

STATE OF MICHIGAN
WORKERS' COMPENSATION APPELLATE COMMISSION

RANDALL G. PAIGE (DECEASED),
PLAINTIFF,

V

DOCKET #03-0085

CITY OF STERLING HEIGHTS,
SELF INSURED,
DEFENDANT.

SUPPLEMENTAL OPINION AFTER REMAND TO MAGISTRATE SLOSS.

STEVEN J. POLLOK FOR PLAINTIFF,
RONALD A. WEGLARZ FOR DEFENDANT.

OPINION

GASPAROVICH, CHAIRPERSON

This case is once again before the Workers' Compensation Appellate Commission (WCAC) after remand to the Board of Magistrates. A detailed summary of the facts of this case is set forth in one of our prior opinions, *Paige v City of Sterling Heights*, 2004 ACO #136. We will not repeat those facts here, except where necessary to the particular points of this opinion.

The Supreme Court had vacated our 2004 decision and remanded for further proceedings, consistent with their reinterpretation of MCL 418.375(2) in *Paige v City of Sterling Heights*, 476 Mich 495 (2006), which overturned the interpretation previously set forth in *Hagerman v Gencorp Automotive*, 457 Mich 720 (1998). We were to address two issues on remand: 1) Determine what the proximate cause of Mr. Paige's death was and; 2) Determine the factual dependency of plaintiff's son at the time of the injury. We remanded to the Board of Magistrates for further proceedings and analysis, consistent with the Supreme Court's remand order.

The magistrate issued an opinion and order on remand, on July 1, 2008. He initially indicated that the dependency issues were rendered moot at the remand hearing on June 2, 2008, when defendant agreed to stipulate to Adam Paige's dependency. At that time the parties presented medical testimony by deposition. There apparently were no other exhibits or lay testimony presented in association with the remand. The magistrate made the following findings:

In this case, I find as fact, Plaintiff's fatal myocardial infarction was caused by a combination of coronary artery disease and the weakened condition of his

heart from the initial work-related myocardial infarction in 1991. Drs. Goldberg and Kelly testified persuasively that Plaintiff's stress on the job, inhalation of smoke over 20 years as a firefighter and damage to his arteries and heart wall from the 1991 myocardial infarction made his cardiovascular system more susceptible to plaque build-up, leading to the blockage that caused the fatal event.

Dr. Levinson testified that Plaintiff's fatal coronary artery disease was caused completely by nonvocational factors. However, I did not find his opinion to be persuasive, since its framework is at odds with Magistrate Miller's initial findings in this case. Accordingly, I have given his opinion no weight.

There is a presumption with firefighters that heart diseases arise out of and in the course of employment in the absence of evidence to the contrary. MCL 418.405(2); MSA 17.237(405)(2). Plaintiff applied for "like benefits" as is required as a condition precedent for application of the above presumption. See MCL 418.405(3); MSA 17.237(405)(3). Plaintiff had some outside risk factors, such as smoking cigarettes, but since I have accepted that the work factors caused Plaintiff's cardiovascular system to be more susceptible to plaque build-up, I necessarily reject that these nonvocational factors played any significant role. Accordingly, the presumption applies, and I find as fact that the one most immediate, efficient, and direct cause preceding Plaintiff's death was his employment with Defendant.

I note parenthetically, as did Magistrate Miller, that even were I to apply the usual "significant manner" test of Section 301(2) of the Act, I would find that Plaintiff's fatal event was work-related. As noted above, Plaintiff's work activities played an overwhelmingly large role in the development of his fatal coronary artery disease, and the nonwork factors were insignificant. [Magistrate's opinion, p 3.]

The Supreme Court had held that the phrase "the proximate cause" as used in § 375(2), "...refers to the sole proximate cause, i.e., 'the one most immediate, efficient, and direct cause preceding an injury.'"¹

Defendant argues in its supplemental brief after remand that the "one most immediate, efficient and direct cause: preceding the death of Mr. Paige in 2001 was not his myocardial infarction of 1991. It further argues that the medical testimony establishes that the proximate cause of the 2001 death was the preexisting, progressive non-occupational coronary artery disease. Defendant refers us to Dr. Levinson's testimony in support of its position. However, Dr. Levinson's testimony was specifically rejected by the magistrate. The magistrate is free to accept the medical evidence he finds most persuasive and where, as here, there is a reasonable basis for his findings, we will not displace them. *Miklik v Michigan Special Machine Company*, 415 Mich 364 (1982).

In discussing Dr. Goldberg's testimony, defendant attempts to equate a "medical cause" with a "legal cause." The doctor had indicated on the death certificate that the cause of death was a

¹ Quoting from *Robinson v City of Detroit*, 462 Mich 439 (2000), which had interpreted the phrase "the proximate cause" as used in MCL 691.1407(2)(c), a section defining governmental immunity from tort actions.

myocardial infarction. To accept that as a legal definition of the proximate cause would be to ignore the overwhelming medical testimony regarding the role Mr. Paige's coronary artery disease played in the whole medical picture. It should be noted that the death certificate also listed coronary artery disease as a cause of death.

Dr. Goldberg, decedent's treating cardiologist, testified that a cardiac catheterization in August 2000, revealed multivessel coronary artery disease with hypokinesis of the inferoapical segment of the left ventricle. He explained that the inferoapical segment was not contracting normally. (Dr. Goldberg's deposition, January 9, 2002, p.9) He went on to testify that this abnormality quite likely was related to the first myocardial infarction which occurred in 1991:

- A. Actually, some abnormality was present on the left ventricular angiogram which was done following his first heart attack, his first myocardial infarction. What was described was a small area of akinesis at the apex of the left ventricle. So this finding is in a similar location in the left ventricle. In 1991 it was described as akinesis which means that that portion of the left ventricle was not contracting at all, it was a small area, whereas in 2000, at the time of the left ventricular angiogram it was described as hypokinesis meaning it's contracting, but not as well as it should.
- Q. So based upon that information, can you determine whether it was related, the left ventricle not contracting normally was related to the first myocardial infarction?
- A. I would say it quite likely was since abnormality was present in 1991 in a similar location.

Dr. Goldberg also opined that the effect of all of the heart attacks (including the work related one in 1991) would increase the risk of further cardiac problems and subsequent cardiac death. Also, the fact that he had coronary artery disease and the fact that he had had the myocardial infarction certainly increased the risk that he would have subsequent infarctions. [Dr. Goldberg's deposition, January 9, 2002, p 24.] An individual who experiences a first heart attack is more inclined to have successive heart attacks, where coronary artery disease is present. [Dr. Goldberg's deposition, January 9, 2002, p 49.]

The magistrate did not specifically mention Dr. Zobl's testimony in his opinion after remand, however, he did rely in part on this cardiologist's testimony in his previous decision. There is nothing in his subsequent opinion which would indicate that said reliance was misplaced. Therefore, we consider Dr. Zobl's testimony also. Dr. Zobl opined that each of the myocardial infarctions left cumulative damage to the left ventricle, further reduced overall cardiac function and was the most significant factor in his prognosis. [Dr. Zobl's deposition, July 30, 2002, p 14.] He further opined that he believed that all three cardiac episodes were part of the same disease process, progressing to Mr. Paige's ultimate demise. The cumulative damage of ischemia, infarcted tissue and remodeling are all part of the same disease process which led to his final demise. [Dr. Zobl's deposition, July 30, 2002, p 15.] Dr. Zobl testified:

A. There is a process which we now recognize called remodeling of the heart. When there is an area of the heart which is nonfunctioning because of destruction, in this case by an infarction, the other areas of the heart have to take up the slack. They have to perform for the deficit produced by the infarction. And over a period of time this causes a thickening or enlarging of those functional areas of the myocardium, and this can produce a limiting factor in eventually reducing the overall function of the myocardium.

Q. And the nonfunction or the interference with functioning of the myocardium, does it produce symptoms? Does it produce further damage? What happens?

A. Not damage in the respect that there is more area of infarction necessarily but it imposes an additional workload on the functional areas of the heart. And over a period of time as the left ventricular wall thickens it can outgrow its blood supply and lead to further scattered areas of ischemia in the myocardium.

Q. And ischemia is?

A. Inadequate blood supply. [Dr. Zobl's deposition, July 30, 2002, p 18.]

He further testified that statistically it has been recognized that there are a number of factors which affect the prognosis and one of the more significant ones is a history of a previous myocardial infarction, which causes an increased mortality or a reduced prognosis. He responded affirmatively when asked if a history of the previous myocardial infarction is a significant risk factor.

When asked about the progression, Dr. Zobl testified:

A. Yes. I think that the left ventricular damage was progressive as well as the coronary disease.

Q. So both progress.

A. Both progressed as evidenced in the autopsy where it shows that his left ventricular wall was thickened to 1.8 centimeters. Normal is 1.1 to 1.2. So 1.8 is a very significant hypertrophy of that wall.

Q. And when that wall hypertrophies, that would require the heart to further remodel or work hard to compensate for that damage; is that a fair statement?

A. That's true.

Q. And lastly, based on the autopsy report the best you can conclude is what

you have concluded here today. There's no other way to make any further findings; is that true?

A. I'm not quite sure I understand the question.

Q. Well, based on the autopsy report your conclusion is that it was the arrhythmia that caused the death.

A. I think it is most likely a lethal arrhythmia based on prior experience. I can't be certain of that. But certainly the cause of death was his coronary artery disease.

Q. And the left ventricular damage?

A. "And the left ventricular damage." [Dr. Zobl's deposition, July 30, 2002, pp 49-50.]

The magistrate also relied on the testimony of Dr. Kelly, an internal medicine physician with a specialty in preventive medicine/occupational medicine. Dr. Kelly reviewed the autopsy report, records from Frank A Baciewicz, Jr., M.D., Huron Valley Hospital and the depositions of Gerald Jay Levinson, D.O., Edlred G. Zobl, M.D., and Mark J. Goldberg, M.D. He reported that the records from the original myocardial infarction in 1991 indicated that the blocked arteries were the same arteries, the left anterior descending and posterior distribution of the right coronary. He discussed his opinion on causation as follows:

Discussion: The autopsy clearly identifies the severity of Mr. Paige's coronary artery disease as witnessed by areas of damaged scar tissue and occlusion in several major vessels. The issue with respect to the cause of death probably reflects more of a malignant dysrhythmia than an ischemic event because there did not appear to be any active thrombus in the coronary arteries or any evidence described in the autopsy report of an acute injury. In all likelihood the dysrhythmia was caused by the lack of oxygen flow to the heart and if a dysrhythmia had not occurred an ischemic event likely would have been identified because of the occlusion of the grafts and the severity of the coronary artery disease.

With respect to the etiologies of coronary artery disease, they of course are multiple and involve many risk factors, however, the single most important factor is the presence of coronary artery disease and a previous myocardial infarction. In Mr. Paige's case there was the clear evidence of the work-related myocardial infarction with coronary artery disease identified in 1991. Those same vessels were and are the same vessels that were involved in the subsequent myocardial infarction and likewise it was these same vessels that were found to be so occluded at the time of his death and autopsy in 2001. There did not appear to be any significant events in between the 1991 and 2000 period although there were some episodes of chest pain. Stress testing and evaluations were unremarkable. The role and mechanism of the previous coronary artery disease with respect to the development of

subsequent coronary artery disease and events has to do with the plaque formation that was present in 1991. Those plaques did not and could not regress. The presence of plaque accelerates the development of additional plaque as the injury itself provides a foci for further deposition of plaque and thrombus. These plaques provide a site for platelet aggravation and the deposition of lipid-laden deposits. There are a variety of other hypothetical mechanisms for Mr. Paige's death, but the facts present from my review of this material clearly indict the work-related injury of 1991 as the most likely. That event and decision regarding the 1991 injury clearly makes the work-related myocardial infarction from 1991 the most immediate and direct cause of the coronary artery disease preceding his death and thus the cause of his death as well. [Plaintiff's deposition exhibit #1, p 2.]

Defendant argues that the "one most immediate, efficient and direct cause" preceding the death of Mr. Paige in 2001 was not his myocardial infarction of 1991. We would agree. Based on the medical testimony, as relied upon and given weight by the magistrate, the "one most immediate, efficient and direct cause" preceding the death of Mr. Paige in 2001 was his coronary artery disease. Doctors Zobl and Kelly explained in great detail how any preexisting coronary artery diseases that he may have experienced prior to 1991 was significantly aggravated by the myocardial infarction he suffered in 1991. That MI was found to be work related. Since the 1991 MI was work related and a significant contributing factor to the coronary artery disease, a § 301(2) work related aggravation of the coronary artery disease has been established. The coronary artery disease, in turn, being work related and the "one most immediate, efficient and direct cause" preceding the death, establishes a compensable death claim.

Defendant argues that the magistrate erred in applying the "firefighters presumption" found in § 405(2) and conditioned by § 405(3):

- (2) Such respiratory and heart diseases or illnesses resulting therefrom are deemed to arise out of and in the course of employment in the absence of evidence to the contrary.
- (3) As a condition precedent to filing an application for benefits, the claimant, if he or she is one of those enumerated in subsection (1), shall first make application for, and do all things necessary to qualify for any pension benefits which he or she, or his or her decedent, may be entitled to. If a final determination is made that pension benefits shall not be awarded, then the presumption of "personal injury" as provided in this section shall apply. The employer or employee may request 2 copies of the determination denying pension benefits, 1 copy of which may be filed with the bureau.

We agree that the magistrate erred. The condition precedent in § 405(3) was not met. Mr. Paige had applied for and was receiving a disability pension prior to his demise. However, the magistrate found, in the alternative, that the decedent's work activities played an overwhelmingly large role in the development of his fatal coronary artery disease, and that the nonwork factors were

insignificant. That finding is supported by the competent, material and substantial evidence set forth above. It is that finding which is the basis of our conclusion to affirm.

Commissioners Grit and Will concur.

Martha M. Gasparovich	Chairperson
Donna J. Grit	Commissioner
Rodger G. Will	Commissioner

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This matter was remanded to the Board of Magistrates on October 17, 2006, ACO #243. Magistrate Andrew G. Sloss' supplemental decision was mailed July 1, 2008. The Commission has considered the record and counsel's briefs, and believes that the magistrate's supplemental decision should be affirmed. Therefore,

IT IS ORDERED that the magistrate's supplemental decision is affirmed.

Martha M. Gasparovich	Chairperson
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Donna J. Grit	Commissioner
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Rodger G. Will	Commissioner
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