

BROWN, J.

{¶ 1} In this original action, relator, PolyOne Corporation, seeks a writ of mandamus from this court ordering respondent, Industrial Commission of Ohio ("commission"), to vacate its order awarding respondent, Twyla Evans ("respondent"), the surviving spouse of Glenn R. Evans ("decedent"), scheduled loss compensation for decedent's loss of use of both arms and legs, and to enter an order denying said compensation.

{¶ 2} Pursuant to Civ.R. 53(C) and Loc.R. 13(M) of the Tenth District Court of Appeals, this court referred the matter to a magistrate who issued the appended decision, including findings of fact and conclusions of law, recommending that this court deny relator's request for a writ of mandamus. The magistrate determined there was some medical evidence to support the commission's finding that decedent's loss of use of his four extremities was permanent rather than temporary, and that there was some medical evidence upon which the commission relied showing that the allowed condition, angiosarcoma, independently caused the permanent loss of decedent's four extremities.

{¶ 3} Relator has filed objections to the magistrate's decision, arguing that R.C. 4123.57(B) does not authorize the benefits sought in this case. Relator contends that the language of that statute does not support an award for partial disability compensation for the loss of use of decedent's arms and legs while in a coma for a short period of time preceding his death; relator maintains that the decedent's failure to survive his comatose condition does not mean his loss of use of limbs was permanent. In support, relator relies upon this court's decision in *State ex rel. Carter v. Indus. Comm.*, 10th Dist. No. 09AP-30, 2009-Ohio-5547.

{¶ 4} In *Carter*, a worker suffered a gunshot wound while employed as a nightclub bouncer, and doctors amputated his right leg. Medical complications developed, and medical personnel sedated and chemically paralyzed the patient as part of the course of treatment, but he died while in the hospital. The relators (his dependents) subsequently filed a claim for scheduled loss compensation for the loss of use of his upper and lower extremities, arguing that the induced paralysis caused a loss of use which became permanent upon his death. The commission awarded loss of use compensation for his amputated right leg, but denied loss of use compensation for his upper extremities

and left leg. In *Carter*, the magistrate concluded that the chemically induced paralysis to the employee's left leg and upper extremities was not permanent and, therefore, found no abuse of discretion by the commission. The relators filed objections to the magistrate's decision. In *Carter*, this court overruled the relator's objections, holding in part that the evidence indicated that the "decendent's induced paralysis was a temporary measure designed to aid in his recovery," and that there was "no evidence that, but for decendent's death, the paralysis would have been permanent." *Id.* at ¶ 5.

{¶ 5} In the present case, the magistrate analyzed the decision in *Carter*, and found relator's reliance upon that case to be misplaced. Specifically, the magistrate noted that, unlike the injured worker in *Carter*, decendent's loss of use was not chemically induced or therapeutic, but, rather, the "natural consequence of his angiosarcoma." Thus, in contrast to the temporary paralysis of the employee in *Carter*, decendent's loss of use was permanent because it was expected to last, and did last, until his death. We agree with the magistrate that the decision in *Carter* is distinguishable and does not preclude an award of benefits under R.C. 4123.57(B).

{¶ 6} Relator's objections also challenge the award under R.C. 4123.57(B) on the grounds that (1) decendent was comatose immediately prior to death and, therefore, unaware of his injury, and that (2) his "purported loss of use" only occurred over a course of a few days. We conclude, however, that the magistrate did not err in finding that the staff hearing officer properly applied the Supreme Court of Ohio's holding in *State ex rel. Moorehead v. Indus. Comm.*, 112 Ohio St.3d 27, 2006-Ohio-6364. In *Moorehead*, an employee fell 15 to 20 feet onto a concrete floor and suffered a severe spinal cord injury; he lived for approximately 90 minutes, but never regained consciousness and was never aware that he had been rendered a quadriplegic. The commission denied the widow's application for loss of use benefits, but the Supreme Court subsequently allowed the writ and remanded for a determination of benefits, holding in part that "R.C. 4123.57(B) does not specify a required length of time of survival after a loss-of-use injury before benefits pursuant to R.C. 4123.57(B) are payable." *Id.* at ¶ 14. The Supreme Court also made clear "there is no language in R.C. 4123.57(B) requiring that an injured worker be consciously aware of his paralysis in order to qualify for scheduled loss benefits." *Id.* at ¶ 16. Accordingly, the commission did not abuse its discretion in applying *Moorehead* to find

that R.C. 4123.57 does not require an injured worker to be cognizant of his loss of use, nor does that decision support relator's duration of survival argument.¹

{¶ 7} Relator challenges the medical evidence in the record, and points to the opinion of its medical expert Dr. Joseph Buell. The magistrate, however, found relator's reference to Dr. Buell's report "problematic" in light of the fact the commission did not rely upon it, and that such report was directly contradicted by the reports of Drs. Matthew Levy and Kevin Trangle. The magistrate further noted that the reports of Drs. Levy and Trangle "could not be clearer" that the allowed condition, angiosarcoma, independently caused the loss of use of all four extremities. The magistrate adequately addressed the medical evidence, and for the reasons set forth, relator's objection as to that issue is not persuasive.

{¶ 8} Relator further contends the magistrate failed to consider the legislative intent of R.C. 4123.57, arguing that the award for loss of use benefits in the instant case essentially represents additional death benefits already provided for the surviving spouse under R.C. 4123.59. More specifically, relator maintains that benefits under R.C. 4123.57(B) are only intended to compensate for an injured worker's presumed loss of earning capacity.

{¶ 9} Relator's contention that an award for loss of use benefits under R.C. 4123.57(B) is duplicative of a death benefit award under R.C. 4123.59 is unpersuasive. It has been noted that the intent of R.C. 4123.59 is to compensate dependents for the "loss of support" resulting from the employee's death. *Fulton, Ohio Workers Compensation Section 11.3 at 531 (4th Ed.2011)*. By contrast, "benefits for partial disability are more akin to damages for work-related injuries." *State ex rel. Gen. Motors Corp. v. Indus. Comm.*, 42 Ohio St.2d 278, 282 (1975). *See also State ex rel. Miller v. Indus. Comm.*, 97 Ohio St.3d 418, 2002-Ohio-6664, ¶ 12 ("partial disability benefits have been compared to damages and are awarded irrespective of work capacity"); *State ex rel. Dudley v. Indus. Comm.*, 135 Ohio St. 121, 125 (1939) (noting scheduled compensation for loss of the sight

¹ We note that relator has filed, as supplemental authority, a recent decision by the Supreme Court, *State ex rel. Smith v. Indus. Comm.*, ___ Ohio St. ___, 2014-Ohio-513. *Smith*, however, involves scheduled loss benefits for loss of sight and hearing, rather than loss of use of extremities, and the *Smith* court does not discuss (or overrule) its decision in *Moorehead*.

of an eye is "arbitrarily fixed, and has nothing whatever to do with impairment of earning capacity").

{¶ 10} Relator also asserts that the award was impermissible under Ohio Adm.Code 4123-3-15(C)(4), arguing that a surviving spouse is only entitled to compensation if the award was made prior to the death of the injured worker. We disagree.

{¶ 11} Ohio Adm.Code 4123-3-15(C)(4) states in relevant part: "Where an award under division (B) of section 4123.57 of the Revised Code has been ordered but not paid prior to the death of an employee, upon application, the award is payable to the surviving spouse." While this administrative code provision addresses an award ordered "prior to the death of an employee," it does not address (nor does it preclude) an application made by a dependent after the death of an employee. R.C. 4123.60, however, states in part:

If the decedent would have been lawfully entitled to have applied for an award at the time of his death the administrator may, after satisfactory proof to warrant an award and payment, award and pay an amount, not exceeding the compensation which the decedent might have received, but for his death, for the period prior to the date of his death, to such of the dependents of the decedent, or for services rendered on account of the last illness or death of such decedent, as the administrator determines in accordance with the circumstances in each such case.

{¶ 12} Here, as noted by the commission, because decedent would have been entitled to have applied for a scheduled loss award at the time of his death, his surviving spouse was entitled to apply for benefits to which he was entitled.² Thus, relator's objection asserting that Ohio Adm.Code 4123-3-15(C)(4) precludes the surviving spouse from recovering benefits in the instant case is not well-taken.

² We note that amicus curiae, Ohio Self-Insurer's Association, argues that respondent (surviving spouse) should only be granted loss of use benefits for the period during which decedent experienced loss of use while alive, i.e., the four days prior to his death. The parties, however, did not raise that argument before either the commission or magistrate. See *Lakewood v. State Emp. Relations Bd.*, 66 Ohio App.3d 387, 394 (8th Dist.1990) ("*Amici curiae* are not parties to an action and may not, therefore, interject issues * * * not raised by parties"). (Emphasis sic.) In any event, such issue would not appear ripe for review. The commission itself maintains it did not mandate the payment of 850 weeks of scheduled loss payments, noting the order of the staff hearing officer indicates: "[p]ayment to be made and processed per statute" (i.e., including the terms of R.C. 4123.60).

{¶ 13} Based upon this court's independent review, we overrule relator's objections and adopt the magistrate's findings of fact and conclusions of law. In accordance with the magistrate's decision, we deny the requested writ of mandamus.

Objections overruled; writ of mandamus denied.

CONNOR and DORRIAN, JJ., concur.

APPENDIX

IN THE COURT OF APPEALS OF OHIO

TENTH APPELLATE DISTRICT

State ex rel. PolyOne Corporation,	:	
Relator,	:	
v.	:	No. 12AP-313
The Industrial Commission of Ohio and Glenn R. Evans/Twyla Evans,	:	(REGULAR CALENDAR)
Respondents.	:	
	:	

MAGISTRATE'S DECISION

Rendered on April 5, 2013

Reminger Co., L.P.A., Martin T. Galvin and Marianne Barsoum Stockett, for relator.

Michael DeWine, Attorney General, and Colleen C. Erdman, for respondent The Industrial Commission of Ohio.

Wincek & DeRosa Co., LPA, Joseph C. DeRosa and Daryl Gagliardi, for respondent Glenn R. Evans/Twyla Evans.

IN MANDAMUS

{¶ 14} In this original action, relator, PolyOne Corporation ("relator" or "PolyOne") requests a writ of mandamus ordering respondent Industrial Commission of Ohio ("commission") to vacate its order awarding to respondent Twyla Evans, the surviving spouse of Glenn R. Evans ("Evans" or "decedent"), R.C. 4123.57(B) scheduled

loss compensation for decedent's loss of use of both arms and legs, and to enter an order denying the compensation.

Findings of Fact:

{¶ 15} 1. Until his retirement in 1994, Evans was employed for many years as a laborer by PolyOne or its predecessor. During his employment, Evans was exposed to vinyl chloride.

{¶ 16} 2. In July 2010, Evans underwent a CT of his chest and mediastinum. He later underwent an MRI. Following a CT-guided biopsy of the liver, Evans was diagnosed with hepatic angiosarcoma. Chemotherapy treatment began in early August 2010. Vinyl chloride exposure is widely known to cause hepatic angiosarcoma.

{¶ 17} 3. In October 2010, Evans filed a workers' compensation claim on a form captioned "First Report of an Injury, Occupational Disease or Death" ("FROI-1"). On the form, Evans alleged an "[o]ccupational exposure to vinyl chloride resulting in angiosarcoma of the liver." August 5, 2010 was listed as the injury date.

{¶ 18} 4. In late October 2010, PolyOne, a self-insured employer, certified the industrial claim (No. 10-848253) for "angiosarcoma."

{¶ 19} 5. The commission officially recognizes the claim for "angiosarcoma." The commission recognizes the injury date as August 5, 2010, which apparently approximates the date of diagnosis.

{¶ 20} 6. On July 8, 2011, Evans died. On the certificate of death, "angiosarcoma liver" is given as the cause of death. The death certificate was completed and certified by attending physician Poornanand Palaparty, M.D.

{¶ 21} 7. Earlier, on July 4, 2011, Evans was examined at his home in the presence of his wife, daughter, and counsel, by orthopedic surgeon Matthew E. Levy, M.D.

{¶ 22} 8. On July 5, 2011, Dr. Levy wrote:

I performed an examination of patient Glenn Evans last night in regards to his diagnosis of angiosarcoma. I found that as of 11:15 p.m. 07/04/2011, he had lost all use of both arms and both legs.

{¶ 23} 9. On July 5, 2011, Evans moved for R.C. 4123.57(B) scheduled loss compensation for the alleged loss of use of both arms and legs. In support, Evans submitted the July 5, 2011 report of Dr. Levy.

{¶ 24} 10. On August 12, 2011, a commission hearing officer mailed an ex-parte order finding that Evans' industrial claim was abated by his death.

{¶ 25} 11. On August 17, 2011, Twyla Evans filed an R.C. 4123.59 death claim on the FROI-1 form.

{¶ 26} 12. Following a November 21, 2011 hearing, a district hearing officer ("DHO") issued an order allowing the death claim.

{¶ 27} 13. Relator administratively appealed the DHO's order of November 21, 2011 allowing the death claim.

{¶ 28} 14. Following a January 20, 2012 hearing, a staff hearing officer ("SHO") issued an order allowing the death claim, but modifying the DHO's order of November 21, 2011. The SHO's order of January 20, 2012 provides in part:

It is found that the decedent's spouse, Twyla Evans, born on 01/16/1936, was wholly dependent upon the decedent for support at the time of death, and that she is entitled to weekly benefits in the amount of \$748.53.

{¶ 29} 15. The record fails to disclose whether the January 20, 2012 order of the SHO allowing the death claim was administratively appealed. Presumably, Twyla Evans is currently receiving weekly benefits under R.C. 4123.59 as a surviving spouse who was wholly dependent upon decedent at the time of his death.

{¶ 30} 16. Earlier, on July 27, 2011, Dr. Levy issued a seven-page narrative report based in part on his July 4, 2011 examination of Evans at his home. In his report, Dr. Levy states:

Mr. Evans is being evaluated in conjunction with his development of angiosarcoma, his current status and for determination of loss of use of certain appendages and faculties.

* * *

CURRENT COMPLAINTS

I was called to evaluate Mr. Evans in his home on 07/04/2011 at 2315 hours. Mr. Evans was evaluated in the presence of his wife, his daughter and counsel, Mr. Joseph DeRosa. At the time of evaluation, Mr. Evans was unable to provide me with any history.

The history was provided by his wife and daughter who note that his condition has taken a precipitous turn for the worst today. He was unable to get out of bed, unable to feed himself, and unable to even participate in his own care and hygiene. He was noted to be moaning in discomfort throughout the entirety of my evaluation.

The remainder of the history was gleaned from the medical records.

PHYSICAL EXAMINATION:

On the physical evaluation, Mr. Evans was found to be in an obtunded state. He was minimally arousable and he had rattling respirations. He had a very limited response even to noxious stimuli. His color was poor[.]

Examination of his upper and lower extremities revealed pitting edema within the lower extremities, pale coloration throughout the extremities, no volitional movement and minimal withdrawal even to noxious stimuli.

I was able to document full passive range of motion in both shoulders, elbows, wrists and fingers in the upper extremities; and hips, knees, ankles and toes in the lower extremities. However, he exhibited no tone in any of the above-mentioned extremities.

Reflexes were symmetrically diminished in both upper and lower extremities. Pathologic reflexes such as Babinski's and Hoffman signs were not observed.

There was no obvious response of the individual to speech, to noises or to the environment from an auditory perspective. Similarly, he did not open his eyes, show any meaningful visual interaction with his environment, track movements or for all intents and purposes, show that he had any vision perception of his surroundings.

ASSESSMENT:

Based upon review of the history and physical examination, medical records and all enclosed documentation, the following opinions are offered with a reasonable degree of medical certainty.

* * *

Diagnoses include:

- a) Dependent edema;
- b) Angiosarcoma;
- c) Fatigue;
- d) Leg weakness;
- e) Elevated liver function tests;
- f) Anemia;
- g) Gastroesophageal reflux disease;
- h) Malnutrition;
- i) Status post congestive heart failure;
- j) Hearing loss;
- k) Cardiac murmur;
- l) Loss of use of all four extremities;
- m) Loss of vision;
- n) Loss of hearing

At the time that he was evaluated, Mr. Glenn Evans was a 74-year-old gentleman with a diagnosis of angiosarcoma of the liver, a result of an occupational environmental exposure sustained in the course of his employment. The diagnosis of angiosarcoma was confirmed through the pathology department at Cedar Sinai Medical Center in October 2010 by Steven Geller. Before I had evaluated him, Mr. Evans was found also to have a medical history of congestive heart failure, hypertension, reflux and anorexia, among his other conditions.

I was called to evaluate Mr. Evans in his home on 07/04/2011 at 2315 hours. Mr. Evans was evaluated in the presence of his wife, his daughter and counsel, Mr. Joseph DeRosa. At the time of evaluation, Mr. Evans was unable to provide me with any history.

Coma is defined as a profound state of deep unconsciousness. It affects any individual's ability to interact with the surrounding environment. In this particular case it

is the direct sequelae of the obfundation and deep unconsciousness caused by his progress fatal cancer. The cancer caused a cascade of events leading to metabolic derangement, lack of oxygenation and in general lack of the necessary physiologic mechanisms sufficient to sustain conscious awareness and bodily function.

It is academic where one draws the line in terms of what sort of responses a patient has to certain stimuli. Mr. Evans was not noted to have any voluntary responses. In fact, he was incapable of any response at all to his surrounding environment. Except for the rare response to noxious stimuli, he was not interactive with his environment.

The patient does have permanent loss of use of various body parts as statutorily determined. Mr. Evans had no functional, meaningful or volitional use of either of his arms or legs. All four limbs can be considered to have no functional use. For actual practical purposes he has permanently lost the use through the central nervous system dysfunction he has of any extremity movement or activity.

Similarly, at the point in time I saw him, his eyes remain closed. He did not respond to stimuli. He did not track or follow and had for all intents and purposes no intentional volitional vision use. This would apply to bilateral use of both eyes.

In regards to his hearing, there was no response to hearing or noise. He did not respond to commands. It was entirely conjectural if there was even any brain stem auditory response that was functioning. There was no conscious level of hearing, interpretation of sound or even a human response to noise stimuli.

SUMMARY:

Mr. Evans, at the time I saw him, suffered from the permanent conditions of loss of use of the following:

[One] Right and left arms;

[Two] Right and left legs;

[Three] Vision comprehension in right and left eyes;

[Four] Hearing comprehension in both the right and left ears.

{¶ 31} 17. On July 28, 2011, Twyla Evans, as surviving spouse, moved for R.C. 4123.57(B) scheduled loss compensation for decedent's alleged loss of use of both arms and legs, vision and hearing. In support, Twyla Evans submitted her marriage certificate, the death certificate, and the July 27, 2011 report of Dr. Levy.

{¶ 32} 18. Following a September 27, 2011 hearing, a DHO issued an order denying the July 28, 2011 motion of Twyla Evans. The DHO's order explains:

Prior to a hearing on the merits, the surviving spouse's counsel withdrew the request for the SCHEDULED LOSS OF VISION COMPREHENSION IN BOTH EYES and SCHEDULE[D] LOSS OF HEARING COMPREHENSION IN BOTH EARS. Therefore, these requests are DISMISSED.

On 07/08/2011, the decedent, Glenn Evans, died. Four days prior [to] his death, on 07/04/2011, the decedent lost consciousness and could no longer move his legs or arms. The surviving spouse is requesting the loss of use of both arms and both legs due to his drastic change in health on 07/04/2011. The report of Dr. Levy dated 07/27/2011 is presented in support of this request.

The District Hearing Officer finds that the medical evidence is insufficient to support the requested loss of use as being related to the allowed condition of ANGIOSARCOMA. Dr. Levy's own report lists the following diagnoses from his 07/04/2011 examination: Dependent edema, Angiosarcoma, fatigue, leg weakness, elevated liver function tests, anemia, gastroesophageal reflux disease, malnutrition, status post congestive heart failure, hearing loss, cardiac murmur, loss of use of all four extremities, and loss of vision. Dr. Levy went on to indicate that the Injured Worker was in a coma, and noted that the Injured Worker had a medical history of congestive heart failure, hypertension reflux and anorexia. Dr. Levy does not sufficiently explain how the allowed condition of angiosarcoma directly caused the damage to the central nervous system that would then cause the loss of use of all four extremities, especially in light of other conditions the Injured Worker had. For these reasons, the District Hearing Officer finds that the surviving spouse has not met the requisite proof necessary to support a finding of a loss of use of the requested four extremities.

The District Hearing Officer has reviewed and considered all evidence prior to rendering this decision.

(Emphasis sic.)

{¶ 33} 19. Twyla Evans administratively appealed the DHO's order of September 27, 2011.

{¶ 34} 20. On November 3, 2011, at the request of counsel for Twyla Evans, Kevin L. Trangle, M.D., issued a seven-page narrative report. Dr. Trangle wrote:

DISCUSSION OF VINYL CHLORIDE-INDUCED HEPATIC ANGIOSARCOMA:

Hepatic angiosarcoma (HAS) is an uncommon mesenchymal malignant neoplasm of the vascular or lymphatic endothelium, accounting for 2% of all soft tissue sarcomas. Angiosarcoma can affect any organ. Although primary HAS is rare and accounts for only 2% of primary hepatic tumors, it is the most common malignant mesenchymal tumor of the liver.

Popper and colleagues, and Gedigt et al. have undoubtedly provided the greatest contribution in the study of the histogenesis and pathohistology of HAS. Vinyl chloride monomers (VCM) are transformed by hepatic microsomal enzymes to toxic metabolites that covalently bind to DNA. After exposure to VCM, hepatocytic proliferation, sinusoidal lining cell proliferation, and focal sinusoidal dilatation occurs; this process leads to angiosarcoma from the sinusoidal lining cells. In a typical histologic picture, there are wide vascular spaces and systems of anastomosed vessel canals lined with atypical endothelium, with marked sarcomatous stroma. Mr. Evans clearly had developed HAS secondary to his work related exposure to Vinyl Chloride.

HAS progresses rapidly; therefore, most cases are discovered at an advanced stage, and less than 20% of the patients can even be conceivably helped by surgery. The lack of specific symptoms and radiological findings leads to the delay of diagnosis resulting in the poor prognosis. Only a few patients have been reported to survive for more than one year after hepatic resection for HAS. HAS usually develops in the sixth decade of life, and is more frequent in males than in females (ratio 3:1). Mr. Evans had a classic presentation of a work related HAS caused by VC exposure.

The prognosis of HAS is dismal. Most patients die within six months of the diagnosis. The most frequent causes of death are hepatic failure and intraabdominal bleeding. Fifty percent of patients develop metastases before death. Very few patients have limited tumor at the time of diagnosis to allow surgical resection.

Hepatic Failure (HF) as a Consequence of Hepatic Angiosarcoma

The liver is commonly involved in metastatic disease, and the degree of liver biochemistry derangement tends to reflect the extent of parenchymal replacement with tumor. Hepatic failure can develop as a consequence of primary or metastatic liver tumors. The mechanism of liver failure is multifactorial. Evidence suggests a combination of hepatic ischemia leading to parenchymal infarction, vascular occlusion of portal vein by tumor thrombi and non-occlusive infarction of liver due to shock from secondary causes such as sepsis or cardiac dysfunction plays an important role in these patients. Typically, replacement of hepatocytes by malignant cells leads to secondary necrosis of hepatocytes with the subsequent development of liver failure.

Numerous authors have reported the development of HF in patients with HAS. As noted above, this is usually the terminal event associated with diffuse involvement of the liver by the HAS.

One study conducted by Myszor et al. looked at the association and presentation of malignant disease of the liver with hepatic failure. The authors described three cases and reviewed the best documented reports in the literature. Their review of 25 patients showed that in most cases, the liver was massively replaced by tumor that often spread in an intrasinusoidal pattern and resulted in HF and subsequent death.

Another study conducted by Dannaher et al. looked at 10 workers from a single vinyl chloride polymerization plant in Louisville, Kentucky that developed HAS. Average survival from diagnosis was about 12 months. Overt liver failure occurred as a preterminal event and was the major cause of death in all of the patients.

Baxter et al. studied 35 cases of HAS occurring in Great Britain. The most common terminal event in these patients

was liver failure and its attendant complications. The duration of symptoms preceding admission to hospital was known for over 30 cases. The median time was about five weeks. The length of survival after admission was known for all adults, the median time being three weeks. Only three cases lived beyond six months after admission to hospital. The two patients with the shortest duration of symptoms died from hemoperitoneum.

In addition to the neoplasm itself, treatment with various chemotherapeutic agents can contribute to further injury to the liver. Gemcitabine represents one of these agents and it has been shown to be hepatotoxic.

Hepatic Encephalopathy and Coma

Hepatic encephalopathy is defined as a spectrum of central nervous system abnormalities in patients with liver dysfunction, after exclusion of other known brain disease. Hepatic encephalopathy is characterized by personality changes, intellectual impairment, and a depressed level of consciousness. The development of hepatic encephalopathy is explained to a large extent by the effect of neurotoxic substances which accumulate as a result of liver failure; additionally brain edema plays a prominent role. The brain edema of hepatic failure is attributed to increased permeability of the blood-brain barrier, impaired osmoregulation within the brain, and increased cerebral blood flow. The resulting brain cell swelling and brain edema cause loss of consciousness and eventually death.

Typically, patients subsequently become hypotensive and tachycardic as a result of the reduced systemic vascular resistance that accompanies hepatic failure, a pattern that is indistinguishable from septic shock. The combination of cerebral edema with resulting increased intracranial pressure and systemic hypotension leads to coma and then death.

Gastrointestinal bleeding can also contribute to the development of hepatic encephalopathy. The presence of blood in the upper gastrointestinal tract results in increased ammonia and nitrogen absorption from the gut. Bleeding may predispose to kidney hypoperfusion and impaired renal function. These metabolic consequences lead to increased toxic ammonium levels in the blood and even further

depression of central nervous system function, loss of consciousness and death.

ANALYSIS AND OPINION:

In the case at hand, Mr. Evans was diagnosed and treated for vinyl chloride-induced hepatic angiosarcoma. He ultimately died on 07/08/2011. His death certificate lists angiosarcoma of the liver as his cause of death.

He was initially diagnosed with hepatic angiosarcoma by CT-guided needle biopsy in July of 2010. He underwent chemotherapy with Taxol, Sorafenib and gemcitabine. He was admitted for liver failure following treatment with gemcitabine. Dr. Palaparthi [sic] noted he had edema in both legs, shortness of breath and icterus. He had elevated liver enzymes including elevated bilirubin, ALT/AST and alkaline phosphatase. His total protein and albumin were significantly diminished and he had evidence of pancytopenia. He experienced a typical side effect of chemotherapy; namely toxic deterioration of liver function.

He subsequently had two episodes of intra-abdominal hemorrhage requiring embolization of the hepatic artery and blood transfusions.

His most recent abdominal CT demonstrated extensive neoplastic infiltration of the liver with extracapsular spread of disease and ascites. There was extensive free fluid noted surrounding the liver, spleen and extending in the mesenteric which was likely hemorrhagic.

He ultimately developed fulminant hepatic failure as a direct result of the extensive neoplastic infiltration of his liver. The repeated intraperitoneal hemorrhages and chemotherapy also contributed to his encephalopathy. The hemorrhages undoubtedly also led to increased blood ammonia levels. The chemotherapeutic agents, particularly gemcitabine, caused hepatic injury which contributed to the development of liver failure.

The medical literature supports this as the most common pre-terminal event in patients suffering from hepatic angiosarcoma. The liver failure led to the development of hepatic encephalopathy which progresses to hepatic coma and death.

While suffering from hepatic encephalopathy and profound central nervous system depression and loss of consciousness, he completely lost the ability to use his upper and lower extremities as well as his ability to hear and see. This was a direct result of a combination of the build-up of neurotoxic substances, cerebral edema with increased intracranial pressure, and cerebral ischemia. His level of consciousness progressively and rapidly diminished to the point of coma.

SUMMARY/CONCLUSION:

Mr. Evans' condition of hepatic encephalopathy as noted above resulted from a combination most likely of liver failure with toxic metabolites circulating in the blood stream and spilling over into the central nervous drainage system and brain fluids due to the abnormal permeability of the blood-brain barrier secondary to his cancer; additionally the same process of his cancer progression led to cerebral edema.

Additionally, it is likely that Mr. Evans also had some degree of cerebral bleeding as the liver is directly responsible for producing coagulation factors as part of the coagulation cascade that prevents an individual from having abnormal bleeding and in particular intercerebral bleeding.

This sequence of events in Mr. Evans was an inexorable, ongoing, worsening situation that had no available treatment. It was undoubtedly a progressive and permanent condition which advanced to the point of his death. There was no temporary, transient or conditional aspect to his cerebral encephalopathy, central nervous system depression and coma.

To explain it perhaps more succinctly, the combination of encephalopathy, cerebral edema and bleeding directly caused a profound central nervous system depression. Profound central nervous system depression is called coma where there is a loss of consciousness. In addition to loss of consciousness there is also loss of use of the extremities as the central nervous system from the brain does control the other parts of the central nervous system including the spinal cord which mediates the function and motion of the extremities. As Mr. Evans' level of central nervous system depression became more deeply affected and his coma continued to permanently deepen, he had permanent loss of use of his extremities.

The reason the terminology permanency is used in this context is simply that unlike a medically induced coma for treatment purposes, loss of consciousness due to central nervous system depression (defined as coma) and loss of use of extremities, are ongoing and irreversible processes with any type of malignancy such as hepatic angiosarcoma where there is no available treatment. The lack of viable treatment alternatives for Mr. Evans had already been proven and accepted. His development of coma and loss of use of his extremities was a one-way street with unfortunately no available or known medical intervention that could reverse this process.

In short his permanent loss of use of his extremities was a direct result of the combination of the buildup of neurotoxic substances, cerebral edema, increased intracranial pressure, cerebral ischemia and most likely even cerebral bleeding. This was an irreversible, permanent progression of events that led to coma which is the definition of profound central nervous system depression with loss of consciousness; and with concomitant inability to use his extremities on a permanent basis. Ultimately, the cerebral pressure and other noted factors built up to the point that the brain stem was almost certainly compressed to the point that he could no longer breathe and this resulted in his ultimate demise. All of these conditions; buildup of neurotoxic substances, cerebral edema, increased intracranial pressure, cerebral ischemia and cerebral bleeding, hepatic failure, hepatic encephalopathy, central nervous system depression, and resultant permanent loss of use of his upper and lower extremities are physical manifestations that are the direct result of the allowed claim for hepatic angiosarcoma.

In my medical opinion, and expressed with a reasonable degree of medical certainty, Mr. Evans succumbed and died secondary to his work-related hepatic angiosarcoma, a claim already allowed, and a cause of death also affirmed on his death certificate. Furthermore, his pre-terminal state resulted from the angiosarcoma which irreversibly and permanently depressed the central nervous system leading to the level of depression which resulted in permanent loss of use of his upper and lower extremities.

{¶ 35} 21. On November 17, 2011,³ at the request of relator, Joseph F. Buell, M.D., issued a two-page narrative report. Dr. Buell is a professor of surgery and pediatrics at Tulane University located at New Orleans, Louisiana. In his report, Dr. Buell opines:

I am in receipt and have reviewed the medical records of Mr. Glen[n] Evans. Mr. Evans was a retired Poly[O]ne worker who was diagnosed with angiosaroma of the liver. I was provided medical records for Mr. Evans, which noted his polyvinyl exposure and identified a distant history of smoking. After diagnosis of angiosarcoma was made, Mr. Evans was started on a T1 inhibitor, and later treated with taxol, gemcitabine and eventually gemzar chemotherapy. The records noted he developed congestive heart failure during this time frame. This claim is not supported by the medical evidence which demonstrated his cardiac function measure by ejection fraction was normal as measured by cardiac ECHO. An initial occupational medical exam was performed by Dr. Darr on 4/11/11 which reported Mr. Evans as "fatigued." At this time Dr. Darr opinioned that Mr. Evans had Class III impairment.

Subsequently, in June of 2011 Mr. Evans presented to the emergency room with a rupture of his liver tumor. This was treated with a radiologic procedure to clot the bleeding. Often radiologic treatment of a liver tumor clots blood flow to the tumor as well as the normal uninvolved liver. After extensive chemotherapy and a delayed treatment of his tumor after rupture Mr. Evans suffered liver decompensation. Mr. Evans was examined by an orthopedic surgeon who claimed four extremity disabilities.

After review of Mr. Evans medical records and my extensive clinical experience with liver disease and management of liver tumors and particularly angiosarcoma, I have formulated the following medical opinions:

[One] There are some concerning irregularities in the opinions and management of Mr. Evans by his physicians during his care and hospitalizations. As examples there is lack of clinical data i[.]e[.] cardiac ECHO, to support his medical diagnosis of congestive heart failure and as another

³ The report of Dr. Buell is incorrectly dated "November 17, 2010." It is obvious that the report is incorrectly dated.

example Mr. Evans did not receive local therapy to prevent tumor rupture.

[Two] Mr. Evans became encephalopathic (unconscious) due to hepatic failure. To physicians unfamiliar with the manifestations of liver disease and decompensated liver patients it might appear that they suffered irreversible damaged [sic] of the central nervous system, but this is purely a reversible condition. Noting again there was no permanent injury resulting in loss of all four extremities. Neither the agiosarcoma nor the treatment of the angiosarcoma can cause permanent damage to the central nervous system. More often than not this state of encephalopathy is completely reversible with appropriate medical therapy. In no way did Mr. Evans ever permanently lose function of all four of his extremities.

[Three] An orthopedic surgeon (bone surgeon) has limited knowledge and experience with liver failure patients let alone management of hepatic encephalopathy. It is my opinion that this was a flawed opinion due to the physician's lack of knowledge.

[Four] Lastly, I have reviewed the summary report from Dr. Trangle who quotes several historic papers. What is not presented is the full spectrum of patient with long-term survival. Several series document that when patients receive aggressive therapy they can survive 2 to 3 years. I again reiterate Mr. Evans suffered from hepatic encephalopathy and decompensated liver disease that was not aggressively treated. At time of his exam by an orthopedic doctor he did not have loss of extremity use but was rather suffering from a reversible medical condition.

{¶ 36} 22. Following a November 21, 2011 hearing, an SHO issued an order that vacates the DHO's order of September 27, 2011 and awards 4123.57(B) scheduled loss compensation for loss of use of both arms and legs. The SHO's order of November 21, 2011 explains:

The Staff Hearing Officer finds that applicant/surviving spouse Twyla B. Evans has withdrawn her requests for awards for the "loss of hearing comprehension in both ears" and for the "loss of vision comprehension in both eyes." The Staff Hearing Officer, therefore, orders that these requests be dismissed.

The Staff Hearing Officer finds, per the 11/03/2011 report of Dr. Trangle and the 07/05/2011 and 07/27/2011 reports of Dr. Levy, that decedent Glenn Evans suffered the total loss of use of his bilateral arms and bilateral legs prior to his death on 07/08/2011. The Staff Hearing Officer further finds that such losses of use were the direct result of the allowed condition "angiosarcoma" and its sequelae. Per the rationale set forth in State, ex rel. Moorehead -v- Industrial Commission (2006), 112 Ohio State 3d 27, 857 North East 2d 1203, the Staff Hearing Officer does not find that O.R.C. 4123.57 requires that an Injured Worker be cognizant of his 'loss of use' in order to receive compensation for same.

In the instant case, decedent Glenn Evan's [sic] comatose condition, during which his loss of use of his arms and legs was present, does not bar an award for same. Additionally, the Staff Hearing Officer finds that speculation that Mr. Evan's [sic] condition or the course of his "angiosarcoma" might have been altered had a different treatment protocol been adopted does not negate the fact that a loss of use of the bilateral arms and bilateral legs existed. Finally, the Staff Hearing Officer finds no persuasive medical evidence that demonstrates that Mr. Evan's [sic] comatose condition and resultant losses of use were temporary or transient (see, State, ex rel. Carter -v- Industrial Commission, 2009 WL 3366373 (Ohio App. 10 Dist)).

The Staff Hearing Officer finds that applicant/surviving spouse Twyla B. Evans is entitled to an award for the losses of use described above (eight hundred and fifty weeks - 4123.57). Start date for the award is 07/05/2011 (Dr. Levy's report). Payment to be made and processed per statute.

All evidence on file and at hearing, including the 11/17/2011 report of Dr. Buell, was reviewed and considered.

{¶ 37} 23. On December 21, 2011, another SHO mailed an order refusing relator's administrative appeal from the SHO's order of November 21, 2011.

{¶ 38} 24. On February 22, 2012, the three-member commission, on a unanimous vote, mailed an order denying relator's request for reconsideration.

{¶ 39} 25. On April 6, 2012, relator, PolyOne Corporation, filed this mandamus action.

Conclusions of Law:

{¶ 40} Two main issues are presented: (1) whether the commission relied upon some medical evidence meeting the statutory requirement that the loss of use of both arms and legs was permanent rather than temporary, and (2) whether the commission relied upon some medical evidence showing that the allowed condition, angiosarcoma, independently caused the permanent loss of use of Evans' four extremities.

{¶ 41} The magistrate finds: (1) there is indeed some medical evidence upon which the commission did rely to support the statutory requirement that the loss of use was permanent rather than temporary, and (2) there is indeed some medical evidence upon which the commission relied showing that the allowed condition, angiosarcoma, independently caused the permanent loss of use of Evans' four extremities.

{¶ 42} Accordingly, it is the magistrate's decision that this court deny relator's request for a writ of mandamus, as more fully explained below.

{¶ 43} R.C. 4123.57(B) provides for weekly scheduled loss compensation for enumerated body parts. It provides as follows:

For the loss of an arm, two hundred twenty-five weeks.

* * *

For the loss of a leg, two hundred weeks.

{¶ 44} The only compensable loss of use under R.C. 4123.57(B) is a permanent and total loss of use. *State ex rel. Welker v. Indus. Comm.*, 91 Ohio St.3d 98 (2001). An injured worker claiming a loss of use under R.C. 4123.57(B) has the burden of showing that his loss of use is permanent. *State ex rel. Carter v. Indus. Comm.*, 10th Dist. No. 09AP-30, 2009-Ohio-5547, citing *Welker*.

{¶ 45} Two cases are instructive to the issues here. They are *State ex rel. Moorehead v. Indus. Comm.*, 112 Ohio St.3d 27, 2006-Ohio-6364 and *Carter*. Accordingly, both cases will be presented here at some length.

The Moorehead Case

{¶ 46} In *Moorehead*, William Moorehead fell approximately 15 to 20 feet head first onto a concrete floor while working on a raised platform at his job site. Upon impact,

he suffered severe spinal cord and other injuries. Unrebuttable evidence established that the spinal cord injury rendered him a quadriplegic. Moorehead never regained consciousness and died 90 minutes after the fall.

{¶ 47} Moorehead's widow applied for death benefits and also for scheduled loss compensation based on loss of use of both arms and legs. The commission denied the application for scheduled loss compensation, observing that scheduled loss benefits may be awarded only to injured workers who experience both a physical and sustained loss of use and also consciously perceive and experience the physical suffering and hardship caused by the loss of use of a body part in the period between injury and death. The commission stated that "the widow-claimant's application for such benefits must fail, as the decedent did not sustain the loss of his extremities, because he was comatose, and completely unaware of the extent of his injuries, for the brief period between the accident and his death." *Id.* at ¶ 3.

{¶ 48} In *Moorehead*, the Supreme Court of Ohio issued a writ of mandamus, explaining:

Similarly, there is no language in R.C. 4123.57(B) requiring that an injured worker be consciously aware of his paralysis in order to qualify for scheduled loss benefits. In an analogous case the Supreme Court of New Hampshire considered a scheduled loss application filed on behalf of a worker whose injury left him in an irreversible vegetative state. *Corson v. Brown Prods., Inc.* (1979), 119 N.H. 20, 397 A.2d 640. The application was denied administratively solely because Corson's vegetative state made him unaware of his loss. The New Hampshire Supreme Court vacated that decision and awarded scheduled loss compensation, writing:

What is of paramount importance in this case is that words such as 'awareness' or 'consciousness' cannot be added under the guise of legislative history to a statute which clearly states that '[t]he scheduled awards under this section accrue to the injured employee simply by virtue of the loss or loss of the use of a member of the body.' * * * When the language used in a statute is clear and unambiguous, its meaning is not subject to modification by construction." *Id.*, 119 N.H. at 23, 397 A.2d 640.

The same rule of statutory construction applies here. When "the meaning of the statute is unambiguous and definite, it must be applied as written and no further interpretation is necessary." *State ex rel. Savarese v. Buckeye Local School Dist. Bd. of Edn.* (1996), 74 Ohio St.3d 543, 545, 660 N.E.2d 463. R.C. 4123.57(B) does not say that compensation is dependent upon a claimant's conscious awareness of his or her loss, whether resulting from amputation or paralysis. Rather, where the requisite physical loss has been sustained, the statute directs that scheduled loss compensation shall be paid.

This court should not graft duration-of-survival or cognizance requirements to R.C. 4123.57(B), because the statute has no text imposing them. Public-policy arguments relative to the requisites of scheduled loss benefits pursuant to R.C. 4123.57 are better directed to the General Assembly, including arguments that a specified time of survival should be mandated after a paralyzing injury and that a worker be cognizant of his or her loss before loss-of-use benefits are payable.

The appellant proffered medical evidence establishing that William Moorehead sustained the physical loss of use of his limbs as a result of his fall. Consciousness of that loss during an extended period of survival is not required by R.C. 4123.57(B), and the commission therefore incorrectly applied the statute when it denied the appellant's application on that basis.

Id. at ¶ 16-20.

The Carter Case

{¶ 49} In *Carter*, the commission denied R.C. 4123.57(B) compensation for the alleged loss of use of the upper extremities and left leg of David E. Carter, who died on October 17, 2006 as a result of an October 14, 2006 gunshot wound to his abdomen while employed as a night club bouncer/security guard. During Carter's hospitalization following the gunshot wound, his right leg was surgically amputated at the knee. Also during the period of hospitalization, Carter underwent a chemically induced paralysis intended to be therapeutic and reversible.

{¶ 50} In *Carter*, the commission denied compensation for the alleged loss of use of the three extremities on grounds that the loss was not permanent, but only temporary in nature. The commission reasoned that, had Carter survived his traumatic injury, he would have recovered from the chemically induced paralysis and would have had full use of the three extremities.

{¶ 51} Carter's dependent children filed a mandamus action in this court challenging the commission's denial of compensation for the alleged loss of use of the three extremities.

{¶ 52} While the relators conceded that the chemical paralysis was intended to be therapeutic and reversible, they posited that the paralysis was rendered permanent by the fact that the paralysis continued up to Carter's death. This court disagreed, stating in its decision:

While the evidence in *Moorehead* showed that the decedent had suffered permanent, albeit brief, paralysis prior to his death, the evidence here indicates that decedent's induced paralysis was a temporary measure designed to aid in his recovery. There is no evidence that, but for decedent's death, the paralysis would have been permanent.

Id. at ¶ 5.

{¶ 53} In *Carter*, this court adopted the magistrate's decision which further explains this court's rationale in holding that Carter's dependents had failed to prove that the loss of use was permanent.

{¶ 54} In the magistrate's decision adopted by the *Carter* court, the magistrate relied upon the definition of "permanent" provided by the syllabus of *Logsdon v. Indus. Comm.*, 143 Ohio St. 508 (1944). The syllabus states:

The term 'permanent' as applied to disability under the workmen's compensation law does not mean that such disability must necessarily continue for the life of a claimant, but that it will, with reasonable probability, continue for an indefinite period of time without any present indication of recovery therefrom.

{¶ 55} Finding the *Logsdon* definition of permanent to be helpful, the magistrate explained why Carter's paralysis was temporary:

In the magistrate's view, the court's discussion of the meaning of the term "permanent" in *DaimlerChrysler* is helpful to the resolution of relator's claim that decedent's death turned a temporary paralysis into a permanent one.

The determination of whether a condition is temporary or permanent, of necessity, involves a determination of the probable future status of the condition based upon current medical information. It is not a determination to be made from hindsight, but a determination of reasonable probability as to the future. *State ex rel. Matlack, Inc. v. Indus. Comm.* (1991), 73 Ohio App.3d 648, 658, 598, N.E.2d 121 ("[C]ourts have held that the permanency is not gauged on the basis of hindsight.").

Thus, the relevant inquiry as to whether the chemically-induced paralysis was temporary or permanent is premised upon events at the time that the paralysis was chemically induced, not upon the hindsight view after decedent's death. *Id.*

Id. ¶ 57-59.

The First Issue: Was the loss of use permanent?

{¶ 56} Here, relying upon this court's decision in *Carter*, relator argues that Evans' death, some four days after Dr. Levy's in-home examination, rendered temporary the observed loss of use of the extremities.

{¶ 57} Clearly, this court's analysis and rationale in the *Carter* case does not compel relator's conclusion that Evans' loss of use was temporary rather than permanent. That is, relator's reliance upon *Carter* is misplaced.

{¶ 58} As indicated by the medical evidence upon which the commission relied, Evans' loss of use was the medically expected result of his angiosarcoma of the liver. Unlike Carter's situation, Evans' loss of use was not chemically induced. Evans' loss of use was not in anyway therapeutic. Rather, Evans' loss of use was the natural consequence of his angiosarcoma. Thus, unlike Carter's temporary paralysis, Evans' loss of use was permanent because it was expected to last, and did last, until Evans' death.

{¶ 59} Here, relying upon the report of its own medical expert, Dr. Buell, relator posits that Evans' loss of use of his four extremities during the days prior to his death was not permanent, but temporary. Dr. Buell opined:

Neither the angiosarcoma nor the treatment of the angiosarcoma can cause permanent damage to the central nervous system. More often than not this state of encephalopathy is completely reversible with appropriate medical therapy. In no way did Mr. Evans ever permanently lose function of all four of his extremities.

{¶ 60} Relator's reference to Dr. Buell's report to support its contention that Evans' loss of use was temporary is problematic given that the commission did not find the report worthy of its reliance.

{¶ 61} Dr. Buell's opinion was directly contradicted by the reports of Drs. Levy and Trangle upon whom the commission did rely.

In his seven-page narrative report, dated July 27, 2011, Dr. Levy states:

The patient does have permanent loss of use of various body parts as statutorily determined. Mr. Evans had no functional, meaningful or volitional use of either of his arms or legs. All four limbs can be considered to have no functional use. For actual practical purposes he has permanently lost the use through the central nervous system dysfunction he has of any extremity movement or activity.

{¶ 62} In his seven-page narrative report dated November 3, 2011, Dr. Trangle states:

To explain it perhaps more succinctly, the combination of encephalopathy, cerebral edema and bleeding directly caused a profound central nervous system depression. Profound central nervous system depression is called coma where there is a loss of consciousness. In addition to loss of consciousness there is also loss of use of the extremities as the central nervous system from the brain does control the other parts of the central nervous system including the spinal cord which mediates the function and motion of the extremities. As Mr. Evans' level of central nervous system depression became more deeply affected and his coma continued to permanently deepen, he had permanent loss of use of his extremities.

{¶ 63} Clearly, the reports of Drs. Levy and Trangle, upon which the commission relied provide the some evidence needed to support the commission's finding that Evans' loss of use of his four extremities was permanent, thus satisfying the statutory requirement for compensation.

The Second Issue: Causation

{¶ 64} As earlier noted, relator contends that Evans' loss of use of his four extremities during the days preceding his death were caused in part by non-allowed conditions and thus the loss of use is not compensable.

{¶ 65} Relator points out that the DHO, following the September 27, 2011 hearing, found that "Dr. Levy does not sufficiently explain how the allowed condition of angiosarcoma directly caused the damage to the central nervous system that would then cause the loss of use of all four extremities."

{¶ 66} Relator points out here, as did the DHO in his order, that Dr. Levy listed multiple "diagnoses" in his July 27, 2011 report.

{¶ 67} It can be noted that the SHO's order of November 21, 2011 vacates the DHO's order of September 27, 2011 and awards compensation based upon Dr. Levy's July 27, 2011 report that the DHO found to be problematical. Also, the SHO's order relies upon the November 3, 2011 report of Dr. Trangle that issued after the DHO's decision.

{¶ 68} Of course, it should be understood that the November 21, 2011 hearing before the SHO was de novo. Thus, it was within the SHO's discretion to reject the DHO's view of Dr. Levy's July 27, 2011 report and to rely upon the report to support an award. That is, the DHO's rejection of Dr. Levy's report was not binding on the SHO.

{¶ 69} A claimant must always show the existence of a direct and proximate causal relationship between his or her industrial injury and the claimed disability. *State ex rel. Waddle v. Indus. Comm.*, 67 Ohio St.3d 452 (1993). Non-allowed medical conditions cannot be used to advance or defeat a claim for compensation. *Id.* The mere presence of a non-allowed condition in a claim for compensation does not in itself destroy the compensability of the claim, but the claimant must meet his burden showing that an allowed condition independently caused the disability. *State ex rel. Bradley v. Indus. Comm.*, 77 Ohio St.3d 242 (1997).

{¶ 70} In his July 27, 2011 report, Dr. Levy could not be clearer that the angiosarcoma independently caused the loss of use of all four extremities during the days prior to death:

At the time that he was evaluated, Mr. Glenn Evans was a 74-year-old gentleman with a diagnosis of angiosarcoma of the liver, a result of an occupational environmental exposure sustained in the course of his employment. * * * Before I had evaluated him, Mr. Evans was found also to have a medical history of congestive heart failure, hypertension, reflux and anorexia, among his other conditions.
* * *

Coma is defined as a profound state of deep unconsciousness. It affects any individual's ability to interact with the surrounding environment. In this particular case it is the direct sequelae of the obfundation and deep unconsciousness caused by his progress fatal cancer. The cancer caused a cascade of events leading to metabolic derangement, lack of oxygenation and in general lack of the necessary physiologic mechanisms sufficient to sustain conscious awareness and bodily function.

{¶ 71} In his November 3, 2011 report, Dr. Trangle could not be clearer that the angiosarcoma independently caused the loss of use of all four extremities during the days prior to death:

[H]is permanent loss of use of his extremities was a direct result of the combination of the buildup of neurotoxic substances, cerebral edema, increased intracranial pressure, cerebral ischemia and most likely even cerebral bleeding. This was an irreversible, permanent progression of events that led to coma which is the definition of profound central nervous system depression with loss of consciousness; and with concomitant inability to use his extremities on a permanent basis. Ultimately, the cerebral pressure and other noted factors built up to the point that the brain stem was almost certainly compressed to the point that he could no longer breathe and this resulted in his ultimate demise. All of these conditions; buildup of neurotoxic substances, cerebral edema, increased intracranial pressure, cerebral ischemia and cerebral bleeding, hepatic failure, hepatic encephalopathy, central nervous system depression, and resultant permanent loss of use of his upper and lower

extremities are physical manifestations that are the direct result of the allowed claim for hepatic angiosarcoma.

{¶ 72} Based on the forgoing analysis, the magistrate concludes that the commission relied upon some evidence supporting a finding that the allowed condition, angiosarcoma, independently caused the loss of use of all four extremities.

{¶ 73} Accordingly, for all the above reasons, it is the magistrate's decision that this court deny relator's request for a writ of mandamus.

/S/ MAGISTRATE
KENNETH W. MACKE

NOTICE TO THE PARTIES

Civ.R. 53(D)(3)(a)(iii) provides that a party shall not assign as error on appeal the court's adoption of any factual finding or legal conclusion, whether or not specifically designated as a finding of fact or conclusion of law under Civ.R. 53(D)(3)(a)(ii), unless the party timely and specifically objects to that factual finding or legal conclusion as required by Civ.R. 53(D)(3)(b).