BEFORE THE INDUSTRIAL COMMISSION OF THE STATE OF IDAHO

DAVE M. MASON, Claimant, v. LITTLE VALLEY ELK RANCH, LLC, Employer, and IDAHO STATE INSURANCE FUND, Surety, Defendants.	**IC 2009-003466** **FINDINGS OF FACT, CONCLUSIONS OF LAW, AND ORDER** Filed December 23, 2015

INTRODUCTION

Pursuant to Idaho Code § 72-506, the Idaho Industrial Commission assigned the above-entitled matter to Referee Alan Taylor, who conducted a hearing in Twin Falls, Idaho on May 28, 2015. Claimant, Dave Mason, was present in person and represented by Clark Jordan, of Salmon. Defendant Employer, Little Valley Elk Ranch, LLC, (Little Valley), and Defendant Surety, Idaho State Insurance Fund, were represented by Jon Bauman, of Boise. The parties presented oral and documentary evidence. No post-hearing depositions were taken. Briefs were submitted and the matter came under advisement on August 21, 2015.

Pursuant to Idaho Code § 72-506(2), the Commission is authorized, indeed, required, to approve and confirm a proposed decision before it can be deemed a finding, order, decision or award of the Commission. The statute was recently construed in Lorca-Merono v. Yokes Washington Foods, Inc., 137 Idaho 446, 50 P.3d 461 (2002). There, the issue before the Commission was whether claimant's pre-existing cervical spine condition was aggravated by her industrial accident. The Referee to whom the case was assigned concluded that the most

persuasive medical evidence was that endorsing a causal relationship between claimant's condition and the accident, and wrote a decision awarding benefits to claimant for her permanent aggravation. The Commission declined to adopt the proposed decision and authored its own decision in which it denied benefits based on other medical evidence of record which it found more persuasive. On appeal, claimant argued that the Commission was not authorized to "simply reject" the findings and conclusions proposed by the Referee. Treating this assertion, the Court stated:

> The findings of fact made by the referee were merely recommendations to the Industrial Commission. Upon reviewing those findings, it could either adopt them or enter its own findings. Idaho Code §§ 72-506(2) & 72-717 (1999). The Commission need not explain why it did not adopt certain findings recommended by the referee. The Industrial Commission, as the factfinder, is free to determine the weight to be given to the testimony of a medical expert. (Citations omitted)
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> Lorca-Merono v. Yokes Washington Foods, Inc., 137 Idaho 446, 50 P.3d 461 (2002).

Here, Referee Taylor authored a proposed decision in which he found that Claimant met his burden of establishing a causal connection between the subject accident and his L3-4 condition. As developed infra, the undersigned Commissioners have reviewed the record and reach a different conclusion. Therefore, the undersigned Commissioners have chosen to adopt only a portion of the Referee's proposed decision and hereby issue their own findings of fact, conclusions of law and order.

ISSUES

The issues to be determined are:

1. Whether Claimant is entitled to a laminectomy and fusion at L3-4; and
2. Whether Claimant is entitled to time loss benefits related to such surgery.

All other issues are reserved.

CONTENTIONS OF THE PARTIES

Defendants acknowledge that Claimant sustained an industrial accident while working for Little Valley on January 25, 2009, when his truck and trailer rolled, resulting in serious injuries. He underwent extensive medical treatment including multiple surgeries. Claimant asserts his present need for L3-4 laminectomy and fusion is related to his industrial accident and also claims temporary disability benefits related to such surgery. Defendants maintain that any need for L3-4 laminectomy is not related to the industrial accident, L3-4 fusion is not warranted, and Claimant is not a good surgical candidate.

EVIDENCE CONSIDERED

The record in this matter consists of the following:

1. The Industrial Commission legal file;
2. Claimant's Exhibits 1-13, admitted at the hearing;
3. Defendants' Exhibits 1-8, admitted at the hearing;
4. The testimony of Claimant taken at the May 28, 2015 hearing;
5. The post-hearing deposition testimony of David B. Verst, M.D., taken by Claimant on June 5, 2015; and
6. The post-hearing deposition testimony of Timothy Doerr, M.D., taken by Defendants on June 19, 2015.

All pending objections are overruled and motions to strike are denied, except Defendants' objection to Claimant's opening brief. Defendants object to the filing of Claimant's opening brief and request sanctions on the grounds that the brief violates the formatting requirements of JRP 11(A). Defendants observe that the Commission has in past decisions stricken the over-

length portions of briefs. Claimant acknowledges his briefing formatting faults but asserts the unintentional violation of the rule does not warrant sanctions.

JRP 11(A) limits a claimant's opening brief to 30 pages and requires double-spaced text, top and bottom margins of one and one-half inches, and side margins of one inch. Adherence to the prescribed font, margins, and double spacing produces approximately 21 lines of text per page, resulting in a brief of approximately 630 total lines (21 lines x 30 pages).

In the present case, Claimant's initial brief utilized one and one half spacing and contains approximately 34 lines per page. While his initial brief is 23 pages long, plus the certificate of service page, it totals approximately 782 lines (34 lines x 23 pages)—substantially more than effectively allowed by JRP 11(A) without prior Commission approval. Thus Claimant's brief is over-length. Limiting Claimant's opening brief to 630 total lines of text at 34 lines per page (considering his one and one-half spacing), equals approximately 18.53 pages (630 total lines ÷ 34 lines per page). Pursuant to JRP 11(A), Defendants' objection is sustained and page 19, l. 20 through page 23 of Claimant's Initial Brief are hereby stricken. Page 1 through page 19, l. 19 and page 24 (certificate of service page) are considered herein.

FINDINGS OF FACT

1. Claimant was born in 1951. He is right-handed. At the time of the hearing he was 63 years old, six feet two inches tall, weighed 285 pounds, and lived in Glenns Ferry.

2. At all relevant times, Little Valley was a 16,000 acre elk ranch in Lemhi Valley.

3. **Background**. While in high school, Claimant began working as a civilian laborer at the Concord Naval Weapons Station in California. He eventually supervised a crew of 25 men transferring ordnance from boxcars onto ships. From 1976 until 1990, Claimant was employed at Pullman Trailmobile in Stockton, California repairing diesel trucks and trailers. He was hired

as a welder and fabricator and eventually became a shop foreman. From 1992 until 2003, Claimant worked for Pend Oreille Telephone Company in Ione, Washington. He was hired as a mechanic, later learned telephone installation, and ultimately became the manager. From 2003 until 2008, Claimant worked at Ken Maupin Construction as a heavy equipment operator, welder, and fabricator.

4. In May 2008, Claimant was hired as the ranch manager and head guide at Little Valley. His responsibilities included irrigating, maintaining fences, and overseeing the care and maintenance of the ranch grounds and compound. He enjoyed his job and had no difficulty performing his work duties prior to January 2009.

5. **Significant prior medical history**. Claimant suffered various accidents and injuries in his prior work and recreational pursuits.

6. In 1973, Claimant's hand was run over by a forklift and he lost his left index finger. In 1990, Claimant stopped to assist the victim of a motor vehicle accident and was himself struck by a car and suffered cervical injury for which he eventually underwent C4-5 fusion in 1993. Claimant developed left carpal tunnel syndrome and underwent release surgery in 1992. In 1999, Claimant sustained a severe concussion when he was struck by a 1,000 pound transformer while working. He initially suffered seizures and thereafter noted recurring headaches. In 2004, he suffered a right knee injury from an ATV accident and underwent partial medial meniscectomy and anterior cruciate ligament debridement. In 2006 and 2007, Claimant presented to Joseph Taylor, M.D., complaining of numbness and tingling in his hands and feet. Dr. Taylor assessed myalgias possibly due to the prescription Lipitor Claimant was taking for hyperlipidemia. He stopped taking Lipitor and the symptoms resolved. In 2007 Claimant underwent cardiac catheterization and was found to have early coronary artery disease.

7. **January 25, 2009 industrial accident and treatment.** On January 25, 2009, Claimant was working for Little Valley driving from Nevada towards Twin Falls on icy roads in a snowstorm. A gust of wind blew his trailer off the road, pulling his pickup off the road and rolling it over. Claimant apparently lost consciousness and awoke with the roof of the cab partially crushed down on top of him. He was trapped in the crushed cab for nearly an hour until emergency responders arrived and extracted him. Claimant reported severe neck, back, and shoulder pain and was taken via ambulance to Twin Falls Regional Medical Center. Diagnostic imaging revealed reversal of the normal cervical lordosis, C5-6 ligamentous disruption, large C5-6 and C6-7 disk herniations with spinal cord compression, and first-degree spondylolisthesis of L4 on L5 but no acute fractures. On January 28, 2009, David Christensen, M.D., performed cervical surgery, including C-6 corpectomy with C5-7 decompression and anterior interbody instrumented fusion. Claimant noted ongoing low back and right leg pain after the accident. He was discharged from the hospital ambulating with a walker.

8. In March 2009, Dr. Christensen released Claimant to light-duty work and Claimant returned to work at Little Valley. His back and right leg hurt constantly. In April 2009, Dr. Christensen released Claimant to full-duty work. His back and right leg continued to hurt constantly; however, he returned to full-duty work.

9. **June 12, 2009 aggravation and further treatment.** On June 12, 2009, Claimant was working at Little Valley putting up new fences. He lifted a heavy 12-foot length of pipe and noted immediate increased back and neck pain. He was unable to work thereafter. Little Valley subsequently terminated his employment. Claimant has not worked since.

10. In July 2009, Claimant moved to Glenns Ferry.

11. On August 4, 2009, Dr. Christensen rated Claimant's cervical impairment at 25% of the whole man and his L4-5 impairment at 12% of the whole man, with 6% attributable to his industrial accident and 6% attributable to pre-existing stenosis. Claimant continued to report debilitating low back pain.

12. On December 2, 2009, David Verst, M.D., examined Claimant at Defendants' request. Dr. Verst recommended L4-5 laminectomy and fusion. Claimant accepted Dr. Verst's recommendation and Defendants authorized the surgery. On February 9, 2010, Dr. Verst performed L4 and L5 laminectomies and foraminotomies and L4-5 posterolateral fusion with cage placement and pedicle screw instrumentation. Claimant's back and right leg pain improved, although his right leg numbness persisted. On June 17, 2010, Dr. Verst found Claimant medically stable from his L4-5 surgery.

13. Claimant's cervical spine symptoms increased and a June 28, 2010 cervical CT scan revealed nonunion at C5-7. Defendants authorized cervical surgery and on November 8, 2010, Dr. Verst performed a posterior C5-7 instrumented fusion. After recuperating from surgery, Claimant's cervical symptoms lessened but did not resolve.

14. In 2010, Claimant began receiving Social Security Disability benefits.

15. Claimant's cervical symptoms increased. On October 24, 2012, Dr. Verst performed C5-6 and C6-7 hemilaminectomies and foraminotomies with hardware removal. Claimant's neck and arm pain improved.

16. In late 2012, Claimant began seeing psychologist Barry Jenks for counseling for depression and anxiety. By the time of hearing, Defendants had ceased paying for these counseling sessions.

17. Claimant developed increasing low back pain and on February 11, 2013, he awoke with burning pain down his left leg. Later that day he presented to the emergency room at the North Canyon Medical Center in Gooding because he fell when his left leg "went out" and he could not stand. He received medications and was directed to follow-up with Dr. Verst.

18. On June 5, 2013, Dr. Verst examined Claimant and noted his cervical symptoms were much improved; however his low back and bilateral leg pain had worsened. Claimant underwent lumbar epidural steroid injections and physical therapy without lasting benefit. An October 7, 2013 lumbar MRI revealed L3-4 stenosis.

19. On February 6, 2014, Dr. Verst recommended L3-4 surgery and fusion, which Defendants did not authorize.

20. On April 1, 2014, Nancy Greenwald, M.D., and neuropsychologist Craig Beaver examined Claimant at Defendants' request. Dr. Beaver diagnosed major depression and post traumatic stress disorder.

21. On April 7, 2014, Claimant completed a functional capacity evaluation at Defendants' request. After the day-long evaluation Claimant felt "wiped out" and rested in bed for two or three days.

22. On May 8, 2014, David Jensen, D.O., performed bilateral EMG testing and noted evidence of peripheral neuropathy.

23. On July 17, 2014, Timothy Doerr, M.D., examined Claimant at Defendants' request and opined further surgery was not indicated.

24. From October 27 through November 21, 2014, Claimant participated in the LifeFit chronic pain program in Boise at Defendants' insistence. He noticed improvement in upper body conditioning, but his legs and back became more painful and limited his

participation. On November 24, 2014, Robert Friedman, M.D., summarized Claimant's progress and completion of the LifeFit program. Dr. Friedman opined Claimant had reached maximum medical improvement and would not benefit from additional lumbar surgery.

25. **Condition at the time of hearing.** Between February 11, 2013, and the time of hearing, Claimant fell approximately 10 times because his legs gave out. At the time of hearing Claimant continued to have low back and bilateral leg pain. He was able to walk for only 10 to 15 minutes before having to stop. Claimant credibly testified that he fell and cut his hand about one and one-half weeks before hearing because his right leg gave out. He did not believe he would be able to return to any work and, given his prescription medications, would be unable to pass a drug test. At the time of hearing Claimant was taking prescription Lisinopril (for hypertension), Pro Xanthine (for hypertension and nightmares), Gabapentin (for lower extremity pain), Diazepam (for anxiety), Hydrocodone (for pain), and a form of Prozac (for depression). He continued to receive Social Security Disability. He counseled periodically with psychiatrist Laura Musteti-Oprea, M.D., for depression, panic attacks, and post-traumatic stress disorder. Defendants authorized this counseling.

26. **Credibility.** Having observed Claimant at hearing and compared his testimony with other evidence in the record, the Referee found Claimant to be a credible witness. The Commission finds no reason to disturb the Referee's findings and observations on Claimant's presentation or credibility. This is a case which turns on the testimony from the medical experts, not on Claimant's observational credibility.

DISCUSSION AND FURTHER FINDINGS

27. The provisions of the Idaho Workers' Compensation Law are to be liberally construed in favor of the employee. Haldiman v. American Fine Foods, 117 Idaho 955, 956, 793

P.2d 187, 188 (1990). The humane purposes which it serves leave no room for narrow, technical construction. Ogden v. Thompson, 128 Idaho 87, 88, 910 P.2d 759, 760 (1996). Facts, however, need not be construed liberally in favor of the worker when evidence is conflicting. Aldrich v. Lamb-Weston, Inc., 122 Idaho 361, 363, 834 P.2d 878, 880 (1992).

28. **Medical benefits.** The primary issue presented is whether Defendants are liable for Claimant's proposed L3-4 laminectomy and fusion. The parties concede that Claimant's L4-5 level was injured in the subject accident, and that the medical care Claimant has received for that injury, to include the L4-5 fusion performed by Dr. Verst, is likewise related to the subject accident. The parties also appear to concede that Claimant had degenerative changes at L3-4 which preceded the subject accident, as revealed on the 2009 MRI. Further, the parties agree that Claimant did not suffer any new injury at the L3-4 level as an immediate result of the subject accident. Finally, the parties appear to be in agreement that between the date of the January 27, 2009 lumbar MRI and the date of hearing, Claimant's degenerative L3-4 condition has progressed, such that Dr. Verst, at least, believes that Claimant is a surgical candidate for laminectomy and fusion at this level. It is Claimant's contention that his L3-4 condition progressed faster than it otherwise would have due to the fact that Claimant underwent accident related L4-5 fusion in early 2010. The theory, endorsed by both Dr. Verst and Dr. Blair, is that the L4-5 fusion subjects the L3-4 level to greater and altered biomechanical stresses, such that disk degeneration is accelerated. This phenomena was identified by Dr. Verst as adjacent segment disorder (ASD). Defendants acknowledge that ASD is a known phenomena but contend that Claimant does not suffer from it. Defendants contend that the progression of disk disease at L3-4 between January 27, 2009 and the date of hearing represents nothing but the natural history

of progressive degenerative changes. Defendants deny that the work-related L4-5 fusion did anything to accelerate the normal progression of Claimant's L3-4 degenerative disk disease.

29. Claimant asks the Commission to first find that Claimant's L3-4 condition is causally related to the subject accident, and second, that the surgery "required" by Dr. Verst is a "reasonable" course of treatment which must be provided by Defendants. We first treat the issue of whether Claimant's L3-4 condition is, in some respect, causally related to the subject accident. Specifically, did the L4-5 fusion accelerate the progression of Claimant's degenerative processes at L3-4 such that a causal relationship between the subject accident and Claimant's L3-4 condition is demonstrated?

30. The claimant in a workers' compensation case carries the burden of proving that the condition for which compensation is sought is causally related to the industrial accident. Duncan v. Navajo Trucking, 134 Idaho 202, 998 P.2d 1115 (2000). The proof required is medical proof, and Claimant bears the burden of demonstrating causation to a "reasonable degree of medical probability". See Anderson v. Harper's, Inc., 143 Idaho 193, 141 P.3d 1062 (2006). To prove that a causal relationship is medically probable requires Claimant to demonstrate that there is more medical evidence for the proposition than against it. Jensen v. City of Pocatello, 135 Idaho 406, 18 P.3d 211 (2000). Where the medical evidence is in equipoise, Claimant has not met his burden of proving medical causation. Seamans v. Maaco Auto Painting Body Works, 128 Idaho 747, 918 P.2d 1192 (1996). The Commission is not required to construe facts liberally in favor of the Claimant when the evidence is conflicting. Aldrich v. Lamb-Weston, Inc., 122 Idaho 361, 834 P.2d 878 (1992).

31. In the instant case, three physicians have rendered opinions on the threshold causation issue before the Commission. Dr. Verst and Dr. Blair have opined that Claimant's

L3-4 disease progressed faster than would otherwise be expected, and that the cause of this accelerated degeneration is the Claimant's fusion at the adjacent L4-5 segment. Dr. Doerr, testifying for Defendants, has opined that the rate of progression of Claimant's L3-4 disk disease is in line with the natural history of progressive disk disease, and that there is no evidence that the progression in this case was accelerated by fusion at the L4-5 level. All three physicians are well-qualified to render opinions on this question. The record further reflects that all three opinions are generally based on an accurate and complete foundation.

32. To resolve the threshold causation question, the Commission must determine which of the positions on medical causation is the most persuasive. This warrants careful review of the records and testimony of Drs. Verst, Blair and Doerr as well as a review of the radiological and EMG testing which informs their opinions.

Radiological Studies

33. Claimant underwent MRI evaluation of his lumbar spine on January 27, 2009. That study, read by Daniel Adler, M.D., demonstrated the following significant findings in Claimant's lumbar spine:

> T11-12, T12-L1, L1-L2 AND L2-L3 LEVEL: No significant disk bulge, protrusion or extrusion. Nerve roots exit through the respective neural foramina above the respective disk levels and do so freely.
>
> L3-4 LEVEL: Mild diffuse disk bulge that abuts but does not compress the L4 nerve roots in the lateral recesses. Disk bulge extends into the foraminal regions but does not compress the L3 nerve roots. The L3 nerve roots exit freely through the respective neural foramina. There is moderate central canal narrowing present and right greater than left facet arthropathy.
>
> L4-5 LEVEL: Diffuse disk bulge and mild thickening of ligamentum flava. The disk bulge causes significant central canal narrowing and flattens the L5 nerve roots in the lateral recesses. This is worse on the right than the left. There is clumping of the nerve roots within the crowded central canal at this level. The L4 nerve roots themselves exit freely through the respective neural foramina above this level. Mild bilateral facet arthropathy.

L5-S1 LEVEL: Diffuse disk bulge with focal central disk protrusion that abuts this S1 nerve root, right more than left. This does not appear to compress the nerve roots. This causes moderate central canal narrowing. The L5 nerve roots exit freely through the neural foramina above this level. The S1 nerve roots exit freely below this level.

IMPRESSION. L4-5 DIFFUSE DISK BULGE CAUSING SIGNIFICANT CENTRAL CANAL NARROWING AND COMPRESSING THE L5 NERVE ROOTS IN THE LATERAL RECESS, RIGHT POSSIBLE MORE THAN LEFT.

L5-S1 DISK BULGE WITH CENTRAL DISK PROTRUSION THAT DISPLACES THE S1 NERVE ROOTS POSTERIORLY, RIGHT MORE THAN LEFT, BUT DOES NOT APPEAR TO COMPRESS THE NERVE ROOTS IN THEIR LATERAL RECESSES. MODERATE CENTRAL CANAL NARROWING.
FACET ARTHROPATHY AT MULTIPLE LEVELS.
L3-4 MILD DIFFUSE DISK BULGE AS DETAILED ABOVE. NO COMPRESSION OF THE NERVE ROOTS IN THE LATERAL RECESSES.

Claimant's Exhibit 2, 37-38. (Emphasis supplied.)

Subsequent to that study, Claimant was taken to surgery by Dr. Verst, who performed an L4-5 fusion.

34. On February 11, 2013, Claimant was seen at North Canyon Medical Center in Gooding, Idaho after an episode of his left leg giving way. Claimant underwent CT evaluation of the lumbar spine on February 11, 2013. That study was read as follows:

At the L3-4 Level, the intervertebral disc appears normal with no evidence of disc herniation. The AP diameter of the spinal canal is normal. There are degenerative changes of facet joints and appears to be bilateral neural foraminal narrowing worse on the right.

At the L4-5 level, laminectomy defect is again noted. There is beam hardening artifact arising from the hardware fixed L4 and L5. There may be mild right neuroforaminal narrowing.

At the L5-S1 level, the intervertebral disc appears normal with no evidence of disc herniation. AP diameter of the spinal canal appears normal. There are mild degenerative changes of facet joints with mild right neural foraminal narrowing.

IMPRESSION:
STATUS POST POSTERIOR L4-5 FUSION WITH HARDWARE IN PLACE. THERE IS SLIGHT ANTERIOR SUBLUXATION OF L4 ON L5.

DEGENERATIVE CHANGES OF FACET JOINTS RESULT I BILATERAL NEURAL FORAMINAL NARROWING, WORSE ON THE RIGHT AT L3-4 AND MILD RIGHT NEUROFORAMINAL NARROWING AT L4-5 AND L5-S1.

IF CLINICALLY INDICATED, MRI OF THE LUMBAR SPINE MAY BE HELPFUL FOR FURTHER EVALUATION.

Defendants' Exhibits, 01527. (Emphasis supplied.)

35. On or about October 8, 2013, Dr. Verst ordered MRI evaluation of Claimant's lumbar spine. That study was read by Larry Poe, M.D., as follows:

L2-3: Spinal canal is developmentally small due to congenitally short pedicles. Disc is maintained in height and signal intensity.

L3-4: Infraspinous laminectomies have been performed. Mild disc bulging with endplate spinal proximal aspect of the right neural foramen is moderately to severely stenotic. Moderate left foraminal stenosis. Mild to moderate lateral recess stenosis. Mass effect of the L4 roots and L3 dorsal root ganglia.

L4-5: Signs of interbody arthrodesis likely utilizing a PLIF. No canal stenosis or obvious neural displacement. Grade 1 spondylolisthesis of L4 on L5.

L5-S1: Tiny right posterolateral protrusion does not displace the right S1 root. Mild right lateral recess stenosis has contribution from ligamentum flavum thickening. Mild left facet arthrorisis.
Conus and intraspinal: No intramedullary or intradural pathology. After contrast is given, no pathologic neural enhancement is seen.

Impression:

1. L3-4: Although infraspinous laminectomies have been performed, substantial bilateral lateral recess stenosis remains and foraminal stenosis persists with mass effect on L4 roots and L3 dorsal root ganglia.
2. L4-5: Signs of interbody arthrodesis. Grade 1 spondylolisthesis of L4 on L5. No canal stenosis.

3. L5-S1: Mild right lateral recess stenosis. Tiny right posterolateral protrusion.

Defendants' Exhibits, 01602-01603. (Emphasis supplied.)

36. On or about January 14, 2015, Claimant underwent another MRI evaluation of the lumbar spine performed at North Canyon Medical Center in Gooding, Idaho, and read by Jeffrey Pugsley, M.D. That study demonstrated the following findings at L3 through S1:

> L3-L4: Disc desiccation with slight posterior disc height loss. Mild central canal narrowing secondary to a small focal central disc protrusion and mild facet arthropathy. Moderate right and mild left neural foraminal narrowing similar to facet arthropathy and foraminal zone this bulge.
>
> L4-L5: Fusion and laminectomies at this level. No narrowing of the thecal sac or foramina.
>
> L5-S1: Disc desiccation with no disc height loss. No central canal narrowing. Mild right foraminal narrowing secondary to facet arthropathy.
>
> CONCLUSION:
>
> Disc degeneration and facet arthropathy in the lumbar spine, as described. Prior L4-5 posterior fusion with laminectomies.

Claimant's Exhibit 3, 138. (Emphasis supplied.)

37. On or about January 29, 2015, Claimant underwent a myelogram and post-myelogram CT, again at North Canyon Medical Center in Gooding. That study was read by Michael Dixon, M.D., who had the opportunity to compare the January 29, 2015 study against the January 14, 2015 MRI. The myelogram findings were as following:

> MYELOGRAM FINDINGS: Contrast is present in the spinal canal, and outlines the nerve roots. At least a moderate spinal canal stenosis is present at L3-L4, visualized on the myelographic images with truncation of the CSF column and displacement and tortuosity of the nerve roots inferior to this level.

Claimant's Exhibit 3, 144.

Dr. Dixon interpreted the post-myelogram CT as follows:

CT FINDINGS: Fusion: Posterior fusion noted at L4-L5, with fixes anterolisthesis of approximate 7 mm. The fusion includes an interbody fusion device, located at the posterior aspect of the disc space in appropriate position. Posterior fusion rods and lateral transpedicular screws at L4 and L5 are noted. There is bony fusion of the posterior elements. The disc space remains visible. There is no evidence of lucency around the hardware or fracture of the hardware. The postoperative changes are associated with laminectomy at L4. There is hypertrophic bone from the right L3 inferior articular facet. There is no evidence of fluid collection in the posterior paraspinal tissues by CT. Subcutaneous edema is noted posteriorly on the back. There is mild atrophy of the paraspinal musculature inferior to the fusion site.

. . .

L2-L3: Mild disc bulge. Mild ligamentum flavum thickening. Question congenitally narrow spinal canal. Mild additional spinal canal narrowing from degenerative change suspected. No evidence of neuroforaminal narrowing.

L3-L4: Mild broad-based disc bulge. Facet hypertrophy and disorganization. Mild ligamentum flavum thickening. This level is just above the fusion. There is moderate spinal canal stenosis with effacement of CSF space noted. There appears to be moderate bilateral neural foraminal narrowing at this level.

L4-L5: Level of the posterior fusion and laminectomy. Mild residual narrowing of the spinal canal is present at the L4 level, however at the disc level there is no spinal canal stenosis. The narrowing at the L4 level is partially due to hypertrophy of the inferior articular facet from the right L3 level. The left neural foramen appears patent. There is mild-moderate narrowing of the right neural foramen.

L5-S1: Mild disc bulge. Facet hypertrophy is mild. Thecal sac tapers normally at this level. Negative for spinal canal stenosis or neuroforaminal narrowing.

IMPRESSION:
POSTOPERATIVE CHANGES L4-L5. NO COMPLICATIONS OBSERVED.

ADDITIONAL DEGENERATIVE CHANGES AS DESCRIBED.

MODERATE SPINAL CANAL STENOSIS AT L3-L4. THIS IS ALSO DEMONSTRATED ON THE MYELOGRAPHIC IMAGES, AND APPEARS MORE SEVERE ON THE MYELOGRAM IN THE PRONE POSITION THAN THE CT IN THE SUPINE POSITION.

MILD SPINAL CANAL STENOSIS L2-L3. QUESTION CONGENITALLY NARROW SPINAL CANAL.

MODERATE NEUROFORAMINAL NARROWING, BILATERAL L3-L4.

MILD TO MODERATE NEUROFORAMINAL NARROWING, RIGHT L4-L5.

Claimant's Exhibit 3, 144-146. (Emphasis supplied.)

38. Therefore, while it seems clear that Claimant's degenerative changes at L3-4 have progressed over time, the radiologists' interpretations, standing alone, leave the Commission altogether unable to determine whether Claimant's disease at L3-4 has progressed faster than it otherwise would have absent the L4-5 fusion. For this determination we look to the opinions of medical experts.

EMG Study

39. At Dr. Greenwald's instance, Claimant underwent EMG and NCV studies on May 8, 2014, performed by David Jensen, D.O. Dr. Jensen noted the following findings on exam:

> **Diagnostic Studies**
> EMG & NCV Findings
> Motor Nerve Conduction Studies
> 1. The right peroneal motor has normal distal latency, reduced amplitude and reduced conduction velocity.
> 2. The left peroneal motor has normal distal latency, amplitude and slightly reduced conduction velocity.
> 3. The left median motor has slightly prolonged distal latency, normal amplitude and normal conduction velocity.
> 4. The right tibial motor has prolonged distal latency, normal amplitude and reduced conduction velocity.
>
> F Wave Studies: All the F wave studies have prolonged latency.
>
> Sensory Nerve Conduction Studies
> 1. The left sural sensory has normal peak latency and borderline low amplitude.
> 2. The right sural sensory has normal peak latency and reduced amplitude.
>
> Needle EMG: Using a disposable concentric needle, there was some spontaneous activity seen in the right dorsal interosseous. The left dorsal interosseous has some reduced recruitment. There was some mild reduced recruitment in the tib anterior, peroneus longus on the right, and a little bit on the right gastroc. The paraspinals there was some CRDs and spontaneous activity around the L5. There

was some spontaneous activity around L4, and just some very slight increased activity at L3 on the right.

Claimant's Exhibit 7, 2.

Dr. Jensen interpreted these test results as follows:

> **Assessment**
> 1. PERIPHERAL NEUROPATHY
>
> **Plan**
>
> Electrodiagnostic Impression
> 1. There is electrodiagnostic evidence of an underlying polyneuropathy effecting motor and sensory fibers, showing demyelinating and likely some axonal loss.
> 2. I do not see compelling evidence of an acute ongoing lumbar radiculopathy.
> 3. There is evidence suggestive of a resolved right L5-S1 radiculopathy.
>
> Of note, the patient presents with a clinical complaint of stenosis. Electrodiagnostically often times we will not find peripheral muscles in the leg with stenosis and may sometimes only find some spontaneous activity in the paraspinals, and not always even that. There was some spontaneous activity in the paraspinals but I could not make a diagnostic call based on those because of the fact that he has had prior surgery which can cause spontaneous activity in and of itself. As such, the diagnosis of spinal stenosis is not ruled out with the electrodiagnostic test.

Claimant's Exhibit 7, 2-3.

Benjamin Blair, M.D.

40. At the instance of his attorney, Claimant was evaluated by Benjamin Blair, M.D. on or about April 29, 2015. Dr. Blair was not deposed by way of post-hearing deposition, but his report has been introduced into evidence as Claimant's Exhibit 12. In formulating his opinions, Dr. Blair had access to the relevant medical records, including the studies referenced above, and the records and reports of Drs. Verst and Doerr. In addition to reviewing the reports of the radiologists who interpreted Claimant's lumbar spine studies, it appears that Dr. Blair personally reviewed the following studies and offered his own comments on what those studies revealed:

REVIEW OF RADIOGRAPHIC STUDIES:

- **01/27/09** – *MRI of the lumbar spine* reveals mild to moderate degenerative changes as the L4-5 level with secondary foraminal narrowing. There are mild degenerative changes at the L3-4 level with minimal central canal narrowing.
- **10/07/13 –** *MRI of the lumbar spine* with and without contrast reveal postoperative changes at the L4-5 level. The canal appears well decompressed at the L4-5 level. There are moderate degenerative changes with associated significant central we well as lateral recess stenosis at the L3-4 level.
- **01/29/15** – *Plain radiographs* of the lumbar spine reveal status post instrumented lumbar fusion at L4-5 which appears well healed. There are mild to moderate degenerative changes at the area above (L3-4). On flexion, extension there appears to be no instability.
- **02/18/15** – *Myelogram, post-myelogram CT scan* reveals status post lumbar interbody fusion at the L4-5 level which appears well healed. Hardware is intact. At the L3-4 level, there is significant narrowing of the central canal consistent with mild to moderate stenosis. In addition, the foramen is significantly narrowed bilaterally at this well.

Claimant's Exhibit 12, 9-10.

41. Dr. Blair opined that Claimant's problems at L3-4 are significant enough to warrant surgical intervention. However, he did not agree with Dr. Verst's observation that Claimant had instability of L3 on L4. Dr. Blair found no evidence of instability, and therefore recommended a simple L3-4 laminectomy versus a laminectomy plus fusion. However, he conceded that fusion might ultimately be necessary, but this is a decision that should be made intra-operatively.

42. Dr. Blair did not believe that the EMG study ruled out lumbar spinal stenosis, noting that electro-diagnostic studies are a fairly poor diagnostic test for stenotic syndrome. Therefore, while the testing did reveal that Claimant has a polyneuropathy, Dr. Blair believed that Claimant's symptoms were also due to ongoing stenosis, notwithstanding that the EMG studies did not demonstrate the existence of stenosis. (See Claimant's Exhibit 12, 10-11.) On

the question of whether the need for an L3-4 laminectomy is causally related to the L4-5 fusion, Dr. Blair stated:

> Yes. I feel the proposed L3-4 laminectomy (and possible fusion) by Dr. Verst is directly related to Mr. Mason's industrial accident and injury of January 25, 2009. Although Mr. Mason certainly had preexisting degenerative changes at the L3-4 level as seen on the initial MRI of 01/27/09, there was no associated neurologic impingement. The MRI and subsequent myelogram, post-myelogram CT scan obtained 10/13 and 1/15 shows significant progression at the L3-4 level of degenerative changes with associated secondary stenosis, both centrally and laterally. Although it is possible that this represents a natural progression of the degenerative changes seen on the initial MRI, it is far more likely that these changes have been accelerated due to the fusion at the L4-5 level. Fusions, because of the altered biomechanics, often lead to associated degenerative changes and need for secondary surgical treatment at the levels adjacent to level of initial fusion in approximately 20% of patients (to the extent that this syndrome actually has a name "adjacent level disease"). Because of this, I believe, within a reasonable degree of medical probability, Mr. Mason's stenosis at the L3-4 level and ongoing symptoms are directly related to the work related injury of 01/25/09 and subsequent fusion at the L4-5 level.

Claimant's Exhibit 12, 11.

Therefore, Dr. Blair acknowledged that Claimant suffered from degenerative changes at L3-4 which pre-dated the subject accident. He proposed that between January of 2009 and October of 2013 these changes progressed in a "significant" fashion. However, as Dr. Doerr has noted, "significant" does not equate with "severe". Dr. Blair acknowledged that the progression seen in these studies could represent a natural progression of Claimant's pre-existing degenerative changes, but thought it "far more likely" that the changes seen were the result of the accelerative effects of the L4-5 fusion. Why? Because fusions "often lead" to degenerative changes in approximately 20% of fusion patients. There are a number of ways to read this sentence. It could mean that 20% of fusion patients go on to develop ASD. It could also mean that only some subset of that 20% of fusion patients go on to develop ASD. However, even if fully 20% of fusion patients go on to develop ASD, it is somewhat difficult to understand how this supports

the proposition that it is more probable than not that the progression of Claimant's pre-existing degenerative disk disease at L3-4 is related to his fusion, instead of the natural history of degenerative joint disease progression. We find nothing in Dr. Blair's report that explains why a 20% incidence of ASD in fusion patients makes it medically probable that Claimant has ASD.

Dr. Verst

43. Dr. Verst first saw Claimant in November of 2009. He later performed three surgeries on Claimant. In February of 2010 he performed an L4-5 laminectomy and fusion on Claimant. In November of 2010 he performed a redo cervical fusion on Claimant's neck. In October of 2012 he performed a keyhole foraminotomy at C6. He reported that Claimant did well following the February 2010 L4-5 laminectomy and fusion until February of 2013, when he presented at the emergency room with complaints of left leg giving way and low back discomfort. Commenting on the results of the February 11, 2013 CT scan, Dr. Verst testified as follows:

> Mr. Bauman:
> Q. What were the results of Mr. Mason's lumbar CT scan that was done on February 11, 2013?
>
> Dr. Verst:
> A. The results demonstrate fusion at the L4-5 level with an associated degenerative disk at the level above the 3-4 level.

Verst Deposition, 11/21-15.

However, the radiologist who read that study did not say anything in his report to suggest that the degenerative changes seen at L3-4 were somehow "associated" with the L4-5 fusion.

44. Concerning the October 8, 2013 MRI, Dr. Verst commented that it demonstrated "instability", in addition to L3-4 spinal stenosis and facet arthropathy. (See Claimant's Exhibit 3, 115). In his February 6, 2014 chart note, he noted that Claimant presented with L3-4

spondylolisthesis, instability and severe spinal stenosis secondary to overgrowth of facet joints, disk protrusion and thickening of the ligamentum flavum. Both Dr. Blair and Dr. Doerr have opined that flexion/extension films failed to show any instability of L3 on L4.

45. Dr. Verst initially agreed that lumbar fusions "often lead to associated degenerative changes and the need for surgical treatment at levels adjacent to the fusion". (See Verst Deposition, 23/2-9). However, on cross-examination, Dr. Verst acknowledged that it was probably accurate to say that only 20 to 25% of those persons who have undergone a lumbar fusion procedure go on to develop ASD. (Verst Deposition, 44/19-45/7).

46. Concerning the EMG study, Dr. Verst stated in a letter dated May 28, 2014, that his review of that study revealed abnormalities. He stated that there was evidence of "polyradiculopathy" involving the roots of L5, L4 and L3. (See Claimant's Exhibit 3, 124). The actual interpretation of the EMG prepared by Dr. Jensen, while referencing "polyneuropathy", makes no reference to "polyradiculopathy". At the time of his deposition, Dr. Verst, like Dr. Blair, stated that EMG testing is rather poor at diagnosing stenotic syndromes. However, he stated that the polyneuropathy identified by Dr. Jensen actually is evidence of spinal stenosis. Per Dr. Verst, a long-standing compressive syndrome like spinal stenosis causes internal intrinsic changes within the nerve roots, which manifests as polyneuropathy. (Verst Deposition, 26/6-28/2.) Therefore, per Dr. Verst, the EMG studies support the proposition that Claimant now suffers from spinal stenosis that is severe enough to produce some part of the polyneuropathy observed by Dr. Jensen.

47. Dr. Jensen's report does not suggest that the polyneuropathy he observed was the result of an underlying spinal stenosis, as proposed by Dr. Verst. Claimant was referred to Dr. Jensen for the purposes of evaluating whether Claimant's stenotic condition was causing a

radiculopathy. The study demonstrated that while Claimant has a polyneuropathy, there was no evidence of spinal stenosis. However, Dr. Jensen cautioned that just because the EMG did not demonstrate evidence of a spinal stenosis does not mean that Claimant does not have a symptomatic spinal stenosis. If spinal stenosis can present as polyneuropathy, it might be supposed that Dr. Jensen would have stated that Claimant's polyneuropathy might be evidence of spinal stenosis. Instead, he only cautioned that the EMG could be a false negative for spinal stenosis.

48. While Dr. Verst expressed his opinion that Claimant's rate of L3-4 degeneration was accelerated by the L4-5 fusion, he could not entirely disagree with the conclusions of a recent article published in the journal of the American Academy of Orthopedic Surgeons that a complete explanation of ASD and the impact of surgical fusion remains elusive. The authors of that article found that although some patients developed symptomatic degenerative disease at levels adjacent to fusions, the effects of the fusion itself and the underlying degenerative disease are difficult to separate. (See Verst Deposition, 46/1-47/24).

49. In the end, Dr. Verst signified his agreement with the conclusion stated by Dr. Blair: While it is possible that the changes as seen in Claimant's low back represent nothing but natural progression of degenerative disk disease at L3-4, it is far more likely that the fusion at L4-5 is responsible for contributing to the changes seen between January of 2009 and the date of hearing. (Verst Deposition, 22/1-23).

Dr. Doerr

50. At the instance of Defendants, Timothy Doerr, M.D., evaluated Claimant pursuant to Idaho Code § 72-433 on July 17, 2014. He performed a clinical exam of Claimant. As well, he had the opportunity to review Claimant's prior medical records, to include the electro-diagnostic

studies and the actual films of the radiological studies referenced above. (Doerr Deposition, 42/24-43/1). Dr. Doerr was aware that both Dr. Verst and Dr. Blair are of the opinion that Claimant's L4-5 fusion accelerated his disk degeneration at L3-4. However, while Dr. Doerr agreed that ASD is a known phenomena, and may be seen in 20% of the fusion patient population, he was in rather vehement disagreement with Dr. Verst and Dr. Blair that Claimant's L4-5 fusion did anything to accelerate the progression of Claimant's L3-4 degenerative changes. (Doerr Deposition, 60/9-65/18). In his review of the imaging studies, particularly the January 27, 2009 MRI, the October 7, 2013 MRI and the February 18, 2015 myelogram and post-myelogram CT, Dr. Doerr found that while Claimant's L3-4 degenerative changes did progress over the time frame represented by the studies, this progression is entirely consistent with what he knows about the natural history of degenerative disk disease. (Doerr Deposition, 7/24-9/5; 22/12-23/17; 25/3-21; 42/19-43/23).

51. Dr. Doerr acknowledged it is sometimes difficult to make a clinical judgment as to whether or not a fusion has caused or accelerated disk disease at an adjacent level. He testified that there are certain factors which make it more likely that disk degeneration will be caused or accelerated by an adjacent fusion. Where the individual has undergone a laminectomy at the segment adjacent to the fusion, ASD is more likely. That did not happen here. Where the fusion is at a transitional segment, such as the transition between the lumbar and thoracic spine, ASD at an adjacent segment is more likely. That did not happen here. Where the patient has had a multi-level fusion, ASD is more likely at the adjacent motion segment. That did not happen here. Finally, where the individual suffers from some type of spinal imbalance, such as scoliosis, that too can make ASD more likely. Dr. Doerr explained that neither is that factor present in the instant matter. (Doerr Deposition, 23/6-28/22).

52. Also, Dr. Doerr explained how the thinking of the medical community about ASD has evolved over the years. When he was in school in the late 1980s, it was thought that adjacent segment disease was a common consequence of a fusion. In fact, the impetus behind the development of artificial disks was to attenuate the risk to an adjacent segment caused by a fusion. Interestingly, however, patients with artificial disks (which preserve the motion segment) do not seem to have a lower risk of disk degeneration at adjacent levels than do fusion patients. This tends to support the proposition that what the medical community once described as disk disease caused by adjacent fusion is really nothing more than the natural progression of the degenerative process in that adjacent segment. (Doerr Deposition, 37/14-38/7; 61/22-63/6).

53. Therefore, while Dr. Doerr freely acknowledged that adjacent segment disease, as described by Dr. Blair and Dr. Verst, can occur, he reaffirmed that Claimant does not have any of the particular risk factors he articulated for the development of the condition, and that Claimant's is not one of those cases in which ASD, as an explanation for Claimant's L3-4 problems, should be entertained:

> Physicians are going to disagree based on their experience. But it's a continuum, and there are situations that fall on one end which are clearly not adjacent segment disease. There are some on the other extreme which clearly are.
>
> I believe that Mr. Mason's case falls very closely to the end of the spectrum that this is not adjacent segment disease.

Doerr Deposition, 38/15-22.

An L4-5 fusion, standing alone, creates only a low risk of adjacent segment disease. (Doerr Deposition, 28/13-19). Dr. Doerr has followed thousands of patients with spinal stenosis in the course of his 20 year plus practice. He testified that he sees nothing in Claimant's films which would suggest to him that the rate of development of Claimant's L3-4 degenerative disk disease

between January of 2009 and February of 2015 is inconsistent with the natural history of the progression of such degenerative changes. (Doerr Deposition, 62/19-63/6).

54. Dr. Doerr also commented on Dr. Verst's belief that electro-diagnostic evidence of polyneuropathy is actually evidence of a long-standing compressive syndrome in Claimant's spine. First, Dr. Doerr is in agreement that the EMG is a relatively poor diagnostic tool to evaluate the existence of spinal stenosis or radiculopathy. In other words, one can have a normal EMG and still have radiculopathy. Dr. Verst took this one step further and asserted that what Dr. Jensen described as polyneuropathy is actually evidence of a stenotic syndrome. With this assertion Dr. Doerr was, once again, in rather emphatic disagreement:

> Dr. Doerr:
> A. I do. I completely disagree. Polyneuropathy is not caused by spinal stenosis.
>
> Mr. Jordan:
> Q. You have never seen that in your practice?
>
> A. I have never seen polyneuropathy as a result of spinal stenosis in my 20 years of practice, nor am I aware of any literature to suggest that spinal stenosis will cause polyneuropathy.
>
> Now, if you want to get down to the semantic of what's the definition of polyneuropathy, polyneuropathy means pathology of multiple nerve roots.
>
> Now, can spinal stenosis affect multiple nerve roots?
>
> Absolutely it can.
>
> But polyneuropathy - - the definition, as we use it in the medical profession, polyneuropathy is a different disease process than spinal stenosis. Polyneuropathy is - - as I mentioned before, it tends to be something that is an intrinsic issue with the nerve roots themselves. It affects the nerve roots distally greater than proximally, so people will get stocking glove distribution symptoms, pins and needles, burning, numbness in a non-dermatomal distribution. It's not a term that is used interchangeably with spinal stenosis, nor, in my experience, has it ever been.

Doerr Deposition, 47/14–48/16.

We believe that Dr. Doerr's explanation is also more consistent with Dr. Jensen's comments offered in interpretation of the EMG. To the extent that the EMG informed Dr. Verst's opinion

that the L4-5 fusion accelerated Claimant's degenerative condition to produce spinal stenosis sooner than it otherwise would have, we believe that the EMG actually does not support this conclusion. As Dr. Doerr explained, polyneuropathy is an entity separate and distinct from spinal stenosis. Polyneuropathy affects the nerves distally, and is ordinarily thought to be the result of a metabolic or systemic disease process, such as diabetes. Per Dr. Doerr, it has nothing to do with spinal stenosis.

55. The Commission is mindful of the fact that the three physicians who have offered opinions on the threshold issue are well-qualified and respected in their areas of practice. All three have treated hundreds of patients with degenerative disease of the lumbar spine. All three had access to Claimant's records and radiological studies; it is difficult to challenge any of the opinions on the basis of inadequate or incorrect foundation. All three physicians recognize that ASD does exist. All recognize that Claimant had pre-existing disease at L3-4 and that it has progressed since January 2009. At the end of the day, the difference of opinion comes down to whether Claimant's pre-existing L3-4 disk disease progressed naturally, or at a faster rate than one would normally expect. Both Dr. Verst and Dr. Blair have stated that the L4-5 fusion accelerated Claimant's progression, although they also acknowledge that ASD is only seen in 20 to 25% of the patients who undergo a lumbar fusion. They have not explained why it is more probable than not that in Claimant's case the degeneration was accelerated. On the other hand, Dr. Doerr has cogently explained why Claimant is at low risk for ASD, and why he finds no evidence of abnormal or accelerated progression of Claimant's underlying L3-4 disease. He has also cogently explained why the EMG does not support the proposition that Claimant's degenerative disease has progressed to cause spinal stenosis.

56. Referee Taylor was somewhat critical of Dr. Doerr's opinion because he thought Dr. Doerr incorrectly questioned certain aspects of Claimant's credibility. Dr. Doerr testified that neither he nor Dr. Blair found that Claimant had any lower extremity weakness on clinical exam. Claimant's counsel asked Dr. Doerr to reconcile these findings with the fact that Claimant had suffered a number of falls, the first occurring on February 11, 2013 when Claimant's left leg gave way in the shower. Dr. Doerr noted that while the ER record does reflect giving way of the left leg, it does not reflect that Claimant fell. (*See* Claimant's Exhibit 3 at 88-90.) Therefore, Dr. Doerr was somewhat skeptical of Claimant's history of falling, particularly when he had exhibited no lower extremity weakness on exam, but did exhibit positive Weddell signs. However, as Referee Taylor noted, there is an additional ER record found in Defendants' exhibits, which does reflect that when Claimant's leg gave way on February 11, 2013, he did fall. (See Defendants' Exhibit 2, 01498.) So, Dr. Doerr appears to be mistaken about the occurrence of the fall. We do not believe that this demonstrates any particular prejudice against Claimant by Dr. Doerr. While Dr. Doerr's mistaken belief about Claimant's history of falling might be relevant to challenging his opinion on whether Claimant's problems are severe enough to make him a surgical candidate, it is not particularly relevant to the issue of whether radiological and electro-diagnostic studies demonstrate that Claimant's L3-4 condition has progressed faster than would be expected in the absence of the L4-5 fusion.

57. Referee Taylor also noted that Dr. Doerr agreed with Dr. Verst that Claimant exhibits bony overgrowth of the facet joints at L3-4. Dr. Verst felt that this finding augured in favor of the existence of ASD. Referee Taylor appears to feel that Dr. Doerr's concession about the existence of bony overgrowth is inconsistent with his opinion that ASD is not extant in Claimant's case. We find nothing in Dr. Doerr's testimony which supports this conclusion:

Mr. Bauman:
Q. In Dr. Verst's depo at page 17, lines 15 to 16, he talked about the adjacent segment disorder adding extra bone growth to the joint.

Did you see any radiological evidence, in this case, that extra bone growth had been added to the joint?

Dr. Doerr:
A. Well, the L3-4 facet joints were arthritic in the January 2009 MRI. When we talk about arthritis in the facet joints, that's bone overgrowth. And that gradually progressed between 2009 and 2015.

Doerr Deposition, 75/14-24.

Claimant's bony overgrowth, i.e. facet arthritis predated the subject accident to some extent.

58. While we acknowledge that this is a close case, we believe that Dr. Doerr's opinion is the more persuasive of the views that have been expressed on the threshold issue of causation. We find that Claimant has failed to meet his burden of proving, to a reasonable degree of medical probability, that his L3-4 lesion is causally related to the subject accident. Therefore, we do not reach the secondary question of whether the surgery required by Dr. Verst is reasonable.

CONCLUSIONS OF LAW AND ORDER

1. Claimant has failed to demonstrate to a reasonable degree of medical probability that there is a causal relationship between the subject accident and his L3-4 condition.

2. All other questions are moot.

3. Pursuant to Idaho Code § 72-718, this decision is final and conclusive as to all matters adjudicated.

DATED this 23rd day of December, 2015.

INDUSTRIAL COMMISSION

Participated but did not sign.
R.D. Maynard, Chairman

___/s/_____
Thomas E. Limbaugh, Commissioner

___/s/_____
Thomas P. Baskin, Commissioner

ATTEST:

__/s/_____
Assistant Commission Secretary

CERTIFICATE OF SERVICE

I hereby certify that on the 23rd day of December, 2015, a true and correct copy of the foregoing **FINDINGS OF FACT, CONCLUSIONS OF LAW AND ORDER** was served by regular United States Mail upon each of the following:

CLARK JORDAN
PO BOX 1015
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JON M BAUMAN
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ka ____/s/_____