

EXXON CORPORATION *v.* MRS. EUGENE V.
FLEMING WIDOW OF EUGENE V. FLEMING, DECEASED

5-6149

489 S.W. 2d 766

Opinion delivered January 22, 1973

[Rehearing denied February 26, 1973.]

WORKMEN'S COMPENSATION—COMMISSION'S FINDINGS AS TO CAUSA-
TION—REVIEW.—Commission's finding that there was a casual con-
nection between worker's injury and a subsequent heart attack re-
sulting in death *held* supported by substantial evidence, including
the attending physicians' opinion that worker's injury contrib-
uted to the heart attack.

Appeal from Pulaski Circuit Court, Second Divi-
sion, *Warren Wood*, Judge; affirmed.

Chowning, Mitchell, Hampton & Chowning, for ap-
pellant.

McMath, Leatherman & Wood, for appellee.

CARLTON HARRIS, Chief Justice. Eugene Fleming, an employee of Exxon Corporation,¹ appellant herein, was injured in the course of his employment on January 10, 1970 when he slipped on ice and received a blow on the back of his head. Medical expenses and compensation benefits were paid to Fleming, who did not return to work. Five months later, on June 12, 1970, Fleming died from an acute myocardial infarct. His widow filed a claim with the Workmen's Compensation Commission for benefits which claim was controverted by appellant. The referee directed the company to pay all death benefits, medical, funeral expenses, and attorney's fee. On appeal to the full commission, the award was affirmed with the modification that the payments to the widow be at the rate of \$35.00 per week instead of \$49.50 per week, as fixed by the referee. The Pulaski County Circuit Court affirmed the opinion of the commission, and from the judgment so entered, appellant brings this appeal. For reversal, it is simply asserted that the commission erred in determining that there was a causal connection between Fleming's injury and his subsequent death.

The testimony reflected that Fleming was 58 years of age, employed at the time of the injury at Tanglewood Esso Service Station at Cantrell & Mississippi in Little

¹At that time Humble Oil & Refining Company.

Rock, and appeared in excellent health before the injury. Both his wife and son testified that, prior to the injury, he had never complained of chest pains, nausea, shortness of breath, or pain in his arms. During his stay at the hospital, and after his release, he suffered chest pains and shortness of breath, his condition progressively growing worse, and he continued to get weaker, he could not walk across the room unless holding on to something; he slept sitting at the table with a pillow in front of him to lean on as he could not lie down.

Dr. Ray Jouett, a neurosurgeon of Little Rock, testified that he first saw Mr. Fleming in the emergency room at Baptist Hospital on January 16, 1970, being called into the case by Dr. Thurston Black, a practicing physician. The doctor described Fleming as "semi-comatose" at that time, agitated, his speech incoherent, and he said the patient would only respond to painful stimulation. The doctor diagnosed a convulsive seizure and a traumatic subarachnoid hemorrhage; medication was commenced to relieve swelling of the brain and prevent further convulsive seizures. The pulse rate was slow and the doctor felt that this was due to some cardiac irregularity, and he said that the type of injury would place a burden on the heart. Subsequent to Fleming's dismissal from the hospital, Dr. Jouett saw the patient on February 25, at which time the latter complained of extreme dizziness, shortness of breath, and other symptoms which the doctor stated led him to believe "that he had what we call postural hypotension which is the lowering of the blood pressure when an individual stands or turns quickly. He got spots in front of his eyes, his vision would decrease, and the fact that he was unsteady, all made me believe that he was having episodes of hypotension." A prescription was given to improve circulation, and he again saw Fleming in March and June.²

Dr. Jouett testified with reference to the autopsy report, that report reflecting atherosclerotic cardiovascular disease, pulmonary edema and congestion, chronic passive congestion of lungs and liver, and focal encephalomalacia of the right frontal lobe. These terms were explained by Dr. Jouett who concluded that, in his opinion,

²Fleming was to see the doctor on May 11 but was unable to go to the office because of chest and abdominal pains.

there was a relationship between the injury and Fleming's death, i.e., the injury contributed to the heart attack which resulted in death. When questioned on cross-examination as to the basis for his opinion, Dr. Jouett stated:

"Assuming that such an individual had an underlying heart disease [and this was established] and underwent a period, first of all, of convulsive seizures such as I witnessed on one occasion—he'd also had seizures before he came in—the man was difficult to control. In fact, I believe he had to be restrained in bed. These alone are two things that could aggravate an existing heart condition. Number two, individuals with preexisting arterial disease such as the patient obviously had that we're talking about, demands more efficient cardiac output to continue his circulation because of hardening of the arteries that develops, the arteriosclerosis that has been described. If there is some physiological means or reason in which this circulation is impaired, then this individual is a good candidate to have a stroke, such as a clot in one of the arteries of the brain because of the hardening roughened areas in the brain, and the same is also true of the heart. I think this man had, and this is my opinion, this man had repeated episodes of hypotension or low blood pressure manifested by the symptoms that he kept repeatedly complaining to me of, that is the dizziness, his positional incoordination, the black spots in front of his eyes, all this sort of thing that lets us know that we are dealing with a hypotensive problem. The slowing of the circulation through diseased vessels lends itself to the formation of a thrombosis. That plus the fact that I observed, if I may refer to the patient, this man deteriorating in front of my eyes over a period of about four months medically, that's how I would arrive at that conclusion."

Under vigorous cross-examination, the doctor stated that, in giving his opinion, he was speaking of possibilities rather than probabilities. Though shown articles written by well known medical authorities wherein they disagreed with some of the views expressed by Dr. Jouett, he still said that he saw nothing in the literature which preclud-

ed the fact that a serious brain injury could be an aggravating factor in a pre-existing heart condition.

Dr. Thurston Black saw Mr. Fleming on January 14, 1970 at the request of appellant company,³ and he also saw Fleming while he was in the intensive care unit at the hospital. He said that Fleming was confused, able to talk, but was not entirely rational; that the patient complained a great deal of headaches and dizziness. He later saw Fleming on February 2 after dismissal from the hospital and again saw him on April 21 when he complained of indigestion, a "burning" sensation that he had after exertion. Fleming stated this would cease after he had rested for five or ten minutes. The doctor described the treatment given and he stated that the autopsy reflected a pre-existing heart disease, though there was no way of knowing how long this had been in effect. Black said, assuming that Fleming was asymptomatic as far as heart disease was concerned up to the time of the injury, and assuming that the cardiac symptoms, such as shortness of breath and pains in the chest, developed subsequent to January 10, and based upon his treatment and the history of the patient, it was possible that the brain injury received by Fleming aggravated a pre-existing condition. Further, "I think it is possible that during the time he was having these convulsions that there was an increase in pressure, particularly from the heart, that could conceivably have broken off or gotten a little rough edge on an arteriosclerotic plaque". He also said that decreased blood pressure would present the possibility of a clot from a pooling of blood or from a slower circulation. Neither doctor stated that there was a probability that the accident triggered or aggravated a pre-existing condition that subsequently caused the death of Fleming; both only used the term "possibility". And both, under cross-examination, somewhat modified or qualified some statements made on direct examination—but the fact remains that both considered the connection between the injury and death to be a distinct possibility.

Appellant contends that the testimony of the doctors does not constitute substantial evidence to support an award but we disagree. The same contention was made in the case of *Kearby v. Yarbrough Brothers*

³Dr. Black testified that he examined the employees of appellant.

Gin Co. and Southern Farm Bureau, 248 Ark. 1096, 455 S. W. 2d 912 where the doctor who testified on behalf of the claimant gave rather indefinite answers, several times, while discussing cause and effect, using the expression "could cause" or "might cause", and not stating a conclusion on the causal relationship between the work and the heart attack with language stronger than there "could be" a connection. We held that the use of these expressions or similar phrases would not bar a finding of causal connection, provided that there was other evidence supporting the conclusion.

In the present case, in addition to the statements of the doctors, one circumstance is conspicuous, viz., that Fleming prior to the brain injury, was working every day, had never suffered from chest pains, shortness of breath, nor had other symptoms indicative of a diseased heart or arteries. The proof is clear that subsequent to the accident, these symptoms were much in evidence and the condition they reflected grew progressively worse from day to day. In fact, Dr. Jouett stated that he observed "this man deteriorating in front of my eyes over a period of about four months medically". Let it also be remembered that there was *no* medical evidence to the effect that the injury did not, or could not, contribute to Fleming's death, and this despite the fact that one of the doctors testifying had served as the company's doctor in examining employees.

We hold that there was substantial evidence to support the findings of the commission.

Affirmed.

BYRD, J. concurs; FOGLEMAN, J. dissents.

CONLEY BYRD, Justice, concurring. In this case the lay testimony shows that prior to his injury on January 10, 1970, Eugene Fleming was an able-bodied man who worked every work day. Thereafter and until the time of his death on June 12, 1970, he had headaches, convulsions, chest pains and shortness of breath. In fact he did not work another day. The doctors stated that he deteriorated be-

fore their very eyes. The fact that the doctors used the word "possibly" instead of "probably" does not make the testimony as to causation speculative nor make heart insurance out of the Workmen's Compensation law. To hold otherwise would require the Commission to disregard all of the lay testimony and the undisputed testimony of the doctors that the man deteriorated before their very eyes.

Therefore I respectfully concur.

JOHN A. FOGLEMAN, Justice. dissenting. Prior to the decisions in *Kearby v. Yarbrough Brothers Gin Company and Southern Farm Bureau*, 248 Ark. 1096, 455 S.W. 2d 912, and *Bradley County v. Adams*, 243 Ark. 487, 420 S.W. 2d 900, the result reached here would not have been possible. My dissent in those cases was prompted by the realization that the court's holding would spawn innumerable cases such as this, where the evidence is, to say the least, no more positive as to the cause of death than it was in those cases. The rules of law and authorities stated and referred to in my dissent in each of those cases should be applied here. No one would advocate that causal connection, in a death case, must be proved to a mathematical certainty. Medical science is not that exact. Reasonable medical certainty should be required and possibilities are simply not sufficient to meet the burden of proof. Otherwise, we tend to convert workman's compensation into insurance against death from a heart attack following compensable injury. Perhaps the breach in the dam resulting from the two cases above cited can never be closed, but it should be.

In a personal injury case, *Jonesboro Coca-Cola Bottling Co. v. Young*, 198 Ark. 1032, 132 S.W. 2d 382, this court quoted the following from 15 Am. Jur. 413, Damages § 22, with approval:

The damages recovered in any case must be shown with reasonable certainty both as to their nature and in respect of the cause from which they proceed. No recovery can be had where it is uncertain whether the plaintiff suffered any damages unless it

is established with reasonable certainty that the damages sought resulted from the act complained of. Hence, no recovery can be had where resort must be had to speculation or conjecture for the purpose of determining whether the damages resulted from the act of which complaint is made or from some other cause, * * *.

We applied the rule to approve the following instruction:

If you should find from the testimony that the illness or injury to the plaintiff, if any, might equally as well have resulted from some cause other than drinking the Coca-Cola, you will find for the defendant.

In *Missouri Pac. R. Co. v. Hampton*, 195 Ark. 335, 112 S.W. 2d 428, we said:

* * * where the testimony leaves the matter uncertain, and shows that any one of two or more things may have brought about the injury and death, for one of which the employer would be responsible, and for the others he would not be responsible, there can be no verdict for the plaintiff. This rule is well established, and if all the evidence showed that the injury and death might have occurred from injury or disease, there could be no recovery; * * *.

In *Denton v. Mammoth Spring Electric Light & Power Co.*, 105 Ark. 161, 150 S.W. 572, although we were speaking of proximate cause, primarily, we quoted and relied upon language from *Coin v. John M. Talge Lounge Co.*, 222 Mo. 488, 121 S.W. 1, 25 L.R.A. (n.s.) 1179, 17 Ann. Cas. 888 (1909), which should have application to cause of injuries (or death), just as fully as to proximate cause. The language quoted from the Missouri case was:

If an accident causing injury to a servant may have resulted from either one of two causes, for one of which the master is liable and for the other of which he is not, the servant, in an action to recover for the injury, must show with reasonable certainty that the cause for which the master is liable pro-

duced the injury; and if the evidence merely leaves this to conjecture the plaintiff must fail in his action. To the same effect, see *Green v. Southern Ry. Co.*, 72 S. C. 398, 52 S. E. 45, 5 Ann. Cas. 165, and case note; *Schultz v. C., M. & St. P.R. Co.*, 116 Wis. 31, 92 N.W. 377.

The above cases are tort cases, but I can conceive of no logical reason why we should have one rule in this regard for tort cases and another for workman's compensation cases.

It should be noted in evaluating the evidence and applying the appropriate rules that this case is not one in which the matter at issue is within the realm of common knowledge. Rather, it is a case in which the answer to the question of causation lies peculiarly and wholly within the realm of medical science. It should further be remembered that this is not a case in which there is an absence of evidence of any other apparent cause which might have resulted in the employee's death. It is clear from the evidence that there were other possibilities and that Fleming's death from the natural progress of his heart disease was just as likely. In this cause, the commission either had to apply its own medical knowledge or resort to speculation and conjecture in order to find that the death was caused by the compensable injuries Fleming received. The commission's findings themselves recited that Dr. Jouett recognized the possibility of other causes of Fleming's death and that Dr. Black admitted there was a *possibility* that there was a relationship between Fleming's injury and his coronary occlusion.

Fleming had been involved in a serious automobile accident in 1950, from which he suffered broken bones and contracted pneumonia. He was then off work a little over a year and one-half. Dr. Jouett related that a lumbar puncture revealed "old" blood in Fleming's spinal fluid. He stated that later while Fleming was in the intensive care ward, his pulse became of concern because it was slow. This neuro-surgeon found the patient's spinal pressure to be inconsistent with his pulse. In retrospect, the doctor testified the the slow pulse was attributable to cardiac irregularity instead of, as he originally thought, an intercranial blood clot, because

it developed that there was no such clot. He said that the type of injury Fleming sustained would put a burden on his heart. Two months after Fleming was discharged from the hospital, Dr. Jouett saw the patient again and attributed the patient's complaints of dizziness and shortness of breath to postural hypotension—i.e., lowering of the blood pressure when the affected individual stands or turns quickly. Other symptoms indicated that Fleming was suffering such episodes. Jouett only saw Fleming one other time. That was in June 1970, when Fleming told Jouett that the symptoms about which he had complained to this doctor had improved, but, in the meantime, he had suffered a heart attack for which he was being treated.

Jouett's opinion that there was an indirect relationship between Fleming's injury and death was based upon the assumption that Fleming had a heart disease at the time of the injury but that he was asymptomatic at that time. He said that an electrocardiogram run on Fleming a little over a week after the injury was normal, and in view of that fact, the slow pulse *probably* did not evidence cardiac irregularity. Blood pressure readings were, he said, within normal limits for a man Fleming's age. Dr. Jouett said that the slowing of blood circulation through diseased vessels lent itself to the formation of a thrombosis which, along with the patient's deterioration over a period of four months in front of the doctor's eyes, caused him to arrive at his conclusion that the injury caused the death. The doctor said that a myocardial infarct caused by a thrombus or blood clot was the actual cause of death. On cross-examination, the doctor made pertinent statements that:

1. He did not think the stress of convulsion had anything to do with the formation of the blood clot that was the cause of death.
2. The autopsy report reflected that Fleming had suffered a heart attack at some time in the past, which could have been one to 10 years earlier.

Upon cross-examination, appellant's attorney went to great lengths to determine the basis of the belief of this expert witness that circulatory difficulties resulting

from the injury had something to do with the death of Fleming. These specific questions and answers appear in the record:

Q. When you speak of belief, do you speak of possibilities?

A. Possibilities.

Q. Not probabilities?

A. Possibilities.

(And following a question pertaining to statements from an article in Vol. 148, Journal of American Medical Association):

A. Not really. This honored gentleman uses an awful lot of probabilities, an awful lot of perhapses—

Q. Well, didn't you recite to me that yours was strictly a possibility?

A. That's right. Therefore, as I said, I cannot really see there is any great difference.

Q. Do you accept those statements?

A. Yes, sir.

Q. And you said his degree of expertise in this field was greater than yours?

A. Yes, sir.

Q. To the extent that there is a conflict, will you accept his views over yours?

A. Yes, I surely would.

* * *

Q. Are you saying that low blood pressure will induce the thrombosis?

A. Yes, sir. I'm saying that if we're dealing with an individual that has arteriosclerotic vessels, it can be a precipitating factor.

Q. Doctor, are you attempting to persuade the Commission that there is a causal relationship between the trauma suffered by the claimant in January and his resultant death from a myocardial infarction?

A. I'm not trying to persuade the Commission of anything, first of all, except what I believe. I think I've stated that I believe that there is an indirect relationship of this man's injury and his demise.

Q. An indirect relationship—

A. An indirect relationship. I do not believe that the fact that this man was hit on the head or fell or whatever his difficulty was in January, was the direct result of him having a coronary five or six months later, but I'm saying that—in postulation—that there is an indirect relationship, and I have gone through those of the condition which this individual exhibited. I went through the method in which I arrived at that conclusion, and that is all that I can tell you. It's my personal opinion and the things that I observed.

Q. By postulation, it's a surmise on your part?

A. Yes, sir.

Q. We are faced with the hard fact that the man did die of a myocardial infarction, aren't we?

A. Yes, sir.

Q. The question is the causation?

A. Yes, sir.

Q. And the best that you're able to present to the Commission is a mere surmise on your part?

A. Of observations. Yes, sir.

Q. This is your opinion, and you're stating it to be a possibility?

A. I'm stating it to be a possibility.

Q. Could there be other possibilities?

A. There could be other possibilities.

Q. Unlimited possibilities?

A. Unlimited possibilities.

Dr. Black testified on cross-examination that the decreased blood supply to Fleming's heart muscles was apparently attributable to severe arteriosclerosis, which would *probably* indicate that this was a degenerative disease built up over several years. Dr. Black accepted the following views of Dr. Arthur M. Master expressed in Vol. 148 of the Journal of the American Medical Association with the qualifications therein stated:

The interval elapsing between an effort or trauma and the onset of coronary episode is of great importance in determining causal relationship. In coronary insufficiency symptoms usually set in immediately; if they appear after one or two hours, it is unlikely they were caused from the strain. In coronary occlusion, the sequence is less direct since it may take some time for a thrombus to form. However, one would expect some symptoms such as pain or weakness within the first day or two, if there has been a change in an artery. If there are no symptoms for 48 hours following effort or trauma, and a coronary occlusion develops later, it was probably not caused by the effort or trauma.

The problem becomes more complex if, following the strain and the subsequent angina, coronary occlusion occurs after several months. It is probable that there is no causal relationship between the effort and the occlusion in these circumstances.

On cross-examination Dr. Black classified his own testimony thus:

I think we would have to speculate as to whether or not the injury had any bearing on his arteriosclerotic process. This, as I say, would be a speculation. It's possible.

Q. I think we are agreed, aren't we, that the arteriosclerotic condition of this man's coronary artery is the result of a pathological disease going back over a good long period of time?

A. Probably.

Q. There's no way that this trauma could ever have contributed to that?

A. Not to have caused it. No.

Q. No. So the only thing that we're really concerned with is whether or not this trauma produced the clot basically, and there's no way that—

A. I don't think the trauma would have produced the clot that killed him.

Q. That's right.

A. The only problem is whether there were an aggravation of his preexisting arteriosclerosis, and as I say, this is a possibility, and I have no way of knowing probabilities or percentages on it.

Q. And there could have been any number of factors or other possibilities?

A. Oh, yes.

Q. He could have gotten excited watching a football game on television—

A. Could have.

Q.—or out in the yard walking around?

A. Yes.

Q. There's no way?

A. This is true.

Q. Doctor, coming back to my last— We were at the point of the gap between the thrombus which we agree caused the infarction and this brain injury to which Mr. McMath just referred. At that time I understood you to say that that point—that is to say the thrombus—and the trauma in January, you couldn't connect up?

A. I don't think you could at that length of time unless you postulate some—some change in his arteriosclerotic process due to the injury.

Q. When you say postulate, again don't we mean surmise—

A. That's right.

Q.—conjecture, speculation?

A. Yes.

Each of the doctors put his own testimony in the realm of speculation and conjecture and admitted of other possibilities. The most outstanding possibility was that Fleming's death was attributable to the natural progress of his existing disease. This simply does not meet the standard of proof required to show causal connection.

I would reverse the judgment.