Cite as 2022 Ark. App. 276

ARKANSAS COURT OF APPEALS

DIVISION IV
No. CV-21-611

MICHAEL BEAN APPELLANT	Opinion Delivered June 1, 2022
V.	APPEAL FROM THE ARKANSAS WORKERS' COMPENSATION COMMISSION [NO. G806384]
REYNOLDS CONSUMER PRODUCTS; INDEMNITY INSURANCE COMPANY OF NORTH AMERICA/SEDWICK CLAIMS MANAGEMENT SERVICES, INC.; AND DEATH & PERMANENT TOTAL DISABILITY TRUST FUND APPELLEES	AFFIRMED

BART F. VIRDEN, Judge

Appellant Michael Bean was diagnosed with a rare condition: pauci-immune anti-neutrophilic cytoplasmic autoantibody (ANCA) vasculitis, also known as granulomatosis with polyangiitis (GPA), or pulmonary vasculitis, and formerly known as Wegener's disease. Bean alleged that the ANCA vasculitis, which caused damage to his kidneys and lungs, resulted from his exposure to silica dust while working for appellee Reynolds Consumer Products. The Arkansas Workers' Compensation Commission denied benefits after finding that Bean failed to prove that he sustained a compensable accidental injury or that he sustained a compensable silicosis injury. Bean argues on appeal that (1) his accidental injury

is compensable; (2) his injury was not "legally idiopathic"; and (3) the Commission arbitrarily disregarded the opinion of his medical expert. We affirm the Commission's decision.

I. *Background*

On May 3, 2018, then thirty-four-year-old Bean was referred by his primary care physician (PCP) to CHI St. Vincent with the chief complaint of abnormal lab results, which he had received earlier that day. Specifically, Bean sought an "evaluation of elevated creatinine and BUNs." It was noted that Bean is a body builder who uses creatinine, protein supplements, and Aleve, along with other NSAIDs (nonsteroidal anti-inflammatory drugs) for his back pain. It was further noted that Bean drinks mostly energy drinks, rather than water, and that he had gone to his PCP that day complaining of indigestion issues. The medical records indicate, "Suspect patient's symptoms are likely coming from his creatinine and protein intake as well as his NSAID abuse. Although intrinsic kidney function cannot be excluded." Bean was admitted to the hospital with acute renal failure—it was noted that he had a gradual onset of symptoms and "duration: > 1 month." The hospital gave him intravenous fluids and conducted studies. Bean was provided a gastroenterology consultation on May 4. Dr. Sanford Henry Benjamin noted that Bean had chronic back pain for which he took nonsteroidal medications several times a day and had developed epigastric pain two months ago. Bean was given a chest x-ray and CT scan on May 5 due to shortness of breath and coughing up blood. The impression was bilateral upper lobe and right lower lobe airspace disease consistent with pneumonia. Bean was ultimately diagnosed with acute renal failure, pulmonary alveolar hemorrhage, and "suspected vasculitis."

On May 5, Bean was transferred to the University of Arkansas for Medical Sciences (UAMS) at his request. Dr. Devendra D. Patel noted the earlier symptoms Bean had described to doctors at St. Vincent, and she pointed out that "[Bean] has exposure to Aluminum as he has been working in Aluminum plant for 2–3 years." Dr. Patel's impression was acute renal failure "probably related to long term NSAID use vs vasculitis (with hemoptysis)." She diagnosed anemia, dyspepsia, and hemoptysis. A kidney biopsy was taken on May 7, the findings of which were consistent with an ANCA-associated disease. A chest x-ray was taken on May 8, and it was noted that Bean had "significant interval worsening of patchy consolidation within both lungs . . . this could reflect other hemorrhage versus multifocal pneumonia." Bean was transferred to the MICU on May 9 due to "worsening hypoxic failure which is thought to be due to DAH secondary for vasculitis (pulmonary renal disease)." Bean was placed in a medically induced coma and later diagnosed with ANCA vasculitis.

On May 15, Dr. Manisha Singh, a board-certified nephrologist and internist, reported that Bean was critically ill. Referring to ANCA vasculitis, Dr. Singh wrote, "This is the one disease that can fully explain this clinical picture along with biopsy readings . . . The driver of his clinical condition has to be pauci immune crescentic GN [glomerulonephritis]." Bean was discharged from UAMS on May 23, 2018; however, Dr. Singh continued to see him for nearly a year. In late August, Dr. Singh reported that Bean's renal failure had suddenly become worse over the last month.

In September 2018, Bean completed a Form AR-C describing an inhalation injury on May 1, 2018, that had damaged his kidneys and lungs. On a "Workers' Compensation First Report of Injury or Illness" form prepared by Georgia Diemer, the same information from the Form AR-C was reported with additional information that Bean had last worked at Reynolds on April 18, 2018, and that his disability had begun April 19. It was also reported that the time of the occurrence could not be determined; that the injury, illness, or exposure did not occur on the employer's premises, yet it did occur at the address for Reynolds; that the specific activity or work process that Bean was engaged in when the accident, illness, or exposure occurred was driving a forklift; and that he notified his employer of his injury on September 24, 2018.

Bean returned to work for Reynolds in January 2019, but his condition deteriorated, and he underwent a kidney transplant in March 2019. Bean again returned to work at Reynolds in March 2020.

On July 25, 2020, Dr. Singh was asked on a questionnaire whether she believed with a reasonable degree of medical certainty that the injuries/conditions for which she had treated Bean were caused by exposure to silica at his workplace. She stated, in part, that

> It is difficult to say exactly what caused the [ANCA] GN, but in his history, the only thing that we were able to find is known to be associated with this condition – was the exposure to silica. This is a rare disease [so] not much is known about it. We concluded that this must be the inciting event.

Dr. William Banner, Jr., was asked to review the case for Reynolds. In a report dated October 14, 2020, he stated, in part, the following

The key question in this case is whether there is a causal relationship between Michael Bean's exposure to silica while working at the Reynolds facility and his development of a pulmonary-renal syndrome. As can be seen in this case with conflicting results, testing for ANCA my yield some difficult to interpret results. Nevertheless, his kidney failure is consistent with a minimal (pauci) immune disorder. As stated in a recent article by Scott et al. (Scott, Hartnett, Mockler & Little, 2020), "Like many autoimmune diseases, the exact etiology of AAV (ANCA associated vasculitis), and the factors that influence relapse are unknown. Evidence suggests a complex interaction of polygenic genetic susceptibility, epigenetic influences and environmental triggers." At this point causal links to these associated "triggers" have not been well defined and at this point remain associations . . . . A thorough evaluation of possible exposure in an individual patient would have to also consider the type of silica including particle size and structure.

. . . .

To assess the role of exposure time, it is important to consider the exposure times associated with the development of silicosis . . . . The term acute silicosis which can occur in shorter periods of time requires extremely high concentrations and produces severe symptoms. (Pollard, 2016) Mr. Bean by his deposition has indicated that he worked in this facility for two years. At no point in time did he complain of acute severe silicosis[,] and his chest CT did not at any point reveal chronic nodular inflammatory changes associated with silicosis.

. . . .

[Mr. Bean] had no indication of clinical silicosis either from a complaint history or from the CT scan that was obtained during his hospitalization. His lung disease was diffuse alveolar hemorrhage which is related to his autoimmune vasculitis. That is not surprising in that exposure to silica takes a much longer exposure time to produce clinical silicosis than Mr. Bean had during his work history. Even without a mechanistic causal link between silica exposure, silicosis and autoimmune disease particularly with the anti-nuclear cytoplasmic antibodies[,] the data support that the pathway to autoimmune disease is via the development of clinical silicosis and not asymptomatic silica exposure.

I would conclude to a reasonable degree of medical certainty that Mr. Bean's autoimmune vasculitis with pauci syndrome crescentric glomerulonephritis is not associated with his exposure to silica crystals at his place of employment but is rather idiopathic. I base this on the length of exposure and the lack of clinical signs and symptoms of pulmonary silica exposure.

> The conclusions reached by Dr. Singh and the clinicians at the University of Arkansas Medical School were based on a simplistic review of the literature and did not take into account the timing or degree of exposure with clinical symptomatology. As exemplified by the list in appendix A, a thorough evaluation would require an in-depth occupational history and even with that there may be no specific etiology that can be determined in many cases.

Dr. Singh was deposed on January 21, 2021. The following exchange occurred during her deposition on direct examination by Bean's lawyer:

> Q: So over this year-long treatment that you'd had with Mr. Bean, your conclusion was that it was his exposure to silica that caused the ANCA, is that correct?
>
> A: That's correct, yes . . . . Most likely, yes. That's my take on this, because I couldn't find anything else.
>
> Q: Okay. Is it more likely than not that the exposure to silica caused the ANCA?
>
> A: At this moment, yes . . . .
>
> Q: Other than his exposure to work, could you find any other explanation for his diagnosis of ANCA?
>
> A: No, I could not . . . .
>
> Q: Is it your testimony that you can state within a reasonable degree of medical certainty that Mr. Bean's exposure to silica at work caused his symptoms and his autoimmune response of ANCA vasculitis?
>
> A: Yes, that's correct.

On cross-examination, the following exchange occurred:

> Q: Do you agree that the cause of ANCA is clinically unknown?
>
> A: Cause of all ANCAs? Yes. It's clinically at this point not known.
>
> Q: Okay. Now, what do you know about the silica exposure in this case?

A: From what Mr. Bean told me, he said that he is, there's a lot of dust around him. He physically handles the – and I'm trying to recollect, because in my mind, my picture was he's lifting sacks really, but I don't think it was sacks. But he said that he was physically in contact with it. It was skin-to-skin contact, and he was inhaling a lot of it, too. The dust was all around him.

Q: And how much of the time during the day did he suffer this exposure?

A: Eight to ten hours.

Q: Okay. Did you ever get a sample of this silica?

A: No.

Q: You've not seen it?

A: No . . . .

Q: So your opinion is based solely on what Mr. Bean told you?

A: Yes, that's correct.

Q: Would your opinion change if, for example, the period of time he was exposed to silica was inaccurate from your information?

A: So if you tell me that he was exposed for one day that would change. But if it's a year, I have no studies that would tell me that how much this much in a year would cause this. You see, for us it's an association. And all the studies that have come off associations have come after, in a different patient group altogether. Those are all much older people working for years and years and years. There aren't studies that are looking at one year or, you know, six months exposure. We don't have studies for that . . . .

Q: And how long was he exposed to silica of any amount?

A: From my understanding, one to two years . . . .

Q: Does Mr. Bean have silicosis?

A: No.

Q: He does not?

A: Not from what we've done. We didn't work him up for silicosis as such. We were treating him for ANCA vasculitis, ANCA G, glomerulonephritis. But when we are saying silicosis, we are specifically looking at a lung, a very specific factor in the lung that happens after years of exposure, acute or chronic silicosis. Basically, it's a family disease, and I'm not an expert on that. It's a lung disease.

At a hearing before an administrative law judge (ALJ) on January 29, 2021, Bean testified that he is still working at Reynolds doing the same job despite being warned by his doctors to avoid exposure to silica. He testified that he was hired to work at Reynolds in June 2016 and that he worked twelve-hour shifts as "general utility" three days on and three days off. His job duties ranged from running a forklift and crane to just cleaning up in the breakroom. Bean said that he also worked on a furnace—but not every single day—adding metal and alloys, including iron, zinc, copper, and silica. He said on cross-examination, "Silicon, silica, I don't know but that's what it says on the bag." Bean testified that Reynolds has eight furnaces and that he worked on one, but not more than two, furnaces at a time. He stated that the alloys are in twenty-five-pound bags and that he added about 300 pounds of each alloy to the furnace using a front-end loader.

Bean said that the furnaces were "always" needing to be replaced and that, since he had been there, every furnace in the cast house had been replaced up to four times. Bean testified that contractors were brought in to do the job and that they used jackhammers to "tear down" the furnaces by breaking bricks to take the refractory lining out and replace it. Bean said that this process takes a few weeks up to a month and that it created a lot of silica

dust. Bean said that the silica dust during a "tear down" floated in the air, that it settled onto surfaces, and that he would sweep it into piles. On cross-examination, Bean conceded that he was not constantly exposed to dust because he walked outside some and stayed in the air-conditioned and air-filtered breakroom. Bean testified that he was working next to a furnace that was being torn down "probably some time in April [2018] maybe" and that he became sick and had difficulty breathing shortly afterward. He said that he had told someone at work that his "breathin' [was] not right" and that, as time went on, he "[didn't] feel good," so he went to see his doctor in early May.

Brian Elliott, the environmental manager at Reynolds, testified that Reynolds makes aluminum sheets and coils. He testified that the cast house is a large building containing eight furnaces. Elliott testified that the bay doors on each end of the cast house typically stay open most of the day, that side doors can be opened, and that the southern wall of the cast house is louvered for ventilation. He insisted that the building has good air circulation. Elliott said that utility workers stayed in the cast house anywhere from three to six hours, depending on how many furnaces they were running but that they also spent a "fair amount of time" in the breakroom. Elliott stated that silicon metal is the material added to the furnaces—not silica—and that this silicon does not have silica granules, particles, or dust. He described the furnace as basically a steel shell with a refractory lining and said that the refractory lining of the furnaces needed to be replaced "periodically." According to Elliott, outside contractors do the job; they cover everything in plastic to contain the dust during the job; and they clean up after the job is complete. Elliott said that, when the furnace lining

is being replaced, the dust is "very minimal." When asked if the alloys are still in the furnace when it is being "torn down," Elliott said that "there shouldn't be anything in there. I mean, there could be very minor amounts of aluminum solidified and stuck to [the refractory lining]."

II. *Appeal to the Commission*

The ALJ addressed four theories of compensability asserted by Bean, which the ALJ described as "somewhat confusing and, apparently at least, mutually exclusive and contradictory." The ALJ addressed accidental injury under Ark. Code Ann. § 11-9-102(4)(a)(i) (Repl. 2012); a pulmonary injury under Ark. Code Ann. § 11-9-114(a) (Repl. 2012); an occupational disease under Ark. Code Ann. § 11-9-601(a) (Supp. 2021); and a silicosis injury under Ark. Code Ann. § 11-9-602(a)(2) (Repl. 2012). The ALJ found on each theory that Bean had failed to prove compensability and, in doing so, repeatedly referred to Bean's "medically and legally idiopathic autoimmune disease, ANCA vasculitis/GPA/pulmonary vasculitis." Bean appealed to the Commission. When a determination by an ALJ is appealed to the Commission, the Commission does not sit as an appellate court to review the ALJ's findings; instead, the Commission makes a de novo determination of the facts. *Jackson v. Smiley Sawmill, LLC*, 2019 Ark. App. 235, 576 S.W.3d 43.

The Commission affirmed the ALJ's decision as modified. At the outset, the Commission noted that, although Bean had initially claimed that he suffered from an occupational disease, he had since claimed that he suffered "a compensable injury, not an

occupational disease." Therefore, the Commission did not address occupational disease in general but did address, specifically, a silicosis injury caused by inhalation of silica dust.[1] The Commission found that Bean failed to prove that he sustained a compensable injury pursuant to section 11-9-102(4)(A)(i) and that, alternatively, he failed to prove a compensable silicosis injury pursuant to section 11-9-602(a)(2). Bean does not challenge the Commission's decision that he failed to prove a compensable silicosis injury.

III. *Standard of Review*

In reviewing decisions from the Commission, we view the evidence and all reasonable inferences deducible therefrom in the light most favorable to the Commission's findings. *Willis v. Ark. Dep't of Corr.*, 2021 Ark. App. 50, 616 S.W.3d 679. When the Commission denies benefits because the claimant has failed to meet his or her burden of proof, the substantial-evidence standard of review requires that we affirm if the Commission's decision displays a substantial basis for the denial of relief. *Id.* The issue is not whether the appellate court might have reached a different result from the Commission but whether reasonable minds could reach the result found by the Commission; if so, the appellate court must affirm. *Id.* Questions concerning the credibility of witnesses and the weight to be given to their testimony are within the exclusive province of the Commission. *Id.* Once the Commission has made its decision on issues of credibility, the appellate court is bound by that decision. *Id.*

[1]A silicosis injury is dealt with separately from a claim for an occupational disease. *Johnson v. Democrat Printing & Lithograph*, 57 Ark. App. 274, 944 S.W.2d 138 (1997).

IV. *Discussion*

A. Accidental Injury

Bean contends that the Commission erred in finding that he failed to prove that he suffered a compensable injury under Ark. Code Ann. § 11-9-102(4)(A)(i). Compensable injury means an accidental injury causing internal or external physical harm to the body, arising out of and in the course of employment and which requires medical services or results in disability or death. *Id*. An injury is "accidental" only if it is caused by a specific incident and is identifiable by time and place of occurrence. *Id*. The phrase "arising out of the employment" refers to the origin or cause of the accident, and the phrase "in the course of the employment" refers to the time, place, and circumstances under which the injury occurred. *J.&G. Cabinets v. Hennington*, 269 Ark. 789, 600 S.W.2d 916 (1980). A compensable injury must be established by medical evidence supported by objective findings. Ark. Code Ann. § 11-9-102(4)(D). Objective findings are those findings that cannot come under the voluntary control of the patient. Ark. Code Ann. § 11-9-102(16)(A)(i). Causation does not need to be established by objective findings, so long as objective medical evidence establishes the injury's existence and a preponderance of other nonmedical evidence establishes a causal relation to a work-related incident. *Wal-Mart Stores, Inc. v. VanWagner*, 337 Ark. 443, 990 S.W.2d 522 (1999). The burden of proving a compensable injury under section 11-9-102(4)(A)(i) is on the employee to prove by a preponderance of the evidence. Ark. Code Ann. § 11-9-102(4)(E)(i).

Bean asserts that he sufficiently identified the time frame of the occurrence as April 2018 because he got sick in early May after a furnace next to him had been "torn down." He relies on *Pafford Medical Billing Services, Inc. v. Smith*, 2011 Ark. App. 180, 381 S.W.3d 921, for the proposition that he did not have to identify the exact date of injury. The Commission acknowledged that a precise time and numerical date is not required; however, Bean had worked at Reynolds for two years and claimed that the furnaces were always being "torn down." The Commission referred to Bean's testimony that he "[did] not believe" that he had been feeling sick prior to the "tear down" of the furnace in April but that his injury did not happen in one day. During its recitation of the history of the case, the Commission pointed out the many inconsistencies in Bean's reporting of the injury to his employer.

The Commission found that Bean failed to prove that he sustained an injury as a result of "acute exposure to the silica dust." Bean continues to argue on appeal, as he did below, that his injury resulted from a "single instance" of exposure from which he developed symptoms within two days. The Commission pointed out that on May 3, Bean sought medical treatment for symptoms ranging from abnormal creatinine levels to indigestion and that neither he nor his medical providers attributed those symptoms to his work at Reynolds. Even on May 4 and 5, Bean's problems were described as epigastric and back pain over the past two months and stomach discomfort for about a month.

Bean insists that Dr. Singh's opinion established that his injuries were caused by exposure to silica at work after eliminating all other potential causes. Dr. Singh's opinion, however, primarily relied on Bean's statements to her about his working conditions, which

were inaccurate in many respects. For example, he was not physically carrying bags of silica such that he had "skin-to-skin contact" with the alloy; rather, he was using a front-end loader. There was even some dispute about whether the substance Bean was exposed to was silica given that Elliott drew a distinction between silicon and silica. Moreover, there is some indication in the record that it is crystalline silica that is dangerous, but Dr. Singh had not examined the substance for herself to determine its type, structure, and size. Also, Bean described dust all around him and on surfaces, but Elliott said that there was not much dust in the air during the tear downs and that the air flow is good in the building. Dr. Singh thought that Bean was exposed to the dust eight to ten hours a day, but Elliott said that, assuming Bean was working two furnaces, he could be working at the furnaces for six hours a day, and Bean himself said that he did not work on a furnace every single day. It seems clear that Dr. Singh considered Bean's exposure to be the two-year period that he had worked for Reynolds and not, as Bean insists, "a single instance" of exposure to silica that caused Bean to develop ANCA vasculitis within two days. In her deposition, Dr. Singh said that her opinion on causation would change if she were told that Bean had been exposed to silica for one day.

Dr. Banner opined that Bean's condition was idiopathic, and Dr. Singh essentially agreed that there is no known cause of ANCA vasculitis. Further, Dr. Banner stated that there was no indication that Bean suffered from clinical silicosis, with which Dr. Singh agreed, and that "the pathway" to development of ANCA vasculitis is through "the development of clinical silicosis and not asymptomatic silica exposure." Bean basically asserts

that it is just common sense that he breathed in silica dust and developed ANCA vasculitis. Speculation and conjecture, even if plausible, cannot take the place of proof. *Kimble v. Lab. Force, Inc.*, 2013 Ark. App. 601, 430 S.W.3d 156.

Bean also argues that the Commission erred in finding that there was no "conclusive evidence" demonstrating that his condition was causally related to the alleged inhalation of silica dust. The Commission made that statement with respect to its finding that Bean failed to prove a silicosis injury. *See* Ark. Code Ann. § 11-9-602(b). Again, that aspect of the Commission's decision is not challenged on appeal. We hold that the Commission's decision displays a substantial basis for the denial of relief.[2]

B. Arbitrary Disregard of Medical Testimony

Bean argues that the Commission arbitrarily disregarded Dr. Singh's medical testimony. Although it is true that the Commission may not arbitrarily disregard evidence, the Commission's failure to specifically discuss conflicting evidence does not mean that it was arbitrarily disregarded where there is substantial evidence to support its decision. *Est. of Bogar v. Welspun Pipes, Inc.*, 2014 Ark. App. 536, 444 S.W.3d 405. In workers'-compensation cases, arbitrary disregard of evidence is demonstrated when, for example,

[2]Bean further argues that he suffered an occupational *injury* as opposed to an occupational *disease*. He draws a distinction between the two and asserts that there is ambiguity in the statutory definitions, which should be resolved in his favor. We note, however, that he specifically abandoned the theory that he suffered an occupational disease pursuant to Ark. Code Ann. § 11-9-601 such that the Commission did not address it. Because the Commission did not address the theory at all, and given that we have affirmed the Commission's decision that Bean failed to prove a compensable injury, we need not further address Bean's point on the distinction between an injury and a disease.

the Commission affirmatively states that there is "no evidence" for a proposition when such evidence has, in fact, been presented in the proceeding. *Id.* Arbitrary disregard has also been described by this court in *Lonoke Exceptional School, Inc. v. Coffman*, 2019 Ark. App. 80, 569 S.W.3d 378, as follows:

> The Commission cannot disbelieve the testimony of a witness for an irrational or whimsical reason; for example, it cannot decide a case on the rationale that witnesses with names beginning in vowels are never credible, or that foreign-born doctors always offer more accurate medical opinions, or that back injuries are never work-related.

Coffman, 2019 Ark. App. 80, at 4, 569 S.W.3d at 381 (quoting *Pyle v. Woodfield, Inc.*, 2009 Ark. App. 251, at 6, 306 S.W.3d 455, at 459 (Pittman, J., concurring)).

We hold that the Commission did not arbitrarily disregard Dr. Singh's opinion. The Commission quoted from both doctors' opinions and simply gave more weight to Dr. Banner's opinion that Bean's injury was idiopathic. The Commission has the duty of weighing the medical evidence as it does any other evidence. *Believ v. Lennox Indus.*, 2010 Ark. App. 112. The Commission further has the authority to accept or reject medical opinions, and its resolution of the medical evidence has the force and effect of a jury verdict. *Id.* The determination of the credibility and weight to be given a witness's testimony is within the sole province of the Commission, which is not required to believe the testimony of the claimant or any other witness but may accept and translate into findings of fact only those portions of the testimony it deems worthy of belief. *Id.*

Moreover, the Commission is not bound by a doctor's opinion that is based largely on facts related to him or her by the claimant where there is no sufficient independent

knowledge upon which to corroborate the claimant's claim. *Id.* Here, as noted during the discussion of Bean's previous point, Dr. Singh in rendering her opinion primarily relied on what Bean had reported to her, some of which was inaccurate and inconclusive.

C. "Legally Idiopathic"

An idiopathic injury is one that is personal in nature or peculiar to the individual; therefore, it is not work-related. *Little Rock Convention & Visitors Bureau v. Pack*, 60 Ark. App. 82, 959 S.W.2d 415 (1997). Bean argues that, while ANCA vasculitis is medically idiopathic because its true cause is unknown, it is not legally idiopathic.[3]

The Commission affirmed but did not adopt the ALJ's decision. While the ALJ called Bean's injury "legally idiopathic," the Commission did not. This court reviews only the findings of the Commission and ignores those of the ALJ. *Graham v. Turnage Emp. Grp.*, 60 Ark. App. 150, 960 S.W.2d 453 (1998). It is well settled that the ALJ's findings are irrelevant for purposes of appeal. *Sheridan Sch. Dist. v. Wise*, 2021 Ark. App. 459, 637 S.W.3d 280. "As such, we do not recount the ALJ's conclusions herein." *Id.* at 5 n.5, 637 S.W.3d at 283 n.5. We give the ALJ's findings no weight whatsoever. *Multi-Craft Contractors, Inc. v. Yousey*,

[3]In his reply brief, Bean raises an argument that this was an aggravation of a preexisting condition, and he also made an "eggshell skull claimant" argument; however, Bean did not raise those arguments before the Commission and therefore obtained no ruling. We have routinely held that we will not consider arguments presented for the first time on appeal. *Harrison v. Street & Performance, Inc.*, 2017 Ark. App. 611, 533 S.W.3d 648. Further, we do not consider arguments raised for the first time in a reply brief because the appellee would have no opportunity to rebut the argument. *Helena/W. Helena Schs. v. Hislip*, 78 Ark. App. 109, 79 S.W.3d 404 (2002).

2018 Ark. 107, 542 S.W.3d 155; *Clark v. Peabody Testing Serv.*, 265 Ark. 489, 579 S.W.2d 360 (1979); *Johnson v. Hux*, 28 Ark. App. 187, 772 S.W.2d 362 (1989).

Affirmed.

HARRISON, C.J., and VAUGHT, J., agree.

Laura Beth York, for appellant.

Michael Ryburn, for appellees.