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Supreme Court of Kentucky

2006-SC-000885-WC

RUSSELLVILLE WAREHOUSING — APPELLANT

V.

APPEAL FROM COURT OF APPEALS
2006-CA-001224-WC
WORKERS' COMPENSATION NO. 02-64829

KHRIS BASSHAM, DECEASED;
GLENDA BASSHAM, SURVIVING SPOUSE
OF KHRIS BASSHAM;
HON. R. SCOTT BORDERS,
ADMINISTRATIVE LAW JUDGE; AND
WORKERS' COMPENSATION BOARD — APPELLEES

OPINION OF THE COURT

AFFIRMING

Among the grounds for reopening a final workers' compensation award are newly discovered evidence and mistake. An Administrative Law Judge (ALJ) dismissed the defendant-employer's motion to reopen, which was based upon evidence that came into existence after an award was rendered and changed a medical expert's opinion regarding causation. Although the Workers' Compensation Board (Board) affirmed insofar as the evidence did not come within the legal concept of newly discovered evidence, a majority reversed on the ground of mistake. The Court of Appeals found the Board's construction of what constituted "mistake" to be so broad as to undermine the doctrine of res judicata and reversed on that basis. We affirm.

Khris Bassham was born in 1952 and graduated from high school with no specialized or vocational training. He was exposed to manganese dust for about seven years in the course of his work, which included loading 50-pound bags of manganese powder into a bin. Early in 2002, he began to lose weight and to have difficulty sleeping and negotiating stairs. Later, he developed a tremor in his hands and greater difficulty walking. He last worked in October 2002 and was referred to a neurologist shortly thereafter. After testing revealed a level of manganese in his blood that was nearly 2 ½ times the normal limit, Bassham filed an application for benefits. His wife testified at the June 2004 hearing on his behalf. She stated that his weight had dropped to 84 pounds. His arms and legs were contracted; he was unable to move by his own volition; and he was on a feeding tube, oxygen, and a catheter.

Dr. Hoos, a neurologist who evaluated Bassham at Vanderbilt University on December 30, 2002, noted that his speech was sometimes slurred. His gait was abnormal. He kept his feet wide apart, and he was unsteady. His main complaint was difficulty walking. Dr. Hoos stated that blood studies ruled out neuropathy, immunologic causes of neuropathy, immunologic conditions that may be associated with an underlying cancer, and myasthenia gravis. He explained that manganese targets the nervous system and that the classic symptoms of manganese toxicity resemble those of Parkinson's disease; however, Bassham had symptoms that involved the cerebellum and were not typical of Parkinson's. Dr. Hoos acknowledged that he had never seen a case of manganese toxicity, did not know the full spectrum of the condition, and would not be surprised to learn that it caused problems with the cerebellum. Nonetheless, he diagnosed an organic ataxia due to a disease of the nervous system. In February 2003

he noted that a brain MRI was normal. He continued to think that the high manganese blood level was work-related but that Bassham's symptoms were not due to manganese toxicity.

Dr. Nausieda specialized in Parkinson's disease and other movement disorders and ultimately became Bassham's treating neurologist. In the first of three depositions, Dr. Nausieda stated that manganese is one of the toxins known to produce Parkinsonian symptoms. He explained that an elevated manganese level is a common feature in Parkinson's disease and that he had studied the relationship since the early 1970s. He noted that the physicians at Vanderbilt had tested Bassham exhaustively for inherited ataxias and syndromes known to occur in some cancer patients but that all tests were negative. Despite finding highly elevated blood manganese levels, they had dismissed the possibility of manganese toxicity. Instead, they diagnosed progressive ataxia, which is an imbalance syndrome related to cerebellar dysfunction. Dr. Nausieda testified that he first saw Bassham in March 2003, at which time he showed a severe gait disturbance, could not walk without full assistance despite keeping his legs wide apart, had a masked face, and appeared to be acutely psychotic. He diagnosed acute manganism with neuropsychiatric manifestations and a spastic ataxic gait and noted that the symptoms resembled those of workers in third-world countries who have been exposed to raw manganese.

Dr. Nausieda noted that Bassham's manganese levels remained abnormally high in April 2003. Biochemical screens were normal; copper, mercury, and lead levels were normal; and there was no evidence of infection or inflammatory disease. An electroencephalogram revealed pathologically slow brain wave activity. The most

recent brain MRI revealed mild generalized atrophy that was consistent with some type of toxic effect. Medication helped reduce the symptoms temporarily, but by mid-June Bassham was significantly worse and totally dependent on others. He had deteriorated even more by September and required round-the-clock care. Dr. Nausieda concluded that his symptoms resulted from manganese toxicity and that all other conditions had been ruled out.

Dr. Gray performed a neuropsychological evaluation in June 2003. He stated that he data he obtained was consistent with a neuro-toxic encephalopathy and that Bassham's neuro-cognitive profile and progressive decline were consistent with the literature concerning manganese toxicity. He acknowledged, however, that his background regarding manganese exposure was limited and deferred to Dr. Nausieda.

Dr. Wolens, a specialist in occupational and environmental medicine, examined Bassham for the employer in February 2004. He stated that he suspected manganese toxicity initially, but his research indicated that Bassham's rapid and profound deterioration and his diffuse cortical atrophy were unprecedented in the medical literature. Dr. Wolens reviewed air sampling data regarding the employer's facility and stated in a supplemental report that the cumulative manganese levels would be expected to be low and that it was unclear why Bassham's levels had been elevated.

Dr. Corwin, a neurologist, reviewed Bassham's medical records at Dr. Wolens' request. He diagnosed an organic ataxia of unknown origin. He also listed numerous differential diagnoses, including chronic meningoencephalitis, lipid storage disorder, Creutzfeldt-Jakob disease, cerebrocerebellar degeneration, Alzheimer's disease, Pick's disease, multi-infarct dementia, head injury, mild chronic epilepsy, neuronal storage

disease, and A.I.D.S. He did not address causation.

Dr. Nausieda testified in a second deposition that, like manganese toxicity, some of the conditions that Dr. Corwin listed could only be excluded on autopsy. Testing had ruled out meningoencephalitis, other infections of the nervous system, a bacterial infectious process, or an immunologic disorder. A test for Creutzfeldt-Jakob disease was indeterminate, but the negative EEG and lack of clear-cut serologic data militated against the diagnosis. Cultures for fungi, anaerobic bacteria, and tuberculosis showed no growth. He stated that Dr. Corwin had listed diseases that can cause progressive cognitive decline and severe gait disturbance but had ignored Bassham's occupational history, which overwhelmingly suggested a more obvious etiology. Likewise, regardless of how safe Dr. Wolens found the workplace to be, the fact remained that Bassham handled manganese and had high blood levels of the substance. He attributed the severity of Bassham's symptoms to the fact that he was exposed to manganese oxide, while the cases in the medical literature involved other forms of manganese.

Dr. Racette, a specialist in movement disorders at the Washington University School of Medicine, testified for the employer based on a medical records review and Dr. Nausieda's deposition. He noted that Bassham's history included a family member who had died from a prion disease, and he disagreed with most of Dr. Nausieda's conclusions. He did not think that Bassham suffered from occupational manganism or that he exhibited either the signs or symptoms of the condition. In his opinion, a more likely diagnosis was a prion disease, caused by a slow virus, the most commonly-known form of which is Mad Cow disease. Dr. Racette stated that Bassham's symptoms were consistent with Creutzfeldt-Jakob disease, a rapidly-progressing prion disease that is

often characterized by ataxia, dementia, and unsteady gait.

After reviewing Dr. Racette's deposition testimony, Dr. Nausieda stated that it raised nothing that he had failed to consider and explained why he continued to think that Bassham's symptoms were due to manganese toxicity. Asked about Mad Cow disease and Creutzfeldt-Jakob disease as possible diagnoses, he stated that the spinal fluid analysis and electroencephalogram ruled them out.

The ALJ reviewed the conflicting evidence regarding causation in great detail and noted that a university evaluation by Dr. Fletcher did not carry presumptive weight because it did not address the issue. Based on the evidence of work-related manganese exposure, the elevated blood levels of manganese, and the testimonies of Drs. Nausieda and Gray, the ALJ determined ultimately that Bassham suffered from an occupational disease that manifested itself in a neurological disorder and resulted in total disability. Neither party appealed; thus, the decision became final.

Bassham died on November 11, 2004, about two months after the award. His widow moved to be substituted as a party, attaching a copy of the certified death certificate. It confirmed that a pathological study was performed at the time of death and listed the cause of death as apparent manganese poisoning due to probable exposure to raw manganese. The employer conceded that the widow was entitled to a continuation of the award under KRS 342.730(3)(a); however, it refused to stipulate that manganese poisoning was the cause of death. Moreover, it requested a copy of the pathology report or permission to have an autopsy performed at its own expense. The ALJ awarded continuation benefits, noting that there had yet to be a finding that Bassham's death was work-related.

In May 2005 the widow filed a motion for death benefits. She attached a report from Dr. Sparks, who had treated Bassham at the Muhlenberg Medical Center since 2003. It indicated that post-mortem pathologic studies were not consistent with Parkinson's disease, which some physicians had diagnosed. It also stated that manganese is a known neuro-toxin, the symptoms of which are seen more often in third-world countries where it is handled in its raw state. Dr. Sparks concluded that Bassham's neurological deterioration and death resulted from manganese toxicity. The ALJ entered an order on September 12, 2005, that confirmed the continuation of benefits under KRS 342.730(3)(a).

On October 31, 2005, the employer filed a motion to reopen, seeking a revocation of all previous awards on the basis of newly discovered evidence or mistake. The employer acknowledged that KRS 342.125 did not permit a reopening to affect sums already paid but requested an order permitting it to terminate payment to Bassham's widow. Attached to the motion was a September 7, 2005, report from Dr. Nausieda, stating that the autopsy data did not support a diagnosis of manganese toxicity and that pathologic examination indicated that Bassham appeared to have suffered from a slow virus. Moreover, the National Prion Laboratory had confirmed the presence of Creutzfeldt-Jakob agent in his nervous system, and no evidence suggested that he had more than one primary neurologic disorder. The employer argued that the autopsy evidence could not have been discovered with the exercise of due diligence in the initial proceeding and, in the alternative, that Slone v. R & S Mining, Inc., 74 S.W.3d 259 (Ky. 2002); Durham v. Copley, 818 S.W.2d 610 (Ky. 1991); and Messer v. Drees, 382 S.W.2d 209 (Ky. 1964), supported a reopening on the ground of mistake.

However, the ALJ concluded that the employer failed to make a prima facie showing under either ground.

As the ALJ noted, Black's Law Dictionary 579 (7th ed. 1999) explains that "newly discovered evidence" is a legal term of art. It refers to evidence that existed but that had not been discovered and with the exercise of due diligence could not have been discovered at the time a matter was decided. Stephens v. Kentucky Utilities Company, 569 S.W.2d 155 (Ky. 1978), explains further that when the term is used in a statute, it may not be construed to include evidence that came into being after a matter was decided. The decisive effect of evidence does not arise unless it is properly viewed as being "newly discovered." See Walker v. Farmer, 428 S.W.2d 26 (Ky. 1968). Bassham's autopsy report was not newly discovered evidence for the purposes of KRS 342.125 because it did not exist when Bassham's award was rendered; therefore, its decisive effect was immaterial unless another ground existed for reopening. The employer argues that mistake was such a ground.

Messer v. Drees, supra, concerned a motion to reopen the claim for a 1960 injury. Messer sustained a work-related blow to the head and alleged that the injury aggravated a degenerative cervical spine condition, causing it to be disabling. The referee awarded a permanent partial disability. While review by the full Board was pending, Messer sought to reopen in order to introduce recently-obtained medical evidence, which indicated that he also suffered from a traumatic neurosis. Attached was a psychiatrist's report and counsel's affidavit that further proof would enable Messer to show a change of conditions and the existence of a mistake in the referee's

disability estimate.[1] The employer objected based on medical evidence that Messer's inability to work did not result from an organic cause. The Board found Messer's evidence to be inadequate and dismissed the motion.

Messer filed a subsequent motion to reopen, again based on change of conditions and mistake. He attached another psychiatrist's affidavit, which indicated that he was permanently and totally disabled due to post-traumatic encephalopathy, an organic brain disease that caused blackouts, severe memory loss, and impaired thinking and concentration. The employer's expert conceded that head trauma could precipitate or aggravate that type of disability. Although the Board found no change of condition or mistake in the initial award and viewed the motion as a belated attempt to submit evidence that should have been produced in the initial proceeding, the court disagreed. It acknowledged that Messer's actual malady and total disability might have existed since his accident but emphasized that the symptoms necessary to diagnose it did not become apparent until after the hearing. Thus, the court found the distinction between mistake and change of conditions to be insignificant in such circumstances and distinguished authority in which there was no change in the worker's condition. It concluded that both of the motions were proper and remanded the claim for further proof on the nature, cause, and extent of disability.

In Fayette County Board of Education v. Phillips, 439 S.W.2d 319 (Ky. 1969), the parties did not dispute that the defendant-employer had knowledge of Phillips' concurrent employment. Phillips failed to introduce any direct evidence of that fact, and the defendant-employer neglected to include the concurrent earnings when submitting

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1 At that time, KRS 342.125 permitted reopening for a "change of conditions" rather than a "change of disability."

average weekly wage information. On that basis, the "old" Board held that the defendant-employer did not know of the concurrent employment and refused to consider the concurrent wages when calculating Phillips' award.

Phillips filed a motion to reopen based on "mistake" and submitted evidence that the defendant-employer knew of the concurrent employment. But the Board dismissed it, reasoning that the "mistake" was Phillips' inadvertent failure to prove employer knowledge, which was not the type of mistake the statute contemplated. The court pointed out, however, that a "mistake" also occurred when the defendant-employer failed to apprise the Board that the wage it submitted did not include Phillips' concurrent earnings. It held that such a mistake warranted reopening and reversed.

Consistent with the principle of res judicata, subsequent decisions make it clear that the "mistake" provision is not an invitation to retry a litigated claim and that litigation must end when a decision becomes final unless extraordinary circumstances exist. Where the parties present conflicting evidence on a question of fact in the initial proceeding and a decision on the matter is final, subsequent evidence that the finding was mistaken does not show a "mistake" within the meaning of KRS 342.125. See Darnall v. Ziffrin Truck Lines, 484 S.W.2d 868 (Ky. 1972); Young v. Harris, 467 S.W.2d. 588 (Ky. 1971). Nor is such evidence the type of "very persuasive reason" to which the court referred in Slone v. R & S Mining, Inc., supra at 261.

In Durham v. Copley, supra, the insurance carrier obtained evidence before the claim was decided, which indicated that the work-related incident caused an injury of which Durham was unaware. Noting that the carrier failed to reveal the injury to opposing counsel until seven weeks after the claim was dismissed, the court permitted

a reopening on the ground of mistake in order to prevent what it considered to be a manifest injustice. This is not such a case. This is also not a case such as Wheatley v. Bryant Auto Service, 860 S.W.2d 767 (Ky. 1992), which involved an obvious mistake of law in awarding total disability benefits for 425 weeks rather than life. Likewise, this is not a case such as Messer v. Drees, supra, or Fayette County Board of Education v. Phillips, supra.

Causation was hotly contested in the initial claim. The parties offered conflicting evidence regarding the cause of Bassham's symptoms, including extensive evidence that a prion disease or some other non-work-related condition was the cause. Based on the evidence, the ALJ found that Bassham suffered from a work-related occupational disease. Under such circumstances, post-award evidence that the finding was mistaken did not show a "mistake" within the meaning of KRS 342.125. Thus, the ALJ did not err in concluding that the employer failed to make a prima facie case for reopening and reviewing the award.

The decision of the Court of Appeals is affirmed.

Lambert, C.J., and Abramson, Cunningham, Minton, Noble and Scott, JJ., concur. Schroder, J., dissents without opinion.

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