoms which may be attributable to exposure to lead.

1910.1025(d)(3)(ii)

Monitoring for the initial determination may be limited to a representative sample of the exposed employees who the employer reasonably believes are exposed to the greatest airborne concentrations of lead in the workplace.

1910.1025(d)(3)(iii)

Measurements of airborne lead made in the preceding 12 months may be used to satisfy the requirement to monitor under paragraph (d)(3)(i) if the sampling and analytical methods used meet the accuracy and confidence levels of paragraph (d)(9) of this section.

1910.1025(d)(4)

Positive initial determination and initial monitoring.

1910.1025(d)(4)(i)

Where a determination conducted under paragraphs (d)(2) and (3) of this section shows the possibility of any employee exposure at or above the action level, the employer shall conduct monitoring which is representative of the exposure for each employee in the workplace who is exposed to lead.

1910.1025(d)(4)(ii)

Measurements of airborne lead made in the preceding 12 months may be used to satisfy this requirement if the sampling and analytical methods used meet the accuracy and confidence levels of paragraph (d)(9) of this section.

1910.1025(d)(5)

Negative initial determination. Where a determination, conducted under paragraphs (d)(2) and (3) of this section is made that no employee is exposed to airborne concentrations of lead at or above the action level, the employer shall make a written record of such determination. The record shall include at least the information specified in paragraph (d)(3) of this section and shall also include the date of determination, location within the worksite, and the name and social security number of each employee monitored.

1910.1025(d)(6)

Frequency.

1910.1025(d)(6)(i)

If the initial monitoring reveals employee exposure to be below the action level the measurements need not be repeated

except as otherwise provided in paragraph (d)(7) of this section.

1910.1025(d)(6)(ii)

If the initial determination or subsequent monitoring reveals employee exposure to be at or above the action level but below the permissible exposure limit the employer shall repeat monitoring in accordance with this paragraph at least every 6 months. The employer shall continue monitoring at the required frequency until at least two consecutive measurements, taken at least 7 days apart, are below the action level at which time the employer may discontinue monitoring for that employee except as otherwise provided in paragraph (d)(7) of this section.

1910.1025(d)(6)(iii)

If the initial monitoring reveals that employee exposure is above the permissible exposure limit the employer shall repeat monitoring quarterly. The employer shall continue monitoring at the required frequency until at least two consecutive measurements, taken at least 7 days apart, are below the PEL but at or above the action level at which time the employer shall repeat monitoring for that employee at the frequency specified in paragraph (d)(6)(ii), except as otherwise provided in paragraph (d)(7) of this section.

1910.1025(d)(7)

Additional monitoring. Whenever there has been a production, process, control or personnel change which may result in new or additional exposure to lead, or whenever the employer has any other reason to suspect a change which may result in new or additional exposures to lead, additional monitoring in accordance with this paragraph shall be conducted.

1910.1025(d)(8)

Employee notification.

1910.1025(d)(8)(i)

The employer must, within 15 working days after the receipt of the results of any monitoring performed under this section, notify each affected employee of these results either individually in writing or by posting the results in an appropriate location that is accessible to affected employees.

1910.1025(d)(8)(ii)

Whenever the results indicate that the representative employee exposure, without regard to respirators, exceeds the permissible exposure limit, the employer shall include in the written notice a statement that the permissible exposure limit was exceeded and a description of the corrective action taken or to be taken to reduce exposure to or below the permissible exposure limit.

1910.1025(d)(9)

Accuracy of measurement. The employer shall use a method of monitoring and analysis which has an accuracy (to a confidence level of 95%) of not less than plus or minus 20 percent for airborne concentrations of lead equal to or greater than 30 ug/m^3.

1910.1025(e)

Methods of compliance -

1910.1025(e)(1)

Engineering and work practice control's.

1910.1025(e)(1)(i)

Where any employee is exposed to lead above the permissible exposure limit for more than 30 days per year, the employer shall implement engineering and work practice controls (including administrative controls) to reduce and maintain employee exposure to lead in accordance with the implementation schedule in Table I below, except to the extent that the employer can demonstrate that such controls are not feasible. Wherever the engineering and work practice controls which can be instituted are not sufficient to reduce employee exposure to or below the permissible exposure limit, the employer shall nonetheless use them to reduce exposures to the lowest feasible level and shall supplement them by the use of respiratory protection which complies with the requirements of paragraph (f) of this section.

1910.1025(e)(1)(ii)

Where any employee is exposed to lead above the permissible exposure limit, but for 30 days or less per year, the employer shall implement engineering controls to reduce exposures to 200 ug/m^3, but thereafter may implement any combination of engineering, work practice (including administrative controls), and respiratory controls to reduce and maintain employee exposure to lead to or below 50 ug/m^3

TABLE I

Industry	Compliance dates(1): (50 UG/m^3)
Lead chemicals, secondary copper smelting.	July 19, 1996.
Nonferrous foundries.	July 19, 1996(2).

Brass and bronze ingot manufacture.	6 years(3).

Footnote(1) Calculated by counting from the date the stay on implementation of paragraph (e)(1) was lifted by the U.S. Court of Appeals for the District of Columbia, the number of years specified in the 1978 lead standard and subsequent amendments for compliance with the PEL of 50 ug/m^3 for exposure to airborne concentrations of lead levels for the particular industry.
Footnote(2) Large nonferrous foundries (20 or more employees) are required to achieve the PEL of 50 ug/m^3 by means of engineering and work practice controls. Small nonferrous foundries (fewer than 20 employees) are required to achieve an 8-hour TWA of 75 ug/m^3 by such controls.
Footnote(3) Expressed as the number of years from the date on which the Court lifts the stay on the implementation of paragraph (e)(1) for this industry for employers to achieve a lead in air concentration of 75 ug/m^3. Compliance with paragraph (e) in this industry is determined by a compliance directive that incorporates elements from the settlement agreement between OSHA and representatives of the injury, are required to comply within five years.

1910.1025(e)(2)

Respiratory protection. Where engineering and work practice controls do not reduce employee exposure to or below the 50 ug/m^3 permissible exposure limit, the employer shall supplement these controls with respirators in accordance with paragraph (f).

1910.1025(e)(3)

Compliance program.

1910.1025(e)(3)(i)

Each employer shall establish and implement a written compliance program to reduce exposures to or below the permissible exposure limit, and interim levels if applicable, solely by means of engineering and work practice controls in accordance with the implementation schedule in paragraph (e)(3).

1910.1025(e)(3)(ii)

Written plans for these compliance programs shall include at least the following:

1910.1025(e)(3)(ii)(A)

A description of each operation in which lead is emitted; e.g. machinery used, material processed, contro's in place, crew size, employee job responsibilities, operating procedures and maintenance practices;

1910.1025(e)(3)(ii)(B)

A description of the specific means that will be employed to achieve compliance, including engineering plans and studies used to determine methods selected for controlling exposure to lead;

1910.1025(e)(3)(ii)(C)

A report of the technology considered in meeting the permissible exposure limit;

1910.1025(e)(3)(ii)(D)

Air monitoring data which documents the source of lead emissions;

1910.1025(e)(3)(ii)(E)

A detailed schedule for implementation of the program, including documentation such as copies of purchase orders for equipment, construction contracts, etc.;

1910.1025(e)(3)(ii)(F)

A work practice program which includes items required under paragraphs (g), (h) and (i) of this regulation;

1910.1025(e)(3)(ii)(G)

An administrative control schedule required by paragraph (e)(5), if applicable;

1910.1025(e)(3)(ii)(H)

Other relevant information.

1910.1025(e)(3)(iii)

Written programs shall be submitted upon request to the Assistant Secretary and the Director, and shall be available at the worksite for examination and copying by the Assistant Secretary, Director, any affected employee or authorized employee representatives.

1910.1025(e)(3)(iv)

Written programs must be revised and updated at least annually to reflect the current status of the program.

1910.1025(e)(4)

Mechanical ventilation.

1910.1025(e)(4)(i)

When ventilation is used to control exposure, measurements which demonstrate the effectiveness of the system in controlling exposure, such as capture velocity, duct velocity, or static pressure shall be made at least every 3 months. Measurements of the system's effectiveness in controlling exposure shall be made within 5 days of any change in production, process, or control which might result in a change in employee exposure to lead.

1910.1025(e)(4)(ii)

Recirculation of air. If air from exhaust ventilation is recirculated into the workplace, the employer shall assure that (A) the system has a high efficiency filter with reliable back-up filter; and (B) controls to monitor the concentration of lead in the return air and to bypass the recirculation system automatically if it fails are installed, operating, and maintained.

1910.1025(e)(5)

Administrative controls. If administrative controls are used as a means of reducing employees TWA exposure to lead, the employer shall establish and implement a job rotation schedule which includes:

1910.1025(e)(5)(i)

Name or identification number of each affected employee;

1910.1025(e)(5)(ii)

Duration and exposure levels at each job or work station where each affected employee is located; and

1910.1025(e)(5)(iii)

Any other information which may be useful in assessing the reliability of administrative controls to reduce exposure to lead.

1910.1025(f)

Respiratory protection.

1910.1025(f)(1)

General. For employees who use respirators required by this section, the employer must provide each employee an appropriate respirator that complies with the requirements of this paragraph. Respirators must be used during:

1910.1025(f)(1)(i)

Periods necessary to install or implement engineering or work-practice controls.

1910.1025(f)(1)(ii)

Work operations for which engineering and work-practice controls are not sufficient to reduce employee exposures to or below the permissible exposure limit.

1910.1025(f)(1)(iii)

Periods when an employee requests a respirator.

1910.1025(f)(2)

Respirator program.

1910.1025(f)(2)(i)

The employer must implement a respiratory protection program in accordance with § 1910.134(b) through (d) (except (d)(1)(iii)), and (f) through (m), which covers each employee required by this section to use a respirator.

1910.1025(f)(2)(ii)

If an employee has breathing difficulty during fit testing or respirator use, the employer must provide the employee with a medical examination in accordance with paragraph (f)(3)(i)(C) of this section to determine whether or not the employee can use a respirator while performing the required duty.

1910.1025(f)(3)

Respirator selection.

1910.1025(f)(3)(i)

Employers must:

1910.1025(f)(3)(i)(A)

Select, and provide to employees, the appropriate respirators specified in paragraph (d)(3)(i)(A) of 29 CFR 1910.134.

1910.1025(f)(3)(ii)(B)

Provide employees with full facepiece respirators instead of half mask respirators for protection against lead aerosols that cause eye or skin irritation at the use concentrations.

1910.1025(f)(3)(ii)(C)

Provide HEPA filters for powered and non-powered air-purifying respirators.

1910.1025(f)(3)(iii)

Employers must provide employees with a powered air-purifying respirator (PAPR) instead of a negative pressure respirator selected according to paragraph (f)(3)(i) of this standard when an employee chooses to use a PAPR and it provides adequate protection to the employee as specified by paragraph (f)(3)(i) of this standard.

1910.1025(g)

Protective work clothing and equipment –

1910.1025(g)(1)

Provision and use. If an employee is exposed to lead above the PEL, without regard to the use of respirators or where the possibility of skin or eye irritation exists, the employer shall provide at no cost to the employee and assure that the employee uses appropriate protective work clothing and equipment such as, but not limited to:

1910.1025(g)(1)(i)

Coveralls or similar full-body work clothing;

1910.1025(g)(1)(ii)

Gloves, hats, and shoes or disposable shoe coverlets; and

1910.1025(g)(1)(iii)

Face shields, vented goggles, or other appropriate protective equipment which complies with 1910.133 of this Part.

1910.1025(g)(2)

Cleaning and replacement.

1910.1025(g)(2)(i)

The employer shall provide the protective clothing required in paragraph (g)(1) of this section in a clean and dry condition at least weekly, and daily to employees whose exposure levels without regard to a respirator are over 200 $\mu g/m^3$ of lead as an 8-hour TWA.

1910.1025(g)(2)(ii)

The employer shall provide for the cleaning, laundering, or disposal of protective clothing and equipment required by paragraph (g)(1) of this section.

1910.1025(g)(2)(iii)

The employer shall repair or replace required protective clothing and equipment as needed to maintain their effectiveness.

1910.1025(g)(2)(iv)

The employer shall assure that all protective clothing is removed at the completion of a work shift only in change rooms provided for that purpose as prescribed in paragraph (i)(2) of this section.

1910.1025(g)(2)(v)

The employer shall assure that contaminated protective clothing which is to be cleaned, laundered, or disposed of, is placed in a closed container in the change-room which prevents dispersion of lead outside the container.

1910.1025(g)(2)(vi)

The employer shall inform in writing any person who cleans or launders protective clothing or equipment of the potentially harmful effects of exposure to lead.

1910.1025(g)(2)(vii)

Labeling of contaminated protective clothing and equipment.

1910.1025(g)(2)(vii)(A)

The employer shall ensure that labels of bags or containers of contaminated protective clothing and equipment include the following information:

DANGER: CLOTHING AND EQUIPMENT CONTAMINATED WITH LEAD. MAY DAMAGE FERTILITY OR THE UNBORN CHILD. CAUSES DAMAGE TO THE CENTRAL NERVOUS SYSTEM. DO NOT EAT, DRINK OR SMOKE WHEN HANDLING. DO NOT REMOVE DUST BY BLOWING OR SHAKING. DISPOSE OF LEAD CONTAMINATED WASH WATER IN ACCORDANCE WITH APPLICABLE LOCAL, STATE, OR FEDERAL REGULATIONS.

1910.1025(g)(2)(viii)(B)

Prior to June 1, 2015, employers may include the following information on bags or containers of contaminated protective clothing and equipment in lieu of the labeling requirements in paragraphs (g)(2)(vii)(A) of this section:

CAUTION: CLOTHING CONTAMINATED WITH LEAD. DO NOT REMOVE DUST BY BLOWING OR SHAKING. DISPOSE OF LEAD CONTAMINATED WASH WATER IN ACCORDANCE WITH APPLICABLE LOCAL, STATE, OR FEDERAL REGULATIONS.

1910.1025(g)(2)(viii)

The employer shall prohibit the removal of lead from protective clothing or equipment by blowing, shaking, or any other means which disperses lead into the air.

1910.1025(h)

Housekeeping -

1910.1025(h)(1)

Surfaces. All surfaces shall be maintained as free as practicable of accumulations of lead.

1910.1025(h)(2)

Cleaning floors.

1910.1025(h)(2)(i)

Floors and other surfaces where lead accumulates may not be cleaned by the use of compressed air.

1910.1025(h)(2)(ii)

Shoveling, dry or wet sweeping, and brushing may be used only where vacuuming or other equally effective methods have been tried and found not to be effective.

1910.1025(h)(3)

Vacuuming. Where vacuuming methods are selected, the vacuums shall be used and emptied in a manner which minimizes the reentry of lead into the workplace.

1910.1025(i)

Hygiene facilities and practices.

1910.1025(i)(1)

The employer shall assure that in areas where employees are exposed to lead above the PEL, without regard to the use of respirators, food or beverage is not present or consumed, tobacco products are not present or used, and cosmetics are not applied, except in change rooms, lunchrooms, and showers required under paragraphs (i)(2) - through (i)(4) of this section.

1910.1025(i)(2)

Change rooms.

1910.1025(i)(2)(i)

The employer shall provide clean change rooms for employees who work in areas where their airborne exposure to lead is above the PEL, without regard to the use of respirators.

1910.1025(i)(2)(ii)

The employer shall assure that change rooms are equipped with separate storage facilities for protective work clothing and equipment and for street clothes which prevent cross-contamination.

1910.1025(i)(3)

Showers.

1910.1025(i)(3)(i)

The employer shall assure that employees who work in areas where their airborne exposure to lead is above the PEL, without regard to the use of respirators, shower at the end of the work shift.

1910.1025(i)(3)(ii)

The employer shall provide shower facilities in accordance with 1910.141 (d)(3) of this part.

1910.1025(j)(3)(iii)

The employer shall assure that employees who are required to shower pursuant to paragraph (j)(3)(i) do not leave the workplace wearing any clothing or equipment worn during the work shift.

1910.1025(j)(4)

Lunchrooms.

1910.1025(j)(4)(i)

The employer shall provide lunchroom facilities for employees who work in areas where their airborne exposure to lead is above the PEL, without regard to the use of respirators.

1910.1025(j)(4)(ii)

The employer shall assure that lunchroom facilities have a temperature controlled, positive pressure, filtered air supply, and are readily accessible to employees.

1910.1025(j)(4)(iii)

The employer shall assure that employees who work in areas where their airborne exposure to lead is above the PEL without regard to the use of a respirator wash their hands and face prior to eating, drinking, smoking or applying cosmetics.

1910.1025(j)(4)(iv)

The employer shall assure that employees do not enter lunchroom facilities with protective work clothing or equipment unless surface lead dust has been removed by vacuuming, down draft booth, or other cleaning method.

1910.1025(j)(5)

Lavatories. The employer shall provide an adequate number of lavatory facilities which comply with 1910.141(d)(1) and (2) of this part.

1910.1025(k)

Medical surveillance -

1910.1025(k)(1)

General.

1910.1025(k)(1)(i)

The employer shall institute a medical surveillance program for all employees who are or may be exposed at or above the action level for more than 30 days per year.

1910.1025(k)(1)(ii)

The employer shall assure that all medical examinations and procedures are performed by or under the supervision of a licensed physician.

1910.1025(k)(1)(iii)

The employer shall provide the required medical surveillance including multiple physician review under paragraph (j)(3)(ii) without cost to employees and at a reasonable time and place.

1910.1025(k)(2)

Biological monitoring -

1910.1025(k)(2)(i)

Blood lead and ZPP level sampling and analysis. The employer shall make available biological monitoring in the form of blood sampling and analysis for lead and zinc protoporphyrin levels to each employee covered under paragraph (k)(1)(i) of this section on the following schedule:

1910.1025(k)(2)(i)(A)

At least every 6 months to each employee covered under paragraph (k)(1)(i) of this section;

1910.1025(k)(2)(i)(B)

At least every two months for each employee whose last blood sampling and analysis indicated a blood lead level at or above 40 ug/100 g of whole blood. This frequency shall continue until two consecutive blood samples and analyses indicate a blood lead level below 40 ug/100 g of whole blood; and

1910.1025(k)(2)(i)(C)

At least monthly during the removal period of each employee removed from exposure to lead due to an elevated blood lead level.

1910.1025(l)(2)(ii)

Follow-up blood sampling tests. Whenever the results of a blood lead level test indicate that an employee's blood lead level is at or above the numerical criterion for medical removal under paragraph (k)(1)(i)(A) of this section, the employer shall provide a second (follow-up) blood sampling test within two weeks after the employer receives the results of the first blood sampling test.

1910.1025(l)(2)(iii)

Accuracy of blood lead level sampling and analysis. Blood lead level sampling and analysis provided pursuant to this section shall have an accuracy (to a confidence level of 95 percent) within plus or minus 15 percent or 6 ug/100 ml, whichever is greater, and shall be conducted by a laboratory licensed by the Center for Disease Control, United States Department of Health, Education and Welfare (CDC) or which has received a satisfactory grade in blood lead proficiency testing from CDC in the prior twelve months.

1910.1025(l)(2)(iv)

Employee notification. Within five working days after the receipt of biological monitoring results, the employer shall notify in writing each employee whose blood lead level is at or above 40 [mu]g/100 g:

1910.1025(l)(2)(iv)(A)

Of that employee's blood lead level; and

1910.1025(l)(2)(iv)(B)

That the standard requires temporary medical removal with Medical Removal Protection benefits when an employee's blood lead level is at or above the numerical criterion for medical removal under paragraph (k)(1)(i) of this section.

1910.1025(l)(3)

Medical examinations and consultations -

1910.1025(l)(3)(i)

Frequency. The employer shall make available medical examinations and consultations to each employee covered under paragraph (l)(1)(i) of this section on the following schedule:

1910.1025(l)(3)(i)(A)

At least annually for each employee for whom a blood sampling test conducted at any time during the preceding 12 months indicated a blood lead level at or above 40 ug/100 g;

1910.1025(l)(3)(i)(B)

Prior to assignment for each employee being assigned for the first time to an area in which airborne concentrations of lead are at or above the action level;

1910.1025(l)(3)(i)(C)

As soon as possible, upon notification by an employee either that the employee has developed signs or symptoms commonly associated with lead intoxication, that the employee desires medical advice concerning the effects of current or past exposure to lead on the employee's ability to procreate a healthy child, or that the employee has demonstrated difficulty in breathing during a respirator fitting test or during use; and

1910.1025(l)(3)(i)(D)

As medically appropriate for each employee either removed from exposure to lead due to a risk of sustaining material impairment to health, or otherwise limited pursuant to a final medical determination.

1910.1025(l)(3)(ii)

Content. Medical examinations made available pursuant to paragraph (l)(3)(i)(A)-(B) of this section shall include the following elements:

1910.1025(l)(3)(ii)(A)

A detailed work history and a medical history, with particular attention to past lead exposure (occupational and non-occupational), personal habits (smoking, hygiene), and past gastrointestinal, hematologic, renal, cardiovascular, reproductive and neurological problems;

1910.1025(l)(3)(ii)(B)

A thorough physical examination, with particular attention to teeth, gums, hematologic, gastrointestinal, renal, cardiovascular, and neurological systems. Pulmonary status should be evaluated if respiratory protection will be used;

1910.1025(l)(3)(ii)(C)

A blood pressure measurement;

1910.1025(l)(3)(ii)(D)

A blood sample and analysis which determines:

1910.1025(j)(3)(ii)(D)(1)

Blood lead level;

1910.1025(j)(3)(ii)(D)(2)

Hemoglobin and hematocrit determinations, red cell indices, and examination of peripheral smear morphology;

1910.1025(j)(3)(ii)(D)(3)

Zinc protoporphyrin;

1910.1025(j)(3)(ii)(D)(4)

Blood urea nitrogen; and,

1910.1025(j)(3)(ii)(D)(5)

Serum creatinine;

1910.1025(j)(3)(ii)(E)

A routine urinalysis with microscopic examination; and

1910.1025(j)(3)(ii)(F)

Any laboratory or other test which the examining physician deems necessary by sound medical practice. The content of medical examinations made available pursuant to paragraph (j)(3)(i)(C) - (D) of this section shall be determined by an examining physician and, if requested by an employee, shall include pregnancy testing or laboratory evaluation of male fertility.

1910.1025(j)(3)(iii)

Multiple physician review mechanism.

1910.1025(j)(3)(iii)(A)

If the employer selects the initial physician who conducts any medical examination or consultation provided to an employee under this section, the employee may designate a second physician:

1910.1025(j)(3)(iii)(A)(1)

To review any findings, determinations or recommendations of the initial physician; and

1910.1025(j)(3)(iii)(A)(2)

To conduct such examinations, consultations, and laboratory tests as the second physician deems necessary to facilitate this review.

1910.1025(j)(3)(iii)(B)

The employer shall promptly notify an employee of the right to seek a second medical opinion after each occasion that an initial physician conducts a medical examination or consultation pursuant to this section. The employer may condition its participation in, and payment for, the multiple physician review mechanism upon the employee doing the following within fifteen (15) days after receipt of the foregoing notification, or receipt of the initial physician's written opinion, whichever is later:

1910.1025(j)(3)(iii)(B)(1)

The employee informing the employer that he or she intends to seek a second medical opinion, and

1910.1025(j)(3)(iii)(B)(2)

The employee initiating steps to make an appointment with a second physician.

1910.1025(j)(3)(iii)(C)

If the findings, determinations or recommendations of the second physician differ from those of the initial physician, then the employer and the employee shall assure that efforts are made for the two physicians to resolve any disagreement.

1910.1025(j)(3)(iii)(D)

If the two physicians have been unable to quickly resolve their disagreement, then the employer and the employee through their respective physicians shall designate a third physician:

1910.1025(j)(3)(iii)(D)(1)

To review any findings, determinations or recommendations of the prior physicians; and

1910.1025(l)(3)(iii)(C)(2)

To conduct such examinations, consultations, laboratory tests and discussions with the prior physicians as the third physician deems necessary to resolve the disagreement of the prior physicians.

1910.1025(l)(3)(iii)(E)

The employer shall act consistent with the findings, determinations and recommendations of the third physician, unless the employer and the employee reach an agreement which is otherwise consistent with the recommendations of at least one of the three physicians.

1910.1025(l)(3)(iv)

Information provided to examining and consulting physicians.

1910.1025(l)(3)(iv)(A)

The employer shall provide an initial physician conducting a medical examination or consultation under this section with the following information:

1910.1025(l)(3)(iv)(A)(1)

A copy of this regulation for lead including all Appendices;

1910.1025(l)(3)(iv)(A)(2)

A description of the affected employee's duties as they relate to the employee's exposure;

1910.1025(l)(3)(iv)(A)(3)

The employee's exposure level or anticipated exposure level to lead and to any other toxic substance (if applicable);

1910.1025(l)(3)(iv)(A)(4)

A description of any personal protective equipment used or to be used;

1910.1025(l)(3)(iv)(A)(5)

Prior blood lead determinations; and

1910.1025(l)(3)(iv)(A)(6)

All prior written medical opinions concerning the employee in the employer's possession or control.

1910.1025(l)(3)(iv)(B)

The employer shall provide the foregoing information to a second or third physician conducting a medical examination or consultation under this section upon request either by the second or third physician, or by the employee.

1910.1025(l)(3)(v)

Written medical opinions.

1910.1025(l)(3)(v)(A)

The employer shall obtain and furnish the employee with a copy of a written medical opinion from each examining or consulting physician which contains the following information:

1910.1025(l)(3)(v)(A)(1)

The physician's opinion as to whether the employee has any detected medical condition which would place the employee at increased risk of material impairment of the employee's health from exposure to lead;

1910.1025(l)(3)(v)(A)(2)

Any recommended special protective measures to be provided to the employee, or limitations to be placed upon the employee's exposure to lead;

1910.1025(l)(3)(v)(A)(3)

Any recommended limitation upon the employee's use of respirators, including a determination of whether the employee can wear a powered air purifying respirator if a physician determines that the employee cannot wear a negative pressure respirator; and

1910.1025(l)(3)(v)(A)(4)

The results of the blood lead determinations.

1910.1025(l)(3)(v)(B)

The employer shall instruct each examining and consulting physician to:

1910.1025(k)(3)(v)(B)(1)

Not reveal either in the written opinion, or in any other means of communication with the employer, findings, including laboratory results, or diagnoses unrelated to an employee's occupational exposure to lead; and

1910.1025(k)(3)(v)(B)(2)

Advise the employee of any medical condition, occupational or nonoccupational, which dictates further medical examination or treatment.

1910.1025(k)(3)(vi)

Alternate Physician Determination Mechanisms. The employer and an employee or authorized employee representative may agree upon the use of any expeditious alternate physician determination mechanism in lieu of the multiple physician review mechanism provided by this paragraph so long as the alternate mechanism otherwise satisfies the requirements contained in this paragraph.

1910.1025(k)(4)

Chelation.

1910.1025(k)(4)(i)

The employer shall assure that any person whom he retains, employs, supervises or controls does not engage in prophylactic chelation of any employee at any time.

1910.1025(k)(4)(ii)

If therapeutic or diagnostic chelation is to be performed by any person in paragraph (k)(4)(i), the employer shall assure that it be done under the supervision of a licensed physician in a clinical setting with thorough and appropriate medical monitoring and that the employee is notified in writing prior to its occurrence.

1910.1025(k)

Medical Removal Protection -

1910.1025(k)(1)

Temporary medical removal and return of an employee -

1910.1025(k)(1)(i)

Temporary removal due to elevated blood lead levels -

1910.1025(k)(1)(i)(A)

The employer shall remove an employee from work having an exposure to lead at or above the action level on each occasion that a periodic and a follow-up blood sampling test conducted pursuant to this section indicate that the employee's blood lead level is at or above 60 ug/100 g of whole blood; and,

1910.1025(k)(1)(i)(B)

The employer shall remove an employee from work having an exposure to lead at or above the action level on each occasion that the average of the last three blood sampling tests conducted pursuant to this section (or the average of all blood sampling tests conducted over the previous six (6) months, whichever is longer) indicates that the employee's blood lead level is at or above 50 (mu)g/100 g of whole blood; provided, however, that an employee need not be removed if the last blood sampling test indicates a blood lead level below 40 (mu)g/100 g of whole blood.

1910.1025(k)(1)(ii)

Temporary removal due to a final medical determination.

1910.1025(k)(1)(ii)(A)

The employer shall remove an employee from work having an exposure to lead at or above the action level on each occasion that a final medical determination results in a medical finding, determination, or opinion that the employee has a detected medical condition which places the employee at increased risk of material impairment to health from exposure to lead.

1910.1025(k)(1)(ii)(B)

For the purposes of this section, the phrase "final medical determination" shall mean the outcome of the multiple physician review mechanism or alternate medical determination mechanism used pursuant to the medical surveillance provisions of this section.

1910.1025(k)(1)(ii)(C)

Where a final medical determination results in any recommended special protective measures for an employee, or limitations on an employee's exposure to lead, the employer shall implement and act consistent with the recommendation.

1910.1025(k)(1)(iii)

Return of the employee to former job status.

1910.1025(k)(1)(iii)(A)

The employer shall return an employee to his or her former job status:

1910.1025(k)(1)(iii)(A)(1)

For an employee removed due to a blood lead level at or above 60 [mu]g/100 g, or due to an average blood lead level at or above 50 [mu]g/100 g, when two consecutive blood sampling tests indicate that the employee's blood lead level is below 40 [mu]g/100 g of whole blood;

1910.1025(k)(1)(iii)(A)(2)

For an employee removed due to a final medical determination, when a subsequent final medical determination results in a medical finding, determination, or opinion that the employee no longer has a detected medical condition which places the employee at increased risk of material impairment to health from exposure to lead.

1910.1025(k)(1)(iii)(B)

For the purposes of this section, the requirement that an employer return an employee to his or her former job status is not intended to expand upon or restrict any rights an employee has or would have had, absent temporary medical removal, to a specific job classification or position under the terms of a collective bargaining agreement.

1910.1025(k)(1)(iv)

Removal of other employee special protective measure or limitations. The employer shall remove any limitations placed on an employee or end any special protective measures provided to an employee pursuant to a final medical determination when a subsequent final medical determination indicates that the limitations or special protective measures are no longer necessary.

1910.1025(k)(1)(v)

Employer options pending a final medical determination. Where the multiple physician review mechanism, or alternate medical determination mechanism used pursuant to the medical surveillance provisions of this section, has not yet resulted in a final medical determination with respect to an employee, the employer shall act as follows:

1910.1025(k)(1)(v)(A)

Removal. The employer may remove the employee from exposure to lead, provide special protective measures to the employee, or place limitations upon the employee, consistent with the medical findings, determinations, or recommendations of any of the physicians who have reviewed the employee's health status.

1910.1025(k)(1)(v)(B)

Return. The employer may return the employee to his or her former job status, and any special protective measures provided to the employee, and remove any limitations placed upon the employee, consistent with the medical findings, determinations, or recommendations of any of the physicians who have reviewed the employee's health status, with two exceptions. If -

1910.1025(k)(1)(v)(B)(1)

the initial removal, special protection, or limitation of the employee resulted from a final medical determination which differed from the findings, determinations, or recommendations of the initial physician or

1910.1025(k)(1)(v)(B)(2)

The employee has been on removal status for the preceding eighteen months due to an elevated blood lead level, then the employer shall await a final medical determination.

1910.1025(k)(2)

Medical removal protection benefits -

1910.1025(k)(2)(i)

Provision of medical removal protection benefits. The employer shall provide to an employee up to eighteen (18) months of medical removal protection benefits on each occasion that an employee is removed from exposure to lead or otherwise limited pursuant to this section.

1910.1025(k)(2)(ii)

Definition of medical removal protection benefits. For the purposes of this section, the requirement that an employer provide medical removal protection benefits means that the employer shall maintain the earnings, seniority and other employment rights and benefits of an employee as though the employee had not been removed from normal exposure to lead or otherwise limited.

1910.1025(k)(2)(iii)

Follow-up medical surveillance during the period of employee removal or limitation. During the period of time that an employee is removed from normal exposure to lead or otherwise limited, the employer may condition the provision of medical removal protection benefits upon the employee's participation in follow-up medical surveillance made available pursuant to this section.

1910.1025(k)(2)(iv)

Workers' compensation claims. If a removed employee files a claim for workers' compensation payments for a lead-related disability, then the employer shall continue to provide medical removal protection benefits pending disposition of the claim. To the extent that an award is made to the employee for earnings lost during the period of removal, the employer's medical removal protection obligation shall be reduced by such amount. The employer shall receive no credit for workers' compensation payments received by the employee for treatment related expenses.

1910.1025(k)(2)(v)

Other credits. The employer's obligation to provide medical removal protection benefits to a removed employee shall be reduced to the extent that the employee receives compensation for earnings lost during the period of removal either from a publicly or employer-funded compensation program, or receives income from employment with another employer made possible by virtue of the employee's removal.

1910.1025(k)(3)(i)

Employees whose blood lead levels do not adequately decline within 18 months of removal. The employer shall take the following measures with respect to any employee removed from exposure to lead due to an elevated blood lead level whose blood lead level has not declined within the past eighteen (18) months of removal so that the employee has been returned to his or her former job status:

1910.1025(k)(3)(i)(A)

The employer shall make available to the employee a medical examination pursuant to this section to obtain a final medical determination with respect to the employee;

1910.1025(k)(3)(i)(B)

The employer shall assure that the final medical determination obtained indicates whether or not the employee may be returned to his or her former job status, and if not, what steps should be taken to protect the employee's health;

1910.1025(k)(3)(i)(C)

Where the final medical determination has not yet been obtained, or once obtained indicates that the employee may not yet be returned to his or her former job status, the employer shall continue to provide medical removal protection benefits to the employee until either the employee is returned to former job status, or a final medical determination is made that the employee is incapable of ever safely returning to his or her former job status.

1910.1025(k)(3)(i)(D)

Where the employer acts pursuant to a final medical determination which permits the return of the employee to his or her former job status despite what would otherwise be an unacceptable blood lead level, later questions concerning removing the employee again shall be decided by a final medical determination. The employer need not automatically remove such an employee pursuant to the blood lead level removal criteria provided by this section.

1910.1025(k)(3)(ii)

Voluntary Removal or Restriction of An Employee. Where an employer, although not required by this section to do so, removes an employee from exposure to lead or otherwise places limitations on an employee due to the effects of lead exposure on the employee's medical condition, the employer shall provide medical removal protection benefits to the employee equal to that required by paragraph (k)(2)(i) of this section.

1910.1025(l)

Employee information and training -

1910.1025(l)(1)

Training program.

1910.1025(l)(1)(i)

Each employer who has a workplace in which there is a potential exposure to airborne lead at any level shall inform employees of the content of Appendices A and B of this regulation.

1910.1025(l)(1)(ii)

The employer shall train each employee who is subject to exposure to lead at or above the action level, or for whom the possibility of skin or eye irritation exists, in accordance with the requirements of this section. The employer shall institute a training program and ensure employee participation in the program.

1910.1025(l)(1)(iii)

The employer shall provide initial training by 180 days from the effective date for those employees covered by paragraph (l)(1)(ii) on the standard's effective date and prior to the time of initial job assignment for those employees subsequently covered by this paragraph.

1910.1025(l)(1)(iv)

The training program shall be repeated at least annually for each employee.

1910.1025(l)(2)(v)

The employer shall assure that each employee is informed of the following:

1910.1025(l)(2)(v)(A)

The content of this standard and its appendices;

1910.1025(l)(2)(v)(B)

The specific nature of the operations which could result in exposure to lead above the action level;

1910.1025(l)(2)(v)(C)

The purpose, proper selection, fitting, use, and limitations of respirators;

1910.1025(l)(2)(v)(D)

The purpose and a description of the medical surveillance program, and the medical removal protection program including information concerning the adverse health effects associated with excessive exposure to lead (with particular attention to the adverse reproductive effects on both males and females);

1910.1025(l)(2)(v)(E)

The engineering controls and work practices associated with the employee's job assignment;

1910.1025(l)(2)(v)(F)

The contents of any compliance plan in effect; and

1910.1025(l)(2)(v)(G)

Instructions to employees that chelating agents should not routinely be used to remove lead from their bodies and should not be used at all except under the direction of a licensed physician;

1910.1025(l)(2)

Access to information and training materials.

1910.1025(l)(2)(i)

The employer shall make readily available to all affected employees a copy of this standard and its appendices.

1910.1025(l)(2)(ii)

The employer shall provide, upon request, all materials relating to the employee information and training program to the Assistant Secretary and the Director.

1910.1025(l)(2)(iii)

In addition to the information required by paragraph (l)(1)(v), the employer shall include as part of the training program, and shall distribute to employees, any materials pertaining to the Occupational Safety and Health Act, the regulations issued pursuant to that Act, and this lead standard, which are made available to the employer by the Assistant Secretary.

1910.1025(m)

Communication of hazards—

1910.1025(m)(1)

Hazard communication—general.

1910.1025(m)(1)(i)

Chemical manufacturers, importers, distributors and employers shall comply with all requirements of the Hazard Communication Standard (HCS) (§ 1910.1200) for lead.

1910.1025(m)(1)(ii)

In classifying the hazards of lead at least the following hazards are to be addressed: Reproductive/developmental toxicity; central nervous system effects; kidney effects; blood effects; and acute toxicity effects.

1910.1025(m)(1)(iii)

Employers shall include lead in the hazard communication program established to comply with the HCS (§ 1910.1200). Employers shall ensure that each employee has access to labels on containers of lead and to safety data sheets, and is trained in accordance with the requirements of HCS and paragraph (l) of this section.

1910.1025(m)(2)

Signs.

1910.1025(m)(2)(i)

The employer shall post the following warning signs in each work area where the PEL is exceeded:

DANGER
LEAD
MAY DAMAGE FERTILITY OR THE UNBORN CHILD
CAUSES DAMAGE TO THE CENTRAL NERVOUS SYSTEM
DO NOT EAT, DRINK OR SMOKE IN THIS AREA

1910.1025(m)(2)(ii)

The employer shall ensure that no statement appears on or near any sign required by this paragraph (m)(2) which contradicts or detracts from the meaning of the required sign.

1910.1025(m)(2)(iii)

The employer shall ensure that signs required by this paragraph (m)(2) are illuminated and cleaned as necessary so that the legend is readily visible.

1910.1025(m)(2)(iv)

The employer may use signs required by other statutes, regulations, or ordinances in addition to, or in combination with, signs required by this paragraph (m)(2).

1910.1025(m)(2)(v)

Prior to June 1, 2016, employers may use the following legend in lieu of that specified in paragraph (m)(2)(i) of this section:

WARNING
LEAD WORK AREA
POISON
NO SMOKING OR EATING

1910.1025(n)

Recordkeeping -

1910.1025(n)(1)

Exposure monitoring.

1910.1025(n)(1)(i)

The employer shall establish and maintain an accurate record of all monitoring required in paragraph (d) of this section.

1910.1025(n)(1)(ii)

This record shall include:

1910.1025(n)(1)(ii)(A)

The date(s), number, duration, location and results of each of the samples taken, including a description of the sampling procedure used to determine representative employee exposure where applicable;

1910.1025(n)(1)(ii)(B)

A description of the sampling and analytical methods used and evidence of their accuracy;

1910.1025(n)(1)(ii)(C)

The type of respiratory protective devices worn, if any;

1910.1025(n)(1)(ii)(D)

Name, social security number, and job classification of the employee monitored and of all other employees whose exposure the measurement is intended to represent; and

1910.1025(n)(1)(ii)(E)

The environmental variables that could affect the measurement of employee exposure.

1910.1025(n)(1)(iii)

The employer shall maintain these monitoring records for at least 40 years or for the duration of employment plus 20 years, whichever is longer.

1910.1025(n)(2)

Medical surveillance.

1910.1025(n)(2)(i)

The employer shall establish and maintain an accurate record for each employee subject to medical surveillance as required by paragraph (j) of this section.

1910.1025(n)(2)(ii)

This record shall include:

1910.1025(n)(2)(ii)(A)

The name, social security number, and description of the duties of the employee;

1910.1025(n)(2)(ii)(B)

A copy of the physician's written opinions;

1910.1025(n)(2)(ii)(C)

Results of any airborne exposure monitoring done for that employee and the representative exposure levels supplied to the physician; and

1910.1025(n)(2)(ii)(D)

Any employee medical complaints related to exposure to lead.

1910.1025(n)(3)(ii)

The employer shall keep, or assure that the examining physician keeps, the following medical records:

1910.1025(n)(3)(ii)(A)

A copy of the medical examination results including medical and work history required under paragraph (j) of this section;

1910.1025(n)(3)(ii)(B)

A description of the laboratory procedures and a copy of any standards or guidelines used to interpret the test results or references to that information;

1910.1025(n)(3)(ii)(C)

A copy of the results of biological monitoring.

1910.1025(n)(3)(iv)

The employer shall maintain or assure that the physician maintains those medical records for at least 40 years, or for the duration of employment plus 20 years, whichever is longer.

1910.1025(n)(3)

Medical removals.

1910.1025(n)(3)(i)

The employer shall establish and maintain an accurate record for each employee removed from current exposure to lead pursuant to paragraph (k) of this section.

1910.1025(n)(3)(ii)

Each record shall include:

1910.1025(n)(3)(ii)(A)

The name and social security number of the employee;

1910.1025(n)(3)(ii)(B)

The date on each occasion that the employee was removed from current exposure to lead as well as the corresponding date on which the employee was returned to his or her former job status;

1910.1025(n)(3)(ii)(C)

A brief explanation of how each removal was or is being accomplished; and

1910.1025(n)(3)(ii)(D)

A statement with respect to each removal indicating whether or not the reason for the removal was an elevated blood lead level.

1910.1025(n)(3)(iii)

The employer shall maintain each medical removal record for at least the duration of an employee's employment.

1910.1025(n)(4)

Availability.

1910.1025(n)(4)(i)

The employer shall make available upon request all records required to be maintained by paragraph (n) of this section to the Assistant Secretary and the Director for examination and copying.

1910.1025(n)(4)(ii)

Environmental monitoring, medical removal, and medical records required by this paragraph shall be provided upon request to employees, designated representatives, and the Assistant Secretary in accordance with 29 CFR 1910.1020 (a)-(e) and (2)-(i). Medical removal records shall be provided in the same manner as environmental monitoring records.

1910.1025(n)(5)

Transfer of records.

1910.1025(n)(5)(i)

Whenever the employer ceases to do business, the successor employer shall receive and retain all records required to be maintained by paragraph (n) of this section.

1910.1025(n)(5)(ii)

The employer shall also comply with any additional requirements involving transfer of records set forth in 29 CFR 1910.1020 (h).

1910.1025(o)

Observation of monitoring.

1910.1025(o)(1)

Employee observation. The employer shall provide affected employees or their designated representatives an opportunity to observe any monitoring of employee exposure to lead conducted pursuant to paragraph (d) of this section.

1910.1025(o)(2)

Observation procedures.

1910.1025(o)(2)(i)

Whenever observation of the monitoring of employee exposure to lead requires entry into an area where the use of respirators, protective clothing or equipment is required, the employer shall provide the observer with and assure the use of such respirators, clothing and such equipment, and shall require the observer to comply with all other applicable safety and health procedures.

1910.1025(o)(2)(ii)

Without interfering with the monitoring, observers shall be entitled to:

1910.1025(o)(2)(ii)(A)

Receive an explanation of the measurement procedures;

1910.1025(o)(2)(ii)(B)

Observe all steps related to the monitoring of lead performed at the place of exposure; and

1910.1025(o)(2)(ii)(C)

Record the results obtained or receive copies of the results when returned by the laboratory.

1910.1025(p)

Appendices. The information contained in the appendices to this section is not intended by itself, to create any additional obligations not otherwise imposed by this standard nor detract from any existing obligation.

[60 FR 52958, Oct. 11, 1995; 61 FR 5507, Feb. 13, 1996; 63 FR 1152, Jan. 8, 1998; 63 FR 20098, April 23, 1998; 70 FR 1142, Jan. 5, 2005; 71 FR 16672 and 16673, April 3, 2006; 71 FR 50169, August 24, 2006; 73 FR 75585, Dec. 12, 2008; 76 FR 33608, June 8, 2011; 76 FR 80740, Dec. 27, 2011; 77 FR 17780, March 26, 2012]

Next Standard (1910.1025 App A)

Electronic Code of Federal Regulations
e-CFR

e-CFR Data is current as of October 30, 2009

Title 29: Labor
PART 1926—SAFETY AND HEALTH REGULATIONS FOR CONSTRUCTION
Subpart D—Occupational Health and Environmental Controls

Browse Previous | Browse Next

§ 1926.62 Lead.

(a) *Scope.* This section applies to all construction work where an employee may be occupationally exposed to lead. All construction work excluded from coverage in the general industry standard for lead by 29 CFR 1910.1025(a)(2) is covered by this standard. Construction work is defined as work for construction, alteration and/or repair, including painting and decorating. It includes but is not limited to the following:

(1) Demolition or salvage of structures where lead or materials containing lead are present;

(2) Removal or encapsulation of materials containing lead;

(3) New construction, alteration, repair, or renovation of structures, substrates, or portions thereof, that contain lead, or materials containing lead;

(4) Installation of products containing lead;

(5) Lead contamination/emergency cleanup;

(6) Transportation, disposal, storage, or containment of lead or materials containing lead on the site or location at which construction activities are performed, and

(7) Maintenance operations associated with the construction activities described in this paragraph.

(b) *Definitions.*

Action level means employee exposure, without regard to the use of respirators, to an airborne concentration of lead of 30 micrograms per cubic meter of air (30 $\mu g/m^3$) calculated as an 8-hour time-weighted average (TWA).

Assistant Secretary means the Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, or designee.

Competent person means one who is capable of identifying existing and predictable lead hazards in the surroundings or working conditions and who has authorization to take prompt corrective measures to eliminate them.

Director means the Director, National Institute for Occupational Safety and Health (NIOSH), U.S. Department of Health and Human Services, or designee.

Lead means metallic lead, all inorganic lead compounds, and organic lead soaps. Excluded from this

definition are all other organic lead compounds.

This section means this standard.

(c) *Permissible exposure limit.* (1) The employer shall assure that no employee is exposed to lead at concentrations greater than fifty micrograms per cubic meter of air (50 $\mu g/m^3$) averaged over an 8-hour period.

(2) If an employee is exposed to lead for more than 8 hours in any work day the employees' allowable exposure, as a time weighted average (TWA) for that day, shall be reduced according to the following formula:

Allowable employee exposure (in $\mu g/m^3$) = 400 divided by hours worked in the day.

(3) When respirators are used to limit employee exposure as required under paragraph (c) of this section and all the requirements of paragraphs (e)(1) and (f) of this section have been met, employee exposure may be considered to be at the level provided by the protection factor of the respirator for those periods the respirator is worn. Those periods may be averaged with exposure levels during periods when respirators are not worn to determine the employee's daily TWA exposure.

(d) *Exposure assessment*—(1) *General.* (i) Each employer who has a workplace or operation covered by this standard shall initially determine if any employee may be exposed to lead at or above the action level.

(ii) For the purposes of paragraph (d) of this section, employee exposure is that exposure which would occur if the employee were not using a respirator.

(iii) With the exception of monitoring under paragraph (d)(3), where monitoring is required under this section, the employer shall collect personal samples representative of a full shift including at least one sample for each job classification in each work area either for each shift or for the shift with the highest exposure level.

(iv) Full shift personal samples shall be representative of the monitored employee's regular, daily exposure to lead.

(2) *Protection of employees during assessment of exposure.* (i) With respect to the lead related tasks listed in paragraph (d)(2)(i) of this section, where lead is present, until the employer performs an employee exposure assessment as required in paragraph (d) of this section and documents that the employee performing any of the listed tasks is not exposed above the PEL, the employer shall treat the employee as if the employee were exposed above the PEL, and not in excess of ten (10) times the PEL, and shall implement employee protective measures prescribed in paragraph (d)(2)(v) of this section. The tasks covered by this requirement are:

(A) Where lead containing coatings or paint are present: Manual demolition of structures (e.g, dry wall), manual scraping, manual sanding, heat gun applications, and power tool cleaning with dust collection systems;

(B) Spray painting with lead paint.

(ii) In addition, with regard to tasks not listed in paragraph (d)(2)(i), where the employee has any reason to believe that an employee performing the task may be exposed to lead in excess of the PEL, until the employer performs an employee exposure assessment as required by paragraph (d) of this section and documents that the employee's lead exposure is not above the PEL the employer shall treat the employee as if the employee were exposed above the PEL and shall implement employee protective measures as prescribed in paragraph (d)(2)(v) of this section.

(iii) With respect to the tasks listed in paragraph (d)(2)(ii) of this section, where lead is present, until the employer performs an employee exposure assessment as required in paragraph (d) of this section, and documents that the employee performing any of the listed tasks is not exposed in excess of 500 $\mu g/m^3$, the employer shall treat the employee as if the employee were exposed to lead in excess of 500 $\mu g/m^3$ and shall implement employee protective measures as prescribed in paragraph (d)(2)(v) of this section. Where the employer does establish that the employee is exposed to levels of lead below 500 $\mu g/m^3$, the

(2) *Change areas.* (i) The employer shall provide clean change areas for employees whose airborne exposure to lead is above the PEL, and as interim protection for employees performing tasks as specified in paragraph (d)(2) of this section, without regard to the use of respirators.

(ii) The employer shall assure that change areas are equipped with separate storage facilities for protective work clothing and equipment and for street clothes which prevent cross-contamination.

(iii) The employer shall assure that employees do not leave the workplace wearing any protective clothing or equipment that is required to be worn during the work shift.

(3) *Showers.* (i) The employer shall provide shower facilities, where feasible, for use by employees whose airborne exposure to lead is above the PEL.

(ii) The employer shall assure, where shower facilities are available, that employees shower at the end of the work shift and shall provide an adequate supply of cleansing agents and towels for use by affected employees.

(4) *Eating facilities.* (i) The employer shall provide lunchroom facilities or eating areas for employees whose airborne exposure to lead is above the PEL, without regard to the use of respirators.

(ii) The employer shall assure that lunchroom facilities or eating areas are as free as practicable from lead contamination and are readily accessible to employees.

(iii) The employer shall assure that employees whose airborne exposure to lead is above the PEL, without regard to the use of a respirator, wash their hands and face prior to eating, drinking, smoking or applying cosmetics.

(iv) The employer shall assure that employees do not enter lunchroom facilities or eating areas with protective work clothing or equipment unless surface lead dust has been removed by vacuuming, downdraft booth, or other cleaning method that limits dispersion of lead dust.

(5) *Hand washing facilities.* (i) The employer shall provide adequate handwashing facilities for use by employees exposed to lead in accordance with 29 CFR 1926.51(f).

(ii) Where showers are not provided the employer shall assure that employees wash their hands and face at the end of the work-shift.

(j) *Medical surveillance*—(1) *General.* (i) The employer shall make available initial medical surveillance to employees occupationally exposed on any day to lead at or above the action level. Initial medical surveillance consists of biological monitoring in the form of blood sampling and analysis for lead and zinc protoporphyrin levels.

(ii) The employer shall institute a medical surveillance program in accordance with paragraphs (j)(2) and (j)(3) of this section for all employees who are or may be exposed by the employer at or above the action level for more than 30 days in any consecutive 12 months;

(iii) The employer shall assure that all medical examinations and procedures are performed by or under the supervision of a licensed physician.

(iv) The employer shall make available the required medical surveillance including multiple physician review under paragraph (j)(3)(iii) without cost to employees and at a reasonable time and place.

(2) *Biological monitoring*—(i) *Blood lead and ZPP level sampling and analysis.* The employer shall make available biological monitoring in the form of blood sampling and analysis for lead and zinc protoporphyrin levels to each employee covered under paragraphs (j)(1)(i) and (ii) of this section on the following schedule:

(A) For each employee covered under paragraph (j)(1)(ii) of this section, at least every 2 months for the first 6 months and every 6 months thereafter;

(B) For each employee covered under paragraphs (j)(1) (i) or (ii) of this section whose last blood sampling and analysis indicated a blood lead level at or above 40 µg/dl, at least every two months. This

frequency shall continue until two consecutive blood samples and analyses indicate a blood lead level below 40 µg/dl; and

(C) For each employee who is removed from exposure to lead due to an elevated blood lead level at least monthly during the removal period.

(ii) *Follow-up blood sampling tests.* Whenever the results of a blood lead level test indicate that an employee's blood lead level exceeds the numerical criterion for medical removal under paragraph (k)(1)(i) of this section, the employer shall provide a second (follow-up) blood sampling test within two weeks after the employer receives the results of the first blood sampling test.

(iii) *Accuracy of blood lead level sampling and analysis.* Blood lead level sampling and analysis provided pursuant to this section shall have an accuracy (to a confidence level of 95 percent) within plus or minus 15 percent or 6 µg/dl, whichever is greater, and shall be conducted by a laboratory approved by OSHA.

(iv) *Employee notification.* (A) Within five working days after the receipt of biological monitoring results, the employer shall notify each employee in writing of his or her blood lead level; and

(B) The employer shall notify each employee whose blood lead level exceeds 40 µg/dl that the standard requires temporary medical removal with Medical Removal Protection benefits when an employee's blood lead level exceeds the numerical criterion for medical removal under paragraph (k)(1)(i) of this section.

(3) *Medical examinations and consultations*—(i) *Frequency.* The employer shall make available medical examinations and consultations to each employee covered under paragraph (j)(1)(ii) of this section on the following schedule:

(A) At least annually for each employee for whom a blood sampling test conducted at any time during the preceding 12 months indicated a blood lead level at or above 40 µg/dl;

(B) As soon as possible, upon notification by an employee either that the employee has developed signs or symptoms commonly associated with lead intoxication, that the employee desires medical advice concerning the effects of current or past exposure to lead on the employee's ability to procreate a healthy child, that the employee is pregnant, or that the employee has demonstrated difficulty in breathing during a respirator fitting test or during use; and

(C) As medically appropriate for each employee either removed from exposure to lead due to a risk of sustaining material impairment to health, or otherwise limited pursuant to a final medical determination.

(ii) *Content.* The content of medical examinations made available pursuant to paragraph (j)(3)(i)(B)–(C) of this section shall be determined by an examining physician and, if requested by an employee, shall include pregnancy testing or laboratory evaluation of male fertility. Medical examinations made available pursuant to paragraph (j)(3)(i)(A) of this section shall include the following elements:

(A) A detailed work history and a medical history, with particular attention to past lead exposure (occupational and non-occupational), personal habits (smoking, hygiene), and past gastrointestinal, hematologic, renal, cardiovascular, reproductive and neurological problems;

(B) A thorough physical examination, with particular attention to teeth, gums, hematologic, gastrointestinal, renal, cardiovascular, and neurological systems. Pulmonary status should be evaluated if respiratory protection will be used;

(C) A blood pressure measurement;

(D) A blood sample and analysis which determines:

(*1*) Blood lead level;

(*2*) Hemoglobin and hematocrit determinations, red cell indices, and examination of peripheral smear morphology;

(*3*) Zinc protoporphyrin;

respirators including skin rash where the facepiece makes contact with the skin, unacceptable stress to breathing in some workers with underlying cardiopulmonary impairment, difficulty in providing adequate fit, the tendency for respirators to create additional hazards by interfering with vision, hearing, and mobility, and the difficulties of assuring the maximum effectiveness of a complicated work practice program involving respirators. Respirators do, however, serve a useful function where engineering and work practice controls are inadequate by providing supplementary, interim, or short-term protection, provided they are properly selected for the environment in which the employee will be working, properly fitted to the employee, maintained and cleaned periodically, and worn by the employee when required.

In its interim final standard on occupational exposure to inorganic lead in the construction industry, OSHA has prohibited prophylactic chelation. Diagnostic and therapeutic chelation are permitted only under the supervision of a licensed physician with appropriate medical monitoring in an acceptable clinical setting. The decision to initiate chelation therapy must be made on an individual basis and take into account the severity of symptoms felt to be a result of lead toxicity along with blood lead levels, ZPP levels, and other laboratory tests as appropriate. EDTA and penicillamine which are the primary chelating agents used in the therapy of occupational lead poisoning have significant potential side effects and their use must be justified on the basis of expected benefits to the worker. Unless frank and severe symptoms are present, therapeutic chelation is not recommended, given the opportunity to remove a worker from exposure and allow the body to naturally excrete accumulated lead. As a diagnostic aid, the chelation mobilization test using CA-EDTA has limited applicability. According to some investigators, the test can differentiate between lead-induced and other nephropathies. The test may also provide an estimation of the mobile fraction of the total body lead burden.

Employers are required to assure that accurate records are maintained on exposure assessment, including environmental monitoring, medical surveillance, and medical removal for each employee. Exposure assessment records must be kept for at least 30 years. Medical surveillance records must be kept for the duration of employment plus 30 years except in cases where the employment was less than one year. If duration of employment is less than one year, the employer need not retain this record beyond the term of employment if the record is provided to the employee upon termination of employment. Medical removal records also must be maintained for the duration of employment. All records required under the standard must be made available upon request to the Assistant Secretary of Labor for Occupational Safety and Health and the Director of the National Institute for Occupational Safety and Health. Employers must also make environmental and biological monitoring and medical removal records available to affected employees and to former employees or their authorized employee representatives. Employees or their specifically designated representatives have access to their entire medical surveillance records.

In addition, the standard requires that the employer inform all workers exposed to lead at or above 30 $\mu g/m^3$ of the provisions of the standard and all its appendices, the purpose and description of medical surveillance and provisions for medical removal protection if temporary removal is required. An understanding of the potential health effects of lead exposure by all exposed employees along with full understanding of their rights under the lead standard is essential for an effective monitoring program.

II. Adverse Health Effects of Inorganic Lead

Although the toxicity of lead has been known for 2,000 years, the knowledge of the complex relationship between lead exposure and human response is still being refined. Significant research into the toxic properties of lead continues throughout the world, and it should be anticipated that our understanding of thresholds of effects and margins of safety will be improved in future years. The provisions of the lead standard are founded on two prime medical judgments: First, the prevention of adverse health effects from exposure to lead throughout a working lifetime requires that worker blood lead levels be maintained at or below 40 µg/dl and second, the blood lead levels of workers, male or female, who intend to parent in the near future should be maintained below 30 µg/dl to minimize adverse reproductive health effects to the parents and developing fetus. The adverse effects of lead on reproduction are being actively researched and OSHA encourages the physician to remain abreast of recent

developments in the area to best advise pregnant workers or workers planning to conceive children.

The spectrum of health effects caused by lead exposure can be subdivided into five developmental stages: Normal, physiological changes of uncertain significance, pathophysiological changes, overt symptoms (morbidity), and mortality. Within this process there are no sharp distinctions, but rather a continuum of effects. Boundaries between categories overlap due to the wide variation of individual responses and exposures in the working population. OSHA's development of the lead standard focused on pathophysiological changes as well as later stages of disease.

1. Heme Synthesis Inhibition: The earliest demonstrated effect of lead involves its ability to inhibit at least two enzymes of the heme synthesis pathway at very low blood levels. Inhibition of delta aminolevulinic acid dehydrase (ALA-D) which catalyzes the conversion of delta-aminolevulinic acid (ALA) to protoporphyrin is observed at a blood lead level below 20 µg/dl. At a blood lead level of 40 µg/dl, more than 20% of the population would have 70% inhibition of ALA-D. There is an exponential increase in ALA excretion at blood lead levels greater than 40 µg/dl.

Another enzyme, ferrochelatase, is also inhibited at low blood lead levels. Inhibition of ferrochelatase leads to increased free erythrocyte protoporphyrin (FEP) in the blood which can then bind to zinc to yield zinc protoporphyrin. At a blood lead level of 50 µg/dl or greater, nearly 100% of the population will have an increase in FEP. There is also an exponential relationship between blood lead levels greater than 40 µg/dl and the associated ZPP level, which has led to the development of the ZPP screening test for lead exposure.

While the significance of these effects is subject to debate, it is OSHA's position that these enzyme disturbances are early stages of a disease process which may eventually result in the clinical symptoms of lead poisoning. Whether or not the effects do progress to the later stages of clinical disease, disruption of these enzyme processes over a working lifetime is considered to be a material impairment of health.

One of the eventual results of lead-induced inhibition of enzymes in the heme synthesis pathway is anemia which can be asymptomatic if mild but associated with a wide array of symptoms including dizziness, fatigue, and tachycardia when more severe. Studies have indicated that lead levels as low as 50 µg/dl can be associated with a definite decreased hemoglobin, although most cases of lead-induced anemia, as well as shortened red-cell survival times, occur at lead levels exceeding 80 µg/dl. Inhibited hemoglobin synthesis is more common in chronic cases whereas shortened erythrocyte life span is more common in acute cases.

In lead-induced anemias, there is usually a reticulocytosis along with the presence of basophilic stippling, and ringed sideroblasts, although none of the above are pathognomonic for lead-induced anemia.

2. Neurological Effects. Inorganic lead has been found to have toxic effects on both the central and peripheral nervous systems. The earliest stages of lead-induced central nervous system effects first manifest themselves in the form of behavioral disturbances and central nervous system symptoms including irritability, restlessness, insomnia and other sleep disturbances, fatigue, vertigo, headache, poor memory, tremor, depression, and apathy. With more severe exposure, symptoms can progress to drowsiness, stupor, hallucinations, delirium, convulsions and coma.

The most severe and acute form of lead poisoning which usually follows ingestion or inhalation of large amounts of lead is acute encephalopathy which may arise precipitously with the onset of intractable seizures, coma, cardiorespiratory arrest, and death within 48 hours.

While there is disagreement about what exposure levels are needed to produce the earliest symptoms, most experts agree that symptoms definitely can occur at blood lead levels of 60

µg/dl whole blood and therefore recommend a 40 µg/dl maximum. The central nervous system effects frequently are not reversible following discontinued exposure or chelation therapy and when improvement does occur, it is almost always only partial.

The peripheral neuropathy resulting from lead exposure characteristically involves only motor function with minimal sensory damage and has a marked predilection for the extensor muscles of the most active extremity. The peripheral neuropathy can occur with varying degrees of severity. The earliest and mildest form which can be detected in workers with blood lead levels as low as 50 µg/dl is manifested by slowing of motor nerve conduction velocity often without clinical symptoms. With progression of the neuropathy there is development of painless extensor muscle weakness usually involving the extensor muscles of the fingers and hand in the most active upper extremity, followed in severe cases by wrist drop or, much less commonly, foot drop.

In addition to slowing of nerve conduction, electromyographical studies in patients with blood lead levels greater than 50 µg/dl have demonstrated a decrease in the number of acting motor unit potentials, an increase in the duration of motor unit potentials, and spontaneous pathological activity including fibrillations and fasciculations. Whether these effects occur at levels of 40 µg/dl is undetermined.

While the peripheral neuropathies can occasionally be reversed with therapy, again such recovery is not assured particularly in the more severe neuropathies and often improvement is only partial. The lack of reversibility is felt to be due in part to segmental demyelination.

3. Gastrointestinal. Lead may also affect the gastrointestinal system producing abdominal colic or diffuse abdominal pain, constipation, obstipation, diarrhea, anorexia, nausea and vomiting. Lead colic rarely develops at blood lead levels below 80 µg/dl.

4. Renal. Renal toxicity represents one of the most serious health effects of lead poisoning. In the early stages of disease nuclear inclusion bodies can frequently be identified in proximal renal tubular cells. Renal function remains normal and the changes in this stage are probably reversible. With more advanced disease there is progressive interstitial fibrosis and impaired renal function. Eventually extensive interstitial fibrosis ensues with sclerotic glomeruli and dilated and atrophied proximal tubules; all represent end stage kidney disease. Azotemia can be progressive, eventually resulting in frank uremia necessitating dialysis. There is occasionally associated hypertension and hyperuricemia with or without gout.

Early kidney disease is difficult to detect. The urinalysis is normal in early lead nephropathy and the blood urea nitrogen and serum creatinine increase only when two-thirds of kidney function is lost. Measurement of creatinine clearance can often detect earlier disease as can other methods of measurement of glomerular filtration rate. An abnormal Ca-EDTA mobilization test has been used to differentiate between lead-induced and other nephropathies, but this procedure is not widely accepted. A form of Fanconi syndrome with aminoaciduria, glycosuria, and hyperphosphaturia indicating severe injury to the proximal renal tubules is occasionally seen in children.

5. Reproductive effects. Exposure to lead can have serious effects on reproductive function in both males and females. In male workers exposed to lead there can be a decrease in sexual drive, impotence, decreased ability to produce healthy sperm, and sterility. Malformed sperm (teratospermia), decreased number of sperm (hypospermia), and sperm with decreased motility (asthenospermia) can all occur. Teratospermia has been noted at mean blood lead levels of 53 µg/dl and hypospermia and asthenospermia at 41 µg/dl. Furthermore, there appears to be a dose-response relationship for teratospermia in lead exposed workers.

Women exposed to lead may experience menstrual disturbances including dysmenorrhea, menorrhagia and amenorrhea. Following exposure to lead, women have a higher frequency of sterility, premature births, spontaneous miscarriages, and stillbirths.

Germ cells can be affected by lead and cause genetic damage in the egg or sperm cells before conception and result in failure to implant, miscarriage, stillbirth, or birth defects.

Infants of mothers with lead poisoning have a higher mortality during the first year and suffer from lowered birth weights, slower growth, and nervous system disorders.

Lead can pass through the placental barrier and lead levels in the mother's blood are comparable to concentrations of lead in the umbilical cord at birth. Transplacental passage becomes detectable at 12–14 weeks of gestation and increases until birth.

There is little direct data on damage to the fetus from exposure to lead but it is generally assumed that the fetus and newborn would be at least as susceptible to neurological damage as young children. Blood lead levels of 50–60 µg/dl in children can cause significant neurobehavioral impairments and there is evidence of hyperactivity at blood levels as low as 25 µg/dl. Given the overall body of literature concerning the adverse health effects of lead in children, OSHA feels that the blood lead level in children should be maintained below 30 µg/dl with a population mean of 15 µg/dl. Blood lead levels in the fetus and newborn likewise should not exceed 30 µg/dl.

Because of lead's ability to pass through the placental barrier and also because of the demonstrated adverse effects of lead on reproductive function in both the male and female as well as the risk of genetic damage of lead on both the ovum and sperm, OSHA recommends a 30 µg/dl maximum permissible blood lead level in both males and females who wish to bear children.

6. Other toxic effects. Debate and research continue on the effects of lead on the human body. Hypertension has frequently been noted in occupationally exposed individuals although it is difficult to assess whether this is due to lead's adverse effects on the kidney or if some other mechanism is involved. Vascular and electrocardiographic changes have been detected but have not been well characterized. Lead is thought to impair thyroid function and interfere with the pituitary-adrenal axis, but again these effects have not been well defined.

III. Medical Evaluation

The most important principle in evaluating a worker for any occupational disease including lead poisoning is a high index of suspicion on the part of the examining physician. As discussed in Section 2, lead can affect numerous organ systems and produce a wide array of signs and symptoms, most of which are non-specific and subtle in nature at least in the early stages of disease. Unless serious concern for lead toxicity is present, many of the early clues to diagnosis may easily be overlooked.

The crucial initial step in the medical evaluation is recognizing that a worker's employment can result in exposure to lead. The worker will frequently be able to define exposures to lead and lead containing materials but often will not volunteer this information unless specifically asked. In other situations the worker may not know of any exposures to lead but the suspicion might be raised on the part of the physician because of the industry or occupation of the worker. Potential occupational exposure to lead and its compounds occur in many occupations in the construction industry, including demolition and salvaging operations, removal or encapsulation of materials containing lead, construction, alteration, repair or renovation of structures containing lead, transportation, disposal, storage or containment of lead or lead-containing materials on construction sites, and maintenance operations associated with construction activities.

Once the possibility for lead exposure is raised, the focus can then be directed toward eliciting information from the medical history, physical exam, and finally from laboratory data to evaluate the worker for potential lead toxicity.

A complete and detailed work history is important in the initial evaluation. A listing of all previous employment with information on job description, exposure to fumes or dust, known exposures to lead or other toxic substances, a description of any personal protective equipment used, and previous medical surveillance should all be included in the worker's record. Where exposure to lead is suspected, information concerning on-the-job personal hygiene, smoking or eating habits in work areas, laundry procedures, and use of any

protective clothing or respiratory protection equipment should be noted. A complete work history is essential in the medical evaluation of a worker with suspected lead toxicity, especially when long term effects such as neurotoxicity and nephrotoxicity are considered.

The medical history is also of fundamental importance and should include a listing of all past and current medical conditions, current medications including proprietary drug intake, previous surgeries and hospitalizations, allergies, smoking history, alcohol consumption, and also non-occupational lead exposures such as hobbies (hunting, riflery). Also known childhood exposures should be elicited. Any previous history of hematological, neurological, gastrointestinal, renal, psychological, gynecological, genetic, or reproductive problems should be specifically noted.

A careful and complete review of systems must be performed to assess both recognized complaints and subtle or slowly acquired symptoms which the worker might not appreciate as being significant. The review of symptoms should include the following:

1. General—weight loss, fatigue, decreased appetite.

2. Head, Eyes, Ears, Nose, Throat (HEENT)—headaches, visual disturbances or decreased visual acuity, hearing deficits or tinnitus, pigmentation of the oral mucosa, or metallic taste in mouth.

3. Cardio-pulmonary—shortness of breath, cough, chest pains, palpitations, or orthopnea.

4. Gastrointestinal—nausea, vomiting, heartburn, abdominal pain, constipation or diarrhea.

5. Neurologic—irritability, insomnia, weakness (fatigue), dizziness, loss of memory, confusion, hallucinations, incoordination, ataxia, decreased strength in hands or feet, disturbances in gait, difficulty in climbing stairs, or seizures.

6. Hematologic—pallor, easy fatigability, abnormal blood loss, melena.

7. Reproductive (male and female and spouse where relevant)—history of infertility, impotence, loss of libido, abnormal menstrual periods, history of miscarriages, stillbirths, or children with birth defects.

8. Musculo-skeletal—muscle and joint pains.

The physical examination should emphasize the neurological, gastrointestinal, and cardiovascular systems. The worker's weight and blood pressure should be recorded and the oral mucosa checked for pigmentation characteristic of a possible Burtonian or lead line on the gingiva. It should be noted, however, that the lead line may not be present even in severe lead poisoning if good oral hygiene is practiced.

The presence of pallor on skin examination may indicate an anemia which, if severe, might also be associated with a tachycardia. If an anemia is suspected, an active search for blood loss should be undertaken including potential blood loss through the gastrointestinal tract.

A complete neurological examination should include an adequate mental status evaluation including a search for behavioral and psychological disturbances, memory testing, evaluation for irritability, insomnia, hallucinations, and mental clouding. Gait and coordination should be examined along with close observation for tremor. A detailed evaluation of peripheral nerve function including careful sensory and motor function testing is warranted. Strength testing particularly of extensor muscle groups of all extremities is of fundamental importance.

Cranial nerve evaluation should also be included in the routine examination.

The abdominal examination should include auscultation for bowel sounds and abdominal

bruits and palpation for organomegaly, masses, and diffuse abdominal tenderness.

Cardiovascular examination should evaluate possible early signs of congestive heart failure. Pulmonary status should be addressed particularly if respirator protection is contemplated.

As part of the medical evaluation, the interim lead standard requires the following laboratory studies:

1. Blood lead level

2. Hemoglobin and hematocrit determinations, red cell indices, and examination of the peripheral blood smear to evaluate red blood cell morphology

3. Blood urea nitrogen

4. Serum creatinine

5. Routine urinalysis with microscopic examination.

6. A zinc protoporphyrin level.

In addition to the above, the physician is authorized to order any further laboratory or other tests which he or she deems necessary in accordance with sound medical practice. The evaluation must also include pregnancy testing or laboratory evaluation of male fertility if requested by the employee. Additional tests which are probably not warranted on a routine basis but may be appropriate when blood lead and ZPP levels are equivocal include delta aminolevulinic acid and coproporphyrin concentrations in the urine, and dark-field illumination for detection of basophilic stippling in red blood cells.

If an anemia is detected further studies including a careful examination of the peripheral smear, reticulocyte count, stool for occult blood, serum iron, total iron binding capacity, bilirubin, and, if appropriate, vitamin B12 and folate may be of value in attempting to identify the cause of the anemia.

If a peripheral neuropathy is suspected, nerve conduction studies are warranted both for diagnosis and as a basis to monitor any therapy.

If renal disease is questioned, a 24 hour urine collection for creatinine clearance, protein, and electrolytes may be indicated. Elevated uric acid levels may result from lead-induced renal disease and a serum uric acid level might be performed.

An electrocardiogram and chest x-ray may be obtained as deemed appropriate.

Sophisticated and highly specialized testing should not be done routinely and where indicated should be under the direction of a specialist.

IV. Laboratory Evaluation

The blood lead level at present remains the single most important test to monitor lead exposure and is the test used in the medical surveillance program under the lead standard to guide employee medical removal. The ZPP has several advantages over the blood lead level. Because of its relatively recent development and the lack of extensive data concerning its interpretation, the ZPP currently remains an ancillary test.

This section will discuss the blood lead level and ZPP in detail and will outline their relative advantages and disadvantages. Other blood tests currently available to evaluate lead exposure will also be reviewed.

The blood lead level is a good index of current or recent lead absorption when there is no anemia present and when the worker has not taken any chelating agents. However, blood lead levels along with urinary lead levels do not necessarily indicate the total body burden of lead and are not adequate measures of past exposure. One reason for this is that lead has a high affinity for bone and up to 90% of the body's total lead is deposited there. A very important component of the total lead body burden is lead in soft tissue (liver, kidney, and brain). This fraction of the lead body burden, the biologically active lead, is not entirely reflected by blood lead levels since it is a function of the dynamics of lead absorption, distribution, deposition in bone and excretion. Following discontinuation of exposure to lead, the excess body burden is only slowly mobilized from bone and other relatively stable body stores and excreted. Consequently, a high blood lead level may only represent recent heavy exposure to lead without a significant total body excess and likewise a low blood lead level does not exclude an elevated total body burden of lead.

Also due to its correlation with recent exposures, the blood lead level may vary considerably over short time intervals.

To minimize laboratory error and erroneous results due to contamination, blood specimens must be carefully collected after thorough cleaning of the skin with appropriate methods using lead-free blood containers and analyzed by a reliable laboratory. Under the standard, samples must be analyzed in laboratories which are approved by OSHA. Analysis is to be made using atomic absorption spectrophotometry, anodic stripping voltammetry or any method which meets the accuracy requirements set forth by the standard.

The determination of lead in urine is generally considered a less reliable monitoring technique than analysis of whole blood primarily due to individual variability in urinary excretion capacity as well as the technical difficulty of obtaining accurate 24 hour urine collections. In addition, workers with renal insufficiency, whether due to lead or some other cause, may have decreased lead clearance and consequently urine lead levels may underestimate the true lead burden. Therefore, urine lead levels should not be used as a routine test.

The zinc protoporphyrin test, unlike the blood lead determination, measures an adverse metabolic effect of lead and as such is a better indicator of lead toxicity than the level of blood lead itself. The level of ZPP reflects lead absorption over the preceding 3 to 4 months, and therefore is a better indicator of lead body burden. The ZPP requires more time than the blood lead to read significantly elevated levels; the return to normal after discontinuing lead exposure is also slower. Furthermore, the ZPP test is simpler, faster, and less expensive to perform and no contamination is possible. Many investigators believe it is the most reliable means of monitoring chronic lead absorption.

Zinc protoporphyrin results from the inhibition of the enzyme ferrochelatase which catalyzes the insertion of an iron molecule into the protoporphyrin molecule, which then becomes heme. If iron is not inserted into the molecule then zinc, having a greater affinity for protoporphyrin, takes the place of the iron, forming ZPP.

An elevation in the level of circulating ZPP may occur at blood lead levels as low as 20–30 µg/dl in some workers. Once the blood lead level has reached 40 µg/dl there is more marked rise in the ZPP value from its normal range of less than 100 µg/dl100 ml. Increases in blood lead levels beyond 40 µg/100 g are associated with exponential increases in ZPP.

Whereas blood lead levels fluctuate over short time spans, ZPP levels remain relatively stable. ZPP is measured directly in red blood cells and is present for the cell's entire 120 day life-span. Therefore, the ZPP level in blood reflects the average ZPP production over the previous 3–4 months and consequently the average lead exposure during that time interval.

It is recommended that a hematocrit be determined whenever a confirmed ZPP of 50 µg/100 ml whole blood is obtained to rule out a significant underlying anemia. If the ZPP is in excess of 100 µg/100 ml and not associated with abnormal elevations in blood lead levels, the laboratory should be checked to be sure that blood leads were determined using atomic absorption spectrophotometry anodic stripping voltammetry, or any method which meets the

accuracy requirements set forth by the standard by an OSHA approved laboratory which is experienced in lead level determinations. Repeat periodic blood lead studies should be obtained in all individuals with elevated ZPP levels to be certain that an associated elevated blood lead level has not been missed due to transient fluctuations in blood leads.

ZPP has a characteristic fluorescence spectrum with a peak at 594 nm which is detectable with a hematofluorimeter. The hematofluorimeter is accurate and portable and can provide on-site, instantaneous results for workers who can be frequently tested via a finger prick.

However, careful attention must be given to calibration and quality control procedures. Limited data on blood lead-ZPP correlations and the ZPP levels which are associated with the adverse health effects discussed in Section 2 are the major limitations of the test. Also it is difficult to correlate ZPP levels with environmental exposure and there is some variation of response with age and sex. Nevertheless, the ZPP promises to be an important diagnostic test for the early detection of lead toxicity and its value will increase as more data is collected regarding its relationship to other manifestations of lead poisoning.

Levels of delta-aminolevulinic acid (ALA) in the urine are also used as a measure of lead exposure. Increasing concentrations of ALA are believed to result from the inhibition of the enzyme delta-aminolevulinic acid dehydrase (ALA-D). Although the test is relatively easy to perform, inexpensive, and rapid, the disadvantages include variability in results, the necessity to collect a complete 24 hour urine sample which has a specific gravity greater than 1.010, and also the fact that ALA decomposes in the presence of light.

The pattern of porphyrin excretion in the urine can also be helpful in identifying lead intoxication. With lead poisoning, the urine concentrations of coproporphyrins I and II, porphobilinogen and uroporphyrin I rise. The most important increase, however, is that of coproporphyrin III; levels may exceed 5,000 µg/l in the urine in lead poisoned individuals, but its correlation with blood lead levels and ZPP are not as good as those of ALA. Increases in urinary porphyrins are not diagnostic of lead toxicity and may be seen in porphyria, some liver diseases, and in patients with high reticulocyte counts.

Summary. The Occupational Safety and Health Administration's interim standard for inorganic lead in the construction industry places significant emphasis on the medical surveillance of all workers exposed to levels of inorganic lead above 30 µg/m^3 TWA. The physician has a fundamental role in this surveillance program, and in the operation of the medical removal protection program.

Even with adequate worker education on the adverse health effects of lead and appropriate training in work practices, personal hygiene and other control measures, the physician has a primary responsibility for evaluating potential lead toxicity in the worker. It is only through a careful and detailed medical and work history, a complete physical examination and appropriate laboratory testing that an accurate assessment can be made. Many of the adverse health effects of lead toxicity are either irreversible or only partially reversible and therefore early detection of disease is very important.

This document outlines the medical monitoring program as defined by the occupational safety and health standard for inorganic lead. It reviews the adverse health effects of lead poisoning and describes the important elements of the history and physical examinations as they relate to these adverse effects. Finally, the appropriate laboratory testing for evaluating lead exposure and toxicity is presented.

It is hoped that this review and discussion will give the physician a better understanding of the OSHA standard with the ultimate goal of protecting the health and well-being of the worker exposed to lead under his or her care.

[58 FR 26627, May 4, 1993, as amended at 58 FR 34218, June 24, 1993; 61 FR 5510, Feb. 13, 1996; 63 FR 1296, Jan. 8, 1998; 70 FR 1143, Jan. 5, 2005; 71 FR 16674, Apr. 3, 2006; 71 FR 50191, Aug. 24, 2006; 73 FR 75588, Dec. 12, 2008]

LEAD CONTROL PROGRAM

I. *Blood Lead Monitoring*

a. **0-19** ug/100 gm whole blood	**Every 6 months**
b. **20-29** ug/100 gm whole blood	**Every 3 months**
c. **30-34** ug/100 gm whole blood	**Every 2 months**
d. **35+** ug/100 gm whole blood	**Every month**

II. *Blood Lead of 35 and above*

a. Employee will be considered on MRP if:
   - 6 month blood lead average (or last 3 samples) is 36 or above
   - One time blood lead reading of 44 or greater
     - MRP Paperwork
       - ✓ Perform detailed history
       - ✓ Check work practices
       - ✓ Personal health
       - ✓ Hygiene consultation
       - ✓ Supply respirator
       - ✓ Wipe samples for Pb analysis
     - Remove from work station, if (lead-in-air over 30 ug/m3) and refer to plant physician

b. Employee will be removed from MRP (return regular job assignment) when two confirmed blood leads are below 33, and 6 month average is 36 or below.

***** Confirmed blood lead of 10 ug/100 gm or more on post-employment physical will be subject to disqualification of employment.***

Signature: Paul Mc[illegible] Date: 1-25-2001

III. *Workplace Safety*

a. Appropriate protective equipment, work apparel, and safety shoes are supplied for all employees
b. Hair protection provided by the company to be worn by all employees in the manufacturing area
c. At no time will work clothes nor safety shoes be taken outside of the plant. This includes
   - Lunch or break areas
   - Home for laundry

***** Uniform laundry service will be provided by plant*****

South Carolina Workers' Compensation Commission
P.O. Box 1715 • 1612 Marion Street
Columbia, South Carolina 29202-1715

WCC File # 7423889
Carrier File #
Carrier Code #
Employer FEIN

Lee Ernest Franklin Claimant's Name SSN	Yuasa Exide, Inc. Employer's Name
729 Miller Road, Sumter, SC 29150 Address City State Zip	P.O. Box 1797, Sumter, SC 29150 Address City State Zip
803-775-8214 Home Phone # Work Phone #	803-779-2300 Insurance Carrier Phone Number
W. Hugh McAngus Preparer's Name	

Complete each information blank. To request a hearing, check box 13 b., indicate the kinds of benefits claimed by checking the box(es) at lines 6, 7, 8, and 9, and file this form in duplicate.

A claim for worker's compensation benefits is made based on the following grounds:

1a. The claimant sustained an accidental injury to his entire body (part of body hurt) from 1974 to 1996 (month day year) in Sumter County, State of South Carolina.

1b. Describe briefly how the accident occurred Claimant was chronically exposed to lead in his employment, resulting in multiple illnesses and injuries.

2. Both the claimant and the employer were subject to the South Carolina Workers' Compensation Act at the time of injury

3. The relationship of the employer and employee existed at the time of injury.

4. At the time of the injury the claimant was performing services arising out of and in the course of employment.

5. Notice of the accidental injury was given to the employer from 1974 forward in the following manner: Employer tested Claimant's blood lead levels and was aware of his ongoing injury. Written notice to Employer of this specific claim was given by letter dated March 20, 1997.

☒ 6. Due to injury, the claimant is in need of (check one):
☒ (a) medical examination and treatment for multiple illnesses and injuries as a result of exposure to lead.
☐ (b) additional medical examination and treatment for occupational disease to kidneys.

☒ 7. Due to injury, the claimant requests temporary total disability benefits because of lost compensable time from work and wages for the period of: From the date of last work ongoing.

☒ 8. Due to the injury, the claimant has permanent disability of the following nature and extent: (check one)
☒ (1)General Disability: ☒ Total ☐ Partial ☒ (2) Specific Disability: ☒ Total ☐ Partial
☒ (3) Permanent disability
Body part(s) affected are: loss of kidneys.

☐ 9. Due to the injury, the claimant has a serious bodily disfigurement consisting of N/A.

10a. At the time of the injury, the claimant was paid weekly wages of $ Requested Form 20; and demands accounting of days worked and wages earned as provide by law.

10b. Give names and addresses of all employers from whom the claimant has worked since the date of the accident.
None.

11a. Further grounds of claim: Claimant requests benefits for total disability and possible lifetime benefits for brain damage as a result of exposure to lead.

11b. List names and addresses of all physicians or other medical specialists who have seen or treated the claimant as a result of the accident: Medical Department at Yuasa Exide; Dr. Pusadee Suchinda; Dr. A. W. Hursey; Dr. Wallace Cone; Dr. W. Bultman; Dr. Capers Hiott

11c. To the best of your knowledge, did you have any prior permanent disability? .
If yes, describe: .

12. Appropriate benefits as provided in the ACT for the above grounds and other relief as the Worker' Compensation Commission may direct as just and proper.

☐ 13a. I am filing a claim. I am not requesting a hearing a this time.
☒ 13b. I am requesting a hearing.

Signature of Claimant/Representative

July 3, 2001
Date

Refer to R.67-205 and R.67-206, R.67-207, and R.67-601 through R.67-615. Questions about the use of this form may be directed to the Commission's Judicial Department.

WCC Form #50. Rev. Date 9/90 **50** **Employee's Notice of Claim and/or Request for Hearing**

STATE OF SOUTH CAROLINA

BEFORE THE WORKERS' COMPENSATION COMMISSION

WCC FILE NO. 7423889

LEE ERNEST FRANKLIN, Claimant, vs. YUASA EXIDE, INC. Employer, and AMERICAN NATIONAL FIRE INSURANCE CO., Carrier, Defendants.	)	**DECISION AND ORDER** COPY

DATE OF HEARING: Hearing held in Sumter, S.C. on September 30, 2004.

APPEARANCES: Claimant appeared and represented by W. Hugh McAngus, Esquire of McAngus, Goudelock & Courie, L.L.C. of Columbia, South Carolina and George C. James, Jr., Esquire, of Lee Law Firm of Sumter, South Carolina.

Defendants Yuasa Exide, Inc. and Great American Insurance Co. represented by Vernon F. Dunbar, Esquire, of Turner, Padget, Graham & Laney, P.A. of Columbia, South Carolina.

PURPOSE OF THE HEARING: To determine issues as set forth on Forms 50 and 51 and any other issues that timely come before the Commission.

COMMISSIONER: George N. Funderburk, Commissioner

FILED: June 10, 2005

STIPULATIONS

The parties stipulated at the hearing to the following:

1. The purpose of the hearing was to determine the issues set forth on Forms 50 and 51, and any other issues that timely come before the Commission.

2. Venue was proper in Sumter County.

3. Notice of the hearing was timely and properly served on all parties of interest.

4. The Claimant seeks a determination of benefits under the South Carolina Workers' Compensation Act; therefore, the Commission has jurisdiction over this claim.

5. The Claimant's average weekly wage and compensation rate are $602.63 and $401.75, respectively.

APA SUBMISSIONS

Pursuant to the South Carolina Administrative Procedures Act and Regulations of the South Carolina Workers' Compensation Commission, the following records and documents were submitted into evidence.

Claimant's submissions:

1.	Article *Occupational Lead Poisoning*, American Family Physician	02/15/98	Pages 1-12
2.	Article *No Safe Level of Lead. Study Suggests*, Associated Press	04/30/01	Pages 13-14
3.	Article *Assessment: Blood Lead Levels*	06/01/99	Pages 15-16
4.	Article *What Lead Does to Your Health*, EPA	12/12/01	Pages 17-20

5.	Article *Management Guidelines for Blood Lead Levels in Children and Adults*	11/27/00	Pages 21-22
6.	Article Isselbacher et al., Harrison's Priniciples of Internal Medicine, Vol. 2, 13th Edition, pp. 2463-2464; pp. 1315-1316		Pages 23-27
7.	Article Hu, Howard, M.D., ScD., et al., *The Relationship of Bone and Blood Lead to Hypertension*, JAMA, Col. 275, No. 15, PP. 1171-1181 8.	04/17/96	Pages 28-38
8.	Article *Information for Consumers: Lead Exposure in Adults - A Guide for Health Care Providers*, New York State Department of Health	01/2001	Pages 39-44
9.	Article *Studies Find Exposure to Lead More Hazardous Than Thought*, U.S. Water News Online	June 1996	Pages 45-46
10.	Letter from Hugh McAngus to Vernon Dunbar	1/15/02	Page 47
11.	Transcript of Hearing before S.C. Workers' Compensation Commission	02/04/02	Pages 48-72
12.	Brief and Memorandum of Law in Support of Defendants American National Fire Insurance Co. and Yuasa Exide, Inc., Appeal (with attachments)	12/29/03	Pages 73-95
13.	Deposition of Lee Ernest Franklin	01/14/99	Pages 96-140
14.	Deposition of Brian Hann	11/19/98	Pages 141-176
15.	Deposition of Dr. Eric Byrd	11/19/98	Pages 177-293
15A.	Deposition of Dr. Eugene Shippen	08/24/04	Pages A1-A60
16.	Records of Carroll H. Crawford, MEd, CRC ProRehab, Inc.	03/19/02	Pages 294-296

17.	Records of Howard Hu, M.D., MPH, ScD Harvard School of Public Health & Medicine dated	07/25/01 - 05/24/04	Pages 297-305
18.	Curriculum Vitae of Howard Hu, M.D., MPH, ScD	07/16/01	Pages 306-332
19.	Records of Richard P. Wedeen, M.D.	01/24/01	Pages 333-334
20.	Curriculum Vitae of Richard Peter Wedeen, M.D.	11/2000	Pages 335-359
21.	Letter from George C. James, Jr., to Richard P. Wedeen, M.D.	01/22/01	Pages 360-361
22.	Chain of Custody Form	01/23/01 - 01/24/01	Pages 362-363
22A.	Records of Michael E. Ullian, M.D. MUSC	09/01/04	Pages 22A1-22A3
22B.	Curriculum Vitae of Michael E. Ullian, M.D.	09/14/04	Pages 22B1-22B7
23.	Exide documentation concerning blood lead levels	08/05/74 - 11/07/95	Pages 364-378
24.	Exide Personal Employee Data Sheets	11/04/91 - 02/05/92	Pages 379-382
25.	Exide Employer Records provided to Attorney McAngus by Attorney Zeigler	08/05/74 - 10/18/96	Pages 383-673
26.	Medical Records from Exide	08/03/74 - 04/23/96	Pages 674-861
27.	Lab Reports from Exide	07/11/89 - 11/19/92	Pages 862-880
28.	Records of Pasadee Suchinda, M.D. Sumter Medical Specialists	04/29/85 - 05/29/91	Pages 881-899
29.	Records of Wallis D. Cone, M.D. Sumter Urological Associates, PA dated	03/10/81 - 05/29/85	Pages 900-904
30.	Tuomey Regional Medical Center	03/10/81 - 05/01/96	Pages 905-1646

31.	Deposition of Howard Hu, M.D., MPH, ScD	9/20/04	

Defendant submissions:

1.	OSHA Regulations (Substance Date Sheet for Occupational Exposure to Lead 1910.1025 etc.)	5/31/99-6/24/93	Pages 1-41
2.	Claimant's Blood Lead Levels reports	7/14/76-7/23/79	Page 45
3.	Records of S.C. Workers Compensation Commission Coverage & Compliance		Pages 46-70

Exhibits:

4.	Records of Exide	12/09/83-10/4/85	
5.	Record of Tuomey Hospital	5/21/88	

STATEMENT OF THE CASE

This hearing comes before the undersigned Commissioner based on the Form 50 filed by Claimant alleging occupational disease as a result of lead exposure during his employment with Yuasa Exide, Inc. Claimant alleges that from 1974 through 1996 while working for Employer, he was continuously exposed to lead which resulted in high blood pressure, kidney disease and failure, and ultimately to carcinoma of the kidneys and removal of his kidneys. Claimant alleges he is permanently and totally disabled as a result of his kidney disease. Claimant contends that Defendants, the carrier on the risk at the date of last injurious exposure, should be liable for this claim. Claimant requests that Utica-Mohawk language be incorporated in the Order.

Defendants contend any responsibility for this claim should lie with the workers' compensation insurance carriers who insured Exide during the earlier parts of his employment and

that Claimant's last injurious exposure occurred during the period of 1974-1985. Defendants contend that any lead exposure during its coverage period (1991-1996) was within OSHA permissible exposure limits and therefore not injurious. Defendants further deny this claim asserting it should be barred by the statute of limitations. Defendants admit Claimant is permanently and totally disabled as a result of his kidney disease.

EVIDENCE OF THE CASE

Claimant testified that he worked for Employer for more than 23 years and had no health problems when his employment began. He initially worked in the assembly area which was considered a high lead area. He first worked for 2 years as a stacking machine operator. This job consisted of working with battery plates stacked on a rack, and Claimant testified that there was lead dust on the plates, which fell as the plates were unloaded. He then worked in pasting operation for 6-7 years mixing lead powder with acid. He testified that in both jobs there was dust in the air and on the floor. He also worked as a winkle burner, welding lead plates with a torch. He then worked as a material handler for 7 years. In this job, he drove a forklift and moved battery plates and elements around the assembly area. He testified that dust blew off the material and back towards him while driving the forklift. At one point, Employer suggested that Claimant try driving his forklift backwards so that the lead dust wouldn't fly in his face. There was a ventilation system installed at some point, but most of the time this did not work right. When it wasn't working, production continued.

Claimant testified that when his employment began, he was provided a paper mask. This changed to a "v-shaped" mask and eventually to a respirator. Regardless of the mask, dust got in on his face and in his nose. The respirator did not stay sealed and would loosen with sweat. At the end

of the day, he would shower in the locker room with special soap to get the lead dust off of his skin and blow his nose to get the dust out. At some point, Claimant's blood pressure and lead levels were high and he was moved to the shipping department, the only low lead area in the plant. Claimant testified that the shipping area was at the back of the plant and he walked through the manufacturing area about 8 times a day to get to and from shipping. The areas were not closed from each other and were only separated by a driveway, with forklifts traveling between the two areas. There was dust in the shipping area which continued to get in his nose.

Claimant testified he first had blood pressure problems in the eighties which continued until he left Employer. He took medication except for periods when his monthly supply ran out before he obtained the next prescription. Claimant began dialysis three times per week for three and a half hours per day in 1993, which continued until his kidneys were removed in 1996. He continued to work during dialysis. He hasn't had any blood pressure problems since his kidneys were removed.

Claimant denied having any history drinking problems. The only moonshine he has ever had was given to him by a supervisor at a company picnic. Claimant also testified he did work around leaded gasoline helping a friend replace engines, but this was not everyday. Four or Five other co-workers were going through dialysis at the same time as Claimant. On cross-examination, Claimant was shown three medical reports: (1) 1983 report which stated he told the doctor about stump hole liquor; (2) 1985 report which states he told the doctor he stopped drinking stump hole liquor in April; and (3) 1988 report where Claimant was told to stop alcohol. Claimant responded that he never denied drinking alcohol, he just denied drinking it on a regular basis, and he only drank stump hole liquor one time.

On cross-examination, Claimant testified that his brother and some cousins have also undergone dialysis, and the cousins also had hypertension and diabetes. His mother has hypertension and diabetes. Claimant took his blood pressure medications except when they ran out, which didn't happen routinely. Employer would tell him during physicals when his blood pressure was high, and his doctors always told him to take his medicine. In the early eighties Claimant was aware that he was loosing protein which was a sign of kidney failure. Initially when Claimant's blood lead levels were high, he was not removed from his work area. Eventually he was removed until his lead levels came back down, then he was put back in his regular job. When his blood lead levels began staying high, he was moved to material handler and eventually to shipping. Claimant testified that his blood pressure was continuously rising and his kidney problems progressively worsening. Once someone is moved to shipping, there is no place left to move. Once in shipping, he continued to be monitored and remembers being told that his blood levels were 50 or above. When he began dialysis in 1993, Claimant was aware Employer didn't want him around high amounts of lead because there were high amounts in his body, but no one told him this was because of his kidney problems. Employer was telling everyone that high lead amounts were not good.

Claimant submitted medical opinions from Dr. Howard Hu and Dr. Richard P. Wedeen. Both confirmed Claimant's disease was causally related to his exposure with Employer. Dr. Howard Hu is a Professor of Occupational and Environmental Medicine who also continues to practice occupational and environmental medicine. In his reports, Dr. Howard Hu opined that Claimant's occupational lead exposure at the Yuasa Exide Battery plant was the major contributing factor towards the causation of his chronic renal failure, end-stage renal failure, need for dialysis, bilateral renal cell carcinomas, and need for bilateral nephrectomies. Dr. Hu concluded that although

Claimant's early employment exposed him to higher levels of lead, Claimant's continued exposure from 1991-1996 more likely than not resulted in additional lead-induced injuries to his kidneys and the onset of renal carcinoma requiring removal of his kidneys in January 1996.

In his deposition, Dr. Hu confirmed that Claimant's condition resulted from his continuous exposure to lead. After Claimant's initial diagnosis of renal disease in 1981, Claimant's exposure lasting until 1996 aggravated or combined with the disease causing renal cell carcinoma. Claimant's high blood pressure and renal failure were first noted in 1981. Claimant's lead exposure directly effected his kidneys and blood pressure, and the high blood pressure in turn continued to contribute to the kidney disease. Claimant's continued exposure to lead until 1996 continued to have an adverse affect on his kidneys and blood pressure.

Dr. Hu testified that the preponderance of the evidence and literature suggest that even a low to moderate lead exposure (exposure causing blood levels of 10-40) will negatively affect renal function, and he cited 9 articles to support this conclusion. Dr. Hu conceded that poorly controlled blood pressure probably exacerbated Claimant's kidney problems, and that African-Americans have a higher incidence of hypertension. However, Dr. Hu asserted that African-Americans also have higher incidences of lead exposure than whites, so he could not conclude that the higher incidences were actually due to genetics versus environmental exposure. When questioned about the "Gerhardsson's study", Dr. Hu admitted that the study showed no adverse effects on the kidneys of lead workers with a blood level of up to 60-70. However, Dr. Hu testified that the study was flawed for two reasons. One, the study is 12 years old and more studies have come out since which do show a connection of low to moderate exposure and renal function. Two, the study suffers from healthy worker bias, meaning only the working population is studied, ignoring those already sick who have

left the workforce.

Dr. Richard P. Wedeen is a Professor in Environmental and Community Medicine and board certified in Internal Medicine with a subspecialty in Nephrology. In his report, Dr. Wedeen concluded that Claimant's lead exposure and medical history were consistent with lead nephropathy as the major cause of his hypertension and progressive renal failure.

Defendants obtained a medical opinion from Dr. Michael E. Ullian, a Professor of Medicine in the Division of Nephrology who is board certified in Internal Medicine with a subspecialty in Nephrology. In his report, Dr. Ullian concluded that lead exposure did initiate Claimant's kidney disease. Dr. Ullian specifically pointed out Claimant's significant lead exposures at work, the "almost simultaneous" beginnings of both high blood pressure and kidney insufficiency, and the fact that no other causes of Claimant's kidney disease had been found. Dr. Ullian concluded that Claimant's kidney cancer was not directly related to lead exposure, stating that "[i]t is fairly common for kidney cancer to develop in patients whose kidneys have failed."

Defendants also relied on the opinion of Dr. Eugene Shippen, an Exide consultant who is board certified in family practice. Dr. Shippen testified that he worked as a consultant for Exide as well as other companies in the lead industry. Claimant objected to Defendants attempt to qualify Dr. Shippen as an expert in lead toxicology. Dr. Shippen admitted that the majority of seminars he has attended were sponsored by the lead industry and that he has no formal medical education in nephrology, environmental medicine, occupational medicine, or epidemiology. Dr. Shippen also admitted he has never done any independent medical or peer-reviewed research on lead toxicity.

In his deposition, Dr. Shippen testified that he examined Claimant once in 1989 during a visit to the Sumter plant, when one of the medical staff pointed out Claimant had elevated blood pressure

and some early renal problems. At the time, Claimant's lead level was around 34 and Dr. Shippen felt that the minor renal dysfunction was not related to his hypertension. He testified that Claimant's early exposure to lead prior to the implementation of OSHA standards aggravated the Claimant's pre-existing hypertension. Although Claimant continued to be exposed to lead after the OSHA standards were implemented in 1979 through 1996, these levels were not enough to cause further damage. The additional damage to Claimant's kidneys was caused by Claimant's noncompliance with treatment for hypertension.

On cross-examination, Dr. Shippen admitted there was no evidence of hypertension, kidney disease, or renal failure when Claimant began working for Exide in 1974. He also testified that Claimant was exposed to high levels of lead at the beginning of his employment. Dr. Shippen testified that lead exposure can cause kidney dysfunction and admitted that Claimant began having renal dysfunction in the 1980's. Although he disagrees with the literature, he admitted that some current literature supports a connection between lead exposure and high blood pressure. He agrees that Claimant is disabled and requires dialysis. He also agrees that some individuals may differ in their susceptible to lead damage.

Dr. Eric Byrd has been the plant physician for Employer since 1988. In his deposition, he testified that he is also plant manager for 4 other companies and practices family medicine. Dr. Byrd testified that lead exposure can cause, aggravate or accelerate kidney disease. In his opinion, a normal blood level is below 10, and levels below 60-70 would not cause significant medical problems. He admitted that he relies on internal industry literature exclusively as the basis for these opinions. He concluded that Claimant's early exposure did affect his kidney function. Dr. Byrd testified that Claimant's continued exposure was not high enough to cause additional damage, but

did testify that exposure levels of 50 could aggravate or accelerate existing kidney disease. Dr. Byrd testified that he believes Claimant was noncompliant with his high blood pressure medication and that his kidney disease would not be as severe had he been compliant.

Dr. Byrd testified that in preparation for his deposition, he spoke with Dr. Shippen who provided him with an article concluding low level lead exposure did not have any affect on renal dysfunction. Dr. Byrd testified he also relied on Harrison's Book of Internal Medicine. He testified that kidney disease can increase blood pressure, that high blood pressure can cause kidney damage, but that lead exposure does not significantly increase blood pressure. However, Dr. Byrd admitted that Harrison's Principles of Internal Medicine, which he believes is an authoritative source, reports an association between hypertension and blood lead levels of 30, and that Claimant's blood levels were consistently above 30 during his employment.

FINDINGS OF FACT

Based on the stipulations of the parties, testimony and evidence received and produced at this hearing, the undersigned Commissioner finds the following facts based upon a preponderance of the evidence:

1. The parties to this proceeding are subject to and bound by the terms and provisions of the South Carolina Workers' Compensation Act. This finding is based upon the stipulations and admissions of the parties.

2. The Claimant's average weekly wage is $602.63 with a compensation rate of $401.75.

3. The date of Claimant's last injurious exposure was his last day of work in January 1996. It is undisputed that Mr. Franklin was exposed to lead up until the last date of his employment.

4. The Claimant suffered a compensable occupational disease as a result of chronic lead exposure, ultimately leading to cancer, removal of Claimant's kidneys and ongoing dialysis. Every medical provider in evidence concluded that Claimant's kidney disease was either caused by, accelerated, or aggravated by his exposure to lead on the job.

5. Claimant is entitled to, and Defendants shall pay, causally related medical treatment, both past and future for Claimant's lifetime.

6. Claimant is permanently and totally disabled as a result of his kidney disease.

7. Claimant shall receive, and Defendants shall pay, 500 weeks of total and permanent disability compensation.

8. Claimant shall receive compensation in a lump sum payment. However, Claimant's request for Utica-Mohawk language is denied. This finding is based on the lack of Commission authority to order such allocation of a lump sum award.

9. Claimant timely filed his claim for occupational disease satisfying the statute of limitations.

CONCLUSIONS OF LAW

Under the foregoing findings of fact, and accordingly, as provided in the South Carolina Code of Laws, as amended, it is concluded as follows:

1. Pursuant to South Carolina Code Ann. §42-1-40 and other applicable law, Claimant's average weekly wage is $602.63 with a compensation rate of $401.75.

2. Pursuant to South Carolina Code Ann. §42-11-10 and other applicable law, Claimant sustained compensable occupational disease as a result of chronic lead exposure during his employment with Employer and its predecessor companies.

3. Pursuant to Hanks v. Blair Mills, Inc. 335 S.E.2d 91, 286 S.C. 378 (Ct. App. 1985) and Glenn v. Columbia Silica Sand Company, 112 S.E.2d 711, 236 S.C. 13 (1960) and other applicable law, Claimant's last injurious exposure was on his last day of employment in January 1996. There is no dispute that Claimant was exposed to lead until his last day of work.

4. Pursuant to Hanks v. Blair Mills, Inc. 335 S.E.2d 91, 286 S.C. 378 (Ct. App. 1985) and Glenn v. Columbia Silica Sand Company, 112 S.E.2d 711, 236 S.C. 13 (1960) and other applicable law, Defendants are liable for this claim in its entirety as the carrier at risk on the date of Claimant's last injurious exposure. Where the employment at the time of disability is of a kind that contributes to the disease, liability for the entire compensation is that of the employer and the carrier at the time of the last exposure causally related to the disease.

5. Pursuant to Hanks v. Blair Mills, Inc. 335 S.E.2d 91, 286 S.C. 378 (Ct. App. 1985), §42-15-40 and other applicable law, Claimant's claim is not barred by the statute of limitations.

6. Pursuant to South Carolina Code Ann. §42-9-10, §42-11-40, §42-9-10 and other applicable law, Claimant is permanently and totally disabled. Defendants shall pay 500 weeks of total and permanent disability compensation.

7. Pursuant to South Carolina Code Ann. §42-15-60 and other applicable law, Defendants shall pay all causally related medical treatment, both past and future for Claimant's lifetime.

8. Pursuant to South Carolina Code Ann. §42-9-301 and other applicable law, Defendants shall pay Claimant's award in a lump sum.

9. Claimant's request for Utica-Mohawk language is denied. Although the Commission may approve settlement with this language, the Commission does not have the authority to "order" this language anymore than it can order settlements themselves.

ORDER

IT IS ORDERED that the Claimant suffered a compensable occupational disease as a result of lead exposure during his employment with Employer.

IT IS FURTHER ORDERED that Claimant is permanently and totally disabled due to his kidney disease and entitled to 500 weeks of disability payments at the rate of $401.75 per week. Payment shall be made in lump sum.

IT IS FURTHER ORDERED that Claimant is entitled to, and Defendants shall pay, causally related medical treatment, past and future for the period of Claimant's lifetime.

AND IT IS SO ORDERED.

George N. Funderburk, Commissioner

South Carolina Workers' Compensation Commission

6/3, 2005

CERTIFICATE OF SERVICE

This is to certify that the undersigned has this date served this order in the above entitled action upon all parties to this cause by depositing a copy hereof, postage paid, in the United States mail addressed to the attorney or attorneys for said parties.

This 10 day of June, 2005.

By Joyce H Master

Administrative Assistant to the Commissioner

WHM
VFD

APPELLATE PANEL DECISION AND ORDER

OF THE

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION

W.C.C. FILE NO. 7423889

LEE ERNEST FRANKLIN,

EMPLOYEE,
CLAIMANT,

-VS-

AFFIRM IN PART/
REVERSE IN PART

YUASA EXIDE, INC.,

EMPLOYER,

AND

AMERICAN NATIONAL FIRE INSURANCE COMPANY,

INSURER,
DEFENDANTS.

Appellate Panel Review held in Columbia, South Carolina on November 22, 2005, timely and properly served upon all parties of interest.

Appellate Panel Decision and Order filed May 3, 2006.

APPEARANCES: Claimant represented by:
MCANGUS, GOUDELOCK & COURIE, LLC with
W. Hugh McAngus appearing

Defendants represented by:
Vernon F. Dunbar

RECEIVED
MAY 08 2006
RECEIVED
2335101

E-MAILED
5/8/06

Appeal Due 6/2/06

STATEMENT OF THE CASE

The parties were heard by Commissioner George N. Funderburk on September 30, 2004 in Sumter, South Carolina. On June 10, 2005, the Hearing Commissioner issued the following Order:

"IT IS ORDERED that the Claimant suffered a compensable occupational disease as a result of lead exposure during his employment with Employer.

IT IS FURTHER ORDERED that Claimant is permanently and totally disabled due to his kidney disease and entitled to 500 weeks of disability payments at the rate of $401.75 per week. Payment shall be made in lump sum.

IT IS FURTHER ORDERED that Claimant is entitled to, and Defendants shall pay, causally related medical treatment, past and future for the period of Claimant's lifetime.

AND IT IS SO ORDERED."

Within the statutory period, counsel for Defendants filed an Application for Review, setting forth grounds for appeal, copies of which were furnished to all interested parties prior to oral argument.

All proper testimony has been taken. Such, together with all documentary evidence has been delivered by argument to the individual members of the Panel and have since been under study and consideration.

By appeal, it is respectfully submitted that the Hearing Commissioner erred in the following:

1. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the date of Claimant's last injurious exposure to lead was on the last day of work in January 1996, when the reliable, probative and substantial evidence of the case does not support such a finding of fact or conclusion of law, the error being that the Claimant did not work in January 1996 and that the last

date of injurious exposure, if any, occurred from 1974 to 1981 or from 1981 through 1989.

2. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the Claimant suffered an occupational disease as a result of chronic lead exposure, the error being that the Claimant failed to prove that he was exposed to lead during the coverage period provided by Great American Insurance Company/American National Fire Insurance Company, which could have resulted in the kidney disease and kidney cancer.

3. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the Defendants are responsible for payment of all causally related medical treatment, both past and future for the Claimant's lifetime, the error being that §42-15-20 states that no compensation shall be payable, which may have accrued to notice being given to an employer.

4. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the Claimant timely filed his claim for occupational disease within two years, when the reliable, probative and substantial evidence of the case clearly reveals that notice was not given within two years from the date of accident nor within two years from the date of disablement or diagnosis.

5. The Hearing Commissioner erred in finding as fact and concluding as a matter of law that the Claimant is entitled to payment of the award in a lump sum, the error being that the Claimant never filed a Form 24 pursuant to Reg. 67-1605 requesting a lump sum nor did the Claimant obtain the Defendants' consent to payment of any award in lump sum pursuant to Reg. 67-1605, the error being that benefits are not owed and due until the promulgation of an award and that such is to be paid in weekly allocations unless a Form 24 has been filed and approved by the Commission; or, there is a finding regarding the date of disability.

6. The Hearing Commissioner erred in failing to find as a fact that the Claimant was continuously exposed to dangerously high levels of lead from the period 1974 through 1983, which exceeded current municipal exposure limits as established by OSHA, the error being that the Claimant's exposure to lead during this period constitutes the last injurious exposure and is the proximate cause of the Claimant's current disability.

7. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the proximate cause or etiology of the Claimant's renal kidney disease is as a consequence of chronic lead exposure, when the reliable, probative and substantial evidence of the case reflects that genetics, medical history, failure to comply with medical treatment regiments and hypertension are proximate causes of Claimant's kidney disease.

8. The Hearing Commissioner erred in failing to consider OSHA Standards and Federal Regulations regarding the permissible exposure limits to lead in his findings and conclusions with respect to what amount of exposure to lead constituted the last injurious exposure during the Claimant's employment.

9. The Hearing Commissioner failed to address the amount of exposure to lead, which can be deemed injurious as opposed to non-injurious, the error being that the Commissioner's finding of compensability is predicated upon speculation, conjecture and surmise.

10. The Hearing Commissioner erred in failing to find the date of Claimant's disability, the error being the date upon which the Claimant became disabled after having been made aware of the possible causal relationship between his employment activities and his kidney disease, the two statute of limitations bars this claim.

11. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the last injurious exposure occurred during the time that Claimant was listed as an employee, as opposed to the time in which the Claimant last worked, with respect to designating Great American Insurance Company/American National Fire Insurance Company as the carrier on risk for payment of the Claimant's disability benefits, the error being that the Claimant's injurious exposure during the time that the Employer's Insurance of Wausau Insurance Company provided coverage during the period from 1974 through 1989, reflecting the period of an injurious exposure to lead.

12. The South Carolina Commissioner legally erred and abused its discretion in preventing the Defendants from adding additional parties to the case with respect to prior insurance companies, the error being that because the last injurious exposure occurred when other insurance carriers were on risk, the Defendant insurance carrier has been unduly prejudiced and ordered to pay benefits when the injury did not occur during its coverage period as a consequence of an injury by accident or occupational disease.

13. The Hearing Commissioner erred in failing to find that the Claimant's disability began in 1992/1993 when he began his dialysis treatment, the error being that prior to beginning dialysis treatment, the Claimant was made aware of the effect of exposure by lead on his kidneys.

14. The Hearing Commissioner erred in failing to place weight upon testimony that the Claimant was provided with safety equipment to minimize his exposure to lead resulting from his employment beginning in 1989 until he last physically worked in 1995, the error being that utilization of recommended OSHA and NIOSH equipment diminishes the possibility that the Claimant suffered an injurious exposure as a consequence of exposure to lead subsequent to 1989.

15. The Hearing Commissioner erred in failing to consider or place any weight upon the purchase agreement of Yuasa Exide, which states that the Exide Corporation and its insurance carrier, the Employer's Insurance of Wausau, will not be responsible for any claims regarding workers' compensation benefits filed by Exide employees, the error being that the Claimant was an Exide employee and has filed a workers' compensation claim.

Pursuant to S.C. Code Ann. §42-17-50 (1985), we, the Appellate Panel, have reviewed the award, weighed the evidence and considered all issues raised on appeal. The Panel may make its own findings of fact and reach its own conclusions of law. Based upon a review of the foregoing, the Panel affirms in part and reverses in part the Order of June 10, 2005 and enters the following findings of fact and conclusions of law.

FINDINGS OF FACT

1. The parties to this proceeding are subject to and bound by the terms and provisions of the South Carolina Workers' Compensation Act. This finding is based on the stipulations and admissions of the parties.

2. The Claimant's average weekly wage is $602.63 with a compensation rate of $401.75. This finding is based on the stipulations and admissions of the parties.

3. Claimant was employed by Yuasa Exide, Inc., a lead battery plant, from 1974 until 1996. American National Insurance Fire Insurance Co. provided workers' compensation coverage to Employer from 1991 through 1998.

4. Claimant suffered a compensable occupational disease as a result of chronic lead exposure, ultimately leading to cancer, removal of Claimant's kidneys and ongoing dialysis. Every medical opinion in evidence concluded that Claimant's kidney disease was either caused, accelerated, or aggravated by his exposure to lead on the job.

5. It is undisputed that Claimant was continuously exposed to lead during his employment up until his last day of work with Employer. The date of last injurious exposure was Claimant's last day of work in January 1996.

6. Claimant became disabled in January 1996, when he left work and underwent the removal of both kidneys for end-stage renal failure.

7. Defendants assertion that consideration must be given to Employer provided, safety equipment, compliance with OSHA guidelines, and overall decrease in lead exposure levels to determine injurious vs. non-injurious exposure is not persuasive. Such a determination is contrary to the rule of Glenn v. Columbia

Silica Sand Company, 236 S.C. 13, 112 S.E.2d 711 (1960), providing that in cases of occupational disease, where the employment is of a kind contributing to the disease, liability is assigned to the carrier who was on the risk when the disease resulted in disability.

8. Claimant's employment was of a kind contributing to kidney disease. Every medical opinion in evidence concludes that lead exposure can cause kidney disease. Claimant was exposed to lead throughout his employment. Therefore, liability for the entire claim falls on Carrier, who was on the risk when Claimant became disabled in January 1996.

9. Claimant's kidney disease was a direct result of chronic lead exposure, a hazard greater than that ordinarily incident to normal employment and peculiar to the lead battery industry.

10. The first definitive diagnosis as to the cause of the Claimant's kidney disease was made on January 24, 2001, by Dr. Richard Wedeen, who concluded that Claimant's "loss of kidney function and the development of end-stage renal disease was, in my opinion, due to lead nephropathy." (APA p.334). Thus, the statute of limitations would not have begun to run until this date of definitive diagnosis.

11. Claimant did not become aware of any definitive diagnosis until January 24, 2001. In his 1999 deposition, Claimant testified that no physician had informed him that his kidney failure or hypertension were a result of his employment, and that when he asked Dr. Suchinda if his condition was work-related she said "there was no medical proof." (APA p 126, lines 17-21).

12. The requirement of notice has been met. The first definitive diagnosis as to the cause of the Claimant's Kidney disease was January 24, 2001. Claimant provided notice well before the definitive diagnosis by filing his initial Form 50 in 1997.

13. Defendant's Motion to Add Parties was correctly denied. Liability in cases of occupational disease is not apportioned among successive carriers. As Carrier was on the risk when Claimant's disease resulted in disability, Carrier is liable for the entire claim.

14. Claimant is permanently and totally disabled as a result of his kidney disease. This finding is based on the stipulations and admissions of the parties.

15. Claimant's kidney disease was not accelerated or aggravated by non-compensable causes which would entitle Defendants to any apportionment of the award under §42-11-90.

16. Claimant shall receive, and Defendants shall pay, 500 weeks of total and permanent disability compensation, paid in weekly allocations. Benefits accrued to date are due and payable immediately. Claimant's request for payment of

compensation in lump sum is denied as it was not properly pled in Claimant's Form 50 or raised before the Hearing Commissioner.

17. The purchase agreement introduced by Defendant is irrelevant in a determination of Carrier's liability under the Act. Any cause of action for alleged indemnification would lie in civil court, and the Commission lacks jurisdiction to address the purchase agreement.

CONCLUSIONS OF LAW

Under the foregoing findings of fact, and accordingly, as provided in the South Carolina Code of Laws, as amended, it is concluded as follows:

1. Pursuant to South Carolina Code Ann. § 42-17-50 and other applicable law, the Commission has the power to affirm, amend, or reverse the decision of a Single Commissioner.

2. Pursuant to South Carolina Code Ann. §42-1-40 and other applicable law, Claimant's average weekly wage is $602.63 with a compensation rate of $401.75.

3. Pursuant to South Carolina Code Ann. §42-1-160 and other applicable law, Claimant sustained compensable occupational disease as a result of chronic lead exposure during his employment with Employer and its predecessor companies.

4. Pursuant to Hanks v. Blair Mills, Inc. 335 S.E.2d 91, 286 S.C. 378 (Ct. App. 1985) and Glenn v. Columbia Silica Sand Company, 112 S.E.2d 711, 236 S.C. 13 (1960) and other applicable law, Claimant's last injurious exposure was on his last day of employment in January 1996.

5. Pursuant to Hanks v. Blair Mills, Inc. 335 S.E.2d 91, 286 S.C. 378 (Ct. App. 1985) and Glenn v. Columbia Silica Sand Company, 112 S.E.2d 711, 236 S.C. 13 (1960) and other applicable law, Defendants are liable for this claim in its entirety as the carrier at risk on the date of Claimant's last injurious exposure.

6. Pursuant to South Carolina Code Ann. §42-15-40 and other applicable law, Claimant filed his claim within the two-year statute of limitations.

7. Pursuant to South Carolina Code Ann. §42-15-20 and other applicable law, Claimant provided timely notice to his employer.

8. Pursuant to South Carolina Code Ann. §42-9-10 and other applicable law, Claimant is permanently and totally disabled. Defendants shall pay 500 weeks of total and permanent disability compensation.

9. Pursuant to South Carolina Code Ann. §42-15-60 and other applicable law, Defendants shall pay all causally related medical treatment, both past and future for Claimant's lifetime.

10. Claimant's request for payment of compensation in lump sum is denied as it was not properly pled in Claimant's Form 50 or raised before the Hearing Commissioner.

11. Pursuant to South Carolina Code Ann. §42-11-90 and other applicable law, Defendants are not entitled to any apportionment of the award.

ORDER

IT IS, THEREFORE, ORDERED the Decision and Order of the Single Commissioner is hereby affirmed in part and reversed in part.

IT IS ORDERED that the Claimant suffered a compensable occupational disease as a result of lead exposure during his employment with Employer.

IT IS FURTHER ORDERED that Claimant is permanently and totally disabled due to his kidney disease and entitled to, and Defendants shall pay, 500 weeks of disability payments at the rate of $401.75 per week.

IT IS FURTHER ORDERED that Claimant is entitled to, and Defendants shall pay, causally related medical treatment, past and future for the period of Claimant's lifetime.

AND IT IS SO ORDERED.

S.C. WORKERS' COMPENSATION COMMISSION

J. Alan Bass, Commissioner

CONCUR:

G. Bryan Lyndon, Commissioner

Susan Barden, Commissioner

CERTIFICATE OF SERVICE

This is to certify that the undersigned has this date served this order in the above case upon all parties to this cause by depositing a copy hereof, postage paid, in the United States mail, addressed to the attorneys of record and said parties.

This 26th day of May, 2016

By C. Mason

Administrative Assistant to the Commissioner

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION

W.C.C. FILE NO.: 9643549

YUASA EXIDE, INC. AND GREAT AMERICAN INSURANCE COMPANY, Plaintiffs/Employer and Carrier, vs. SOUTH CAROLINA SECOND INJURY FUND Defendant. **IN RE:** LEE ERNEST FRANKLIN, Employee/Claimant, v. YUASA EXIDE, INC., Employer.	**DECISION AND ORDER**

HEARING: Held in Sumter, South Carolina on November 27, 2007.

APPEARANCES: Plaintiff/Employer and Carrier were represented by Vernon F. Dunbar, Esquire, of TURNER, PADGET, GRAHAM & LANEY, P.A.

Defendant, South Carolina Second Injury Fund was represented by Latonya D. Edwards, Esquire.

PURPOSE OF THE HEARING: To determine issues as set forth on the Forms 54 and 55.

HEARD BY: The Honorable George N. Funderburk, Commissioner

FILED: January 31, 2008

RECEIVED
FEB 1 2008
RECEIVED

STIPULATIONS

1. The purpose of the hearing is to determine the issues as set forth on the Forms 54 and 55. Also, whether the claim filed by Great American Insurance Company a/k/a American National Fire Insurance Company is barred by Section 15-3-600, which provides that any action for relief must be commenced within ten years after the cause of action has accrued.

2. Notice of the hearing was timely and properly provided to all parties of interest.

3. Proper venue is in Sumter County, South Carolina as agreed upon by all parties.

4. The South Carolina Worker's Compensation Commission has subject matter jurisdiction over this proceeding.

5. Without objection, the commission file was made a part of the record excluding any unstipulated medical reports, self-serving statements and hearsay testimony.

APA SUBMISSIONS

APA#	NAME OF PROVIDER/OTHER	DATE(S) OF RECORD(S)	PAGE NUMBERS
1	Letters from Dr. Hu	05/24/04 & 07/25/01	1 – 9
2	Dr. Wedeen Report	01/24/2001	10 - 11
3	Portion of Claimant's Employment file	1983 – 1985	12 - 20
4	Industrial Hygiene Laboratory Studies	1974 – 1985	21 - 25
5	Report of Dr. ER Shippen	09/14/2004	26 - 30
6	Report of Carroll Crawford	03/19/2002	31 - 33

STATEMENT OF THE CASE

Plaintiffs, Yuasa Exide, Inc., the employer, and Great American Insurance Company, the worker's compensation insurance carrier, by and through its attorney argued that the worker's compensation claim of Lee Ernest Franklin with respect to lead exposure resulting in renal kidney failure was subject to Second Injury Fund reimburseiment. The plaintiffs contend that because the South Carolina Worker's Compensation Commission had ruled as a matter of law Lee Ernest Franklin's subsequent exposures to lead below OSHA's permissible exposure guidelines constituted subsequent accidents, Lee Ernest Franklin's last accidental injury occurred on his last date of employment in November 1995.

Lee Ernest Franklin, the claimant, had been exposed to high levels of lead that exceeded OSHA's permissible exposure guidelines between the years 1974 and 1985. During the 1980's, Mr. Franklin was diagnosed with Stage I renal kidney disease that continuously progressed to Stage V. The claimant's kidney disease was as a consequence of his continuous exposure to lead. The plaintiffs argued that had the claimant ceased working at Yuasa Exide at the time of his first diagnosis around 1982, the preexisting Stage I renal kidney disease would not have been aggravated by the claimant's subsequent exposures to lead.

The plaintiffs further argue that as a result of the claimant's exposure to lead, he developed hypertension. The claimant's hypertension was certainly aggravated throughout his employment with the employer because of his continuous exposure to lead. Again, relying upon the expert testimonies of Dr. Howard Hu, Associate Profession

of Harvard Schools of Public Health & Medicine, Richard P. Wedeen, and Eugene Shippen, M.D., neither the claimant's hypertension nor chronic renal disease would have been continuously aggravated but for the claimant's continuous exposure to lead.

Accordingly, the plaintiffs assert that because the disease or condition of heavy metal poisoning is listed as a condition that is presumed to be an impairment that results in 78 weeks or more of disability and constitutes a hindrance and obstacle to employment pursuant to Section 42-9-400 (d)(23), the claim is subject to reimbursement.

In the alternative, the plaintiffs assert that Dr. Eugene Shippen's testimony and his medical questionnaire corroborate that this claim is reimbursable for both heavy lead metal poisoning and the preexisting condition of hypertension. Dr. Howard Hu's testimony corroborated this conclusion. It is clear that Mr. Franklin's hypertensive state preexisted his last accidental injury or exposure in 1995; and that such was constantly aggravated as a result of subsequent exposures to lead. Moreover, Mr. Franklin's hypertensive state constituted a serious condition and a hindrance or obstacle to employment and reemployment. Because the claimant's hypertensive condition results in 78 weeks or more disability and combined with the renal kidney failure caused by heavy metal poisoning, the claim is also subject to reimbursement as a consequence of the diagnosis of hypertension.

The South Carolina Second Injury Fund, by and through its attorney, Latonya D. Edwards, first contended that the plaintiff's claim for reimbursement was barred by the ten year statute of limitations as set forth in Section 15-3-600 *S. C. Code Annot.*

§15-3-600 (2007). Specifically, the Fund avers that because an action for relief was not commenced within ten years after Lee Ernest Franklin's injury or contraction of an occupational disease, the plaintiff's claim must be barred as a matter of law. The Fund does not dispute that the plaintiffs placed it on notice of a potential claim for reimbursement in 1998. The Fund also concedes that the claim was not deemed compensable until after all appeals had been exhausted and abandoned by the parties in 2006.

Nonetheless, the Fund argues that the ten year statute of limitations should be applied despite the fact that Title 42 does not expressly set forth a statute of limitations on Second Injury Fund claims. The Fund's avers a delay in timely filing reimbursement claims often creates administrative confusion and unnecessary costs.

Last, Attorney Edwards contends that the claim is not subject to reimbursement due to the fact that Mr. Franklin suffered with an occupational disease, which is simply a progression of the initial or Stage I of the chronic renal disease that first began in the 1980's. Therefore, the Fund contends that only one injury occurred during the 1980's. There was no second accident, but merely a progression of the renal kidney disease caused by heavy metal poisoning.

In response to the Fund's arguments, the plaintiffs contend that Section 15-3-600 is not applicable and Section 42-9-400 does not set forth a statute of limitations by which to commence an action against the South Carolina Second Injury Fund. In short, the plaintiffs assert that because it properly and timely placed the Fund on notice of a claim while litigation involving compensability was still ongoing, it had

complied with the requirements as set forth in Section 42-9-400 *S.C. Code Annot. §42-9-400 (2007).*

Additionally, the plaintiffs noted that the Appellate Panel Decision and Order filed on May 3, 2006 found as a fact and concluded as a matter of law that the claimant was not aware of the compensability of this claim until January 24, 2001, when he was definitively diagnosed as having an occupational disease or suffered from an accidental injury as a consequence of his employment duties. Dr. Howard Hu provided the definitive diagnosis. Therefore, the plaintiffs argued that even if a ten year statute of limitations statute is applicable in this case pursuant to Section 15-3-600, such did not begin to run until January 24, 2001, which is the earliest date the parties became aware of a potential compensable claim.

Finally, with respect to the Fund's assertion that the claimant did not suffer subsequent injuries by accident, but rather his disability was as a result of a progression of a progressive occupational disease, it was noted that the Hearing Commissioner and Appellate Panel concluded as a matter of law that the claimant's disability was clearly as a consequence of continuous exposure to lead or from heavy metal lead poisoning, which "either caused, accelerated, or aggravated" the claimant's kidney disease.

EVIDENCE OF THE CASE

Lee Ernest Franklin, the claimant, is a 54-year-old married male, who graduated from high school. Mr. Franklin also attended technical college for four years. Mr. Franklin testified that he was employed at Yuasa Exide, Inc. for 23 years and nine

months. Prior to his employment at Yuasa Exide, Inc., the claimant had worked at V. B. Williams Furniture Company.

The claimant testified that when he began working at Yuasa Exide, Inc., formerly known as The Exide Plant and Exide Battery in 1974, he was given a physical. The claimant stated that he no health problems when he began his work at Exide with respect to high blood pressure or kidney problems.

Mr. Franklin stated that he first began working as a stacking machine operator, which required him to stack negative and positive plates in the battery manufacturing process. The claimant testified that he was commonly exposed to lead dust when he began his work at The Exide Plant as a stacking machine operator in 1974 (Hrg. Tr. pp. 14-15).

Subsequently, the claimant was assigned to work in the pasting operation department. The pasting operation required Mr. Franklin to mix powdered lead with a formula of acid into a paste. The paste was then compressed into the grids, which in turn were utilized to manufacture a plate (Hrg. Tr. p. 15, ll. 3-11). The claimant further testified that the lead powder was stored in drums, which was dumped into a large mixer. According to the claimant, when the drums of lead were dumped, lead dust was in the environment (Hrg. Tr. p. 15, ll. 12-25). The claimant stated that during this operation, dust would collect on him and on the floor. Mr. Franklin testified that he worked as a pasting operator for approximately seven years (Hrg. Tr. p. 15-16).

The claimant was later transferred to the material handlers operation (Hrg. Tr. p. 16). In this manufacturing process, the claimant welded lead plates with a torch. The

claimant indicated that when welding lead plates, smoke and dust was created (Hrg. Tr. p. 17, ll. 2-12). Furthermore, the claimant indicated that as a material handler, he was required to transport material to other departments (Hrg. Tr. p. 17, ll. 13-25). During the transporting of various materials, he was exposed to dust. The claimant worked in the materials handling department for approximately seven years.

The claimant testified that his employment in the stacking, pasting and materials handlers departments were all considered high lead levels. The claimant last worked in the shipping department, which is a low lead area (Hrg. Tr. p. 21, ll. 22-25; p. 22, l. 1).

The claimant testified that he began having kidney problems in the early 1980's (Hrg. Tr. p. 32, ll. 21-25). As a matter of fact, the claimant admitted on cross-examination that when he was informed that he was losing protein, he knew it was a sign of kidney failure (Hrg. Tr. p. 32, ll. 21-25; p. 33, ll. 1-7). Upon being told that the claimant was having kidney failure, the claimant was moved from the pasting department to a less lead-laden environment (Hrg. Tr. p. 33, ll. 2-7). The claimant further admitted that he did not have to be moved while he was employed in the stacking department due to high lead levels, but was first moved once he began working in the pasting department during the late 1970's through 1983 (Hrg. Tr. p. 33).

The claimant testified that over the course of his employment at Yuasa Exide, Inc., he was removed from his employment duties in a particular department and reassigned to another department until his lead levels returned to an acceptable level (Hrg. Tr. p. 34-36).

MEDICAL EVIDENCE

Dr. Eric Byrd is an occupational medical physician, who provided medical services for Yuasa Exide's employees. Dr. Byrd testified via deposition that he has attended seminars concerning lead and lead exposure. Dr. Byrd further testified that he has reviewed studies regarding lead exposure and kidney function as a consequence of his work duties as an occupational medicine physician (Depo. Tr. pp. 17-19).

Dr. Byrd testified that over a three-year period from July 1976 through July 1979, the claimant's blood lead levels range from 60 to 91 micrograms per deciliter of blood (Depo. Tr. p. 23, ll. 9-17). According to Dr. Byrd, during this three year period of time, Mr. Franklin's blood lead levels generally were above 70 micrograms per deciliter of blood (Depo. Tr. p. 23, ll. 18-19).

Dr. Byrd also testified that from May 1980 through August 1984, Mr. Franklin's blood lead levels ranged from 40 to 60 micrograms per deciliter of blood (Depo. Tr. p. 23, ll. 20-25 and p. 24, ll. 1-8). Finally, from November 1983 through January 1989, the claimant's blood lead levels range from 31 to 57 micrograms per deciliter of blood.

Dr. Byrd testified that Yuasa Exide, Inc. monitored the employees' blood lead levels to prevent any type of illnesses that result from lead exposure. The removal process begins when an individual's blood lead levels reach 50 micrograms per deciliter of blood or greater (Depo. Tr. p. 28).

Dr. Byrd testified that beginning in 1981, it was discovered that Mr. Franklin had abnormalities of BUN or creatinine with respect a potential problem with his kidneys (Depo. Tr. p. 43). The claimant was requested to make an appointment with an

urologist, Dr. Cone, to discuss the elevation of BUN. Dr. Byrd testified that he was concerned that the claimant's kidney problems were related to lead exposure and discussed such with him and made an appointment for him to see Dr. Pusadee Suchinda, a nephrologist (Depo. Tr. p. 43 and 44, ll. 1-25; p. 45, ll. 1-10).

Dr. Byrd further testified that in June 1985, Mr. Franklin had stated in a roundtable discussion with former plant physician, Dr. Hursey. Dr. Hursey told the claimant to seek other employment because of his chronic renal condition and continuous lead exposure. Dr. Byrd testified from his review of the notes that Dr. Hursey had indicated that he had discussed the claimant's employment duties and his kidney condition on numerous occasions and that the claimant should not work around lead (Depo. Tr. p. 47, ll. 2-25). Dr. Byrd went on to explain that he was of the opinion that Mr. Franklin's exposure to lead in the early years of employment first caused or contributed renal problems. However, he was of the opinion that Mr. Franklin's subsequent employment duties, which did not result in his blood lead levels exceeding 50 or 60 micrograms per deciliter of blood, had little affect upon the claimant's kidney function (Depo. Tr. p. 50, ll. 3-18). According to Dr. Byrd, there is no doubt that the claimant's exposure to high levels of lead in the 1970's and early 1980's caused damage to the claimant's kidneys (Depo. Tr. p. 54, ll. 16-23).[1]

Next, Dr. Eugene R. Shippen, a board certified physician and expert in lead toxicology, had testified in the merits hearing on behalf of the defendants. According to Dr. Shippen's testimony, he has approximately 25 years of practical experience in

[1] By 1988, Mr. Franklin had already been removed to a low lead level area wherein the lead in the air is less than 30 micrograms per cubic meter.

dealing with lead toxicology. Dr. Shippen is a member of the American Occupation Society Environmental Clinic Panel, which consists of nationally known American lead experts. Dr. Shippen completed a medical questionnaire about the aggravating effect of lead upon the claimant's health maladies.

The claimant retained Dr. Howard Hu, a Professor of Occupational and Environmental Medicine at the Harvard School of Public Health. In a report dated July 25, 2001, Dr. Hu noted that the claimant began with a pre-employment blood lead level of 28 ug/dL. When the claimant's blood lead level was next measured on February 11, 1975, it was 50 ug/dL. Dr. Hu noted that this substantial increase resulted from the claimant's occupational lead exposure. Dr. Hu noted that the claimant's blood lead levels fluctuated between 36 and 69 ug/dL until March 22, 1978, when it increased to 80 ug/dL. A peak blood lead level of 91 ug/dL was observed on August 21, 1978. Subsequently, the blood lead levels of Mr. Franklin fell to 70 ug/dL and below.

Dr. Hu opined that Mr. Franklin's occupational lead level exposure at Yuasa Exide, Inc. was a major contributing factor towards the causation of his renal failure. In particular, Dr. Hu opined that the claimant was exposed to lead at a level that exceeded OSHA Regulations and is associated with a toxic effect on the kidney. According to Dr. Hu, the OSHA standard requiring medical removal protection is 50 ug/dL. Dr. Hu noted that the claimant's blood lead levels exceeded the OSHA Regulations for most of the time that he worked at Yuasa up until the point where the first elevated serum creatinine was detected in 1981. Dr. Hu noted that this excessive exposure to lead in violation of OSHA Regulations continued during the 1980's; and that this "high

occupational exposure over 10+ years that result in body burdens typically associated with chronic renal failure..."

Dr. Hu later opined that subsequent exposures to lead at work aggravated the renal kidney disease and accelerated kidney failure.

Based upon the foregoing arguments, a review of the deposition testimonies, and the entire record, the following findings of fact are made:

FINDINGS OF FACT

1. Yuasa Exide, Inc. and Great American Insurance Company, the plaintiffs, clearly proved by a preponderance of the evidence that it is subject to reimbursement from the South Carolina Second Injury Fund with respect to the accidental injury to the kidneys or occupational disease suffered by Lee Ernest Franklin during the course and scope of his employment with Yuasa Exide, Inc. from 1974 through 1996.

2. The plaintiffs' claim is not barred by Section 15-3-600. First, there is no case law or any statutory law reflecting that a ten year statute of limitation is applicable in claims for reimbursement against the South Carolina Second Injury Fund. The only requirement in order to obtain funds from the Second Injury Fund with respect to a time limitation is set forth in Section 42-9-400. Specifically, if an employer and carrier does not file a claim prior to paying 78 weeks of benefits, the claim is deemed barred.

3. Assuming *arguendo* that a ten year statute of limitations defense is applicable with respect to Second Injury Fund claims, the instant case would not be subject to the ten year statute of limitations for the following reasons. The employer, carrier and claimant did not know whether the claim would be deemed compensable

until after all appeals had been exhausted. A notice of intent to appeal was filed with the South Carolina Court of Appeals but was subsequently withdrawn in 2007. Therefore, once the plaintiffs withdrew its appeal, the Order of the Circuit Court affirming the Commission's finding of compensability became the law of the case. The Circuit Court's Decision was promulgated on August 21, 2006. It appears that at the latest the parties recognized that the claim was, indeed, compensable and subject to reimbursement occurred on August 21, 2006. Therefore, the ten year statute of limitations would not begin to run until the date of the Circuit Court Order.

On the other hand, the earliest date upon which the ten year statute of limitations could potentially begin to run would be when the claimant first became aware as a matter of law and from a medical standpoint that he had sustained a compensable injury or malady as a consequence of his employment duties at Yuasa Exide, Inc. According to the Orders of the Commission and the Court, Lee Ernest Franklin was unaware of a compensable malady until January 24, 2001. The Full Commission Appellate Panel concluded that January 24, 2001 constituted the date of a definitive diagnosis. Prior to January 24, 2001, there was absolutely no evidence of a compensable accidental injury or occupational disease. Therefore, there was no basis for the plaintiff's to have filed a claim for reimbursement. Accordingly, the earliest date by which a theoretical ten year statute of limitations could be applied would be from January 24, 2001. Clearly, the plaintiffs filed a claim for reimbursement well before the expiration of a ten year statute of limitations if either the dates of 2001 and 2006 is utilized.

3. The plaintiffs proved by a preponderance of the evidence that it is entitled to reimbursement from the Second Injury Fund for benefits paid and payable to Lee Ernest Franklin as a consequence of heavy metal poisoning resulting from continuous lead exposure from 1974 through 1995. These exposures resulted in continuous and aggressive injuries. The continued exposure to lead accelerated the claimant's kidney failure. According to the Full Commission Appellate Panel's Decision and Order the subsequent exposures to lead accelerated and aggravated the claimant's renal kidney disease and the damage resulting from renal kidney disease. The subsequent exposures to lead was a direct cause of the renal kidney disease progressing from Stage I to Stage V. Absent the claimant's employment duties effect on his hypertension and renal kidney disease, the claimant's disease would not have progressed in the manner which it did as a result of his continuous exposure to lead.

4. Because the claimant suffered from renal kidney failure caused by heavy metal (lead) poisoning, the claim is subject to reimbursement. Because heavy metal poisoning is a listed disease, it is presumed that it constitutes a hindrance and obstacle to employment; and that such a condition leads to substantially higher medical costs and disability in excess of 78 weeks.

5. The claim for reimbursement from the Second Injury Fund can also be predicated upon the claimant's preexisting disease of hypertension, which was clearly aggravated and accelerated by his constant subsequent exposures to lead until he last worked at Yuasa Exide, Inc. The hypertensive condition clearly qualifies this claim for

reimbursement as is evidenced by the Second Injury Fund medical questionnaire completed by Dr. Eugene Shippen; and through his testimony.

16. The Second Injury Fund's claim that there was no subsequent accidents which aggravated or accelerated the claimant's renal kidney disease is not supported by the evidence or the law. The Fund introduced no evidence to contradict the opinions of Drs. Richard Wedeen, Shippen, Hu, and Byrd. Additionally, in the case of the State Worker's Compensation Fund v. The South Carolina Second Injury Fund in RE: Hunt v. S.C. State Forestry Commission, 313 S.C. 536, 443 S.E.2d 546 (1994), the Court noted that a claimant's arteriosclerosis and cardiac disease qualified as a subsequent disability when the worker became disabled as a consequence of the preexisting malady. In short, the Court rejected the Fund's argument that an employer and carrier are only entitled to reimbursement if a subsequent injury takes the form of some observable event producing an injury. 313 S. C. 536, 539, 443 S.E.2d 546, 548.

17. The record is replete with evidence that the goals of the Second Injury Fund were met in this case. Yuasa Exide retained Lee Ernest Franklin in employment after knowledge of his prior impairments caused by renal kidney failure beginning in Stage I and continuing through Stage V. The claimant constantly was moved to various departments to minimize his exposure to lead. Moreover, the employer provided additional safety measures to reduce the claimant's exposure to lead and to minimize or eliminate subsequent accidental injuries.

18. During the claimant's continued employment he incurred a subsequent disability from the injury by accident and/or by occupational disease arising out of and

in the course of his employment, which ultimately resulted in end stage renal kidney failure.

9. The claimant's last injury resulting from lead exposure combined with the last stage of renal kidney disease prior to Stage V and/or aggravated the preexisting condition to cause a substantially greater disability and would have been caused from the subsequent injury alone; which was as a result of being exposed to lead within permissible guidelines as established by OSHA.

10. The end stage renal kidney disease most probably would not have occurred but for the preexisting hypertensive conditions and the preexisting first four stages of renal kidney disease.

11. The claimant's extensive hypertension would qualify for payment of 78 weeks or more in disability benefits.

CONCLUSIONS OF LAW

1. S.C. Code Annot. §42-1-160 (2007) governs accidental injuries.

2. S.C. Code Annot. §42-11-90 (2007) governs occupational diseases.

3. S.C. Code Annot. §42-9-400 (2007) governs South Carolina Second Injury Fund claims.

ORDER

IT IS HEREBY ORDERED that the South Carolina Second Injury Fund shall reimburse Yuasa Bdde, Inc. and Great American Insurance Company and its assigns or

agents for all benefits paid to and payable to Lee Ernest Franklin in accordance with the statute.

AND IT IS SO ORDERED.

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION

George N. Funderburk, Commissioner

Columbia, South Carolina

January 24, 2008

CERTIFICATE OF SERVICE

This is to certify that the undersigned has this date served this order in the above entitled action upon all parties to this cause by depositing a copy hereof, postage paid, in the United States mail addressed to the attorney or attorneys for said parties.

This 31 day of January, 2008

Elaine M. Boyd

Administrative Assistant to the Commissioner

VPO
LDE

GRN 222645v1

Switch Client | Preferences | Help | Sign Out

Search | Get a Document | Shepard's® | More | History | Alerts

FOCUS™ Terms | Search Within: Original Results (1 - 100) | View Tutorial

Advanced...

Source: Legal > States Legal - U.S. > South Carolina > Find Cases > SC State Cases, Combined

Terms: yuasa exide and fund (Suggest Terms for My Search | Feedback on Your Search)

Select for FOCUS™ or Delivery

*389 S.C. 422, *; 699 S.E.2d 687, **;*
*2010 S.C. LEXIS 320, ****

Transportation Insurance Company and Flagstar Corporation, Carrier/Respondent, v. South Carolina Second Injury **Fund**, Appellant. Travelers Insurance Company and Barclays American, Carrier/Respondent, v. South Carolina Second Injury **Fund**, Appellant. Travelers Insurance Company and It's Fashion, Carrier/Respondent, v. South Carolina Second Injury **Fund**, Appellant. Great American Insurance Co. and **Yuasa Exide**, Inc., Carrier/Respondent, v. South Carolina Second Injury **Fund**, Appellant. Travelers Insurance Company and Marc Knapp DBA, Carrier/Respondent, v. South Carolina Second Injury **Fund**, Appellant. Travelers Insurance Company and All American Termite & Pest, Carrier/Respondent, v. South Carolina Second Injury **Fund**, Appellant.

Opinion No. 26880

SUPREME COURT OF SOUTH CAROLINA

389 S.C. 422; 699 S.E.2d 687; 2010 S.C. LEXIS 320

May 12, 2010, Heard
September 13, 2010, Filed

SUBSEQUENT HISTORY: Rehearing denied by Transp. Ins. v. SC Second Injury **Fund**, 2010 S.C. LEXIS 337 (S.C., Oct. 6, 2010)

DISPOSITION: **[***1]** REVERSED.

CASE SUMMARY

PROCEDURAL POSTURE: Respondent insurers brought proceedings against appellant Second Injury **Fund** (**fund**), seeking reimbursement in cases in which more than ten years passed since claimants' date of injury. The South Carolina Workers' Compensation Commission determined that the statute of limitations found in S.C. Code Ann. § 15-3-600 did not apply to reimbursement cases. The **fund** appealed.

OVERVIEW: The **fund** argued that the ten-year limitations period of § 15-3-600 applied to reimbursement cases brought pursuant to S.C. Code Ann. § 42-9-400. The appellate court agreed. S.C. Code Ann. §§ 15-3-20(A), 15-3-600 mandated that reimbursement actions had to commence within ten years after the cause of action accrued. Because a reimbursement action was an action for relief not provided for in the chapter and there was no statute of limitations in S.C. Code Ann. tit. 42 that applied to claims for reimbursement, § 15-3-600 applied to claims for reimbursement. The **fund** also argued that a cause of action for

reimbursement accrued on the date of the claimant's injury. The appellate court disagreed. The proper time for accrual to begin was the date notice is given to the **fund.** The equitable doctrine of laches did not bar reimbursement in this case. The **fund's** laches arguments were not properly before the appellate court either because the issue was not preserved or the issue was unappealed and was now the law of the case.

OUTCOME: The decisions of the Workers' Compensation Commission were reversed.

CORE TERMS: reimbursement, statute of limitations, laches, carrier, citation omitted, civil actions, claimant's, notice, cause of action, preserved, date of injury, full commission, accrued, default, accrual, accrue, Reform Act, proper notice, entitled to reimbursement, abandoned, pursued, notice requirement, whole record, express terms, unambiguous, prescribed, unappealed, insured, insures, notify

LEXISNEXIS® HEADNOTES Hide

Governments > Legislation > Statutes of Limitations > Time Limitations

Workers' Compensation & SSDI > Benefit Determinations > Cumulative & Successive Disabilities

HN1 See S.C. Code Ann. § 15-3-600 (2005).

Civil Procedure > Appeals > Standards of Review > De Novo Review

Governments > Legislation > Interpretation

HN2 Statutory interpretation is a question of law subject to de novo review. More Like This Headnote | *Shepardize:* Restrict By Headnote

Administrative Law > Judicial Review > Standards of Review > Statutory Interpretation

Governments > Legislation > Interpretation

HN3 The construction of a statute by the agency charged with its administration will be accorded the most respectful consideration and will not be overruled absent compelling reasons. More Like This Headnote

Administrative Law > Judicial Review > General Overview

Workers' Compensation & SSDI > Administrative Proceedings > Judicial Review > General Overview

HN4 The Administrative Procedures Act governs judicial review of the Workers' Compensation Commission's decisions. More Like This Headnote | *Shepardize:* Restrict By Headnote

Administrative Law > Judicial Review > Standards of Review > Substantial Evidence

Workers' Compensation & SSDI > Administrative Proceedings > Judicial Review > Standards of Review > Substantial Evidence

HN5 Substantial evidence is not a mere scintilla of evidence nor evidence viewed from one side, but such evidence, when the whole record is considered, as would allow reasonable minds to reach the conclusion the Full Commission

reached. More Like This Headnote | *Shepardize:* Restrict By Headnote

Governments > Legislation > Statutes of Limitations > General Overview

HN6 Statutes of limitations are not simply technicalities. Statutes of limitations embody important public policy considerations in that they stimulate activity, punish negligence, and promote repose by giving security and stability to human affairs. Statutes of limitations relieve courts of the burden of trying stale claims of those who have slept on their rights. The purpose of statutes of limitation is to ensure litigation is brought within a reasonable time in order that evidence be reasonably available and there be some end to litigation. More Like This Headnote

Governments > Legislation > Statutes of Limitations > General Overview

Governments > Legislation > Statutes of Limitations > Time Limitations

HN7 See S.C. Code Ann. § 15-3-600.

Governments > Legislation > Statutes of Limitations > General Overview

Governments > Legislation > Statutes of Limitations > Time Limitations

HN8 So that other unnamed civil actions were not excluded from having a limitations period, the legislature created the S.C. Code Ann. § 15-3-600 ten-year default statute of limitations provision for other causes of action not specifically enumerated in S.C. Code Ann. tit. 15. More Like This Headnote

Governments > Legislation > Statutes of Limitations > General Overview

HN9 See S.C. Code Ann. § 15-3-20(A) (2005).

Governments > Legislation > Interpretation

Workers' Compensation & SSDI > Administrative Proceedings > Judicial Review > General Overview

HN10 When reading a workers' compensation statute, the Supreme Court of South Carolina will strictly construe its terms, leaving it to the legislature to amend and define any ambiguities. More Like This Headnote | *Shepardize:* Restrict By Headnote

Governments > Legislation > Interpretation

HN11 The cardinal rule of statutory construction is to ascertain and effectuate the intent of the legislature. The text of a statute as drafted by the legislature is considered the best evidence of the legislative intent or will. If a statute's language is plain, unambiguous, and conveys a clear meaning, then the rules of statutory interpretation are not needed and a court has no right to impose another meaning. A court will give words their plain and ordinary meaning, and will not resort to a subtle or forced construction that would limit or expand the statute's operation. More Like This Headnote | *Shepardize:* Restrict By Headnote

Workers' Compensation & SSDI > Benefit Determinations > Cumulative & Successive Disabilities

HN12 See S.C. Code Ann. § 42-9-400(f).

Governments > Legislation > Statutes of Limitations > Time Limitations

Workers' Compensation & SSDI > Administrative Proceedings > Claims > Time Limitations > General Overview

Workers' Compensation & SSDI > Administrative Proceedings > Claims > Time Limitations > Notice Periods

Workers' Compensation & SSDI > Benefit Determinations > Cumulative & Successive Disabilities

HN13 S.C. Code Ann. § 42-9-400(f) is not a statute of limitations, but a notice requirement. Moreover, there is no statute of limitations in S.C. Code Ann. tit. 42 that applies to claims for reimbursement. Reimbursement actions are actions for relief not provided for in the chapter. S.C. Code Ann. § 15-3-600 (2005). Hence, S.C. Code Ann. §§ 15-3-20(A), 15-3-600 mandate that reimbursement actions must commence within ten years after the cause of action shall have accrued. More Like This Headnote

Governments > Legislation > Statutes of Limitations > General Overview

Workers' Compensation & SSDI > Administrative Proceedings > Claims > Time Limitations > General Overview

HN14 The Court of South Carolina has rejected the contention that the two-year statute of limitations for employees' workers' compensation claims contained in S.C. Code Ann. § 42-15-40 (Supp. 2009) also governed claims for reimbursement. More Like This Headnote

Governments > Legislation > Statutes of Limitations > General Overview

Workers' Compensation & SSDI > Administrative Proceedings > Claims > Time Limitations > General Overview

HN15 Because a reimbursement action is an action for relief not provided for in the chapter and there is no statute of limitations in S.C. Code Ann. tit. 42 that applies to claims for reimbursement, S.C. Code Ann. § 15-3-600 applies to claims for reimbursement. For these reasons, § 15-3-600 applies to claims for reimbursement. More Like This Headnote

Governments > Legislation > Statutes of Limitations > Time Limitations

Workers' Compensation & SSDI > Administrative Proceedings > Claims > Time Limitations > General Overview

Workers' Compensation & SSDI > Benefit Determinations > Cumulative & Successive Disabilities

HN16 The proper time for accrual of a claim for reimbursement begins is the date notice is given to the Second Injury **Fund.** More Like This Headnote

Civil Procedure > Appeals > Reviewability > Preservation for Review

HN17 Issues and arguments are preserved for appellate review only when they are raised to and ruled on by the lower court. An unappealed ruling is the law of the case and requires affirmance. More Like This Headnote | *Shepardize:* Restrict By Headnote

Civil Procedure > Appeals > General Overview

Civil Procedure > Appeals > Briefs

Civil Procedure > Appeals > Reviewability > Preservation for Review

HN18 An issue is deemed abandoned where the appellant fails to provide arguments or supporting authority for his assertion. Short, conclusory statements made without supporting authority are deemed abandoned on appeal and therefore not preserved for review. More Like This Headnote | *Shepardize:* Restrict By Headnote

COUNSEL: Latonya Dilligard Edwards, of Columbia, for Appellant.

Deborah Casey Brown and Kyle A. Hougham, both of Gallivan, White & Boyd, Jason Alexander, Griggs, of Wilson, Jones, Carter & Baxley, and Vernon F. Dunbar, of Turner, Padget, Graham & Laney, all of Greenville; Stephen L. Brown, William L. Howard, and Russell G. Hines, all of Young, Clement & Rivers, of Charleston, for Respondents.

JUDGES: CHIEF JUSTICE TOAL. KITTREDGE and HEARN, JJ., concur. PLEICONES, J., concurring in part and dissenting in part in a separate opinion in which Acting Justice James E. Moore, concurs.

OPINION BY: TOAL

OPINION

[*425] [688] CHIEF JUSTICE TOAL:** We granted South Carolina Second Injury **Fund's (Fund)** motion to transfer several appeals from the circuit court to this Court. We reverse the decisions of the South Carolina Workers' Compensation Commission.

[*426] [689] FACTS/PROCEDURAL HISTORY**

On June 20, 2007, the South Carolina General Assembly passed the Workers' Compensation Reform Act (Reform Act), which provides for the winding down of the **Fund** by June 30, 2013. *See* S.C. Code Ann. § 42-7-320(A) (Supp. 2009). **[***2]** Subsequent to the passage of the Reform Act, several carriers requested reimbursement for claims in which more than ten years had passed since the claimant was injured. The **Fund** denied reimbursement in those cases based on the general ten-year statute of limitations, which provides *HN1* "[a]n action for relief not provided for in this chapter must be commenced within ten years after the cause of action shall have accrued." S.C. Code Ann. § 15-3-600 (2005). Carriers in the present action pursued reimbursement in cases in which more than ten years passed since claimant's date of injury.

Travelers Insurance Company (Travelers) insured four of the six employers involved in this action. The four employers insured by Travelers are Barclay's American, It's Fashions, All American Termite & Pest, and Marc Knapp. [1] Travelers is requesting reimbursement for claimants with the following dates of injury: December 19, 1989; July 18, 1994; August 19, 1996; and August 8, 1997. Travelers provided timely and proper notice to the **Fund** pursuant to South Carolina Code Ann. § 42-9-400 (Supp. 2009) in all four of these claims.

FOOTNOTES

1 The **Fund** believes Marc Knapp, It's Fashions, and All American Termite & Pest **[***3]** are no longer in business because no information was discovered on the Secretary of State's database for these businesses.

Transportation Insurance Company (Transportation) insures Flagstar Corporation and is seeking reimbursement for a claimant with a March 19, 1993 date of injury. Transportation provided **[*427]** proper notice of a possible claim to the **Fund** pursuant to section 42-9-400.

Great American Insurance Company (Great American) insures **Yuasa Exide**, Inc., which is now known as EnerSys Corporation. Great American is seeking reimbursement for a January 1, 1996 date of injury. [2] Great American provided proper notice of a possible claim to the **Fund** pursuant to section 42-9-400. [3]

FOOTNOTES

[2] The commission found the date of injury to be January 24, 2001. January 1, 1996 is the **Fund's** position for the date of injury.

The cases referenced above have been adjudicated by the Workers' Compensation Commission, which ultimately determined the statute of limitations found in section 15-3-600 did not apply to reimbursement cases. The **Fund** appealed these cases to the circuit court. However, while the appeals were pending, the **Fund** sought review by this Court and we transferred the appeals from the circuit court **[***4]** to this Court.

ISSUES

I. Does the statute of limitations provision in section 15-3-600 apply to reimbursement cases pursued against the **Fund?**

II. When does time begin to accrue for claims brought under section 42-9-400?

III. Does laches apply such that the carriers outlined above are not entitled to reimbursement?

IV. Under section 42-9-400 is **Yuasa Exide** entitled to reimbursement?

STANDARD OF REVIEW

HN2 Statutory interpretation is a question of law subject to de novo review. *Town of Summerville v. City of N. Charleston*, 378 S.C. 107, 110, 662 S.E.2d 40, 41 (2008) (citation omitted). HN3 "The construction of a statute by the agency charged with its administration will be accorded the most respectful consideration and will not be overruled absent compelling reasons." *Dunton v. S.C. Bd. of Exam'rs In Optometry*, 291 S.C. 221, 223, 353 S.E.2d 132, 133 (1987) (citations omitted). HN4 The Administrative Procedures Act governs judicial review of the Workers' Compensation Commission's decisions. *See Lark v. Bi-Lo, Inc.*, 276 S.C. 130, 276 S.E.2d 304 (1981). This Court can modify the commission's decision in this case only if the **Fund's** substantial rights have been prejudiced because the decision is affected by **[***5]** an **[**690]** error of law or is clearly erroneous in view of the reliable, probative, and substantial evidence on the whole record. See S.C. Code Ann. § 1-23-380(5)(d), (e) (Supp. 2009); *see also* **[*428]** *Shealy v. Aiken County*, 341 S.C. 448, 454-55, 535 S.E.2d 438, 442 (2000). HN5 "Substantial evidence is not a mere scintilla of evidence nor evidence viewed from one side, but such evidence, when the whole record is considered, as would allow reasonable minds to reach the conclusion the Full Commission reached." *Shealy*, 341 S.C. at 455, 535 S.E.2d at 442 (citation omitted).

LAW/ANALYSIS

I. Statute of Limitations

The **Fund** argues the ten-year statute of limitations period outlined in section 15-3-600 applies to reimbursement cases brought pursuant to section 42-9-400. We agree.

HN6 "Statutes of limitations are not simply technicalities." *Moates v. Bobb*, 322 S.C. 172, 176, 470 S.E.2d 402, 404 (Ct. App. 1996). "Statutes of limitations embody important public policy considerations in that they stimulate activity, punish negligence, and promote repose by giving security and stability to human affairs." *Id.* (citation omitted). Statutes of limitations relieve courts of the burden of trying stale claims of those who have **[***6]** slept on their rights. *See McKinney v. CSX Transp., Inc.*, 298 S.C. 47, 49-50, 378 S.E.2d 69, 70 (Ct. App. 1989). "The purpose of statutes of limitation is to ensure litigation is 'brought within a reasonable time in order that evidence be reasonably available and there be some end to litigation.'" *Hooper v. Ebenezer Senior Servs. & Rehab. Ctr.*, 377 S.C. 217, 227, 659 S.E.2d 213, 218 (Ct. App. 2008) (quoting *City of North Myrtle Beach v. Lewis-Davis*, 360 S.C. 225, 231, 599 S.E.2d 462, 464 (Ct. App. 2004)).

Section 15-3-600 is found in chapter 3 of title 15 which addresses "Limitations of Civil Actions." Section 15-3-600 states, HN7 "An action for relief not provided for in this chapter must be commenced within ten years after the cause of action shall have accrued." HN8 So that other unnamed civil actions were not excluded from having a limitations period, the legislature created this ten-year default statute of limitations provision for other causes of action not specifically enumerated in title 15. South Carolina Code Ann. § 15-3-20(A) (2005) provides, HN9 "Civil actions may only be commenced within the periods prescribed in this title after the cause of action has accrued, **[*429]** except when, in special **[***7]** cases, a different limitation is prescribed by statute."

HN10 When reading a workers' compensation statute this Court will strictly construe its terms, leaving it to the legislature to amend and define any ambiguities. *Wigfall v. Tideland Utils., Inc.*, 354 S.C. 100, 110, 580 S.E.2d 100, 105 (2003). HN11 "The cardinal rule of statutory construction is to ascertain and effectuate the intent of the legislature." *Hodges v. Rainey*, 341 S.C. 79, 85, 533 S.E.2d 578, 581 (2000) (citation omitted). The text of a statute as drafted by the legislature is considered the best evidence of the legislative intent or will. *See id.* "If a statute's language is plain, unambiguous, and conveys a clear meaning, then the rules of statutory interpretation are not needed and a court has no right to impose another meaning." *Strickland v. Strickland*, 375 S.C. 76, 88, 650 S.E.2d 465, 472 (2007) (citation omitted). "The Court will give words their plain and ordinary meaning, and will not resort to a subtle or forced construction that would limit or expand the statute's operation." *Harris v. Anderson County Sheriff's Office*, 381 S.C. 357, 362, 673 S.E.2d 423, 425 (2009) (citation omitted).

Section 42-9-400 is plain and unambiguous. **[***8]** Section 42-9-400(f) states in pertinent part:

> HN12 An employer or his carrier must notify the Workers' Compensation Commission and the Director of the Second Injury **Fund** in writing of any possible claim against the **fund** as soon as practicable but in no event later than after the payment of the first seventy-eight weeks of compensation. . . . Failure to comply with the provisions of this subsection shall bar an employer or his carrier from recovery from the **fund.**

HN13 This is not a statute of limitations, but a notice requirement. Moreover, there is no statute of limitations in title 42 that applies to claims for reimbursement. Reimbursement

[691]** actions are actions "for relief not provided for in this chapter." S.C. Code Ann. § 15-3-600 (2005). Hence, sections 15-3-20(A) and 15-3-600 mandate that reimbursement actions must commence within ten years after the cause of action shall have accrued. If this were not so, then so long as the notice requirement of 42-9-400(f) was met, these actions could theoretically **[*430]** extend endlessly into the future, thwarting the intent of the legislature's passing of section 15-3-600.

In *Greenwood Mills, Inc. v. Second Injury* **Fund,** 315 S.C. 256, 433 S.E.2d 846 (1993), HN14 this **[***9]** Court rejected the contention that the two-year statute of limitations for employees' workers' compensation claims contained in S.C. Code Ann. § 42-15-40 (Supp. 2009) also governed claims for reimbursement. *Greenwood Mills,* 315 S.C. at 259, 433 S.E.2d at 848. The question presented in this case was not before the Court in *Greenwood Mills. Greenwood Mills* merely addressed whether section 42-15-40 applied to claims for reimbursement. We found that, on its face, section 42-15-40 was inapplicable to claims for reimbursement. *Id.* at 259, 433 S.E.2d at 847. The Court noted that section 42-15-40 "applies to claims for *compensation,* not *reimbursement;* it governs claims addressed to the *Commission* as opposed to those addressed to the ***Fund;*** it speaks to an *employee's* injury, not to an *employer's* notice of injury." *Id.* (emphasis in original). Like this Court did in *Greenwood Mills,* we must apply the default statute of limitations according to its express terms. HN15 Because a reimbursement action is an "action for relief not provided for in this chapter" and there is no statute of limitations in title 42 that applies to claims for reimbursement, section 15-3-600 applies to claims for reimbursement. **[***10]** For these reasons, section 15-3-600 applies to claims for reimbursement, and the decisions of the Workers' Compensation Commission are reversed.

II. Accrual of Time

The **Fund** argues a cause of action for reimbursement accrues on the date of claimant's injury. We disagree.

The **Fund** contends there are three possible dates on which time starts to accrue: (1) date of claimant's injury; (2) date carrier provides notice to the **Fund;** and (3) when the underlying claims conclude. The date the claimant is injured is not the proper time because at that time, in most instances, carriers would not be aware of a possible claim to the **Fund.** We find HN16 the proper time for accrual to begin is the date notice is given to the **Fund.** By the time notice is given, carriers are aware of a possible claim and have up to 78 weeks after paying benefits to notify the **Fund** of a claim. Hence, a cause of **[*431]** action for reimbursement begins to accrue on the date a carrier provides notice to the **Fund.**[3]

FOOTNOTES

3 The result in this case of accrual beginning when notice is provided is that Great American's claim is the only one that is not precluded by the running of the ten-year statute of limitations.

III. Laches

The **Fund** argues that if **[***11]** the ten-year statute of limitation does not apply, the equitable doctrine of laches bars reimbursement in this case. We disagree.

HN17 "Issues and arguments are preserved for appellate review only when they are raised to and ruled on by the lower court." *Elam v. S.C. Dep't of Transp.,* 361 S.C. 9, 23, 602 S.E.2d 772, 779-80 (2004) (citations omitted). An unappealed ruling is the law of the case and requires affirmance. *See Charleston Lumber Co., Inc. v. Miller Housing Corp.,* 338 S.C. 171, 175, 525 S.E.2d 869, 871 (2000) (citations omitted).

The **Fund's** laches claim against Flagstar is not preserved because it failed to plead laches and the full commission did not rule on it. The **Fund's** laches arguments against Barclay's American, Marc Knapp, and It's Fashions are not preserved because it did not raise laches in the courts below. The **Fund's** laches argument regarding **Yuasa Exide** is not preserved because the issue was not pled below and neither the single commissioner nor the full commission ruled on laches. The single commissioner made the following finding of fact in All American Termite & Pest's case, "The SIF waived any defense based on the equitable doctrine of laches by failing to assert **[***12]** the affirmative defense on their Form 55 or Form 58." The **[**692]** **Fund** failed to appeal the single commissioner's finding to the full commission; thus, it is the law of the case. In summary, the **Fund's** laches arguments are not properly before this Court either because the issue is not preserved or the issue was unappealed and is now the law of the case. [4]

FOOTNOTES

4 At oral argument there was much discussion regarding laches. While laches does not apply to any of the cases in this appeal, laches could certainly be applicable to cases where carriers seek reimbursement from the **Fund.**

[*432] IV. Yuasa Exide

The **Fund** contends Great American is not entitled to reimbursement because claimant's, hypertension and liver failure did not preexist claimant's occupational exposure. We disagree.

The **Fund** failed to cite any authority for its position. Moreover, the half-page argument made by the **Fund** falls far short of overcoming the substantial evidence standard of review. Hence, the **fund** has abandoned this issue. *See First Sav. Bank v. McLean,* 314 S.C. 361, 363, 444 S.E.2d 513, 514 (1994) (HN16 issue deemed abandoned where appellant failed to provide arguments or supporting authority for his assertion); *Eaddy v. Smurfit-Stone Container Corp.,* 355 S.C. 154, 164, 584 S.E.2d 390, 396 (Ct. App. 2003) **[***13]** ("[S]hort, conclusory, statements made without supporting authority are deemed abandoned on appeal and therefore not preserved for our review.").

CONCLUSION

For the foregoing reasons, the decisions of the Workers' Compensation Commission are reversed.

KITTREDGE, and HEARN, JJ., concur. PLEICONES, J., concurring in part and dissenting in part in a separate opinion in which Acting Justice James E. Moore, concurs.

CONCUR BY: PLEICONES (In Part)

DISSENT BY: PLEICONES (In Part)

DISSENT

JUSTICE PLEICONES: I dissent in part and concur in part, and would affirm the decisions of the Workers' Compensation Commission (Commission) holding that S.C. Code Ann. § 15-3-600 (2005) is inapplicable to these reimbursement requests.

Chapter 3 of Title 15, entitled "Limitation of Civil Actions, "is concerned with the time periods,

i.e. statutes of limitation, within which a 'civil action' can be commenced. A "civil action" within the meaning of Title 15 is brought in a court, and is "commenced when the summons and complaint are filed with the clerk of court..." S.C. Code Ann. § 15-3-20(B) (2005). **[*433]** See also Rule 2, SCRCP: "There shall be one form of action to be known as civil action"; compare McDowell v. S.C. Dep't of Soc. Serv., 304 S.C. 539, 405 S.E.2d 830 (1991) **[***14]** (administrative proceeding is a civil action for purposes of the attorney's fees statute once it reaches circuit court, but not while before agency). On the other hand, a claim for reimbursement from the **Fund** is made by written notice to the Commission and to the Director of the **Fund.** S.C. Code Ann. § 42-9-400(f) (Supp. 2009). I agree with the Commission that S.C. Code Ann. § 15-3-600, the default statute of limitations provision for civil actions, is irrelevant to the question whether carriers timely pursued their reimbursement claims from the **Fund.**

The majority also holds that it is following Greenwood Mills, Inc. v. Second Injury **Fund,** 315 S.C. 256, 433 S.E.2d 846 (1993) by applying "the default statute of limitations [i.e. § 15-3-600] according to its express terms." As I read Greenwood Mills, it holds that the statute of limitations found in S.C. Code Ann. § 42-15-40 did not apply to **Fund** reimbursement claims, but that those claims were timely so long as they are made within the period provided by S.C. Code Ann. § 42-9-400(f) (Supp. 2009). Here, all the carriers gave proper and timely notice as required by § 42-9-400. In my opinion, Greenwood Mills does not support the majority's **[***15]** conclusion that a **Fund** reimbursement request is a "civil action" within the meaning of Title 15.

In light of my view that the Commission correctly found § 15-3-600 inapplicable, I would not reach the accrual issue. Moreover, I agree with the majority that neither **[**693]** the laches question nor the **Yuasa Exide** issue is preserved for our appellate review.

I concur in part and dissent in part.

Acting Justice James E. Moore, concurs.

Source: **Legal > States Legal - U.S. > South Carolina > Find Cases > SC State Cases, Combined**
Terms: **yuasa exide and fund** (Suggest Terms for My Search | Feedback on Your Search)
View: Full
Date/Time: Friday, July 27, 2012 - 10:34 AM EDT

* Signal Legend:
- Warning: Negative treatment is indicated
- Questioned: Validity questioned by citing refs
- Caution: Possible negative treatment
- Positive treatment is indicated
- Citing Refs. With Analysis Available
- Citation Information available

* Click on any *Shepard's* signal to *Shepardize*® that case.

About LexisNexis | Privacy Policy | Terms & Conditions | Contact Us
Copyright © 2012 LexisNexis, a division of Reed Elsevier Inc. All rights reserved.

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION
W.C.C. FILE NO: 9930399

YUASA-EXIDE, INC., Claimant, vs. SOUTH CAROLINA SECOND INJURY FUND, Defendant, IN RE: James W. Huggins C.L. Williams Joe Mathis, Jr. Mary C. McConico, et al.,	**MEMORANDUM OF LAW**

OVERALL CASE SUMMARY ON CHRONIC LEAD EXPOSURE

In 1961, a battery manufacturing facility began operations in Sumter, South Carolina. The company was first known as E.S.B. International Corp. The battery manufacturing facility first produced nickel plated batteries then later converted its manufacturing operations to the production of lead batteries. The company was sold and became known as the Exide Alkaline Battery Division. Over the years, the facility changed ownership numerous times. With the sales of the battery manufacturing operation, it became known by various names such as Exide Power Systems, Exide Corporation, Exide, Yuasa-Exide, Inc., Yuasa, Inc., and EnerSys. (Tab 1A, p. 6).

The lead battery manufacturing operations operated in the same facility from 1965 through December 31, 2001. At the time of the plant's closure in 2001, it was

commonly referred to as "Yuasa-Exide" although the corporate entity operating the facility was known as EnerSys.

Employees working in Yuasa-Exide's grid casting department, battery assembly, formation departments, plate cleaning and pasting departments were exposed to the greatest amounts of lead. There is no dispute that Yuasa-Exide employees were exposed to various amounts of lead on a daily basis through inhalation, ingestion and dermal contact.

Prior to 1980, the Federal Government had not established permissible exposure limits regarding lead. Further, there were no universally recognized industry standards regarding permissible exposure limits to lead. Thus, prior to 1980, chronic exposure to lead often resulted in workers' blood lead levels routinely exceeding 100 micrograms per deciliter of blood (ug/dl). (Tab 2A).

Initially, OSHA had recommended permissible exposure limits not to exceed 80 micrograms per deciliter of blood in 1978. the recommendation was rejected by labor. In 1980, OSHA reduced the permissible exposure limit to 70 micrograms per deciliter of blood after further input from labor and industry. Later, in 1983, OSHA promulgated legally enforceable regulations to reflect that if an individual's blood lead level reached or exceeded 60 micrograms per deciliter of blood, that individual had to be instantly removed from the work environment. The regulations also required that if an individual's blood lead level exceeded 50 micrograms per deciliter of blood over a six month average, that individual had to be removed from the work place or placed into a

medical removal program ("MRP"). Id. See also, *29 C.F.R. 1910-1025(k)(1)(i)(a-b).* (Tab 2A).

OSHA's current permissible exposure limit guidelines require an employer to immediately remove a worker from a lead laden environment when his/her blood lead levels reach 60 micrograms per deciliter of blood. *29 C.F.R. 1926.62 Appendix C (I), et seq.* (Tab 2A). Moreover, if an employee has a blood lead levels of 50 micrograms per deciliter of blood or more for two consecutive glood sampling tests, the worker is to be removed from the work environment in placed in a MRP.

Presently, there is a debate among lead industry experts as to whether the current permissible exposure limit of 60 micrograms per deciliter of blood or a sustained blood lead level of 50 micrograms per deciliter of blood is sufficient to avoid various health maladies associated with chronic lead exposure. A normal blood lead level is less than 10 ug/dl. (Tab 2B, pp. 23 and 2078); (Claimants' experts state average blood lead level for the general population is 3 ug/dl)(Tab 1A, p. 23).

In an effort to better protect its employees, Yuasa-Exide reduced the permissible exposure limit below the OSHA threshhold. An employee would be placed on MRP if a six month lead average or three samples registered greater than 36 micrograms per deciliter of blood. A Yuasa-Exide employee was also subject to MRP if there was a one time blood lead level reading of 44 micrograms per deciliter of blood or greater. (Tab 2B).

There are approximately 378 claims which have been filed by former Yuasa-Exide employees. Most of the claims have been resolved via mediation. There is only one reported case regarding the impact of lead exposure on Yuasa-Exide's employees

that has been adjudicated by the Commission. On June 10, 2005, The Honorable George N. Funderburk adjudicated the worker's compensation claim of *Lee Ernest Franklin v. Yuasa-Exide* compensable. (Tabs 3A-B). Yuasa-Exide argued that Mr. Franklin had not sustained a compensable work related injury which resulted in kidney failure at the time of the date of his last employment in January 1996; because his blood lead levels (less than 30 micrograms per deciliter of blood) were not sufficient to have caused injury to the kidneys or aggravated a renal kidney preexisting condition. Yuasa-Exide argued that OSHA's regulations regarding permissible exposure limits should determine what constituted an injurious exposure.

By contrast, Franklin argued that the South Carolina Workers' Compensation Commission was not bound by OSHA's regulations, and was not required to follow such. The Commission relied upon Franklin's experts in adjudicating Mr. Franklin's claim compensable. In particular, Franklin's experts opined that significant research had shown lead related health effects in individuals with blood lead levels in the range of 10 to 19 micrograms per deciliter of blood. Claimant's experts noted that prior research had previously concluded that blood lead levels of 10 to 19 would not result in any ill health effects. Claimant's experts now conclude that recent studies have shown low to moderate lead exposure or exposure causing blood lead levels ranging from 10 to 40 micrograms per deciliter of blood will negatively affect an individual's neurological system, kidneys, cardiopulmonary systems, gastrointestinal systems and hematological systems and certain muscular skeletal systems.

The Decision and Order of Commissioner Funderburk was appealed the South Carolina Worker's Compensation Full Commission Appellate Panel. The Appellate Panel noted in its Findings of Fact that Mr. Franklin was continuously exposed to lead during his employment until his last date of work in January, 1996. (Tab 3C). The Panel further found that compliance with OSHA's guidelines as a determinative factor in deciding whether exposure could be deemed injurious or non-injurious exposure was not persuasive. In particular, the Commission relied upon the Supreme Court holding in the case of *Glenn v. Columbia Selica Sand Co.*, 236 S.C. 13, 112 S.E.2d 711 (1960) which provides that the last exposure constitutes an injurious exposure. See also, *Hanks v. Blair Mills, Inc., 286 S.C. 378; 335 S.E.2d 91 (Ct. Apps., 1985).*

The Commission's Decision was appealed to the Sumter County Court of Common Pleas. The compensability of Mr. Franklin's lead exposure claim was deemed affirmed.

Yuasa-Exide filed a claim for Second Injury Fund reimbursement. The Fund denied the claim for reimbursement. Yuasa-Exide argued that the Commission had ruled as a matter of law that Mr. Franklin's subsequent exposures, although below OSHA's permissible exposure guidelines, constituted subsequent aggravating accidents of heavy metal poisoning. Yuasa-Exide stated it was entitled to reimbursement for aggravation of maladies associated with exposure to lead, i.e., hypertension and heavy metal poisoning. Yuasa-Exide further argued that Mr. Franklin's chronic exposure to lead had caused and/or aggravated Franklin's preexisting kidney disease and hypertension.

The Fund argued that Yuasa-Exide was not entitled to reimbursement because there were no subsequent accidents which aggravated or accelerated Mr. Franklin's kidney disease or hypertension.

Commissioner Funderburk found Yuasa-Exide's claim was subject to reimbursement because of the opinions of Mr. Franklin's experts that subsequent exposures to lead aggravated Mr. Franklin's preexisting renal kidney disease and hypertension. (Tab 3D). Moreover, Commissioner Funderburk found that the goals of the Second Injury Fund were met when Yuasa-Exide retained Mr. Franklin in his employment after knowledge of his prior impairments and an exacerbation of those medical impairments and diseases associated with lead exposure (heavy metal poisoning). The South Carolina Workers' Compensation Commission's Appellate Panel affirmed Commissioner Funderburk's Decision.

The Second Injury Fund appealed the Commission's Decision award and reimbursement to Yuasa-Exide to the South Carolina Supreme Court. In the case of *South Carolina Second Injury Fund v. Transportation Ins. Co.; Travelers' Ins. Co., and Great American Ins. Co.,* 389 S.C. 422, 699 S.E.2d 687 (2010), the South Carolina Supreme Court affirmed the decision to award reimbursement to Yuasa-Exide. (Tab 3E).

LEGAL ARGUMENTS

I. WHETHER YUASA-EXIDE, INC. HAS MET THE REQUIREMENTS AS SET FORTH IN SECTION 42-9-400 ET SEQ. IN ORDER TO OBTAIN REIMBURSEMENT FOR INDEMNITY BENEFITS PAID?

During an individual's employment tenure at Yuasa-Exide, the company maintained an in-house medical department. Employees received medical treatment from Drs. A. W. Hursey and Eric Byrd for non-occupational maladies and occupational accidental injuries and diseases. The medical records kept in ordinary course and scope of business contains health history medical questionnaires, diagnostic reports, in-take notes and other medical reports. From these documents, Yuasa-Exide assessed whether a claim is subject to Second Injury Fund reimbursement.

In order for an employer or insurance carrier to be reimbursed from the Second Injury Fund, knowledge of a permanent physical impairment must be proven. In the instant cases, there is no doubt that workers working in the plate cleaning, pasting, formation, battery assembly, plate wrapping and grid casting departments all suffered with heavy metal poisoning. The experts in the *Franklin* case and OSHA's regulation set forth the adverse effects of chronically elevated blood lead levels. The adverse effects ranged from the inhibition of enzymes associated in the synthesis of hemoglobin to cerebral edema. The Commission has established in prior Orders that blood lead levels of 10 micrograms per deciliter of blood or more is sufficient with respect to an aggravation of a preexisting condition.

Under Section 42-9-400(d)(23) when an employer establishes prior knowledge of a permanent impairment, then there is a presumption that the condition is permanent and that a hindrance or obstacle to employment or reemployment exists. This burden is met with respect to the undisputed preexisting permanent impairment of heavy metal

poisoning and the subsequent accidental exposures to lead. *S.C. Code Ann. §42-9-400(d)(23) (2001).*

Second, after establishing an employer's knowledge of a preexisting permanent impairment, the employer must prove a subsequent disability from an injury by accident. The Commission has ruled that a worker's continued employment at Yuasa-Exide results in a subsequent disability by virtue of a recurring injury by accident. This Finding was appealed to the South Carolina Supreme Court, which affirmed the Commission's findings. *S.C. Second Injury Fund v. Transportation Ins. Co., et al., supra.* Hence, the Court's ruling constitutes the law of the case regarding lead exposure cases.

Yuasa-Exide's request for reimbursement is supported by medical questionnaires required by the Fund, Claimant's Knowledge Affidavits, and Employer Health, Safety and Medical Records. Moreover, claimant's varying blood lead levels reflecting chronic exposure to lead, safety precautions undertaken with regard to improving ventilation systems and requiring the use of respirators and placement in the medical removal program is evidence of undertakings to avoid subsequent disabilities or an aggravation of an employee's physical condition.

II. WHETHER THE FILING OF AN ILLEGIBLE ELECTRONIC RECORD CONTAINING MEDICAL RECORDS SUBSEQUENT TO AN EMPLOYEE'S LAST DATE OF WORK AT YUASA-EXIDE WARRANTS A DENIAL OF REIMBURSEMENT?

Although there is no case law directly on point regarding the issue raised by the Fund, an analysis of a similar case involving improper service and improper filing is helpful.

The United States District Court for the Eastern District of North Carolina denied entry of default where service upon Plaintiff was improper. *Dalenko v. News and Observer Publishing Co.*, U.S. District Lexis 136787 (2010). In *Dalenko*, Plaintiff sought the entry of default because Defendants' Answer had not been timely filed and/or was filed improperly. In the argument against the entry of default, Defendants argued that to the best of their knowledge, copies of the legal pleadings were mailed to Plaintiff but that an oversight could have possibly occurred. Defendants had filed pleadings with the Court through electronic service, but had not attached the required Certificate of Service, thereby making its responsive pleadings untimely.

The Defendants in *Dalenko* timely submitted their pleadings, but erroneously relied on electronic service. *Id.* Consequently, Plaintiff was not served with the responsive pleadings in a timely fashion. In the instant cases, Yuasa-Exide timely submitted all required documents to the Fund before June 30, 2011, but also mistakenly relied on technology when it included an illegible electronic record containing medical reports subsequent to claimants' employment at Yuasa-Exide. The Fund acknowledges receiving all medical reports, employment files that were generated during claimant's employment tenure. They further admit receiving the medical questionnaires and claimant knowledge affidavits. Under the circumstances, the Commission should find that the Defendants deficient and electronic submission

resulted solely from a technical error, which presumably was not egregious enough to result in a denial by the Fund based upon the volumes of other evidence submitted in support of Yuasa-Exide's request for reimbursement.

The Court in *Dalenko* refused to enter an Order of Default arguing that while Defendants may have mistakenly relied upon electronic service, the error did not rise to the level of bad faith requiring entry of default. The Court noted that "**if there was any deficiency, it was technical in nature,** and was rectified without prejudice to Plaintiff or other injustice;" *Id.*

Finally, in the case at bar, Yuasa-Exide immediately rectified its mistake by hand delivering another disc containing legible medical records containing records generated after claimant last worked at Yuasa-Exide. The records were submitted to the Fund in an effort to provide the Fund with an overview of the case and efforts undertaken to defend the claim, so that no assertions could be made to challenge the basis of the settlement. The documents were delivered within three days after the Fund notified the undersigned that the records and documents contained on the first disc were not legible. Similar to the Plaintiff in *Dalenko,* the Fund has failed to show bad faith, willful action on part of Yuasa-Exide, prejudice or injustice.

CONCLUSION

Medical records and employment files generated during claimant's employment at Yuasa-Exide clearly show a permanent impairment caused by heavy metal poisoning. Lead exposure over and above the normal blood lead level of 10 micrograms per deciliter of blood certainly constitutes heavy metal poisoning.

Because the results of heavy metal poisoning can be eliminated and reversed by removing one from a lead laden environment, the employee's daily entrance into the Yuasa-Exide Manufacturing facility constituted a new exposure or injury by accident. This matter was the conclusion and findings of the Workers' Compensation Commission, which have been affirmed by the South Carolina Supreme Court in the case of *Second Injury Fund v. Transportation, Inc., et al., supra.* In this holding, not only did the subsequent exposure to lead on a daily basis constitute a new injury by accident, but also resulted in an aggravation of an underlying condition, namely increased health maladies resulting from consequential episodes of heavy metal poisoning, aggravation of hypertension, cardiopulmonary problems, cardiovascular disease, and various peripheral neuropathies. This fact is further corroborated by the only medical evidence contained in the file with respect to the reports of Drs. Howard Hu, Barry Weissglass, Eugene Shippen, J. Routt Reigart, and Edward L. Baker.

Aside from the overwhelming evidence, the Fund has presented absolutely no evidence to refute the medical opinions of the medical providers. Accordingly, Yuasa-Exide, Inc. has proven its entitlement to reimbursement. See, *Carolinas Recycling Group v. S.C. Second Injury Fund, Opinion No.: 4987 (Ct. Apps. June 13, 2012).*

Respectfully submitted,

VERNON F. DUNBAR
TURNER, PADGET GRAHAM & LANEY, P.A.
Post Office Box 1509
Greenville, SC 29602
ATTORNEY FOR DEFENDANT

(864) 552-4601

Greenville, South Carolina

August 10, 2012