IN THE WORKERS' COMPENSATION COURT OF THE STATE OF MONTANA

2001 MTWCC 45

WCC No. 2000-0173

MARILEE DAVIS
Petitioner

vs.

INSURANCE COMPANY OF THE STATE OF PENNSYLVANIA
Respondent/Insurer for

WAL-MART STORES, INCORPORATED
Employer.

FINDINGS OF FACT, CONCLUSIONS OF LAW AND JUDGMENT

Summary: While working in the stationary department at Wal-Mart, claimant was injured when a paper shredder fell and struck the *left side* of her head. A CT head scan taken that day indicated no hemorrhage or lesions. However, she thereafter developed headaches, memory loss, unsteady gait, and other symptoms. Subsequent MRIs disclosed a mass on the right side of the brain which was larger with each subsequent MRI. Finally, approximately 18 months post-injury, claimant underwent brain surgery which disclosed an anaplastic astrocytoma, which is a glioma-type tumor. Although claimant's treating neurologists initially opined that the tumor was unrelated to her injury, during deposition they changed their opinions and testified that it was caused by the trauma. Claimant also called a non-treating neurosurgeon who testified similarly. However, another neurologist who had examined claimant, testified that there was no relationship between the trauma and the tumor. More importantly, Dr. Peter Burger, a neuropathologist on the faculty at Johns Hopkins, with 30 years experience in the field of brain tumors, testified on behalf of the insurer that that causation was not possible based on everything currently understood about glioma-type tumors, emphasizing in particular that brain tumors do not grow as quickly as would have been necessary in this case. He also testified there is no evidence suggesting trauma *impacts* or *accelerates* the growth of an existing tumor.

Held: Dr. Burger was far more qualified than all of the other medical witnesses in the area of brain tumors and their causation. His opinions were supported by medical literature and were persuasive.

Topics:

> **Benefits: Medical Benefits.** Claimant suffering from right-sided glioma-type brain tumor failed to persuade the Court her tumor was caused by work-related blow to the left side of her head, therefore she is not entitled to medical benefits for treatment of the tumor.

Causation: Medical Condition. Claimant suffering from right-sided glioma-type brain tumor failed to persuade the Court her tumor was caused by work-related blow to the left side of her head. The testimony of a tumor specialist, who was far more qualified than other testifying physicians, was persuasive that no link has been established between trauma and glioma-type brain tumors and that in any event glioma-type tumors develop slowly and could not have possibly developed in the short time in this case.

Evidence: Expert Testimony: Physicians. The testimony of tumor expert, with over 30 years experience and who is well versed in current medical research and literature as to cause of a brain tumor, is more persuasive than the testimony of far less qualified physicians.

Physicians: Conflicting Evidence. The testimony of tumor expert, with over 30 years experience and who is well versed in current medical research and literature as to cause of a brain tumor, is more persuasive than the testimony of far less qualified physicians.

¶1 The trial in this matter commenced on December 14, 2000, in Kalispell, Montana. Petitioner, Marilee Davis (claimant), was unable to be present but was represented by Mr. Garry D. Seaman. Respondent, Insurance Company of the State of Pennsylvania (Insurance of Pennsylvania), was represented by Mr. Donald R. Herndon. Through agreement of the parties, further telephone testimony was taken on January 25, 2001.

¶2 Exhibits: Exhibits 1 through 44, 46 and 47 were admitted without objection. Exhibit 45 was not offered.

¶3 Witnesses and Depositions: The parties agreed that the depositions of Robert Hollis, M.D., Ethan Russo, M.D., Bret D. Lindsay, M.D., and Douglas A. Nelson, M.D. shall be considered by the Court. Peter C. Burger, M.D., testified at trial on December 14, 2000. On January 25, 2001, Robert D. Schimpff, M.D., testified by telephone with the Court participating.

¶4 Issues: As rephrased by the Court, the issues are:

¶4A Whether the brain tumor leading to claimant's June 2000 surgery and responsible for her current medical condition and prognosis was caused or aggravated by the head injury she suffered on December 29, 1998, entitling her to benefits for medical expenses and disability.

¶4B Whether claimant is entitled to an award of costs and/or attorney fees.

¶5 Having considered the Pretrial Order, the testimony presented at trial, the demeanor and credibility of the witnesses, the depositions and exhibits, and the arguments of the parties, the Court makes the following:

FINDINGS OF FACT

¶6 On the morning of December 29, 1998, claimant was working as the manager of the stationary department at the Wal-Mart store in Kalispell. While she was moving paper shredders on a shelf, one fell and struck the left side of her head near her temple. (Ex. 1 at 1.)

¶7 Claimant reported the accident to her employer, which was insured by Insurance Company of the State of Pennsylvania. (*Id.*; Ex. 14 at 1.) A First Report of Injury was completed the day of the injury. Insurance of Pennsylvania accepted liability for the claim[(1)] but disputes liability for a brain tumor that was diagnosed a year-and-a-half after the injury. It alleges that the tumor is unrelated to the industrial accident.

¶8 On the day of her accident, claimant went to the emergency room of the Kalispell Regional Hospital. (Ex. 14 at 1.) She reported headache, disorientation, and difficulty walking (gait disturbance) but no nausea or vomiting. (*Id.*) A small lump was noted on her left temporal/parietal scalp but with no break in the skin. (*Id.*) She underwent an unenhanced CT brain scan which was reported as normal. (Ex. 17 at 1.)

¶9 On January 18, 1999, claimant visited Dr. Douglas A. Nelson, her internist. (Nelson Dep. at 5, 8; Ex. 10 at 6.) She reported:

> Persistent left temporal headache and tenderness to touch there. She noted she was not as mentally sharp as usual. Often lost her train of thought. Got, quote, spaced out, unquote, on sitting. Sometimes felt sleepy, sometimes had difficulty sleeping. She stated that she lacked much of the intuitive sense that she had had previously and had been more emotional than usual, crying or easily losing her temper more easily.
>
> (Nelson Dep. at 11.) Dr. Nelson characterized her as "more emotional than she had been previously." (*Id.*) His physical and mental examination disclosed "mild tenderness over her left temple and superior orbit region . . . where the box struck her head." (*Id.* at 11-12.) The only other abnormal finding "was on a Rhomberg test, which is a test of her balance, and she was quite unsteady with that and had a tendency to fall back and her gait was unsteady." (*Id.* at 12.) He diagnosed "a post-concussive syndrome/minimal brain injury, post blunt head trauma, with some associated deficits in her cognitive functions and neurologic functions." (*Id.* at 14.)

¶10 Dr. Nelson next saw claimant on February 15, 1999. He testified:

> Overall she felt she was improved since the prior visit. She was still having left temporal headaches occurring two to four times per week, which is somewhat less frequent than they had been previously. She was having what she called, quote, black out spells, unquote, which were brief, several second episodes, where she was unaware, was how she described it. When she would come to she would feel panicked and that she had been unaware. On average, these were occurring daily to every other day.
>
> She said that these episodes had not improved since her last visit. She denied at that point weakness, numbness, tingling, or focal neurologic symptoms.

(*Id*. at 15.) He noted claimant's "affect was less emotional and labile than it had been previously." (*Id*.) Her cognition appeared normal but no specific cognitive testing was performed. Dr. Nelson noted continued tenderness over her left temple and that Rhomberg testing and gait were "again somewhat unsteady and unstable." (*Id.* at 15-16.) His impression continued to be "post concussion syndrome/minimal brain injury." (*Id.* at 17.) He believed claimant was slowly recovering but ordered an electroencephalogram (EEG) to further evaluate her. (*Id.*)

¶11 The EEG was done on February 17, 1999, by Dr. Patrick J. Burns, a neurologist. It was abnormal:

> **IMPRESSION:** This is an abnormal EEG characterized by episodes of generalized dysrhythmic activity associated with hyperventilation as well as rare components of frontal sharp activity and focal left temporal slowing as well as occipital theta activity. This tracing is nondiagnostic in nature, but is highly suggestive of an underlying cerebral disturbance. The possibility of seizure disorder can not be excluded with this tracing alone and clinical correlation is advised. Other potential abnormalities would include vascular disease, degenerative processes and metabolic activity.

(Ex. 5 at 1.)

¶12 After reviewing the EEG report, Dr. Nelson considered it likely that claimant had a serious brain injury. He ordered another MRI, which was done on February 26, 1999. The MRI was read as normal by Dr. Michael B. Wickersham, a radiologist. (Nelson Dep. at 18; Ex. 17 at 2.)

¶13 Dr. Nelson next saw claimant on March 3, 1999. She reported:

> That her blackout spells were still occurring every other day to every day. She often - I believe she was back working at Wal-Mart at this point and she was doing inventory work and she noted that she was losing count when she was doing inventory work, which was atypical for her.
>
> She noted that she was having brief memory loss episodes occurring several times daily. Her friends thought that she was getting worse. She often lost her train of thought in the midst of conversation. Still complained of left temporal headaches and tenderness. And she did state specifically at that time that all of these symptoms dated specifically from the date of her head trauma.

(Nelson Dep. at 18-19.) His diagnosis "was still a post-concussive syndrome with minimal brain injury post her blunt head trauma." (*Id.* at 19.) However, given the abnormal EEG, he was concerned about the possibility of a seizure disorder. He referred claimant to Dr. Bret D. Lindsay, a neurologist on staff at the Kalispell Regional Hospital. (*Id.* at 20; Lindsay Dep. at 5.)

¶14 Dr. Lindsay first examined claimant on March 17, 1999. He reviewed the December 29, 1998 CT scan, which he read as normal. (Lindsay Dep. at 7.) He noted the EEG's indication of dysrhythmic activity was not specific for any particular brain disorder. (*Id.* at

8.) Finally, he reviewed the February 26, 1999 MRI. Although the radiologist had read the scan as normal, Dr. Lindsay observed "a small area of abnormality in the posterior temporal lobe on the **right** side; it was subtle but there." (*Id*. at 7, emphasis added.)

¶15 Dr. Lindsay discussed the MRI with Dr. Robert Hollis, a neurosurgeon. Dr. Hollis testified the MRI was also reviewed "at a neuroimaging conference that the neurologists, the neurosurgeon and other ancillary services have once a month." (Hollis Dep. at 8-9.) Their "feeling was, based on the preceding history of head injury, that it represented a contusion - - an old contusion at that point." (*Id.*) A biopsy was considered, but "it was felt that it most likely represented a contusion and that biopsy was not indicated and that Dr. Lindsay would follow her with serial imaging." (*Id.* at 10.)

¶16 Claimant was seen by Dr. Nelson on April 6, 1999. Dr. Nelson testified:

> Marilee stated at that point that she seemed more off balance intermittently, the headaches were somewhat worse, still often relieved with aspirin. Her off-balance sensation was worse when she would look up and her symptoms were worse when she worked a long shift, the 11:00 a.m. to 8:00 p.m. shift. Her balance difficulties worsened in that situation.

(Nelson Dep. at 21.) Dr. Nelson believed claimant's affect and cognition "grossly seemed normal," but he was concerned with her report of memory and cognitive deficits. (*Id.* at 22.) He recommended she continue seeing Dr. Lindsay. (*Id.*)

¶17 On June 15, 1999, claimant underwent neuropsychological testing by Edward H. Trontel, Ph.D. Dr. Trontel reported that the testing "suggested the presence of acquired cognitive difficulties against a background of superior intelligence." (Ex. 2 at 5.) Problems included persistent disruption of working memory, object recognition, and "the emergence of phonemic paraphasias and greater attentional and working memory problems." (*Id.*) He diagnosed a cognitive disorder, citing "disrupted working memory and subtle object recognition problems." (*Id.*) Dr. Trontel also recorded claimant's report of emotional incontinence, diminished taste, and altered sleep patterns. (*Id.*) In his summary of Dr. Trontel's oral report to him, Dr. Lindsey noted that the dysfunction was in the left hemisphere, however, Dr. Trontel's written report does not distinguish between left and right hemispheric function. (*See also* ¶19.)

¶18 Another MRI was done June 17, 1999. Dr. Lindsay read the MRI as showing a subtle lesion in the right posterior temporal lobe. (Lindsay Dep. at 9.) He reported:

> When compared to the prior study of 2/26/99 there has been a slight, equivocal interval increase in the size of this lesion and it is better appreciated. Appearance is nonspecific but differential would include post-traumatic lesion, sequella of ischemia, inflammation and low-grade glioma. Clinical correlation is warranted and if indicated a repeat scan at the appropriate interval would be helpful in further evaluatin [sic] this lesion. No appreciable change in the size of this lesion.

(Ex. 3 at 13; Hollis Dep. at 12.)

¶19 A repeat EEG was done on June 28th, which Dr. Lindsey characterized as "borderline in that it did demonstrate some generalized slowing in the background rhythm characterized as occipital theta activity, but there was no evidence of frontal sharp wave activity or focal left temporal slowing as her previous EEG had demonstrated." (Ex. 3 at 16.)

¶20 In late June 1999, claimant ceased working at Dr. Lindsay's recommendation. (*Id.* at 15.)

¶21 Dr. Lindsay continued to monitor claimant. On August 11, 1999, and September 9, 1999, she reported that she was not improving. A trial of the drug Neurontin helped her headaches, but provided only temporary assistance with her other symptoms. (*Id.* at 19.)

¶22 In October 1999, Dr. Lindsay ordered a third MRI, which he read as showing a "subtle increase in size [of the lesion] over the interval and certainly, when compared to her first MRI scan of 2/26/99, there has been significant increase in size." (*Id.* at 21.) Dr. Lindsay was "suspicious that this likely represents low grade astrocytoma or oligodendroglioma given the interval increase in size over the past ten months." (*Id.*)

¶23 Dr. Lindsay reviewed all three MRI scans with Drs. Hollis, Sorini, and Martini. (*Id.* at 22.) He summarized:

> The concern is that the right posterior temporal lesion appears to be growing in size subtly over the past eight months. The two primary differentials would be evolving contusion secondary to trauma and slowly growing glioma such as a low grade astrocytoma or low grade oligodendroglioma. Neither of the two neurosurgeons feel that biopsy or resection would be indicated yet as the lesion is quite small and it has demonstrated only very mild changes over the interval. We have hoped to obtain a functional scan either with SPECT or PET to help elucidate whether this is an area of encephalomalacia or if this represents neoplasm and the patient will be referred to Dr. Lynne Taylor in Seattle for a second opinion from a neuro-oncologist and, hopefully, functional scanning. Additionally, I will have her follow-up with Dr. Hollis for review of her case as well.

(Ex. 3 at 22.)

¶24 Claimant was examined by Dr. Robert Hollis, a neurosurgeon, on October 28, 1999. (Hollis Dep. at 6.) On October 28, 1999, Dr. Hollis wrote to Dr. Lindsay noting the following "Impression/Plan":

> Closed head injury, post-concussive syndrome and intercerebral mass (? [questionable] Tumor vs. contusion). Through your office endeavoring to set up an appointment for her, she will go to Seattle for functional studies. If this proves to be a mass, then we will proceed with open biopsy/resection. If this proves to be a "cold" lesion then we will continue to watch it. If it is equivocal, I told her we would continue to watch.

(Hollis Dep. Exhibits.)

¶25 On November 10, 1999, claimant was seen by Dr. Lynne Taylor in Seattle. (Ex. 19 at 1.) Dr. Taylor took claimant's history, including her report that "cognitive disruptions have increased rather than decreased." (*Id*.) On review of the diagnostic studies, Dr. Taylor observed the "vague area of T2 abnormality in the posterior aspects of the right temporal lobe," but stated "it is not at all clear to me that it is actually growing in overall size." (*Id.* at 3.) Because the area was either stable or possibly slightly enlarging, Dr. Taylor noted "**post traumatic change is felt to be less likely, as that would produce some atrophy over time**." (*Id.*, emphasis added.) She considered differential possibilities to include "low-grade glioma, cortical tuber, or post-traumatic change." (*Id.*)

¶26 A SPECT scan was performed and reviewed by Dr. Taylor, who found the study "entirely unremarkable" and, ironically, "confidently excluded a malignant neoplasm or anaplastic astrocytoma . . . ." (*Id.* at 3-4.)

¶27 Dr. Lindsay then recommended repeat neuropsychological testing with Dr. Trontel. (Ex. 3 at 25; Lindsay Dep. at 11.) Dr. Trontel examined claimant in February of 2000 and found improvement in "cognitive control, working memory, and 'sequencing'." (Ex. 2 at 10.) However, he also found "[l]ower than expected scores on tests sensitive to nonverbal memory and spatial-relational processing persisted, but she could not be said to be functionally impaired." (*Id*.) He noted other impairments - nonverbal memory and spatial relation - which he did not relate to claimant's head injury. These impairments persisted and were considered of "unknown etiology." (*Id*. at 11.)

¶28 During March 2000, an independent medical examination (IME) was conducted by Dr. Dana Headapohl, who specializes in Occupational Medicine, Dr. Robert Velin, a Clinical Psychologist, and Dr. Ethan Russo, a board certified specialist in neurology. (Ex. 6; Russo Dep. at 5.) The panel's impressions included:

> a. History of closed head injury with post traumatic headaches and reported cognitive impairment.
>
> b. Poorly circumscribed non-enhancing lesion in the right temporal parietal lobe just superior to the tentorium, of unknown etiology. Available evidence points away from an injury effect, given the lack of surrounding atrophy and slight interval increase in size. A repeat MRI is pending.

(Ex. 6 at 4.) The panel opined that the "right temporal lobe mass was not likely caused by the 12/29/98 injury," but suggested further MRI to clarify "whether or not the findings are more consistent with post trauma findings or a temporal lobe mass." (*Id.* at 5.) The panel placed claimant at maximum medical healing from her head trauma. (*Id.*)

¶29 A fourth MRI was done May 31, 2000. Dr. Lindsay noted a "[f]airly dramatic increase in size in the lesion in the right temporal lobe compared with the prior study." (Lindsay Dep. at 14.) He felt the "lesion almost undoubtedly represents a low grade glioma." (Ex. 3 at 34.)

Dr. Lindsay and claimant discussed her industrial accident as the possible cause of the tumor:

> Marilee reports significant change in her neurologic symptoms with more difficulty with sequencing, disequilibrium and right temporal headaches. **She does inquire with regard to the coincidence of head trauma and occurrence of her tumor and I discussed that I felt cause and effect were unlikely.**

(*Id.*, emphasis added.)

¶30 On June 23, 2000, Dr. Hollis operated, removing the tumor. (Ex. 18 at 4-6; Hollis Dep. at 14.) Pathology reported the tumor as an anaplastic astrocytoma. (Lindsey Dep at 14; Hollis Dep. at 14.) Dr. Hollis explained that this type of tumor is "an intermediate grade primary glial cell tumor which does not exhibit frank malignancy but does possess an intermediate clinical course and does exhibit brain invasiveness." (Hollis Dep. at 15.)

¶31 Claimant has treated with Dr. Lindsay following surgery. (Ex. 3 at 38-39.) She has undergone radiation and chemotherapy, as well as physical therapy. (*Id.*) Tragically, her prognosis is poor.

¶32 The issue raised by the claimant's petition is whether her tumor was caused or materially aggravated by her December 29, 1998 industrial accident. The record contains conflicting medical opinions regarding the issue.

¶33 The expertise of each of the physicians who rendered an opinion concerning causation is important to determining whose testimony the Court should give the most weight. I therefore preface my survey of each physician's opinions with a summary of his medical qualifications, beginning with Dr. Lindsay.

Dr. Lindsay

¶34 Dr. Lindsay, as already noted, is a neurologist. He studied medicine at the University of Washington, then did his internship at Virginia Mason in Seattle, a neurologic residency at Stanford, and finally a neuroimaging fellowship in New York. He has been in medical practice in Kalispell for four years. He is not board certified in neurology but has passed the first part of the board test and was scheduled to take the second part of the test in January 2001. (Lindsay Dep. at 5-6.)

¶35 As noted in paragraph 29, prior to the taking of his deposition Dr. Lindsay opined that it was unlikely claimant's tumor was related to her head trauma. On June 20, 2000, he wrote a note to the insurer, saying, "I agree [with] IME; need for surgery not related to injury of December 29, 1998." (Lindsay Dep. Ex. 1 at 75.)

¶36 By the time of his deposition on October 12, 2000, Dr. Lindsay had changed his mind. He testified that "based on the temporal occurrence of the tumor and its rate of progression, and based on my understanding of the literature, I would say that it is more likely than not related to the trauma." (Lindsay Dep. at 15.) He explained what he meant by "temporal relationship:"

> The fact that the lesion appeared to grow at a relatively slow rate initially and that growth rate would have been commensurate with beginning around the time of the trauma, so that one could easily suggest that the tumor or the neoplasm

began growing around the time of the trauma, based on the evolution of the lesion on serial MRI scans.

(*Id*.) Dr. Lindsay's "impression of the imaging is that her tumor began growing right around the time that she incurred trauma." (*Id.* at 27-28.) He agreed he could not exclude the possibility that the tumor pre-existed the trauma. He testified: "I'm saying that 51 percent of my opinion lies with the fact that they are temporally and causally related and 49 percent, which is a large percent, suggests that they could not be. I don't know." (*Id.* at 29.)

¶37 After claimant brought him some Internet articles, he did his own literature review, primarily over the Internet. (*Id*. at 19-20.) He claimed to have "reviewed various articles that suggested a correlation between trauma and the occurrence of malignant transformation in glial cells." (*Id.* at 20.) When asked if he saved his research, Dr. Lindsay testified that he had not. (*Id*. at 20.) When asked if he could identify the articles, Dr. Lindsay testified: "You know, I don't specifically remember the articles, you know, per the authors' names, the studies' names. I don't think I would be able to identify them specifically." (*Id.*) He also did not retain the articles claimant showed him. (*Id.* at 20-21.) He conceded his research on the issue of causation of glial-type tumors was not exhaustive. (*Id.* at 33.)

Dr. Hollis

¶38 Dr. Hollis studied medicine at the University of Illinois at Chicago from 1988 to 1992, did his internship at that school's hospital from 1992 to 1993, and completed neurosurgery training at Loyola University in Chicago in 1998. (Hollis Dep. at 5.) He has since practiced in Kalispell. (*Id*. at 5.) At the time of his deposition, he was board eligible in neurosurgery, but had not completed a two-year practice requirement which is prerequisite to board certification. (*Id.* at 6.)

¶39 At deposition, Dr. Hollis opined "that it is more probable than not that [claimant's] tumor was induced by her head injury." (*Id*. at 15.) His opinion was based on the timing of the tumor, clinical symptoms, the imaging studies, and his research of medical literature. (*Id.* at 24.)

¶40 As did Dr. Lindsay, Dr. Hollis gave great weight to the temporal relationship of the tumor to the trauma, i.e., to the fact that the tumor and her symptoms appeared to arise coincidentally with the industrial accident. He believed that the tumor and claimant's clinical symptoms both arose *after* claimant's December 1998 head trauma. (*Id*. at 21-22.) .

¶41 As to his literature review, he testified: that he did "a review of the medical literature to find some anecdotal evidence of this phenomenon happening." (*Id.* at 24.) When asked to identify the medical literature on which he relied, he responded, "There are a few citations from the British neurosurgical journals as well as the Italian neurosurgical journals that have had anecdotal cases and review of cases which appear to correlate head injury and the development of primary intra-axial glial tumors." (*Id.* at 15-16.) However, he agreed that no "clear-cut association" has been formed between trauma

and glial-type tumors (*id.* at 20), which include anaplastic astrocytoma tumors. He testified:

> Right now we have only citations in the literature that suggest the possibility of development. Some of our older neurosurgeons that are no longer with us felt that relationship was stronger than has now been - that is now felt to be represented.

(*Id.*) He agreed that "the causal connection by medical research seems to be weaker than it was thought to be in the past." (*Id.*)

¶42 When asked for citations to the literature, he stated: "All of these papers can be found in PubMed, which is an Internet based search site which searches Medline, PremedLine, and other ancillaries, such as radiation oncology and occupational health sites." (*Id.* at 25.) He testified to finding "about four or five citations," none of which he had preserved. (*Id.*) Most of his research had been done on the day of the deposition, although he said he had conducted some research earlier in response to claimant's insistence there was a connection. (*Id.* at 26.)

Dr. Nelson

¶43 Dr. Nelson is board certified in internal medicine and pediatrics. He has practiced medicine in Kalispell for over eight years. He did not express an opinion but indicated that there was

> nothing in my earlier training in internal medicine and pediatrics to suggest that malignant gliomas occurred as a result of traumatic injuries.

(Nelson Dep. at 39.)

Dr. Russo

¶44 Dr. Russo is a board certified neuorologist. There is nothing in the record to indicate that he has specialized training in glioma-type tumors.

¶45 Dr. Russo opined that claimant's tumor was not related to the head trauma. (Russo Dep. at 21, 38-39.) When asked whether medical science knew the causes of glioma-type tumors, Dr. Russo testified, "Epidemiologically, we don't know of a relationship on an etiologic basis that tells us that trauma makes a glioma where there was not one before. But best evidence, in fact, is against it." (*Id.* at 25.) He acknowledged that "there's a possibility" that head trauma led to claimant's tumor, "but our best understanding is that traumatic closed head injuries don't produce malignant deterioration." (*Id.* at 39; and *see Id.* at 37.)

¶46 He suspected that claimant's lesion had been present for some period of time prior to the head trauma. (*Id.* at 38.) While he agreed that the CT scan taken just after the injury did not show a tumor, he pointed out that CT scans are primarily used to rule out bleeding following head trauma and "just doesn't show this kind of thing [tumor] as well

as an MRI scan does." (*Id*. at 24.) The fact that the CT scan did not disclose a tumor "doesn't mean it wasn't there." (*Id*.)

Dr. Robert D. Schimpff

¶47 Dr. Robert D. Schimpff is board certified in neurology and neuropathology. (Transcript of Proceedings dated January 25, 2001, hereinafter Tr. II, at 38.) His education includes "three years of fellowship in neurology," followed by "two years of neuropathology fellowship and concurrent with that three years of tumor biology" in the late 1960s and early 1970s. (*Id*. at 6.) During the 1970s, Dr. Schimpff was on the faculty at the University of Florida, where he was jointly appointed to the Department of Pathology and the Department of Neurology. He served as a neuropathologist there from 1972 through 1975, then as chief of neuropathology from 1975 through 1979. In 1979 he left academic medicine to begin a neurology practice in Kalispell, where he still practices. (*Id.*)

¶48 While on the faculty of the University of Florida in the 1970s, Dr. Schimpff did cancer research, the exact nature of which was not disclosed. (*Id.* at 37.) He has not done research since he left the University of Florida in 1979 and agrees that cancer research has progressed rapidly since that time. (*Id.*) He has not had a medical article published since the 1970s. (*Id.* at 38.)

¶49 In preparation for his testimony, Dr. Schimpff reviewed claimant's medical records, imaging studies, EEGs, pathology slides, and the "medical literature with regard to the relationship of trauma to brain tumors, in general gliomas, specifically meningiomas." (*Id.* at 7, 11.) He also reviewed the testimony of Dr. Hollis, Dr. Lindsay, and Dr. Burger. (*Id.* at 8.)

¶50 Based on the EEGs and his review of medical records, Dr. Schimpff believed claimant

> was suffering from a condition called complex partial seizures, and that her medical complaints of blackouts and disturbed cognitive function occurring periodically were secondary to abnormal electrical activity which was being generated in her brain passing through a portion of the brain and causing disruption of her cognitive processes.

(*Id.* at 16.) Dr. Schimpff testified this condition was "known to be a possible sequella of head injuries, and ultimately she was treated with an anticonvulsant by Dr. Lindsay." (*Id.*) He opined that the blow to claimant's head was substantial and "played a definite role in the subsequent medical difficulty which I've termed complex partial seizures." (*Id.* at 11, 17.)

¶51 Dr. Schimpff noted that claimant's symptoms "happened very proximally to the occurrence of the head injury" but testified it was unlikely that the tumor, "generate[d] a complex partial seizure." (*Id.* at 87.) According to Dr. Schimpff, the complex partial seizure disorder is a separate condition from the claimant's tumor, however, in his opinion, claimant's head injury caused "the development of a complex partial seizure disorder" and her need for treatment and was also "related to the genesis of this tumor."

(*Id.* at 30.) He testified that claimant's disability is due to both the tumor and the complex partial seizure disorder. (*Id.* at 30-31.)

¶52 He also believed it was not clear that the abnormality on the February 1999 MRI was then an incipient tumor (*id.* at 64), as it "can be associated with a lot of different types of pathology, and the scans are in no way explicit with regard to what is causing that." (*Id.* at 25.) He thought it possible that it was evidence of injury from trauma, with the tumor developing later.

¶53 Dr. Schimpff discussed medical literature concerning the relationship between glioma-type brain tumors and trauma. According to the doctor, the literature contains anecdotal case reports of trauma inducting brain tumors and epidemiological evidence either showing no apparent association between trauma and gliomas or a weak association. (*Id.* at 21-22.) He also said that there is epidemiological evidence which appears to show an association between trauma and meningiomas. (*Id.)* Claimant, of course, had a glioma-type tumor, not a meningioma.

¶54 Dr. Schimpff articulated a *possible* mechanism by which trauma might cause the emergence of a glioma.

> [W]hen a person has an injury to the brain, it can result in a type of scar, and the type of scar which occurs in the brain can be associated with a condition called gliosis.
>
> And to try and keep it simple, gliosis simply refers to a reactive change which occurs in the astrocytes of the brain or other glial cells in the brain, and that reactive change is a change in the biology of the cells. The glial cells, the astrocytes, are sitting in the brain carrying out certain basic functions, but in the face of trauma, their biology changes. They can reproduce; that is, they can undergo cell division. There's all types of experimental work which shows that there's a change in the types of microbiologic substances which they produce. There can be change in growth factors. There can be a change in the type of metabolic activity which these cells undergo.
>
> Because there is this reactive change which can occur as a result of traumatic injury, people have taken that one step further and said, "Gee, during this reactive change that occurs in these cells, could a certain percentage of patients, a small percentage or a subpopulation of patients, then go on to the development of uncontrolled cell division of the type which would then pathologically be identifiable?"
>
> So that's sort of - I hope I didn't digress too much, but that's sort of a basic thinking which has gone on and been reported in the literature. That's the kind of thinking which I've indulged in thinking about this case, about the possibility that a reactive astrocytosis, secondary to a blow to the head and injury to the brain, could then, in a subpopulation of people, go on to establish actually a tumor.

(*Id.* at 23-24; and *see id.* at 32-33.)

¶55 As for why epidemiological studies have not linked trauma to glioma-type brain tumors, Dr. Schimpff noted that if trauma causes tumors only in a subpopulation who have another specific trait, then looking at the entire population of people with trauma would not prove the connection. (*Id.* at 28.) As an example, he noted that looking at the entire population of women taking birth control pills may not show a statistically significant link to strokes, but if the population of women taking birth control pills who smoke is examined, the link may be shown. (*Id.* at 27-28.) For that reason, Dr. Schimpff believed that epidemiological studies "are a weak tool in this regard." (*Id.* at 32.)

¶56 Cross-examination undermined Dr. Schimpff's opinions. He agreed that a CT scan is less sensitive than an MRI and that it was possible that if a tumor was present in December 1998 the CT scan would not have detected it. (*Id.* at 40.) He conceded that most of the anecdotal cases in which authors suggest a link between trauma and the development of a glioma-type tumor involved "long latency periods," but he did not believe this ruled out the possibility of a short latency period as in claimant's case. (*Id.* at 61.)

¶57 He was specifically cross-examined about seven medical articles he said he reviewed in formulating his opinions. He testified that these were not inclusive in that he had read other articles over the years on the subject, however, he did not identify the articles and therefore could not be cross-examined about them. (*Id.* at 45-46.) As to the seven articles, which were introduced as Exhibits 49A through 49G, the following was pointed out through cross-examination:

> ¶57A Exhibit 49A is a 2000 journal article of an anecdotal report of an astrocytoma associated with an area of gliosisis secondary to a head injury, however, the latency period, i.e., the period between the head injury and discovery of the tumor was 19 years. (*Id*. at 52.) Dr. Schimpff also pointed out in his testimony that the author of the article concluded that the head injury was "incidental rather than [a] causative finding" but then criticized the author's finding by saying, "he did not convince me that he had a strong position in that regard." (*Id*. at 50.) Also, there is no evidence of gliosisis in the area of claimant's tumor. (See ¶ 67.)
>
> ¶57B Exhibit 49B is a 1998 anecdotal report of a case of a post-traumatic glioma. The latency period was two years (the tumor appearing two years after prior surgery which disclosed no tumor at the time). (*Id*. at 52, 56-57.) Dr. Schimpff was asked what in the article supported his opinion in this case and he referred to two things. The first was the introductory statement, "Trauma and scarring have been known to be predisposing factors for the development of meningiomas." (*Id.* at 53.) When counsel pointed out that the present case does not involve a meningioma, the doctor pointed out that the fact that there is a relationship between one type of brain tumor and trauma raises the possibility that a relationship could exist with respect to another type. (*Id*. at 53-54.) The second was reference in the article to a German experiment on rats which suggested "that under certain conditions cranial trauma may act as a cocarcinogen and enhance the rate of glioma formation in rats exposed to a potent carcinogen." (*Id*. at 54.) Dr. Schimpff had not read the German study and was relying on the report of it in the article in question.

¶57C Exhibit 47C is a 1989 journal article reporting an epidemiological study into risk factors for glioma. The study actually supports the proposition that there is no association between trauma and gliomas. It concludes "that head injury appeared **not** to be associated with glioma risk." (*Id*. at 58-59, emphasis added.) Dr. Schimpff, however, felt that the article overall supported his opinion because the mere fact of the study points out that there may be groups of people who on account of some exposure or biological susceptibility may be "susceptible to trauma as a trigger for glioma." (*Id*. at 58.) Dr. Schimpff's reasoning is illogical. The study, as he concedes, fails to find any relationship between the risk factors and subgroups identified by the authors and gliomas. The fact that some associative risk factors not identified in the study "might exist" is not proof that such risk factors in fact exist, it is merely an acknowledgment that lots of things are possible, or, as many experts concede in response to not-so-clever cross-examination, "Anything may be possible." The doctor's sloppy thinking, as manifested in his analysis of this article, is a factor I have considered in resolving the conflicting opinions rendered in this case.

¶57D Exhibit 47D is another epidemiological study published in 1994, however, Dr. Schimpff conceded that it did not find a correlation between head trauma and gliomas. (*Id.* at 66-67.) The doctor argued that the finding "doesn't mean . . . that those things do not play a role." *(Id.* at 67.) I am not impressed by his logic in this regard: The epidemiological finding, no matter how you twist it, does **not support** a conclusion that head trauma can cause a glioma. Dr. Schimpff also testified that he relied on a citation by the authors at page 65 of their article (Ex. 47D at 5) to another paper which found a positive association between head trauma and glioblastomas. (*Id.* at 66.) His testimony is, quite frankly, astonishing. The authors specifically state that "most studies have failed to support this finding " and "[n]o such association emerged in our series either . . . ." (Ex. 47D at 5.) The passage reads in its entirety:

> A positive association between head trauma and glioblastomas was reported by Hochberg, but most studies have failed to support this finding [Choi 19, Annegers 20, Kurland 21, Codd 7]. No such association emerged in our series either, whether cerebral gliomas were considered as a whole or evaluated separately according to their malignancy; nor was any correlation found when mild traumas were distinguished from severe lesions.

(*Id*.)

¶57E Exhibit 47E is an article published in the late 1920s or early 1930s about the possible relationship between trauma and brain gliomas. (*Id.* at 77.) Dr. Schimpff acknowledged that the author concluded that he could not say that trauma caused tumors, but noted that the article cited "several people" who had "formed opinions that trauma could be the initiating factor of cerebral tumors." (*Id*. at 78.)

¶57F Exhibit G added nothing since it was the same article as Exhibit B, thus Dr. Schimpff referenced only six articles.

Dr. Peter C. Burger

¶58 Dr. Peter C. Burger is board certified in neuropathology and anatomic pathology. His training included "a combined neuropathology fellowship and anatomic pathology residency at Duke University in North Carolina." (Transcript of December 14, 2000 Proceedings, hereinafter "Tr. I," at 14.) Since 1970 he has held academic positions involving research on brain tumors. (*Id.* at 14-15.) After residency, Dr. Burger became a faculty member at the Department of Pathology at Duke University, (*Id.* at 15.) He is currently at Johns Hopkins in Baltimore, Maryland, where he holds a regular professorship in the Department of Pathology, along with a joint professorship in the Oncology and Neurosurgery Departments. (*Id.* at 16.) Dr. Burger currently teaches pathology to medical students, medical residents, and other medical clinicians. (*Id.*) His research at Johns Hopkins has focused on the genetics of brain tumors, including the "causation of brain tumors in regard to radiation injury, and we have looked at attempts to prognosticate brain tumors and the basis of their genetic - - molecular genetic changes." (*Id*.) He has been "studying gliomas since 1969 or 1970. It's probably the thing I've spent the most time studying and writing about." (*Id.* at 22.)

¶59 Dr. Burger is on the editorial board or on call for the editorial board of several medical journals, including *Surgical Pathology, Surgical Neurology, Journal of Neuropathology & Experimental Neurology,* and *Brain Pathology*. (*Id.* at 18; Ex. 46.) His curriculum vitae lists 247 published peer-reviewed articles, with another several awaiting publication. (Ex. 46.) He co-authored two books used widely in the field of neuropathology, *Surgical Pathology of the Nervous System and Its Coverings* and *Tumors of the Central Nervous System*. (*Id.* at 21; Ex. 46.) He is working on the Fourth Edition to *Surgical Pathology* and will also work on a new edition of *Tumors of the Nervous System*. (*Id.* at 21.) Dr. Burger testified both books are standard works in the field, meaning one would expect to find them in the office of any pathologist in the United States. (*Id.*) Many of the articles, books, and abstracts that appear in his curriculum vitae deal with glioma-type tumors. (*Id.*)

¶60 Respondent requested Dr. Burger to review the causation issue and testify at trial. In arriving at his opinions, the doctor reviewed claimant's medical records, examined the histologic sections of claimant's tumor, reviewed claimant's various MRI and CT scans, along with the reports of the scans, and read the depositions of Drs. Hollis and Lindsay. (*Id.* at 23-25.) He also listened to the testimony of Dr. Schimpff. Finally, he conducted a medical literature review, first looking through his own files on the subject, then the computerized database of the National Library of Medicine. (*Id.* at 25.) He searched for articles describing the relationship, if any, between trauma and brain tumors. (*Id.* at 26.)

¶61 Dr. Burger testified unequivocally that the claimant's tumor was not causally related to her head trauma. His opinions were based not only on his review of medical literature but also upon his extensive personal research and study.

¶62 He found and reviewed several articles on the subject. He summarized them as follows:

> Well, the articles can be summarized by saying that there are occasional cases where there is a patient that has a brain tumor that has a history of trauma, and

> these articles that were written would attempt to determine whether this was causally related. My review of the literature convinced me, which I already believed anyway, that these two things were not related in any way, shape or form. It's pure coincidence.

(Tr. I at 26.) He acknowledged that some of the articles claim there is a relationship between trauma and glioma tumors but, in his opinion, the articles failed to prove the assertion, while "most of the articles come to just the opposite conclusion." (*Id.* at 43.) When recalled to testify after the testimony of Dr. Schimpff (see below), Dr. Burger pointed out that not one of the articles referenced by Dr. Schimpff supported a causal connection. (Tr. I at 93.)

¶63 Dr. Burger explained why anecdotal case reports of tumors following trauma do not support a conclusion that there is a cause and effect relationship. "Obviously, trauma is not uncommon, and there will be some patients with brain tumors that, by chance, will have had a traumatic episode in the past. That, statistically, has to happen. There's nothing to suggest that this is anything other than just pure chance." (Tr. I at 28.) Dr. Burger also testified there is no evidence suggesting trauma *impacts or accelerates* the growth of an existing tumor. (*Id.* at 63.)

¶64 He acknowledged that some medical articles raise the "possibility" of an association between trauma and brain tumors, but nothing more than a possibility. (*Id.* at 28.) Moreover, he testified that when association between trauma and brain tumors has been hypothesized, it was "generally felt that it takes years or decades for this effect, if there is such a thing, which I don't think there is, to exert its effect, and, in accord, the concept of cancers as induced by something is that it takes years or decades." (*Id.* at 29.) Dr. Burger noted that cancers developing from carcinogens, for instance, take decades to develop. (*Id.*)

¶65 Dr. Burger's own tumor research indicates there is a relationship between radiation and tumors such that "you can be reasonably convinced that in some patients, although a minority, that it does induce the tumor." (*Id.* at 30.) However, the average length of time between radiation and diagnosis of tumor is ten years. He testified: "It may be as low as five, but, generally, 8, 9, 10 years or 20 years." (*Id.*) He then pointed out that physical trauma may disrupt cells, but does not interfere with the basic biology of a cell in the way that radiation and other cancer-inducing exposures are known to do. (*Id.* at 31.)

¶66 From his review of MRI scans in this case, Dr. Burger opined that claimant's glioma was clearly present in the February 1999 scan. (*Id.* at 32.) Based on that scan alone, he agreed that it could not initially have been identified as anything more than an abnormality, but given the subsequent images, and the actual tumor removal, Dr. Burger was "quite convinced that that was a tumor when it was visualized in February." (*Id.* at 33.)

¶67 Dr. Burger unequivocally opined that neither the "lesion" depicted on the February 1999 MRI nor the pathological slides ultimately prove a tumor had anything to do with trauma. He stated: "The sections don't show trauma. The lesion doesn't look like trauma

on the film, and it gets gradually bigger and ends up a brain tumor. I think it [the trauma] is irrelevant." (*Id.* at 66.)

¶68 Importantly, Dr. Burger testified it was not medically possible -- "Not by anything we understand about the genesis of tumors in humans" -- for the claimant's tumor to have developed between the time of her injury and the February 1999 scan because there was simply not enough time involved. (*Id.* at 32.) He testified, "I think I would say it's impossible . . . ." (*Id.* at 33.) He explained further:

> The concept is that, **in brain tumors, like many others, by the time you see it on a scan, it's been there for years. It takes a long time for tumors to get to a certain stage.**
>
> This has been studied mathematically. If you take two cells, which are microscopic, and double them every so often, for a long time it doesn't get to be anything. Once it gets to be a certain size, doubling something that big becomes quite significant. When you see something, it's not like it just all of a sudden showed up. It's been there for awhile. When they first present, you're seeing things which, in a way, are in the late stages of the development.

(*Id.* at 67, emphasis added.) Although it is not known how long it takes for glioma-type tumors to present on an MRI, he opined it would take at least a year in the case of the most rapidly growing tumor and, in some, probably five years. (*Id.* at 68.) He believed growth in the period of two months was not possible. (*Id.* at 69.) Dr. Burger later conceded that as a scientist, "I have to say anything is possible," but he continued: "I don't think it is possible, but I have to grant you that in biology things are complex, and maybe we don't know everything we think we do, but by all of the standards, there's no reason to suspect that [trauma] would produce brain tumors." (*Id.* at 48.)

¶69 When asked why claimant's tumor did not appear on the CT scan taken just after the injury, Dr. Burger testified:

> It's actually very easy to explain. The CT scan is a much less sensitive test than MR by far. You would not expect to see this lesion on the CT when it was seen later in February. CT by today's MR standards is a pretty crude test. You cannot say that this was not there in December because it wasn't seen by CT. It wasn't a sensitive test . . . CTs are done for emergency, often for emergency purposes for head trauma, things like that.

(*Id.* at 69-70.)

¶70 On cross-examination, Dr. Burger was asked to review a synopsis of an article purporting to argue a relationship between trauma and brain tumors. Dr. Burger commented: "They're presenting a single case of anaplastic astrocytoma in a patient that had a head injury 19 years earlier, and the claim is that this meets accepted criteria for brain tumor associated with head trauma." (*Id.* at 46.) In the doctor's opinion, this was merely a case with "both things, but they're not correlated." (*Id.* at 47.) In addition, in that case, "[i]t's 19 years later." (*Id.*)

¶71 After the testimony of Dr. Schimpff, Dr. Burger was asked about Dr. Schimpff's retrospective diagnosis of claimant as suffering from complex partial seizure syndrome. Dr. Burger noted he was a pathologist, not a neurologist, stating:

> But on patients having complex partial seizure as an expression of a tumor, I suppose trauma could do that. Some patients, it's never clear what the cause of it is, but it's a little bit out of my area of speciality.

(Tr. II at 94.)

Resolution

¶72 I am **not** persuaded that claimant's brain tumor was caused by the head injury she suffered at Wal-Mart. Initially, I found the testimony of Drs. Lindsay and Hollis wholly unpersuasive. They both lacked any particular expertise concerning glioma-type tumors. Moreover, their literature review appeared cursory and they did not bother to print out or save their results. I was also unpersuaded by Dr. Schimpff's testimony. While he did some cancer research in the 1970s, he has been out of that field for more than 20 years and has been out of academia for the same time period. He has authored no articles in over 20 years and the medical literature he identified as supporting his opinions was limited to six articles which were either anecdotal or actually concluded that there is no proven link between trauma and glioma tumors. Dr. Schimpff's interpretation of two of the articles as supporting a relationship (see paragraphs 70C and 70D) strained credulity and undermined his credibility. Ultimately, it was clear to me that Dr. Schimpff failed to appreciate the standard for opinion testimony, confusing what is medically "probable" with what is medically "possible."

¶73 By far, the most persuasive testimony was that of Dr. Burger. Dr. Burger had far greater expertise than any of the other doctors, indeed he has decades of experience in tumor research, including gliomas, and has published extensively in the field. His testimony concerning the prevalent medical opinion regarding trauma and gliomas was persuasive; he had far greater familiarity with the medical literature on the subject and his interpretation of the articles which Dr. Schimpff said he relied upon is more consistent with the articles than was Dr. Schimpff's interpretation. But of even greater significance was his testimony concerning the latency period for gliomas, i.e., the time it takes for a glioma to develop into a detectable mass. He was unequivocal in stating that the claimant's glioma manifested itself far too quickly for it to have been triggered by trauma. The fact that the December 1998 CT scan did not disclose a tumor or other lesion does not undermine his opinion: Dr. Burger was not the only physician who pointed out that the CT scan is not as sensitive to tumors as is MRI and that the tumor could have been present but not detected by the scan. (*See* Drs. Russo and Schimpff testimony, ¶¶ 46, 56.) His analysis of the pathology slides from the tumor site disclosed no evidence of traumatic injury or lesion, thus undermining Dr. Schimpff's theory that the glioma could have developed from scar tissue caused by the trauma. Moreover, Dr. Burger testified that the February 1999 abnormality on the MRI "doesn't look like trauma on the film, and it gets bigger and ends up a brain tumor." (Tr. I at 66.) Finally, Dr. Burger also indicated that there is no credible scientific support for the notion that trauma *accelerates* the growth of a tumor.

¶74 The best case for a causal connection between the claimant's head trauma and her tumor is the temporal relationship between her injury and the tumor and symptoms. However, as Dr. Burger pointed out, temporal coincidences do **not** prove causation; it can be expected that in any population of people with brain tumors, some will have a history of head trauma even without a causal connection. The fact that claimant's symptoms followed her head injury does not provide a scientific basis for causation of the tumor. Moreover, some of her symptoms appear to have been caused by the head trauma, while others are due to the tumor.

¶75 This case is complicated by the fact that some of claimant's symptoms were due to the tumor, while others were due to the closed head injury she suffered as a result of her industrial accident. Dr. Schimpff testified that she developed a "complex partial seizure" disorder. On this point, his testimony was unrebutted. However, he acknowledged that any tumor existing in early 1999 would not have been sufficient in size to create the EEG patterns and symptoms underlying his diagnosis. Moreover, there are other indications in claimant's medical records indicating she had an overlapping disorder caused by the trauma. The **left** temporal headaches she reported after the accident, which seem to have eventually resolved, likely resulted from the injury. (*See* Nelson Dep. at 11, 15.) On the other hand, in June 2000, the claimant had developed **right** temporal headaches (the tumor was in the right hemisphere). (Ex. 3 at 34.)

¶76 Dr. Trontel's evaluations also lend credence to the existence of two separate conditions. When Dr. Trontel first evaluated claimant, he noted persistent disruption of working memory, object recognition difficulty, word use problems, and difficulty with working memory. (Ex. 2 at 5.) In the second evaluation, Dr. Trontel found that cognitive control, working memory, and sequencing, which he believed "were most likely secondary to the blow on her head," had shown improvement. (*Id.* at 10.) However, nonverbal memory and spatial-relational processing were now impaired. Dr. Trontel viewed these latter problems as having "unknown" etiology. (*Id.* at 11.) In a March 2000 report, Dr. Lindsay adopted Dr. Trontel's suggestion that claimant presently suffered from symptoms "perhaps consistent with her right temporal lesion," while "post concussive type symptomatology had improved." (Ex. 3 at 27.)

CONCLUSIONS OF LAW

¶77 The 1997 version of the Workers' Compensation Act applies to this case since that was the law in effect on the date of the injury at issue in this case. *Buckman v. Montana Deaconess Hospital*, 224 Mont. 318, 321, 730 P.2d 380, 382 (1986).

¶78 Claimant bears the burden of proving by a preponderance of the evidence that she is entitled to the benefits she seeks. *Ricks v. Teslow Consolidated,* 162 Mont. 469, 512 P.2d 1304 (1973); *Dumont v. Wicken Bros. Construction Co.*, 183 Mont. 190, 598 P.2d 1099 (1979).

¶79 Section 39-71-407, MCA (1997), provides:

> (1) Each insurer is liable for the payment of compensation, in the manner and to the extent provided in this section, to an employee of an employer that it insures

who receives an injury arising out of and in the course of employment or, in the case of death from the injury, to the beneficiaries, if any.

(2)(a) An insurer is liable for an injury, as defined in 39-71-119, if the injury is established **by objective medical findings and if the claimant establishes that it is more probable than not that:**

(i) a claimed injury has occurred; or

(ii) a claimed injury aggravated a preexisting condition.

(b) **Proof that it was medically possible that a claimed injury occurred or that the claimed injury aggravated a preexisting condition is not sufficient to establish liability.** [Emphasis added.]

¶80 Montana case law has also long held that testimony in the form of ***medical possibility*** does not by itself prove a claimant's entitlement to benefits. *Matthews v. State Compensation Insurance Fund*, 1999 MT 225, ¶ 15-17; *Caekaert v. State Compensation Mutual Insurance Fund*, 268 Mont. 105, 114, 885 P.2d 495, 501 (1994); *Kramer v. EBI Companies*, 265 Mont. 525, 533, 878 P.2d 266, 270 (1994) ("Mere medical possibility without supporting evidence is insufficient to establish compensability.").

¶81 In this case I am affirmatively persuaded that claimant's tumor was **not** caused or accelerated by the blow to her head occurring at Wal-Mart during December 1998. Therefore, respondent is **not** liable for medical bills or compensation benefits related to the tumor and subsequent surgery.

¶82 In her post-hearing brief, claimant cites several cases from other states for the proposition that cancer, including a brain tumor, is compensable if a workplace injury caused, accelerated, or aggravated a tumor. *See, e.g., Reynolds Metals Co. v. Industrial Commission of Arizona*, 98 Ariz. 97, 402 P.2d 414 (1965); *United Electric Co. v. Myers*, 134 So.2d 7 (Fla. 1961); *Kling v. Central Lumbar & Millwork Co.*, 3 N.J. 151, 65 A.2d 772 (1949); *Calabretta v. Lanorith*, 90 App.Div.2d 608, 456 N.Y.S.2d 175 (1982); and additional cases noted in Petitioner's Brief in Support of Benefits at pages 16-18. Each of these decisions, of course, turns upon its own facts and medical testimony. As noted, on the record presented, and given current medical knowledge, I am **not** persuaded claimant's brain tumor was caused, accelerated, or aggravated by her work accident. I note that the most recent decision cited by claimant was decided in 1982, with the other decisions falling within the years 1939 through 1967. None of these decisions reflect current medical knowledge.

¶83 The questions presented for trial were whether claimant's tumor was related to her industrial accident and whether she is entitled to temporary total disability on account of the tumor. I have found adversely to her on those issues. However, since the issue was not presented, I do not determine whether she is entitled compensation benefits on account of her closed head injury, including the partial complex seizure disorder Dr. Schimpff believed to exist. Moreover, the evidence presented does not provide good guidance for determining whether absent the tumor she would be disabled.

JUDGMENT

¶84 Claimant's brain tumor was not caused or aggravated by her industrial accident. She is not entitled to medical or disability benefits on account of the tumor.

¶85 Claimant is not entitled to costs or attorneys fees.

¶86 Any party to this dispute may have 20 days in which to request a rehearing from these Findings of Fact, Conclusions of Law and Judgment.

¶87 This JUDGMENT is certified as final for purposes of appeal pursuant to ARM 24.5.348.

DATED in Helena, Montana, this 20th day of August, 2001.

(SEAL)

/s/ Mike McCarter
JUDGE

c: Mr. Garry D. Seaman
Mr. Donald R. Herndon
Submitted: March 2, 2001

1. The parties have not submitted evidence regarding acceptance of the claim, however, the Court infers that the claim was accepted from Uncontested Facts 1 and 2 in the Pretrial Order, which state that claimant was injured in the course and scope of her employment and that her employer was insured by Insurance of Pennsylvania. In addition, the petitioner's second listed issue, to which there is no objection by respondent, is whether claimant is entitled to "reinstatement" of temporary total disability benefits. (Pretrial Order at 2.)