BEFORE THE ARKANSAS WORKERS' COMPENSATION COMMISSION

CLAIM NO. G806384

MICHAEL BEAN, EMPLOYEE	CLAIMANT
REYNOLDS CONSUMER PRODUCTS, EMPLOYER	RESPONDENT
INDEMNITY INS. CO. OF NORTH AMERICA/ SEDGWICK CLAIMS MG'T SERVICES, INC., INSURANCE CARRIER/TPA	RESPONDENT NO. 1
DEATH & PERMANENT TOTAL DISABILITY TRUST FUND	RESPONDENT NO. 2

OPINION FILED SEPTEMBER 1, 2021

Upon review before the FULL COMMISSION in Little Rock, Pulaski County, Arkansas.

Claimant represented by the HONORABLE LAURA BETH YORK, Attorney at Law, Little Rock, Arkansas.

Respondents No. 1 represented by the HONORABLE MICHAEL E. RYBURN, Attorney at Law, Little Rock, Arkansas.

Respondents No. 2 represented by the HONORABLE DAVID L. PAKE, Attorney at Law, Little Rock, Arkansas.

Decision of Administrative Law Judge: Affirmed as modified.

OPINION AND ORDER

The claimant appeals an administrative law judge's opinion filed April 29, 2021. The administrative law judge found that the claimant failed to prove he sustained a compensable injury. After reviewing the entire record *de novo*, the Full Commission affirms the administrative law judge's opinion as modified. The Full Commission finds that the claimant did not prove he sustained a compensable injury in accordance with Ark. Code Ann. §11-9-

102(4)(A)(i)(Repl. 2012). We find that the claimant did not prove he sustained a compensable "silicosis" injury in accordance with Ark. Code Ann. §11-9-602(a)(2)(Repl. 2012).

I. HISTORY

The record indicates that Michael Brady Bean, now age 38, became employed with the respondents, Reynolds Consumer Products, on June 13, 2016. The claimant's Occupation/Job Title was General Utility. The claimant testified on direct examination:

> Q. What were your job duties?
> A. That could – that could be from running a fork truck, running a crane, running a furnace, possibly just cleaning up in the break room….
> Q. What were you doing at a furnace?
> A. Well, in the furnace you load approximately 75,000 pounds into a furnace with plates. Your plates weigh 1,500 to 1,800 pounds depending on the weight of the piece of metal. You would actually heat that up for approximately six to eight hours….
> Q. How many furnaces are there at Reynolds?
> A. Eight.
> Q. Okay. And are all eight of those furnaces running at the same time?
> A. Most of time but not always.
> Q. Okay. Are you – when you're working on a furnace, are you working on one furnace or all eight furnaces?
> A. Either one or two, no more than two….
> Q. So in my mind what I'm imagining is a big brick furnace that has cement in it and there's a fire going on in it.
> A. Yeah, that's correct.
> Q. Okay. And what are you placing into the furnace?
> A. Other than metal?
> Q. Well, metal or what else?
> A. Alloys such as zinc, copper, silica, and iron. Those are our main alloys….

Q. Now, the alloy that you're putting in; zinc, iron, silica, copper, those alloys, are you putting each one of these alloys in at a time or are you putting one? How – describe to us what you're putting into this furnace.
A. Well, they're 25-pound bags. And most of the alloy that we run, it'll be iron and silica. Those are your two main ingredients that they use. And usually it'll be 300 pounds of each….
Q. Now, as far as you know, you've testified that the four alloys that you utilize are zinc, iron, silica, and copper.
A. Yes.
Q. Are those the only ones that you know of?
A. I believe so….
Q. Now, have there ever been furnaces replaced?
A. Always. There's always gonna have to be a furnace that's gonna have to be replaced in time….
Q. So when you are replacing a furnace, what they do (sic)? What is that process?
A. Well, we don't – we don't do it our self. They usually have contractors come in. They will come in there and they'll jackhammer. They use cutting – cutting materials to cut a lot of your bricks up to basically take that refractory out, pull all that stuff out, and try to rebuild from scratch….
Q. Now, when they do that, what's your environment like to work in?
A. Well, it all depends. It depends on if you're right beside the furnace where this work is takin' place. You're fixin' to see a lot of silica dust on the ground, a lot. There's so much of it that I swept it up in piles.
Q. Okay. So during your time at Reynolds, you had the occasion to be working next to one of these furnaces that was being torn down. Is that correct?
A. Yes. Back when I was – the day I actually went, you know I got sick, it – it was takin' place right beside me. I was on the #5 furnace and the #4 furnace was the one bein' worked on, and I'm sure there's paperwork that can show that.
Q. Okay. So this #4 furnace that was being torn down – and you say you were sweeping up silica?
A. Yes. Well, it's all in the air. In fact, it's still on railing right now. I can take you there today and show you.
Q. Okay. So there's a lot of debris in the air?

> A. Yes. Not all – not all the time but when that – that kind of stuff there is takin' place, yes, there is a lot.
> Q. Okay. And do you recall approximately when #4 furnace was being torn down?
> A. It was before May of '18, so I'm gonna say probably some time in April, maybe.
> Q. Do you have an exact date on that?
> A. I – I don't have an exact date. No, I don't.
> Q. Okay. Did you start feeling sick – were you feeling sick at any point prior to tearing down of furnace #4?
> A. I do not believe so….I was there several days when that was takin' place.
> Q. Okay. This didn't just happen one day?
> A. No. No. No….
> Q. Now Mr. Bean, tell us about 2018 and the symptoms that you started to experience.
> A. Well, it wasn't like I got by myself, "Oh, my God, I can't breathe." I just noticed, I thought maybe – I actually said somethin' to somebody at work that my breathin' is not right, and they asked me what was wrong, and I said I don't know….But as time went on I just felt – I was like I don't feel good, so, you know, that's when I actually went to the doctor, which would have been May the 2nd maybe, 3rd, 5th, along through there.

The parties stipulated that the employment relationship existed at all pertinent times, including May 1, 2018. The claimant contended that he "began having symptoms of an occupational illness or disease" on May 1, 2018 "which caused permanent damage to his kidneys and lungs."

The respondents' attorney examined Brian Elliott, the respondent-employer's Environmental Manager:

> Q. What is the product that is made there?
> A. The product that is made there is aluminum sheet and aluminum coils.
> Q. And those aluminum coils eventually get processed somewhere else into aluminum foil?

A. The bulk of it does, yes….
Q. Describe, if you will, the cast house.
A. The cast house itself is large, metal walled and roofed building, concrete floors. It consists of eight total melting furnaces, seven of which would be called legacy furnaces….
Q. Okay. Now, are both ends of this large building open?
A. Yes, there is a large bay door on the east end, which we call Door 2. They're numbered sequentially. Actually, that's Door 1. The one on the west side is Door 4. There's also doors on the south end of that which would be Door 2 and also Door 3, but all of 'em are large bay roll-up doors that typically stay open most of the time that large mobile equipment's goin' in and out of a good part of the day.
Q. Is the air circulation good because these doors are open?
A. Yeah. I mean, yeah, and there's man doors that typically can be opened, too. I mean, it's – air's movin' freely through there….
Q. Are you familiar with Mr. Michael Bean?
A. Yes, I know Brady….
Q. Are you familiar with his job?
A. He's general utility….
Q. On a day-to-day basis, how much time does Mr. Bean spend in the cast house?
A. I'm goin' to speak for just the average general utility there, typically their day's gonna consist of probably – actually have to be in the cast house but depends on whether they're workin' one cast line or two cast lines. Some of this – some time periods they have worked general utility working two cast lines, other times we've have (sic) to let employees that they typically work one. So if you were workin' one, probably a little over three hours. If you were workin' two, you know, obviously there'd be a little over six hours that you would be in the cast house or have to be in the cast house….
Q. So it's your testimony then that Mr. Bean is not exposed to the silica dust in the cast house for an entire shift every day?
A. No….
Q. Mr. Bean testified about some bags of silica that he puts in the furnace.
A. Okay. I think it may be the wrong terminology there. It's – it's actually – what we use is alloy – alloy material that our employees place bags into the furnace is silicon metal.
Q. It's silicon –

A. Yeah.
Q. with a C-O-N, right?
A. S-I-L-I-C-O-N.
Q. Not silica.
A. No, sir.
Q. And this silicon is not silica granules or silica particles or silica dust.
A. No, sir.
Q. It's an integral part of the process.
A. Yes. It is [an] alloy material for the aluminum….
Q. Now, Mr. Bean also testified about the tearing down of the furnaces and rebuilding them.
A. Okay.
Q. Is that something that goes on continuously?
A. No. That's periodically, I mean, very episodic.
Q. Okay. And when that is done, is it done by an outside contractor?
A. Yes.
Q. Is that something that they do at night or at some time when the workers aren't around the furnaces?
A. It could happen at night, at times. It could go day or night shift.
Q. And do they enclose these furnaces with plastic when they do that?
A. Yes. Typically they do and they'll – there's a large furnace door that's basically a whole front. It's the middle refractory line door and it raises up by hydraulics. That furnace opening, they would drape a sheet of visqueen across that opening to contain any of that dust if they tore it out….
Q. Now, then out in the cast house can you see silica particles or stuff in the air, or is it, like, obvious?
A. I don't – I mean, I don't see stuff in the air just on a day-to-day basis, no. No.

According to the record before the Commission, the claimant treated at CHI St. Vincent Hospital Hot Springs on May 3, 2018:

> 34-year-old male presents for evaluation of elevated creatinine and BUNs. The patient is a body builder and uses creatine, protein supplements and uses Aleve and NSAIDs frequently. The patient says that he also drinks mostly energy

> drinks and is not drinking much water. The patient went to see his primary care physician today for indigestion type symptoms and blood work indicated a creatine of 4 and BUN of nearly 100. The patient has no evidence of edema, or other electrolyte disturbance. He is otherwise healthy. Denies steroid or testosterone injection….
> 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. CK and urine studies have been added and the patient will be admitted for IV fluids.
> Further workup shows no elevation of CK but the patient's urine studies do show blood and protein which may be indicative of an intrinsic nephropathy. [He will be] admitted for further workup, IV fluid replacement.

The claimant was admitted to St. Vincent Hospital for "ARF (acute renal failure)." It was noted, "Onset of symptoms: gradual….Duration:>1 month[.]"

An x-ray of the claimant's kidneys was taken on May 3, 2018 with the impression, "No hydronephrosis bilaterally. Bilateral ureteral jets are identified. Both kidneys are slightly enlarged with slightly increased echogenicity and loss of cortical medullary differentiation. The findings are nonspecific but may relate to the history of acute kidney injury."

Dr. Sanford Henry Benjamin provided a Gastroenterology Consult on May 4, 2018:

> 34-year-old gentleman admitted with new onset renal failure. The patient has had chronic back pain for which he takes nonsteroidal medications several times a day. 2 months ago he started getting epigastric pain. The pain was more like an epigastric gurgling discomfort with associated nausea, worse after meals and at night. He has tried multiple over-the-

> counter medications none of which seem to help too much. He has lost 25 pounds over the past 2 months. Over the past several years, he has lost more starting at over 300 pounds. He has no family history of gallbladder disease. He does not smoke, drink alcohol to excess and has no history of peptic ulcer disease. His bowel movements have been dark from Pepto-Bismol. Guaiac testing has not yet been done. His iron studies are normal although he has a mild anemia. He takes multiple nutritional supplements and has a low carbohydrate diet.
> CT scan of the abdomen and pelvis last year for hematuria was unremarkable.

An x-ray of the claimant's chest was taken on May 5, 2018 with the impression, "Confluent airspace opacities in the right upper lobe and scattered patchy airspace opacities diffusely, bilaterally. The findings may relate to multifocal pneumonia although underlying pulmonary nodules/masses are not excluded."

A CT of the claimant's chest was taken on May 5, 2018 with the following interpretation:

> The heart is normal in size. The aorta is normal in caliber. No suspicious mediastinal adenopathy is identified. There is airspace disease in the right upper lobe and in the superior segment of the right lower lobe as well as patchy airspace disease in the left upper lobe. There are air bronchograms. No pleural fluid collections are identified. The adrenal glands are normal. The osseous structures are intact.
> IMPRESSION: Bilateral upper lobe and right lower lobe airspace disease consistent with pneumonia.

The claimant was discharged from St. Vincent Hospital on May 5, 2018 with the diagnoses of ARF, Pulmonary alveolar hemorrhage, and

"Suspected vasculitis – [was] scheduled for renal biopsy: requested transfer to UAMS."

The claimant began treating at University of Arkansas for Medical Sciences on May 5, 2018. Dr. Devendra D. Patel reported at that time:

> Michael Bean is a very pleasant 34 y.o. male with PMH of chronic low back pain, who was transferred from CHI SVI Hot Spring to UAMS on 5/5/2018 for worsening renal failure and as per family request. Patient initially admitted at OSH on 5/3/18 for abnormal labs. He has been having stomach discomfort (abnormal feeling in epigastric area) for about a month. This discomfort usually gets worse with eating and somewhat better with acid medications. His symptom continued to worsen, so he saw a doctor, where he had labs done, and was found to have significantly abnormal labs (elevated Cr), so he was referred to OSH ED for admission. He also mentions feeling nauseated, but denies for vomiting. He was feeling short of breath, which has resolved and denies having chest pain. He has been having low grade fever up to 100 F, and chills. He was feeling dizzy, which has resolved as well. He had intentional weight loss of about 30 lb over past few months….
>
> He never had similar symptom in past. He has never been hospitalized. He has low back pain, for which he was using OTC Aleeve (sic) 2 tablets everyday since 2012, and recently started taking Ibuprofen 800 mg bid, along with Tramadol. He has been using workout supplements – protein powders with caffeine. He has exposure to Aluminum as he has been working in Aluminum plant for 2-3 years.
>
> He also had an episode of hemoptysis this morning, and it contained bright red blood. He denies having any similar episode in past….

Dr. Patel's impression included "Acute renal failure: -Probably related to long term NSAID use vs vasculitis (with hemoptysis)." Dr. Patel's impression was also "Anemia," "Dyspepsia," and "Hemoptysis."

The claimant underwent a CT-guided kidney right biopsy on May 7, 2018 with the following diagnosis and result:

> Kidney, percutaneous needle biopsy:
> - Diffuse crescentic and segmentally necrotizing glomerulonephritis, consistent with pauci-immune type (ANCA-associated). See comment.
> - Patchy acute tubular injury.
> - 3/24 glomeruli with global sclerosis.
> - Interstitial fibrosis and tubular atrophy, mild.

It was commented, “The findings are consistent with ANCA – associated disease, especially P -ANCA, myeloperoxidase positive, since lesions in glomeruli are in various stages ranging from active necrosis to chronic sclerosis. Approximately 15% of pauci-immune crescentic glomerulonephritis are ANCA negative but are treated the same as ANCA positive cases.”

An x-ray of the claimant’s chest was done on May 8, 2018 with the impression, “Significant interval worsening of patchy consolidation within both lungs, particularly within the right mid and lower lung as well as in the left upper and mid lung. In a patient with hemoptysis, this could reflect other hemorrhage versus multifocal pneumonia.”

Dr. Nikhil K. Meena noted on May 9, 2018, “Mr. Bean was transferred to the MICU due to worsening hypoxic failure which is thought to be due to DAH secondary for vasculitis (pulmonary renal disease). He has already started on Plex and steroids. Will monitor in the MICU for

improvement of the oxygen requirements. No current plans to do a bronchoscopy. AKI due to glomerulonephritis."

Dr. Manisha Singh reported on May 15, 2018:

> I saw the patient myself with renal team. I have reviewed note and agree with the assessment and plan of care as documented in the note….
> Pt has pauci immune crescentic GN with DAH, negative anti-GBM, no infections found, and is critically ill. This disease condition carries very high mortality risk. No matter what we do. Given his young age, we are trying to do the maximum we know – and I am of the opinion that at current dosages side effects will come only after we have the desired effect, and they may not be as bad as dreaded in oncology patients where much higher dosages are used.
> I am treating him as an ANCA negative pauci-immune crescentic GN (if I decide to negate the OSH ANCA positive status) ANCA levels wax and wane thus the titers are not followed at this time.
> This is the one disease that can fully explain this clinical picture along with biopsy readings. I am not aware of any other condition that presents like this and could be as devastating as this is. Pt had history of ibuprofen use which might explain the podocytopathy (and the proteinuria), which is easily treatable with steroids and does not cause pulmonary renal syndrome.
> Some degree of podocyte effacement is seen with ANCA GN, however, severe Proteinuria is rare but has been reported with crescentic GN, MPO-ANCA can do this too – however, this would be an academic discussion as none of these disease features would be the clinical driver of his current critical state. An underlying minimal change disease will not be life threatening, and can explain the nephrotic range proteinuria.
> The driver of his clinical condition has to be pauci immune crescentic GN.
> Anemia from blood loss, and from renal failure would be amenable to supportive treatments – EPO levels will not tell us any thing that will change management.

> Currently PLEX is helping him a lot, as is the steroid regimen. I consider he has had adequate high dose steroids, and would agree with starting a taper or transition to oral doses (80 mg po daily) when possible….

The claimant was discharged from UAMS on May 23, 2018. Dr. Singh noted on August 23, 2018, "Pt reports feeling well, but has been drinking more protein shakes, having red meat, and increased workouts. His renal failure has suddenly become worse over last month." The claimant continued to follow up at UAMS, and the claimant's kidney condition continued to worsen.

The claimant's attorney signed a Form AR-C, Claim For Compensation, on September 20, 2018. The Accident Information section of the Form AR-C indicated that the Date of Accident was May 1, 2018 and that the cause of injury was "inhalation of chemicals causing lung and kidney failure and other whole body." The claimant contended on the Form AR-C that he was entitled to indemnity benefits and medical expenses.

On September 25, 2018, Georgia Diemer prepared a Workers Compensation – First Report Of Injury Or Illness. The First Report Of Injury Or Illness indicated that the claimant alleged an "Inhalation" injury, and that the parts of body affected were "lungs and kidneys." The First Report indicated that the Date of Injury/Illness was May 1, 2018, that the claimant's Last Work Date was April 18, 2018, and that the Date Employer Notified was September 24, 2018. The First Report indicated that the claimant was

"driving forklift" when the accident or illness exposure occurred, "EE alleges inhalation of chemicals at work causing lung and kidney failure."

The respondents' attorney examined Brian Elliott, the respondent-employer's Environmental Manager:

> Q. Now, Mr. Bean notified the Commission of his alleged work-related injury in September of 2018. Your name is on this first report.
> A. Probably….I probably entered that information.
> Q. You got the information probably from the insurance carrier?
> A. I believe so….
> Q. Okay. Then you knew nothing about this being a work-related incident until this report?
> A. No. I mean, ever – you know, everything I heard was just from other workers in the plant, you know, that it was a personal illness.
> Q. He had some type of personal illness?
> A. Yes, and then, you know, that was just hearsay-type stuff you hear.

The claimant testified that he returned to work for the respondent-employer in approximately January 2019 but that his physical condition deteriorated. The claimant underwent a procedure on March 21, 2019: "1) Deceased donor kidney transplant." The pre- and post-operative diagnosis was "End stage renal disease." The claimant testified that he again returned to work for the respondents in March 2020.

On July 25, 2020, Dr. Singh answered the following questionnaire provided by the claimant's attorney:

What injuries/conditions did you treat Mr. Michael Bean after

5/1/2018?

Mr. Bean was admitted to UAMS in May 2018 after having microscopic hematuria (blood in urine)(was treated earlier for about a week at an outside hospital) for the management of kidney failure. He was transferred to UAMS after he developed hemoptysis (blood in sputum). The patient had a very complicated hospital course with worsening hemoptysis, spontaneous pneumothorax (rupture in lung tissue), severe respiratory failure needing intubation. Renal biopsy was done that showed crescentic glomerulonephritis with fibrous, fibro-cellular and cellular crescents. He also had evidence of tubular injury. The biopsy findings along one positive ANCA serology and the clinical picture was indicative of pauci immune rapidly progressive glomerular nephritis – a bad and progressive renal disease. I was on service at that time during his hospitalization and was one of the treating physicians for his renal disease. With time his kidneys did not recover, though we tried the best treatments including immunosuppression and plasmapheresis. We had to start him on dialysis and then, after discharge from hospital, I took care of him at the dialysis unit. Mr. Bean's care was transitioned to the transplant doctors after he received a kidney transplant on 3/21/2019.

Do you believe, within a reasonable degree of medical certainty, that the injuries/conditions you treated Mr. Michael Bean for (after the 5/1/2018 occupational exposure) were caused by the exposure to silica while at work?

It is difficult to say exactly what caused the anca GN, but in his history, the only thing that we were able to find that 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. (Gomez-Puerta JA, Gedmintas L. Costenbader KH. The association between silica exposure and development of ANCA-associated vasculitis: systematic review and meta-analysis. Autoimmun Rev. 2013; 12(12):1129-1135. Doi:10.1016/j.autrev.2013.06.016)

> Are the injuries that you treated Mr. Michael Bean for (after the 5/1/2018 occupational exposure) permanent in nature? Please explain why or why not.
>
> Yes he had permanent injury to his kidney thus needing the dialysis and then the transplant.
>
> As of date, what is Mr. Michael Bean's prognosis?
>
> After transplant he is expected to do better than he was doing on dialysis. However, he is going to need anti-rejection medications for life and will be immune-compromised for life.
>
> In your opinion, does Mr. Michael Bean require any additional medical treatment (as a result of the injuries/conditions that you provided medical treatment to him for after the 5/1/2018 occupational exposure) from you or any other medical provider? If so, what type of additional medical treatment will Mr. Michael Bean require? What will be the approximate costs of such additional treatment?
>
> Mr. Bean will require lifelong care from renal doctors specially transplant nephrologists. I do not know how much that costs.

A pre-hearing order was filed on September 29, 2020. According to the text of the pre-hearing order, the claimant contended, "The claimant contends that on or about May 1, 2018, claimant began having symptoms which lead (sic) to a diagnosis of Anca Vasculitis and Silicosis. Claimant underwent a kidney transplant. Claimant's resulting kidney failure and lung failure was the direct result of exposure to elements at respondent's employer. Claimant contends that he sustained a compensable injury, that he is entitled to TTD, medical benefits, and that his attorney is entitled to an attorney fee. All other issues are reserved."

The parties stipulated that the respondents "have controverted this claim in its entirety." The respondents contended, "Respondent No. 1 contends the claimant alleges he has Wegner's Vasculitis that was caused by silica exposure at work. He did not give the employer notice of the alleged injury within 90 days. Wegner's Vasculitis has no known cause. There is no proof the condition is work-related. The claimant does not have a compensable occupational disease."

The pre-hearing order indicated that the parties agreed to litigate the following issues:

> 1. Whether the claimant has sustained a compensable occupational injury or disease injury within the meaning of the Arkansas Workers' Compensation Act (the Act), the symptoms of which began on or about May 1, 2018.
> 2. If the claimant's alleged occupational injury or disease is deemed compensable, whether and to what extent the claimant is entitled to medical and indemnity benefits.
> 3. Whether the claimant's attorney is entitled to a controverted fee on these facts.
> 4. The parties specifically reserve any and all other issues for future litigation and/or determination.

Dr. William Banner, Jr. corresponded with the respondents' attorney on October 14, 2020. Dr. Banner reported in part:

> Michael Brady Bean was born on May 17, 1983. Based on his deposition he worked for a variety of employers until June 2016 when he went to work for Reynolds Consumer Products. He had long standing complaints of degenerative disc disease and was taking nonsteroidal anti-inflammatory drugs and a creatine supplement for body building. He was hospitalized on May 3, 2018 and at that time was found to be mildly anemic but with evidence of acute renal failure. At the time,

the initial diagnosis was that this renal failure was probably secondary to his nonsteroidal anti-inflammatory drug use and the impact of creatine supplementation. After two days in the hospital he started to cough up blood and had a CT scan of the chest that was consistent with a right-sided diffuse alveolar hemorrhage. This altered the differential diagnosis to include an autoimmune pulmonary – renal syndrome and a number of blood studies were obtained which were pending at the time that he was transferred to the University of Arkansas Medical School.

Following the transfer, he continued to have progressive renal failure. The studies from CHI St. Vincent's Hospital demonstrated that he was positive for the P form of anti-nuclear cytoplasmic antibodies (P-ANCA). He was negative for C ANCA. He was positive for myeloperoxidase antibody and did not have any elevations of complement. Other studies were negative. At UAMS they performed an IgG study for anti-nuclear cytoplasmic antibodies which was negative at less than 1:20. Renal biopsy demonstrated pauci immune crescentric glomerulonephritis. While his pulmonary hemorrhage has, with chemotherapy, resolved his renal failure evolved into end-stage kidney disease that was managed with transplantation.

Analysis: Exposure to silica has been intensely studied as an occupational disorder. There are any number of polymorphs of the crystalline structure of silica that vary in their impact. In order for silica to produce pulmonary disease, the particles must be of a size that are small enough to reach the alveoli where they can be ingested by macrophages which may then initiate the cascade of inflammatory response that over time produces scarring and fibrosis characteristic of pulmonary silicosis. A number of factors seem to play an important role in this inflammatory response including individual genetics. Key to our understanding is that progression of autoimmune disease and silica exposure must begin with activation and inflammation in the lungs. (Pollard, 2016)

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 may 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….

Silicosis produces inflammatory changes and scarring particularly in the upper lobes of the lungs. (Pollard, 2016) Chronic simple silicosis occurs after roughly 15 to 20 years of moderate to low exposures after breathing in crystals of silica of the right structure and particle size. Accelerated or more severe silicosis is associated with 5 to 10 years of exposure at higher concentrations. 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….he 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 immune disease is via the development of clinical silicosis and not aymptomatic 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.
> Should further evidence become available I would be glad to review that and supplement my report.

The parties deposed Dr. Singh on January 21, 2021. The claimant's attorney examined Dr. Singh:

> Q. Can you describe to us what an ANCA-associated disease is?
> A. So an ANCA is an auto-antibody. That is basically the body starts making antibodies against itself, and this antibody's target is specifically to white cells. White cells are all over your body. They are trying to protect our body usually, but what happens is for some reason if the white cells break down and the body is able to see what's inside the cell. That's usually either the cytoplasm or the nucleus, body cells fighting that. This is generating antibodies against that, and that becomes a very destructive process overall. The blood vessels start getting involved, there is fundament, what they call necrotizing injuries. There's multiple areas that can be affected for this patient and for a patient who has his own body fighting against itself….So in this case it can be lungs, it can affect lungs, it can affect kidneys, and that's the most common presentations what we see….So that would be ANCA vasculitis. That is the antibodies started attacking the blood vessels.
> Q. So ANCA vasculitis is a form of vasculitis.
> A. Yes….
> Q. Is lung damage usually part of the symptoms that the patient is having when they have an ultimate diagnosis of ANCA vasculitis?
> A. It can be. Every organ need not be always involved, but the severity of the disease is more when the lung is involved….

Q. And Mr. Bean had both lungs and kidney damage, is that correct?
A. Yes. Yes, he did, yes….
Q. Now Mr. Bean went on to have a kidney transplant, is that correct?
A. Yes….
Q. So you treated Mr. Bean from approximately May 5 of 2018 through March 21 of 2019, is that correct?
A. That's correct, yes….
Q. So over this year-long treatment that you've 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….
Q. So is it your opinion that it was his exposure to silica that caused his ANCA?
A. 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.

The respondents' attorney cross-examined Dr. Singh:

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.

A hearing was held on January 29, 2021. At that time, counsel for the claimant asserted that the claimant was exposed to silica at the respondent-employer's plant. The claimant testified on direct examination:

> Q. Other than the exposure to silica at work, do you have any other explanation as to why you developed ANCA vasculitis?
> A. There is no other explanation. You don't just up and get sick – not – not like that sick you don't. And I mean when you look at – it's – it's already been established. Silica is a known environmental factor, and I was around a large quantity of it in a short period of time. I have no doubt in my mind, no matter what happens here today.

An administrative law judge filed an opinion on April 29, 2021. The administrative law judge found, among other things, that the claimant failed to prove he sustained a compensable injury. The administrative law judge therefore denied and dismissed the claim. The claimant appeals to the Full Commission.

II. ADJUDICATION

A. Compensability

The pre-hearing order in the present matter provided that the parties agreed to litigate the issue, "1. Whether the claimant has sustained a compensable occupational injury or disease injury within the meaning of the Arkansas Workers' Compensation Act (the Act), the symptoms of which began on or about May 1, 2018." Compensation for an alleged occupational disease is governed by Act 796 of 1993 as codified at Ark. Code Ann. §11-9-601(e)(Repl. 2012) *et seq*. According to his brief on appeal, however, the claimant contends that he sustained a "Back Injury." The claimant expressly contends that he "suffered a compensable injury,

not an occupational disease." The claimant cites as controlling authority Ark. Code Ann. §11-9-102(4)(Repl. 2012) which provides, in pertinent part:

> (A) "Compensable injury" means:
> (i) 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. An injury is "accidental" only if it is caused by a specific incident and is identifiable by time and place of occurrence[.]

A compensable injury must be established by medical evidence supported by objective findings. Ark. Code Ann. §11-9-102(4)(D)(Repl. 2012). "Objective findings" are those findings which cannot come under the voluntary control of the patient. Ark. Code Ann. §11-9-102(16)(A)(i)(Repl. 2012).

The employee has the burden of proving by a preponderance of the evidence that he sustained a compensable injury. Ark. Code Ann. §11-9-102(4)(E)(i)(Repl. 2012). Preponderance of the evidence means the evidence having greater weight or convincing force. *Metropolitan Nat'l Bank v. La Sher Oil Co.*, 81 Ark. App. 269, 101 S.W.3d 252 (2003).

In the present matter, the Full Commission finds that the claimant did not prove by a preponderance of the evidence that he sustained a compensable injury in accordance with Ark. Code Ann. §11-9-102(4)(A)(i)(Repl. 2012). The claimant became employed as a General Utility worker for the respondents, Reynolds Consumer Products, on June

13, 2016. The claimant testified that much of his employment for the respondents required working around extremely hot furnaces. The claimant testified that he worked around the alloys zinc, iron, silica, and copper. The claimant testified that he began getting sick in approximately April 2018. The claimant asserted that he was exposed to excessive silica dust while "the #4 furnace" was being torn down. The claimant contended that he began suffering from disease symptoms on May 1, 2018.

The evidence does not demonstrate that the claimant sustained an accidental injury causing internal or external physical harm to the body which arose out of and in the course of employment. The phrase "arising out of the employment" refers to the origin or cause of the accident and the phrase "in the course of 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). When the claimant in the present matter began seeking medical treatment on May 3, 2018, he did not attribute any of his symptoms to his work for the respondents. A physician at CHI St. Vincent noted on May 3, 2018, "Suspect patient's symptoms are likely coming from his creatinine and protein intake as well as his NSAID abuse." These symptoms were not causally related to the claimant's work for the respondents, either as the result of working near hot furnaces or purportedly breathing in excessive amounts of silica dust. The

claimant's symptoms did not arise out of the claimant's employment or in the course of employment. Dr. Benjamin reported on May 4, 2018 that the claimant had been suffering from back pain and epigastric pain for approximately two months. Dr. Benjamin did not causally relate the claimant's condition to any circumstance related to the claimant's work for the respondents. Dr. Patel reported on May 5, 2018 that the claimant had been suffering from stomach discomfort for about a month. Dr. Patel did not causally relate the claimant's stomach discomfort to any aspect of the claimant's work for the respondents.

Nor did the claimant prove by a preponderance of the evidence that he sustained a compensable injury which was caused by a specific incident or was identifiable by time and place of occurrence. The Full Commission recognizes that the statute does not require, as a prerequisite to compensability, that the claimant identify the precise time and numerical date upon which an alleged accidental injury occurred. *Edens v. Superior Marble & Glass*, 346 Ark. 487, 58 S.W.3d 369 (2001). Instead, the statute only requires that the claimant prove that the occurrence of the injury is capable of being identified. *Id*. The claimant in the present matter did not prove that there was a specific-incident injury which was capable of being identified. The Full Commission reiterates the claimant's direct examination testimony:

> Q. Did you start feeling sick – were you feeling sick at any point prior to tearing down of furnace #4?
> A. I do not believe so….I was there several days when that was takin' place.
> Q. Okay. This didn't just happen one day?
> A. No. No. No….

Even if the evidence did demonstrate that the claimant sustained an injury which arose out of and in the course of the claimant's employment with the respondents, the claimant's testimony did not show that there was a specific incident which was identifiable by time and place of occurrence. *See Edens, supra*. The Full Commission finds in the present matter that the claimant did not prove he sustained a compensable injury in accordance with Ark. Code Ann. §11-9-102(4)(A)(i)(Repl. 2012). The probative evidence does not support the claimant's contention that he sustained a compensable injury as the result of alleged "acute exposure to the silica dust."

B. Compensation for silicosis

The claimant in the alternative contends that he sustained a compensable injury in accordance with Ark. Code Ann. §11-9-602(Repl. 2012). The administrative law judge found that the claimant failed to prove he sustained a compensable injury pursuant to Ark. Code Ann. §11-9-602(Repl. 2012). It is the duty of the Full Commission to enter findings in accordance with the preponderance of the evidence and not on whether there is substantial evidence to support the administrative law judge's

findings. *Roberts v. Leo Levi Hospital*, 8 Ark. App. 184, 649 S.W.2d 402 (1983). The Full Commission reviews an administrative law judge's opinion *de novo*, and it is the duty of the Full Commission to conduct its own fact-finding independent of that done by the administrative law judge. *Crawford v. Pace Indus.*, 55 Ark. App. 60, 929 S.W.2d 727 (1996). The Full Commission enters its own findings in accordance with the preponderance of the evidence. *Tyson Foods, Inc. v. Watkins*, 31 Ark. App. 230, 792 S.W.2d 348 (1990).

Ark. Code Ann. §11-9-602(Repl. 2012) provides, in pertinent part:

> (a) As used in this subchapter, unless the context otherwise requires:
> (2) "Silicosis" means the characteristic fibrotic condition of the lungs caused by the inhalation of silica dust.
> (b) In the absence of conclusive evidence in favor of the claim, disability or death from silicosis or asbestosis shall be presumed not to be due to the nature of any occupation within the provision of this subchapter unless during the ten (10) years immediately preceding the date of disablement the employee has been exposed to the inhalation of silica dust or asbestos dust over a period of not less than five (5) years, two (2) years of which shall have been in this state, under a contract of employment existing in this state. However, if the employee has been employed by the same employer during the whole of the five-year period, his or her right to compensation against the employer shall not be affected by the fact that he or she had not been employed during any part of the period outside of this state.

A silicosis claim is dealt with separately from a claim for an occupational disease. *See Johnson v. Democrat Printing & Lithograph*, 57

Ark. App. 274, 944 S.W.2d 138 (1997); *Tyson Foods, Inc. v. Watkins*, 31 Ark. App. 230, 792 S.W.2d 348 (1990).

The credibility of witnesses and the weight to be given to their testimony are matters exclusively within the province of the Commission. *Johnson v. Democrat Printing & Lithograph*, 57 Ark. App. 274, 944 S.W.2d 138 (1997), citing *James River Corp. v. Walters*, 53 Ark. App. 59, 918 S.W.2d 211 (1996). The Commission 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. *Jackson v. Circle T. Express*, 49 Ark. App. 94, 896 S.W.2d 602 (1995).

In the present matter, the claimant became employed as a general laborer with the respondents, Reynolds Consumer Products, on June 13, 2016. The claimant testified that he became ill in approximately April or May 2018. The claimant testified that he breathed in silica dust which allegedly permeated the air while a work crew was demolishing a furnace on the respondents' premises. Regarding whether he sustained a kidney or lung injury as the result of breathing silica dust, the Full Commission finds that the claimant was not a credible witness. The evidence of record did not corroborate the claimant's testimony. The claimant began receiving medical treatment on May 3, 2018. The claimant did not attribute his

condition to breathing in silica fumes at work. Instead, the claimant's symptoms were causally related to "elevated creatinine" and other factors with no connection to the claimant's work for the respondents. The claimant complained of "epigastric pain" with no connection to work. The claimant was treated for "Acute Renal Failure" with no stated connection to the claimant's work. The claimant reported stomach problems which had been occurring for approximately two months. The probative evidence of record does not demonstrate that the claimant's kidney or lung condition was causally related to breathing noxious fumes at work.

The claimant began treating with Dr. Singh on or about May 15, 2018. Dr. Singh noted, "Pt has pauci immune crescentic GN with DAH, negative anti-GBM, no infections found, and is critically ill." Dr. Singh did not opine that the claimant's serious condition was work-related. The claimant was discharged from UAMS on May 23, 2018. The claimant returned to work for the respondent-employer in about January 2019 but underwent a kidney transplant on March 21, 2019. The post-operative diagnosis was "End stage renal disease." The evidence does not demonstrate that the claimant's end stage renal disease was causally connected in any form to the claimant's work for the respondents.

Dr. Singh opined on July 25, 2020 that the claimant's alleged exposure to silica at Reynolds Consumer Products "must be the inciting

event." The Commission has the authority to accept or reject a medical opinion and the authority to determine its medical soundness and probative force. *Green Bay Packaging v. Bartlett*, 67 Ark. App. 332, 999 S.W.2d 692 (1999). It is within the Commission's province to weigh all of the medical evidence and to determine what is most credible. *Minnesota Mining & Mfg. v. Baker*, 337 Ark. 94, 989 S.W.2d 151 (1999).

In the present matter, the Full Commission finds that the opinion of Dr. Banner is entitled to more evidentiary weight than the opinion of Dr. Singh. Dr. Banner provided an expert medical review and stated on October 14, 2020, "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 Full Commission finds that Dr. Banner's opinion is supported by the evidence of record and is entitled to significant weight. The evidence does not support Dr. Singh's deposition testimony that alleged workplace exposure to silica was "more likely than not" the cause of the claimant's illness. There was no "conclusive evidence" in the present matter demonstrating that the claimant's condition was causally related to alleged inhalation of silica dust. *See* Ark. Code Ann. §11-9-

602(2)(b)(Repl. 2012). The claimant had been employed with the respondents for approximately two years when he began suffering from symptoms related to his kidneys and lungs. The claimant's disability "shall be presumed not to be due to the nature of any occupation within the provision of this subchapter unless during the ten (10) years immediately preceding the date of disablement the employee has been exposed to the inhalation of silica dust or asbestos dust over a period of not less than five (5) years, two (2) years of which shall have been in this state, under a contract of employment existing in this state." Ark. Code Ann. §11-9-602(2)(b)(Repl. 2012).

The Full Commission finds in the present matter that the claimant did not prove he sustained a compensable injury in accordance with Ark. Code Ann. §11-9-602(2)(b)(Repl. 2012). Moreover, Dr. Singh was not even sure of the alleged origin of the claimant's illness. Dr. Singh expressly testified that the claimant did not have silicosis. Ark. Code Ann. §11-9-602(2)(Repl. 2012) provides for compensation for "silicosis."

After reviewing the entire record *de novo*, the Full Commission finds that the claimant did not prove he sustained a compensable injury in accordance with Ark. Code Ann. §11-9-102(4)(A)(i)(Repl. 2012). We find that the claimant did not prove he sustained a compensable "silicosis" injury in accordance with Ark. Code Ann. §11-9-602(a)(2)(Repl. 2012). The Full

Commission therefore affirms the administrative law judge's opinion as modified, and this claim is respectfully denied and dismissed.

IT IS SO ORDERED.

_____
SCOTTY DALE DOUTHIT, Chairman

_____
CHRISTOPHER L. PALMER, Commissioner

Commissioner Willhite concurs and dissents.

CONCURRING AND DISSENTING OPINION

After my *de novo* review of the entire record, I concur in part but must respectfully dissent in part from the majority opinion. I concur with the majority's finding that the claimant did not prove he sustained a compensable "silicosis" injury in accordance with Ark. Code Ann. §11-9-602(a)(2)(Repl. 2012). However, I must dissent from the majority opinion finding that the claimant did not prove he sustained a compensable injury in accordance with Ark. Code Ann. §11-9-102(4)(A)(i) (Repl. 2012).

Factual and Medical Background

The claimant, now 38 years old, worked for the respondent-employer as a utility worker. The claimant testified that in or about April or May of 2018, he sustained an injury to his lungs and kidneys. The claimant attributes his injuries to being exposed to silica while a furnace was being

torn down. The claimant explained that the work incident occurred as follows:

> Q Okay. So during your time at Reynolds, you had the occasion to be working next to one of these furnaces that was being torn down; is that correct?
>
> A Yes. Back when I was – the day I actually went, you know I got sick, it – it was takin' place right beside me. I was on the #5 furnace and the #4 furnace was the one bein' worked on, and I'm sure there's paperwork that can show that.
>
> Q Okay. So this #4 furnace that was being torn down – and you say you were sweeping up silica?
>
> A Yes. Well, it's all in the air. In fact, it's still on railing right now. I can take you there today and show you.
>
> Q Okay. So there's a lot of debris in the air?
>
> A Yes. Not all – not all the time but when that – that kind of stuff there is takin' place, yes, there is a lot.
>
> Q Okay. And do you recall approximately when #4 furnace was being torn down?
>
> A It was before May of '18, so I'm gonna say probably sometime in April, maybe.
>
> Q Do you have an exact date on that?

A I – I don't have an exact date. No, I don't.

…

Q Okay. Now, when you were working there that day and they're tearing down this –

A I was there several days when that was takin' place.

Q Okay. This didn't just happen one day?

A No. No. No.

Q About how long does it take to tear down a furnace?

A To tear down a [sic] rebuild, a few weeks, I believe, maybe a month. I'm not real sure on that either.

The claimant first sought treatment at the Emergency Department of CHI St. Vincent – Hot Springs on May 3, 2018. The claimant was diagnosed with and treated for Acute Renal Failure, Pulmonary Alveolar Hemorrhage, and suspected vasculitis. On May 5, 2018, the claimant was transferred to UAMS. While at UAMS, the claimant came under the care of Dr. Manisha Singh. When asked, "Do you believe, within a reasonable degree of medical certainty, that the injuries/conditions you treated Mr. Michael Bean for (after the 5/1/2018 occupational exposure)

were caused by the exposure to silica while at work?", Dr. Singh responded as follows:

> It is difficult to say exactly what caused the [ANCA] GN, but in his history, the only thing that we were able to find that 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 that must be the inciting event. …

In a deposition conducted on January 12, 2021, Dr. Singh testified as follows:

> Q Doctor Banner concludes that Mr. Bean's vasculitis is idiopathic. Do you agree with this?
>
> A No. I mean, again, it's a matter of opinion, no. It's a matter of opinion that it's a rare presentation of a rare disease.
>
> 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.

Dr. William Banner, Jr. also provided an opinion regarding causation. Dr. Banner opined:

> I would conclude to a reasonable degree of medical certainty that Mr. Bean's autoimmune vasculitis with pauci immune 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.

Opinion

For the claimant to establish a compensable injury as a result of a specific incident, the following requirements of Ark. Code Ann. §11-9-102(4)(A)(i) (Repl. 2012), must be established: (1) proof by a preponderance of the evidence of an injury arising out of and in the course of employment; (2) proof by a preponderance of the evidence that the injury caused internal or external physical harm to the body which required medical services or resulted in disability or death; (3) medical evidence supported by objective findings, as defined in Ark. Code Ann. §11-9-102 (4)(D), establishing the injury; and (4) proof by a preponderance of the evidence that the injury was caused by a specific incident and is identifiable by time and place of occurrence. *Mikel v. Engineered Specialty Plastics*, 56 Ark. App. 126, 938 S.W.2d 876 (1997).

An idiopathic injury is one whose cause is personal in nature, or peculiar to the individual. See *Kuhn v. Majestic Hotel*, 324 Ark. 21, 918 S.W.2d 158 (1996). Injuries sustained due to an unexplained cause are different from injuries where the cause is idiopathic. *ERC Contractor Yard & Sales v. Robertson*, 335 Ark. 63, 977 S.W.2d 212 (1998). Where a

claimant suffers an unexplained injury at work, it is generally compensable. *Little Rock Convention & Visitors Bur.*, 60 Ark. App. 82, 959 S.W.2d 415 (1997). Because an idiopathic injury is not related to employment, it is generally not compensable unless conditions related to the employment contribute to the risk of injury or aggravate the injury. *Id*. See also *Crawford v. Single Source Transp.*, 87 Ark. App. 216, 189 S.W.3d 507, 2004 Ark. App. LEXIS 549 (2004).

My review of the record indicates that the claimant suffered injuries as the result of an acute exposure to silica rather than because of an occupational disease. Dr. Singh clearly and unequivocally states that the claimant was not treated for silicosis (which is an occupational disease) but instead was treated for ANCA vasculitis. Dr. Singh further opined that the ANCA vasculitis was caused by the claimant's exposure to silica at work.

In light of the aforementioned assessment, I believe that the claimant's lung and kidney injuries are compensable. The claimant was injured in a specific incident while performing employment services in April or May of 2018. The claimant testified that he was exposed to silica while a furnace was being torn down. The claimant began experiencing symptoms, which he had never experienced before, shortly after the completion of the tear down.

There were objective findings of the kidney injury in the form of diffuse crescentic and segmentally necrotizing glomerulonephritis, consistent with pauci-immune type (ANCA-associated) as shown in a renal biopsy taken on May 7, 2018. There are also objective findings of the bilateral lung injury in the form of bilateral upper lobe and right lower lobe airspace disease consistent with pneumonia shown on a chest CT taken on May 3, 2018. In addition, this injury required medical treatment in the form of, *inter alia*, prescription medications, chemotherapy, dialysis, and a kidney transplant.

The primary issue here is causation, i.e., whether the claimant's injuries were caused by his exposure to silica or were they idiopathic in nature. Here, there are conflicting medical opinions. When medical opinions conflict, the Commission may resolve the conflict based on the record as a whole and reach the result consistent with reason, justice and common sense. *Barksdale Lumber v. McAnally*, 262 Ark. 379, 557 S.W.2d 868 (1977). A physician's special qualifications and whether a physician rendering an opinion ever actually examined the claimant are factors to consider in determining weight and credibility. *Id*.

As indicated above, Dr. Singh opined that the claimant's injuries were caused by his exposure to silica at work. Dr. Singh, a board-certified nephrologist, was the claimant's treating physician for almost one

year and had treated the claimant on multiple occasions prior to forming her opinion. It was through this extended course of treatment that Dr. Singh was able to form a more complete picture of the claimant's history and reach the conclusion that this exposure to silica was responsible for the claimant's condition. Therefore, based on Dr. Singh's expertise and experience treating the claimant, I assess great weight to Dr. Singh's opinion.

Conversely, Dr. Banner, an expert hired by the respondents, never treated the claimant. Dr. Banner's opinion was based on a review of the claimant's medical records, other documents provided by the respondents, and scientific publications. As medical experts are hired to limit the respondent's liability, each one must be viewed for what they are, i.e., a money-saving tool, and weighed accordingly. Thus, I assess little weight to the opinion offered by Dr. Banner.

It is important to note that the burden of proof, which is the preponderance of the evidence, is merely 51% more probable than not. If we take Dr. Singh's deposition testimony at face value (which I do), the claimant's burden has been met.

Based on the foregoing, I find that the claimant has established by a preponderance of the evidence that he sustained compensable lung and kidney injuries.

For the foregoing reasons, I concur in part and dissent in part from the majority opinion.

_____
M. SCOTT WILLHITE, Commissioner