**IN THE SUPREME COURT OF THE STATE OF IDAHO**

**Docket No. 35157**

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| **MELINDA COOMBS, natural mother of MICHAEL HALL, deceased, and THE ESTATE OF MICHAEL HALL, as represented by MELINDA COOMBS, personal representative,** | ) | **Boise, August 2009 Term** |
| **Plaintiffs-Appellants,** | ) | **2009 Opinion No. 124** |
| **v.** | ) | **Filed: October 13, 2009** |
| **ADRIAN CURNOW, M.D., and RUSSELL GRIFFITHS, M.D.,** | ) | **Stephen W. Kenyon, Clerk** |
| **Defendants-Respondents.** | ) | |

Appeal from the District Court of the Fourth Judicial District of the State of Idaho, Ada County. Hon. Deborah A. Bail, District Judge.

Order granting judgment notwithstanding the verdict is vacated and the case is remanded.

Lojek Law Offices, Chtd., Boise, for appellants. Donald W. Lojek argued.

Moffatt, Thomas, Barrett, Rock & Fields, Chtd., Boise, for respondents. Patricia M. Olsson and Nancy J. Garrett argued.

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J. JONES, Justice.

This is an appeal from the district court's entry of judgment notwithstanding the verdict (j.n.o.v.) in favor of respondents in a medical malpractice case after the district court found that the appellants' expert testimony regarding causation was scientifically unreliable. Because we find that the district judge impermissibly weighed the evidence in considering respondents' motion and that the evidence was legally sufficient to support the jury's verdict, we vacate the j.n.o.v. and remand with instructions to reinstate the jury verdict.

## I.

On June 22, 2002, Michael Hall, then nearly three years old, and his family were attending a picnic at a family member's home. While at the picnic, Michael was feeding potato chips to the host's dog, a golden retriever. In the course of feeding the dog, Michael spilled some of the chips on the ground. Michael and the dog both attempted to retrieve the dropped chips at the same time and, when Michael tried to take the chips away from the dog, the dog bit him on the face. The bite left Michael with a "deep gouging wound" in his cheek, approximately two inches by two inches in size.

Michael was taken to St. Alphonsus Regional Medical Center for treatment of the bite, which was characterized as "very serious but not life-threatening." Dr. Adrian Curnow, a pediatric surgeon and intensivist specializing in pediatric critical care, was assigned as Michael's primary physician. At the time, Dr. Curnow was one of only two pediatric surgeons in Idaho.[1] To assist in treating Michael, Dr. Curnow contacted Dr. Russell Griffiths, a pediatric craniofacial plastic surgeon. Dr. Griffiths was the only pediatric craniofacial plastic surgeon in Idaho at that time.

After discussing the various treatment options with Drs. Curnow and Griffiths, Michael's mother, Melinda Coombs, opted to have the severed tissue reimplanted on Michael's face.[2] Mrs. Coombs consented to the surgery after being advised of the attendant risks, including the risk of death. Upon obtaining Mrs. Coombs' consent, Dr. Griffiths performed an exploratory surgery to determine the likelihood of successful reattachment. During the surgery, Dr. Griffiths discovered only one artery and no vein, making successful reimplantation less likely. Dr. Griffiths informed Mrs. Coombs of his discovery, at which point she reaffirmed her consent to the reimplantation surgery. Dr. Griffiths then performed the surgery and was able to reattach the tissue to Michael's face and reconnect it to the blood supply.

Throughout Michael's surgery, he was sedated with the sedative agent Propofol (Diprivan). Pursuant to a "bridging order"[3] issued by Dr. Griffiths, Michael was to

[1] Dr. Curnow's partner, Dr. Ellen Reynolds, was the other pediatric surgeon.

[2] Michael's face was seriously deformed from the bite and, without successful reimplantation, he would have been required to undergo multiple surgeries throughout the course of his life and would likely have suffered "significant psychological harm from being visibly deformed."

[3] Michael's primary physicians had left the hospital and the bridging order was a directive for his care until they returned.

remain sedated after his surgery while he was recovering in the intensive care unit (ICU). Prolonged sedation was deemed necessary because there was a significant risk the reimplantation would fail if there was any disturbance to the reattached tissue. In light of Michael's young age, there was concern he would not remain still if conscious and thereby cause disturbance to the tissue. In deciding on the sedative to be administered, Dr. Griffiths consulted with Dr. Smagula, the anesthesiologist assigned to Michael's case. Based on the consultation, Dr. Griffiths ordered Propofol to continue to be administered.[4] Although Propofol was not indicated for long-term sedation of pediatric patients in the ICU, and Dr. Griffiths had not previously administered the drug under such circumstances, he elected to use Propofol because the alternative sedative, Versed, carried a risk of idiosyncratic reaction.[5]

After Michael's surgery, Dr. Curnow and his partner, Dr. Reynolds, resumed primary responsibility for Michael's treatment. Upon doing so, they left the existing Propofol order in place and ordered Michael's blood pressure, lipid metabolism, and hemodynamic status to be frequently monitored. Pursuant to the order, Michael was receiving between 100 and 150 micrograms per kilogram per minute of Propofol. Although Dr. Curnow had previously administered Propofol for short-term sedation and had participated in a team that used it for long-term sedation in one patient, he did not use the drug regularly in his practice.

Despite efforts to promote reattachment of the tissue, Michael's reimplantation ultimately failed. Dr. Griffiths removed the tissue on June 25, 2002, and Michael was returned to the ICU. To assist in Michael's recovery, Dr. Griffiths ordered that Michael continue to be sedated with Propofol and placed him on Fentanyl for pain management. Dr. Griffiths then "signed off" of the case and Drs. Curnow and Reynolds resumed providing for Michael's care. Although Dr. Curnow originally intended to lower Michael's Propofol level, he decided to maintain the existing dose to lessen the pain Michael would experience when the dressings on the wound were changed.

Once Michael was returned to the ICU, he began encountering various problems. His blood pressure and hemoglobin levels became very low, he experienced accelerated

[4] In addition, he ordered that Michael be given a blood thinner, Heparin, and that medical leeches be used to improve blood flow to the reattached tissue.

[5] An idiosyncratic reaction occurs when the patient becomes excited rather than sedated.

heart rates (ventricular tachycardia), and he began suffering from a high fever. A transfusion successfully treated the low hemoglobin levels, but did not assist with his low blood pressure or abnormal heart rate. As a result, Dr. Reynolds ordered additional tests and that Propofol be discontinued. The test results indicated Michael had metabolic acidosis (low albumin and calcium levels), which was successfully treated. Michael's ventricular tachycardia also temporarily ceased.

Michael's improved condition did not last long. During an examination on the afternoon of June 27, a nurse discovered Michael's pupils were fixed and dilated. A computerized axial tomography (CT) scan was taken, revealing tissue death in Michael's cerebellar hemisphere, frontal lob, and several arterial distributions. It also indicated that Michael's brain was swollen and suffering from cytotoxic damage. Based on these findings, Michael was determined to be brain dead. He was subsequently removed from life support and passed away on June 28, 2002.

Dr. Glenn Groben, a forensic pathologist, conducted Michael's autopsy. According to Dr. Groben, Michael died from swelling of the brain (cerebral edema). He also noted evidence of lack of oxygen supply throughout the brain (global hypoxic changes). Aside from these abnormalities, the autopsy did not reveal any irregularities in Michael's other organs or systems.

On December 17, 2004, Mrs. Coombs brought a medical malpractice claim against Drs. Curnow and Griffiths (collectively "the doctors"). The case proceeded to a jury trial, commencing on September 4, 2007. At trial, it was undisputed that Michael died from cerebral edema—the sole issue was what caused the cerebral edema. Mrs. Coombs argued that the doctors' prolonged administration of Propofol was the cause of the cerebral edema. In support of her theory, Mrs. Coombs relied on the opinion of Dr. Gregory Hammer. Over the doctors' objections, the district court allowed Dr. Hammer to testify as an expert regarding the applicable standard of care, breach, and causation. According to Dr. Hammer, the doctors violated the applicable standard of care in Boise, Idaho in 2002, by ordering the long-term, high-dose use of Propofol to sedate a pediatric patient in the ICU. In Dr. Hammer's opinion, the doctors' negligent use of Propofol was the proximate cause of the cerebral edema that resulted in Michael's death. He testified

that the long-term, high-dose sedation with Propofol produced hypotension and lipemia,[6] which, in combination with Michael's low hemoglobin levels, resulted in decreased blood flow and oxygen to the brain. The lack of oxygen, in turn, caused the cerebral edema. Dr. Hammer was the only expert to associate Propofol with Michael's death. The doctors' expert witnesses testified that it was not possible to determine the cause of the cerebral edema.

After the close of evidence, the doctors filed motions for entry of a directed verdict. The district court reserved its rulings on the motions and submitted the case to the jury. The jury subsequently rendered a verdict against the doctors, finding them equally liable, and awarding Mrs. Coombs $750,000.00 in damages. According to the jury's special verdict, both doctors breached the applicable standard of care in treating Michael and their breach was the proximate cause of his death.

On September 28, 2007, the doctors filed motions for judgment notwithstanding the verdict, which the district court granted on March 28, 2008. The court concluded that the doctors were entitled to judgment because there was "no substantial evidence from which a reasonable jury could conclude that the long term use of [P]ropofol was the proximate cause of Michael Hall's cerebral edema." In reaching its conclusion, the court pointed out that there was no scientifically reliable evidence indicating a link between long-term, high-dose Propofol use and cerebral edema resulting in death. Rather, the only cases connecting Propofol use with death involved Propofol Related Infusion Syndrome (PRIS). At the time of treatment, symptoms associated with PRIS included cardiovascular instability, evolving rhabdomyolysis,[7] decreasing renal function, and refractory metabolic acidosis—no documented cases involved cerebral edema alone. Because there were no peer-reviewed, published journal articles directly supporting Dr. Hammer's testimony, the court was unwilling to accept his opinion as reliable. In the absence of Dr. Hammer's testimony, the court concluded that there was not substantial and competent evidence to support the jury's verdict.

Mrs. Coombs now appeals the district court's decision granting the doctors' motions for j.n.o.v. to this Court, arguing that there was substantial and competent

[6] "Lipemia," "hyperlipemia," "hyperlipidemia," and "lipidemia" are all terms used to describe a buildup of fatty acids in the blood, which impacts the energy generation of cells.

[7] Rhabdomyolysis is the breakdown and release of muscle fibers into the bloodstream.

evidence to support the jury's verdict. She contends that the district court impermissibly weighed the evidence in ruling on the motions and failed to draw all reasonable inferences in her favor. She asks this Court to reverse the district court's decision and remand the case with instructions to enter judgment in her favor consistent with the jury's verdict.

**II.**

The following issues are presented: (1) whether the district court may properly reconsider the admissibility of evidence on a j.n.o.v. motion; (2) whether there was substantial and competent evidence to support the jury's verdict; and (3) whether the doctors are entitled to attorney's fees on appeal.

**A.**

In determining whether a district court should have granted a j.n.o.v. motion, this Court employs the same standard the district court used in ruling on the motion. *Jeremiah v. Yanke Mach. Shop, Inc.*, 131 Idaho 242, 247, 953 P.2d 992, 997 (1998). An order granting a j.n.o.v. is appropriate when "the facts are undisputed" and "there can be but one conclusion as to the verdict that reasonable minds could have reached"—namely, that the moving party should prevail. *O'Neil v. Schuckardt*, 112 Idaho 472, 480, 733 P.2d 693, 701 (1986). On the other hand, a verdict will be upheld when it is supported by substantial and competent evidence. *Jeremiah*, 131 Idaho at 247, 953 P.2d at 997. Substantial evidence is evidence of "such sufficient quantity and probative value that reasonable minds could conclude that the verdict of the jury was proper." *Karlson v. Harris*, 140 Idaho 561, 567, 97 P.3d 428, 434 (2004). Evidence may be substantial even though it is contradicted. *Watson v. Navistar Int'l Transp. Corp.*, 121 Idaho 643, 658, 827 P.2d 656, 671 (1992).

In determining whether substantial evidence exists, this Court may not weigh the evidence, attempt to judge the credibility of the witnesses, or compare its factual findings with those of the jury. *Hall v. Farmers Alliance Mut. Ins. Co.*, 145 Idaho 313, 324, 179 P.3d 276, 287 (2008). Instead, the Court must review the evidence as a whole, drawing all inferences "in the light most favorable to the non-moving party" and "view[ing] the facts as if the moving party has admitted the truth of all the non-moving [party's] evidence." *Jeremiah*, 131 Idaho at 247, 953 P.2d at 997. The Court will "not examine any

conflicting evidence presented by the moving party to refute the non-moving party's claims." *Karlson*, 140 Idaho at 567, 97 P.3d at 434. Whether there was sufficient evidence to create an issue of fact for the jury is a pure question of law over which this Court exercises free review. *O'Neil*, 112 Idaho at 480, 733 P.2d at 701.

**B.**

The district court in this case admitted Dr. Hammer's testimony as evidence of the applicable standard of care, breach, and causation after concluding the "threshold that is necessary for the admissibility of testimony in a medical malpractice case has been reached." It did so over the doctors' strenuous objections.[8] After admitting the evidence, the court did not strike it from the record or instruct the jury not to consider it. Instead, the district court essentially re-opened the record in ruling on the motions for j.n.o.v. and concluded Dr. Hammer's testimony was unreliable and, thus, inadmissible. In doing so, the court noted it was justified in ruling on the admissibility of Dr. Hammer's testimony at that point in the proceedings because it would have been too costly for the parties to "try the medical proof twice."

When considering whether to grant a motion for j.n.o.v., Idaho courts are bound by the record submitted to the jury. Although the United States Supreme Court has held that, under the Federal Rules of Civil Procedure, federal courts are not bound by the record submitted to the jury when considering whether judgment as a matter of law is appropriate, that holding is not in accord with Idaho precedent and is not binding on this Court. *See Weisgram v. Marley Co*., 528 U.S. 440, 454 (2000). Under Idaho law, in moving for a j.n.o.v., the moving party admits the truth of all of the non-moving party's evidence. *Hudson v. Cobbs*, 118 Idaho 474, 478, 797 P.2d 1322, 1326 (1990). Once an expert's opinion is admitted, it is up to the trier of fact to weigh the opinion against any conflicting testimony. *City of McCall v. Seubert*, 142 Idaho 580, 585, 130 P.3d 1118, 1123 (2006). The jury's weighing of conflicting, admitted opinions will not be second-guessed on appeal. *Id.* at 586, 130 P.3d at 1124. Moreover, while Idaho Code section 1-1603 acknowledges a court's power "[t]o amend and control its process and orders, so as to make them conformable to law and justice," which includes the power to make the

[8] The doctors objected to the admissibility of Dr. Hammer's opinion in motions for summary judgment, as well as pre-trial motions in limine.

record conform to the facts, a court does not have the power to amend the record to "correct a judicial error." *Donaldson v. Henry*, 63 Idaho 467, 473, 121 P.2d 445, 447 (1941). In light of these well-established legal principles, ruling on the admissibility of evidence at the j.n.o.v. stage of the proceedings is not permitted in Idaho.

Moreover, allowing courts to reconsider the admissibility of evidence at the j.n.o.v. stage of the proceedings is undesirable for several reasons. First, doing so would subject all previously admitted or excluded evidence to reconsideration, thereby placing the party who relied on the evidence at a substantial disadvantage. *See Jackson v. Pleasant Grove Health Care Ctr.*, 980 F.2d 692, 696 (11th Cir. 1993). The disadvantages associated with allowing such reconsideration are aptly demonstrated by the proceedings in this case. In opposing the doctors' motions for summary judgment, Mrs. Coombs relied on Dr. Hammer's affidavit, which the district court ruled to be admissible expert testimony despite the doctors' objections. Before trial, the doctors renewed their objections to the testimony, but the objections were overruled by the district court. Each time Dr. Hammer's opinion was objected to, it was ruled admissible by the district court. As such, Mrs. Coombs relied on Dr. Hammer's testimony to establish her case before the jury. It was not until over six months after the jury rendered its verdict that the district court determined that Dr. Hammer's opinion was unreliable and granted the doctors' motions for j.n.o.v. Had the inadmissibility determination been made earlier, Mrs. Coombs may have had an opportunity to remedy the deficiency in her case by finding another expert. *See Puckett v. Verska*, 144 Idaho 161, 166, 158 P.3d 937, 942 (2007) (holding that a district court may consider affidavits submitted in support of a request for reconsideration even when they are submitted after summary judgment has been granted); *Jackson*, 980 F.2d at 696 ("If evidence is ruled inadmissible during the course of the trial, the plaintiff has the opportunity to introduce new evidence. However, when that evidence is ruled inadmissible in the context of deciding a motion for JNOV, the plaintiff, having relied on the evidence already introduced, is unable to remedy the situation."), *abrogated by Weisgram*, 528 U.S. at 454. If Dr. Hammer's testimony is excluded on the motion for j.n.o.v., Mrs. Coombs would have no evidence to support her claim and would have no opportunity to re-establish her case.

Second, the issue of whether evidence was properly admitted is more appropriately suited for consideration in the context of a motion for a new trial. *Kinser v. Gehl Co*., 184 F.3d 1259, 1269 (10th Cir. 1999); *see also* Idaho R. Civ. P. 59. Idaho Rule of Civil Procedure 59(a) specifically permits the granting of a new trial based on insufficient evidence and errors in law occurring at trial.[9] Idaho R. Civ. P. 59(a). Granting a new trial, while not ideal for the party who prevailed before the jury, at least gives that party the opportunity to repair the deficiencies in its case.[10] A j.n.o.v., on the other hand, forecloses the matter. As stated by the New Mexico Supreme Court:

> A motion for [j.n.o.v.], like a motion for a directed verdict, does not raise questions relating to the competency or admissibility of evidence. Therefore, in considering a motion for [j.n.o.v.], the evidence must be taken as it existed at the close of the trial, and evidence admitted over objection cannot be excluded nor can evidence be included which was improperly rejected. Whether competent or incompetent, all evidence submitted to the jury must be considered by the court in ruling on a motion for [j.n.o.v.], and such a judgment cannot be entered on a diminished record after the elimination of incompetent evidence. The proper remedy for disposing of evidence erroneously admitted during the course of the trial is a new trial where motion therefor has been made.
> . . .
>
> If, after the return of the verdict, the court had been of the opinion that it was based upon incompetent testimony erroneously admitted during the course of the trial, the court had no alternative but to grant a new trial rather than the motion for [j.n.o.v.].

*Townsend v. United States Rubber Co*., 392 P.2d 404, 406–07 (N.M. 1964).

Third, it seems paradoxical that a court could conclude, at the j.n.o.v. stage, that previously admitted evidence was actually inadmissible even though the court deemed the evidence admissible for purposes of summary judgment and at trial. *See* Idaho R. Evidence 104 (directing district courts to make preliminary determinations regarding the qualifications of witnesses). Under such circumstances, the district court has been given several previous opportunities to become familiar with the substance of the evidence and

[9] The qualification of an expert "is a preliminary question of law" and, thus, it is more appropriately addressed in the context of a motion for a new trial. *Kinser v. Gehl Co*., 184 F.3d 1259, 1269 (10th Cir. 1999).

[10] Here, however, there is no record of either party filing a motion for a new trial or of the court granting such a motion.

determine whether it was admissible. Allowing reconsideration of the evidence on a j.n.o.v. motion only creates greater expense for the parties by requiring them to try the evidence several times—during summary judgment proceedings, motions in limine, at trial, and then in the j.n.o.v. proceedings. It also forces the parties to participate in an unnecessary trial. In addition, in denying a motion for summary judgment, a district court acknowledges that admissible evidence exists, creating a genuine issue of material fact for the jury. *Weeks*, 143 Idaho at 837–38, 153 P.3d at 1183–84 (2007) ("Admissibility of evidence within depositions and affidavits in support or in opposition to a motion for summary judgment is a threshold question to be addressed before a court can determine the outcome of the summary judgment motion."). Accordingly, the summary judgment stage would be a more appropriate time to address the reliability of an expert's opinion, as opposed to six months after the jury renders its verdict, which happened here. The reliability of expert testimony should be determined before submitting the case to the jury.

Our holding, precluding courts from reconsidering the record submitted to the jury, promotes certainty and fairness. *See Kinser*, 184 F.3d at 1267. As the Tenth Circuit stated:

> This rule promotes certainty: litigants need not supplement conditionally admitted evidence, perhaps unnecessarily; and district courts need not speculate as to what other evidence might have been offered if the evidence had been excluded at trial. The rule promotes fairness: punishing a litigant for the court's erroneous admission of evidence is unfair; and the remedy of a new trial is available to put both sides on an equal footing.

*Id*. Therefore, we hold that trial courts may not reconsider the record submitted to the jury when determining the propriety of a j.n.o.v.

**C.**

The district court went on to hold that the jury's verdict was not supported by substantial and competent evidence. The court first determined that Dr. Hammer's testimony was scientifically unreliable, primarily because it was not supported by scientific studies. The court then concluded that there was no substantial and competent evidence in the record to support the verdict.

Mrs. Coombs argues the district court erred in concluding that she did not present scientifically reliable evidence establishing that the negligent administration of Propofol was the cause of Michael's death. She contends the court ignored the foundation of Dr. Hammer's opinion and improperly weighed the evidence presented at trial. According to Mrs. Coombs, Dr. Hammer's opinion was supported by basic medical principles and, therefore, the absence of peer-reviewed articles was irrelevant. Alternatively, she maintains the lack of articles establishing a link between long-term, high-dose Propofol use in pediatric patients and death is to be expected given the ethical constraints associated with testing Propofol in pediatric patients.

The doctors argue that Mrs. Coombs failed to present reliable evidence establishing causation between the administration of Propofol and Michael's death. They argue that scientific or specialized knowledge was necessary to assist the trier of fact in determining whether long-term sedation with Propofol caused Michael's death. According to the doctors, Dr. Hammer's opinion was pure speculation and, therefore, would not assist the trier of fact in reaching its conclusion. In making this argument, they point out that, at the time they treated Michael, there was no medical research directly supporting Dr. Hammer's opinion that Propofol could cause death due to cerebral edema alone. Even if the medical evidence Dr. Hammer relied on had been available, they contend it did not support his ultimate conclusion.

In treating a patient, a provider is not obligated to provide optimal care or employ the highest degree of skill. *Dekker v. Magic Valley Reg'l Med. Ctr.*, 115 Idaho 332, 334, 766 P.2d 1213, 1215 (1988). "Negligence on the part of a physician consists in his doing something which he should not have done, or in omitting to do something which he should have done." *Willis v. W. Hosp. Ass'n*, 67 Idaho 435, 442, 182 P.2d 950, 954 (1947). The plaintiff must show more than a mere possibility that a provider may have been negligent. *Id.*

In addition to proving a provider failed to use ordinary care, the plaintiff must demonstrate that the provider's "failure to use ordinary care was the proximate cause of damage to the plaintiff." *Pearson v. Parsons*, 114 Idaho 334, 339, 757 P.2d 197, 202 (1988). "[T]he mere fact that [a procedure does] not result in a favorable outcome does not establish—or even constitute evidence of—negligence or proximate causation."

*Campbell v. United States*, 904 F.2d 1188, 1194 (7th Cir. 1990). To establish proximate cause, a plaintiff must demonstrate that the provider's negligence was both the actual and legal (proximate) cause of his or her injury. *Munson v. Dep't of Highways*, 96 Idaho 529, 531, 531 P.2d 1174, 1176 (1975). Actual cause "is a factual question focusing on the antecedent factors producing a particular consequence." *Id*.

Legal cause, on the other hand, "focuses upon legal policy in terms of whether responsibility will be extended to the consequences of conduct which has occurred." *Id*. Legal cause only exists when "it [is] reasonably foreseeable that such harm would flow from the negligent conduct." *Cramer v. Slater*, 146 Idaho 868, 875, 204 P.3d 508, 515 (2009). The relevant inquiry is "whether the injury and manner of the occurrence are 'so highly unusual that we can say, as a matter of law that a reasonable [person], making an inventory of the possibilities of harm which his conduct might produce, would not have reasonably expected the injury to occur.'" *Id.* (quoting *Doe v. Sisters of the Holy Cross*, 126 Idaho 1036, 1041, 895 P.2d 1229, 1234 (Ct. App. 1995)). Proximate cause in medical malpractice cases involving more than one possible cause of injury will be established if it is shown that the defendant's conduct "was a substantial factor in bringing about the injury suffered by the plaintiff." *Munson*, 96 Idaho at 531, 531 P.2d at 1176. This may be proven by direct evidence or by showing a "chain of circumstances from which the ultimate fact required to be established is reasonably and naturally inferable." *Weeks*, 143 Idaho at 839, 153 P.3d at 1185. "The question of proximate cause is one of fact and almost always for the jury." *Cramer*, 146 Idaho at 875, 204 P.3d at 515.

Nothing in Idaho Code sections 6-1012 or 6-1013 requires that proximate cause be proved by expert testimony—those statutes only address the applicable standard of care and breach of that standard. *See Sheridan v. St. Luke's Reg'l Med. Ctr.*, 135 Idaho 775, 785, 25 P.3d 88, 98 (2001). As such, the admission of testimony to prove proximate cause in medical malpractice cases is governed solely by the Idaho Rules of Evidence. *Id*. Although the Idaho Rules of Evidence do not require expert testimony to establish causation in medical malpractice cases, such testimony is often necessary given the nature of the cases. Expert testimony is generally required because "the causative factors are not ordinarily within the knowledge or experience of laymen composing the jury." *Flowerdew v. Warner*, 90 Idaho 164, 170, 409 P.2d 110, 113 (1965).

Under the rules, expert opinion testimony is only admissible when "the expert is a qualified expert in the field, the evidence will be of assistance to the trier of fact, experts in the particular field would reasonably rely upon the same type of facts relied upon by the expert in forming his opinion, and the probative value of the opinion testimony is not substantially outweighed by its prejudicial effect." *Ryan v. Beisner*, 123 Idaho 42, 47, 844 P.2d 24, 29 (Ct. App. 1992); *see also* Idaho R. Evidence 702, 703, & 403. "[E]xpert opinion which is speculative, conclusory, or unsubstantiated by facts in the record is of no assistance to the jury in rendering its verdict, and therefore is inadmissible." *Ryan*, 123 Idaho at 46–47, 844 P.2d at 28–29; *see also* Idaho R. Evidence 702. Testimony is speculative when it "theoriz[es] about a matter as to which evidence is not sufficient for certain knowledge." *Karlson v. Harris*, 140 Idaho 561, 565, 97 P.3d 428, 432 (2004). Conversely, expert testimony will assist the trier of fact when the reasoning or methodology underlying the opinion is scientifically sound and "based upon a 'reasonable degree of medical probability'"—mere possibility is insufficient. *Bloching v. Albertson's, Inc*., 129 Idaho 844, 846–47, 934 P.2d 17, 19–20 (1997) (quoting *Roberts v. Kit Mfg. Co.*, 124 Idaho 946, 948, 866 P.2d 969, 971 (1993)).

In determining whether expert testimony is admissible, a court must evaluate "the expert's ability to explain pertinent scientific principles and to apply those principles to the formulation of his or her opinion." *Ryan*, 123 Idaho at 46, 844 P.2d at 28. Admissibility, therefore, depends on the validity of the expert's reasoning and methodology, rather than his or her ultimate conclusion. *Id*. at 46–47, 844 P.2d at 28–29. So long as the principles and methodology behind a theory are valid and reliable, the theory need not be commonly agreed upon or generally accepted. *Weeks*, 143 Idaho at 838, 153 P.3d at 1184. While the court must "distinguish scientifically sound reasoning from that of the self-validating expert, who uses scientific terminology to present unsubstantiated personal beliefs," it may not "substitute its judgment for that of the relevant scientific community." *Ryan*, 123 Idaho at 46, 844 P.2d at 28.

Relevant considerations in determining whether the basis of an expert's opinion is scientifically valid include "whether the theory can be tested and whether it has been

subjected to peer-review and publication."[11] *Weeks*, 143 Idaho at 838, 153 P.3d at 1184; *see also Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 589 (1993). Other indicia of reliability include "the close oversight and observation of the test subjects, the prospectivity and goal of the studies, . . . the presence of safeguards in the technique, . . . analogy to other scientific techniques whose results are admissible, . . . the nature and breadth of inferences drawn, . . . the extent to which the basic data are verifiable by the court and jury, . . . [the] availability of other experts to test and evaluate the technique, [and] the probative significance of the evidence in the circumstances of the case." *State v. Konechny*, 134 Idaho 410, 417–18, 3 P.3d 535, 542–43 (Ct. App. 2000); *see also Daubert*, 509 U.S. at 589–94 (1993) (noting also the potential rate of error and general acceptance of the theory).

An expert's opinion does not meet the requisite standard of reliability when it is based on the mere temporal connection between the administration of a drug and a particular consequence. *Swallow v. Emergency Med. of Idaho, P.A.*, 138 Idaho 589, 593, 67 P.3d 68, 72 (2003). In *Swallow*, a patient suffered a heart attack after taking an erroneously prescribed dose of Cipro. *Id.* at 591, 67 P.3d at 70. The district court excluded the plaintiffs' proffered expert testimony regarding causation between Cipro and the heart attack and the plaintiffs appealed. *Id.* On appeal, this Court upheld the district court's evidentiary ruling on the ground that the expert's opinion was unreliable. *Id.* at 592–93, 67 P.3d at 71–72. Because there was no evidence that an overdose of Cipro could cause a heart attack, and because the expert's opinion was based on statistically insignificant information and "the mere temporal connection between the drug and a certain consequence," the opinion was not reliable enough to be considered by the jury.[12] *Id.*

[11] However, "[i]n instances of 'a rare occurrence' where there are few opportunities for scholarly research, the lack of published studies should not bar otherwise scientifically valid testimony." *Weeks*, 143 Idaho at 838–39, 153 P.3d 1180, 1184–85. The reasoning behind admitting expert opinions under such circumstances is that there may be justifiable reasons for the lack of research on a subject. *Id.*

[12] The expert was uncertain whether Cipro had either of the effects necessary to cause a heart attack, there were no studies indicating that it did, the Physicians' Desk Reference merely indicated that less than 1% of patients in a clinical study suffered heart attacks that may or may not have been related to the drug, and he was not sure of the exact role the drug would play in causing a heart attack. *Swallow*, 138 Idaho at 592–93, 67 P.3d at 71–72.

However, expert testimony linking the administration of a drug to a certain consequence does not need to be directly supported by medical evidence so long as the "expert . . . is able to determine conclusively the effect the medication had on the patient." *Weeks*, 143 Idaho at 838, 153 P.3d at 1184. In *Weeks*, a patient died after medications were infused into her brain through a catheter that was intended to drain excess fluid from the brain. *Id*. at 836, 153 P.3d at 1182. The patient's family brought a medical malpractice claim against the hospital. *Id*. The district court excluded the plaintiffs' proffered expert testimony that indicated the infusion was a substantial factor in causing the patient's death. *Id*. 837, 153 P.3d at 1183. The plaintiffs appealed to this Court, which held the expert's testimony should have been admitted. *Id*. at 838, 153 P.3d at 1184. The Court reasoned that, although research had not been done on the exact type of occurrence or the effects of administering medication in the particular manner, the expert's opinion was admissible because it was not based merely on speculation or a temporal occurrence. *Id*. at 839, 153 P.3d at 1185. Instead, it was based on the doctor's experience and research. *Id*. The Court stated:

> While there is no exact known effect of the combination of chemicals infused into [the patient's] brain, there is scientifically reliable evidence regarding the effect of increased intracranial pressure. [The expert] based a portion of his opinion, dealing with the mechanical effect of the increased amount of fluid in [the patient's] brain, upon such scientifically reliable information. However, he testified that these effects could not be separated from the effects of the medications, for which there is no peer-reviewed, published information.

*Weeks*, 143 Idaho at 839, 153 P.3d at 1185. As such, the opinion was admissible even though the expert was "unable to determine the exact effect" the medication had on the patient. *Id*.

Here, the district court, in ruling on the doctors' motions for j.n.o.v., concluded that Dr. Hammer's testimony did not meet the standards of scientific reliability. It pointed out that there were no studies available in 2002 connecting Propofol use with death caused by cerebral edema absent any other signs of PRIS. In fact, there were no studies showing Propofol even caused PRIS.[13] Because Propofol had been in use for a substantial

[13] Even if there were, Michael did not experience any of the hallmarks of the syndrome. Michael did experience acidosis at one point during sedation, but it responded to treatment with sodium bicarbonate and

period of time, the court regarded the lack of the studies as very significant. In addition, the data in the only source Dr. Hammer relied on that was available in 2002, the Physicians' Desk Reference (PDR), was statistically insignificant[14] and did not identify cerebral edema as a cause of death associated with Propofol. The other source Dr. Hammer relied on was not published until 2003 and, therefore, was irrelevant to the doctors' treatment of Michael in 2002. Finally, the other experts who testified at trial indicated that it was not possible to determine the cause of Michael's cerebral edema. Concluding that causation could not be established solely from the temporal association between the use of a drug and a negative health consequence, the court held that Dr. Hammer's testimony was unreliable. Although it did not explicitly state it was doing so, the court effectively ruled Dr. Hammer's testimony as to breach and causation inadmissible and, therefore, concluded there was no evidence to support the jury's verdict.[15]

We conclude that Dr. Hammer's testimony was sufficiently reliable to sustain the jury's verdict. At trial, Dr. Hammer testified that his opinion was based on basic principles of medicine that he learned in medical school and while working in the ICU. Dr. Hammer is a board certified pediatrician and anesthesiologist with a sub-board in pediatric critical care medicine. He is also a professor of anesthesiology and pediatrics at Stanford Medical School. He has authored between 70 and 80 journal articles, a book, and 25 to 30 textbook chapters on various subjects including the use of Propofol, the post-operative use of anesthesia in children, and the use of anesthesia in pain management treatment. His professional focus is on pediatric pharmacology, or drug disposition in children. Based on these facts, it is reasonable to conclude Dr. Hammer was familiar enough with basic principles of medicine to form his opinion.

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thus was not the type of refractory acidosis associated with PRIS. No other signs of PRIS were discovered during Michael's autopsy. Instead, the autopsy indicated that Michael was in "good condition" and was not suffering from any disease or sickness.

[14] The court's finding of statistical insignificance related to a study noted in the PDR where 9% of critically ill pediatric patients in the ICU who were sedated with Propofol died compared to only 4% of similar patients sedated with "standard sedative agents." The court concluded the study was statistically insignificant "to establish a causal, rather than an associational link between the use of [P]ropofol and the deaths." It based its conclusion on the testimony of Dr. Reed, the author of the study.

[15] The court did not, however, reconsider its ruling regarding Dr. Hammer's qualification to testify as to the applicable standard of care.

There was also evidence that Dr. Hammer was familiar with the use of Propofol in pediatric patients for post-operative treatment. Dr. Hammer testified that he works primarily in the ICU and in the operating room doing pediatric anesthesia. He indicated that he is familiar with post-operative treatment and works with various pediatric surgical specialists, including plastic surgeons and general pediatric surgeons. Dr. Hammer himself has used Propofol for short-term sedation of children and has read several articles over the years regarding the use of Propofol. Based on his acquired knowledge and review of the Diprivan entry in the PDR, Dr. Hammer concluded the doctors' negligent administration of Propofol was the proximate cause of Michael's death.

In light of these facts, Dr. Hammer's opinion as to the cause of Michael's death was scientifically reliable. *See Weeks*, 143 Idaho at 837, 153 P.3d at 1183 (holding that a medical doctor's "education, specialized knowledge, and thirty years of experience establishes that he met the test outlined in I.R.E. 702 to establish him as an expert"). Although his opinion was not directly supported by any peer-reviewed articles, that support is not a necessary finding for scientific reliability. *See Thomson v. Olsen*, 147 Idaho 99, 108, 205 P.3d 1235, 1243 (2009) (holding that the lack of specific language in medical literature on a subject is not dispositive). Dr. Hammer explained that there were no articles directly supporting his opinion because "[t]here would be nothing interesting or new about that to publish." In addition, he indicated he was not aware of anyone who used Propofol for long-term, high-dose sedation of pediatric ICU patients and, thus, there were no instances of such use to study. This testimony is consistent with the ethical considerations associated with testing potentially lethal doses of medication on pediatric test subjects. In any event, according to Dr. Hammer, there were several articles establishing connections between each cause and effect in the chain of events that led to his conclusion. Specifically, there were studies indicating Propofol was toxic and tended to cause hypotension, which, in turn, could cause cerebral edema. Thus, like the testimony at issue in *Weeks*, there was direct scientific evidence to support Dr. Hammer's opinion. Unlike the situation in *Swallow*, Dr. Hammer did not rely solely on the temporal proximity between the administration of Propofol and Michael's death. Instead, he was able to provide a scientific explanation of the effect Propofol had on Michael and how it caused his death. He explained the chain of circumstances leading to the cerebral edema

and the large role Propofol played in those events. Accordingly, under *Weeks* and *Swallow*, Dr. Hammer's testimony was sufficiently reliable to prove the doctors' negligence proximately caused Michael's death.

Mrs. Coombs further contends that there was substantial and competent evidence to support the jury's verdict that the doctors' prolonged administration of Propofol was negligent.[16] Specifically, Mrs. Coombs argues the doctors were negligent because they were unfamiliar with using the drug for long-term sedation of children and failed to research such use, misused the drug, dismissed warnings from a nurse about using the drug, did not ask the anesthesiologist to check on Michael after the surgery or ask his advice regarding long-term sedation, failed to notice the distress Michael was in while on Propofol, failed to relieve the distress by discontinuing Propofol, and failed to control Michael's extremely low blood pressure. According to Mrs. Coombs, the doctors' negligent administration of Propofol was the proximate cause of Michael's death.

The doctors argue that there was no evidence they were negligent or that their negligence caused Michael's death. They point out that, at the time they were treating Michael, there were no reported medical data showing Propofol had ever caused death from cerebral edema alone, describing cerebral edema associated with PRIS, or indicating that long-term, high-dose use of Propofol in children may be fatal in the absence of the signs of PRIS. As such, they contend Mrs. Coombs failed to meet her burden as to both breach and causation.

The district court concluded Mrs. Coombs did not present sufficient evidence to support the jury's verdict.[17] After weighing the expert testimony admitted at trial, the court concluded Mrs. Coombs failed to prove that the doctors' negligence was the actual cause of Michael's death. Because Dr. Hammer's hypothesis had not been tested, the court reasoned there was not substantial evidence for the jury to conclude the doctors' long-term administration of Propofol caused Michael's death. Alternatively, it concluded that even if there was evidence of proximate cause, there was no evidence the doctors breached the applicable standard of care. It reasoned there was no evidence indicating

[16] She does not dispute the use of the drug during surgery or immediately thereafter.

[17] In the court's view, the evidence presented was "grossly insufficient." Yet it denied the doctors' motions for summary judgment on the grounds that Dr. Hammer's testimony created a genuine issue of material fact for the jury.

Propofol created the risk of cerebral edema absent the other signs of PRIS. Thus, the doctors were under no obligation to monitor for cerebral edema and they acted in accordance with the standard of care by monitoring for the known signs of PRIS.

As mentioned above, the district court's grant of a j.n.o.v. will only be upheld if it is supported by substantial and competent evidence. *Jeremiah*, 131 Idaho at 247, 953 P.2d at 997. Because Dr. Hammer's testimony was properly part of the record to be considered by the jury, there was substantial and competent evidence to support the jury's verdict. Dr. Hammer testified that he held an opinion regarding the doctors' failure to meet the standard of care, that he could testify to his opinion with reasonable medical certainty, and that he had actual knowledge of the applicable community standard of care in Boise, Idaho, in 2002. He further testified that it was his opinion that the doctors breached the applicable standard of care and that their negligence was the proximate cause of Michael's death.

Dr. Hammer testified that the applicable standard of care for the long-term administration of Propofol in pediatric ICU patients in Boise, Idaho, in 2002, was the same as the national standard. Under that standard, doctors prescribing Propofol should be aware of the consequences of the drug and its indications for use and should not use the drug "overnight or for more prolonged periods, say, exceeding 12 hours." In the uncommon event prolonged administration is necessary, it should be done "under very proscribed dosing and very close monitoring of a variety of laboratory tests." In addition, no more than 67 micrograms per kilogram per minute were recommended for long-term use.

In regards to Dr. Griffiths' violation of the standard of care, Dr. Hammer testified that Dr. Griffiths was:

> principally . . . responsible for the use of Propofol in a relatively high dose for a prolonged period of time in a critically ill child without titrating or modifying the dose of the drug when the blood pressure was low, for example, and when there were signs of organ toxicity, and also did not meet the standard of care with respect to ordering lab tests that would serve to monitor organ toxicity related to Propofol over time.[18]

[18] He also testified Dr. Griffiths breached the standard by not asking Dr. Smagula more specifics on long-term Propofol use.

Dr. Hammer testified he had the same opinion with respect to Dr. Curnow because he "was sharing responsibility for the child." According to Dr. Hammer, Dr. Curnow violated the standard of care by choosing Propofol "in the dose used, for the duration of time used, without appropriate laboratory monitoring and adjustments in the dose, according to what was happening with the child over time."

Next, Dr. Hammer testified that, in his medical opinion, the doctors' negligent administration of Propofol was the proximate cause of Michael's death. In reaching his conclusion, Dr. Hammer thoroughly analyzed the role Propofol played in causing Michael's death. He noted three consequences of long-term Propofol use that contributed to the cerebral edema that resulted in Michael's death. First, he testified that Propofol tends to cause blood pressure to drop below a safe threshold, if "not used very carefully," by depressing heart function and dilating blood vessels, arteries, and veins.[19] In pediatric patients, this was especially problematic given their size, age, and "physiologic circumstances" because it results in inadequate blood flow to "vital organs in the body, including the brain." Low blood pressure (hypotension) associated with "Propofol can cause and . . . exacerbate hypoxic ischemia" resulting from an "inadequate amount of blood, and therefore, oxygen delivery to cells." Consequently, when a patient on Propofol experiences low blood pressure, the infusion rate should be decreased.

Second, administering Propofol over a prolonged period of time (over twelve hours) could result in the build up of lipids, or fat, in the blood. Because Propofol is "not very soluble in the blood," it is mixed with an emulsion that contains significant amounts of fat that is administered with the drug through an I.V. Administering the drug over a prolonged period of time causes the fat in the patient's blood stream to rise.[20] When the fat breaks down, it produces dangerous fatty acids, causing lipidemia and fatty acid toxicity, which poisons mitochondrion and damages the cells. As Dr. Hammer explained:

> [W]hen there's a lot of fatty acids, the mitochondrion, which are the little machines inside the cells that produce energy for the cells, don't function normally, so they may suppress energy production in the cell, and that energy source, which is called ATP, is very vital for the cells to stay alive

[19] Although Dr. Hammer admitted other sedatives could also decrease blood pressure (Versed and morphine), the decrease would not be to the same degree or have such an impact on heart contractility.

[20] Prolonged administration is problematic because "the toxicity and adverse effects associated with Propofol are related to the dose and the duration of the administration, so the lower the dose and for the shorter the duration, the safer the drug is."

> and do all the things that they do that require energy. So fatty acids may actually suppress energy production in cells and cause cells to malfunction.

Third, Propofol interferes with the mitochondrial function directly and, therefore, "decreases the amount of ATP . . . that cells produce." When this occurs, it diminishes the integrity of the cell by preventing it from pumping water out, resulting in the cell becoming swollen and potentially dying. Dr. Hammer explained long-term Propofol use causes:

> mitochondrial dysfunction, lack of energy production, cellular swelling or edema, and potentially cell death, especially if those cells are in a closed compartment like the head, the brain swells, then the pressure inside the head goes up, and that becomes a runaway cycle of swelling, pressure, death, et cetera.

In Dr. Hammer's opinion, these disadvantages of long-term Propofol use combined and were "the No. 1 factor" in causing Michael's death. Dr. Hammer did not, however, attribute Michael's death to PRIS.[21] Instead, it was his opinion that the well-established adverse effects of Propofol, including hypotension and its resulting adverse effects on the organs, hyperlipidemia, and the toxic effects of high levels of triglycerides in the bloodstream caused the child's death. He testified in relevant part:

> I think the arrhythmias and the brain swelling were both caused by the same process, which is hypoxia ischemia, and cellular injury, toxicity.
>
> So I think there was a period of injury to those organs and other organs in the body because of inadequate blood flow, inadequate or low hemoglobin, and also, I think, toxic effects of the Propofol combined.
>
> . . . .
>
> The Propofol definitely caused or contributed to the low blood pressure hypotension. The Propofol definitely caused the lipemia, and in all likelihood, caused toxicity to cells in the body. The low hemoglobin was caused by bleeding from the surgical site.
>
> . . . .
>
> Propofol caused or contributed significantly to the hypotension, and therefore, decrease in blood flow to vital organs in the body, including the brain, the heart, the liver, and other vital tissues.

[21] According to Dr. Hammer, PRIS "relates to a specific mitochondrial toxicity; not the absence of oxygen or glucose delivered to the mitochondrion related to hypotension."

> It also caused the lipemia that in all likelihood contributed to his demise because of the fatty acids that would be produced. And in all likelihood, the Propofol also had a negative effect on the energy generation of cells in the body.
>
> And these are all in combination, especially with the low hemoglobin that the Propofol did not cause, but these events taking place simultaneously all resulted like a triple or quadruple whammy, where they all resulted in diminished oxygen delivery to the brain, which ultimately caused the brain swelling, and also in the body, for which there's evidence, so I think all these things happened together in a predictable way to produce his death.
>
> The low hemoglobin, again, was caused by bleeding. I would emphasize that in the presence of bleeding and the low blood pressure that's caused or contributed to by the bleeding itself, one would have to be particularly mindful of the Propofol-related drop in blood pressure, so if a person is bleeding and their hemoglobin is low and their blood pressure is low, that's the time to decrease the Propofol or turn it off.
> . . . .
> I think almost every article about Propofol or every textbook reference to Propofol indicates that Propofol causes hypotension, or may cause hypotension, and there are textbook chapters and lots of other articles written about the association between hypotension and hypoxia ischemia of the brain and subsequent brain swelling and brain death. So Propofol causes hypotension, hypotension is associated with hypoxia ischemia, hypoxia ischemia is associated with cerebral edema. So it goes without saying, for physicians, at least, that Propofol can cause that series of events leading to cerebral edema.

By failing to adequately monitor and account for the disadvantages associated with long-term, high-dose Propofol use, Dr. Hammer testified that the doctors breached the applicable standard of care. Dr. Hammer noted that Michael's blood pressure was critically low for long periods of time. He indicated such low blood pressure was a danger signal that should have been, but was not, responded to by lowering the Propofol dose. Michael's high fever exacerbated the danger associated with the low blood pressure because it was well known at that time that fever increases the need for blood flow to the brain. In addition, the heart arrhythmias Michael experienced were likely caused by ischemia, which provided the doctors with an additional indication of oxygen deprivation associated with low blood pressure. In Dr. Hammer's opinion, Propofol should have been stopped or the dosage lowered once the tissue was detached and when the fever set in

because, at that point, Michael did not need to be immobilized.[22] Doing so would have resolved the hypotension Michael was experiencing. Alternatively, even assuming the high dosage of Propofol being administered was necessary, the doctors should have given Michael another medication to increase his blood pressure.

In addition to their failure to account for Michael's low blood pressure, Dr. Hammer testified that the doctors should have taken additional measures to monitor Michael and respond to the conditions revealed by such monitoring. First, he testified that the physicians acted negligently by setting and maintaining a minimum order of Propofol with no caveats for low blood pressure or lipidemia. Even after a nurse indicated to Dr. Curnow that there were consequences of using Propofol under the circumstances, he did not change the order. Second, they should have monitored the muscle enzyme (CK) levels inside Michael's cells, which would have been indicative of muscle damage associated with Propofol.[23] Third, because Michael was receiving four to six times the amount of lipids Dr. Hammer thought were justified under the circumstances, the doctors should have also measured triglyceride levels daily. Neither doctor, however, ordered either to be monitored. Fourth, Michael should have received additional glucose because his levels were at less than half of what was required. Administering more glucose was necessary to provide energy that would prevent lipids from breaking down and creating toxic fatty acids. Fifth, Dr. Hammer indicated Propofol should have been periodically "lightened up" to make sure Michael was responsive and neurologically intact. For all of these reasons, and in light of warnings contained in the PDR,[24] Dr. Hammer concluded

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[22] Michael was receiving a general anesthetic dose of 100 to 150 micrograms per kilogram of Propofol per minute over a period of four days. Dr. Hammer testified:

> I don't see any indication for keeping the child under general anesthesia during this time, and there were lots of indications for decreasing the dose or turning the drug off altogether over time, so that the dose that was used was excessive and was not adjusted according to basic abnormalities that were observed, or should have been observed.

[23] The autopsy, however, indicated Michael did not suffer from rhabdomyolysis, the muscle damage associated with Propofol.

[24] The warnings cautioned against the use of Propofol to sedate pediatric patients in the ICU and identified the drug's adverse effects. Relevant passages of the PDR indicate:

> [T]itration to clinical response and daily evaluation of sedation levels are important during use of [Propofol] for ICU sedation, especially of long duration.
>
> . . .

that the doctors negligently administered Propofol to Michael and that their negligence was the proximate cause of his death.

In light of this evidence, the district court's decision, granting the doctors' motions for j.n.o.v., invaded the province of the jury. Although the doctors point to evidence in the record tending to contradict Dr. Hammer's opinion, the jury was entitled

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> With medical ICU patients or patients who have recovered from the effects of general anesthesia or deep sedation, the rate of administration of 50mg/kg/min or higher may be required to achieve adequate sedation. These higher rates of administration may increase the likelihood of patients developing hypotension.
> . . .
> [Propofol] is not indicated for use in Pediatric ICU sedation since the safety of this regimen has not been established.
> . . .
> Patients should be continuously monitored for early signs of significant hypotension and/or bradycardia. Treatment may include increasing the rate of intravenous fluid, elevation of lower extremities, use of pressor agents, or administration of atropine.
> . . .
> Since [Propofol] is formulated in an oil-in-water emulsion, elevations in serum triglycerides may occur when [Propofol] is administered for extended periods of time. Patients at risk of hyperlipidemia should be monitored for increases in serum triglycerides or serum torbidity. Administration of [Propofol] should be adjusted if fat is being inadequately cleared from the body. A reduction in the quantity of concurrently administered lipids is indicated to compensate for the amount of lipid infused.
>
> In pediatric patients, administration of fentanyl concomitantly with [Propofol] may result in serious bradycardia.
>
> [Propofol] is not indicated for use in pediatric patients for ICU sedation . . . for surgical, nonsurgical or diagnostic procedures as safety and effectiveness have not been established.
> . . .
> In one multicenter clinical trial of ICU sedation in critically ill pediatric patients that excluded patients with upper respiratory tract infections, the incidence of mortality observed in patients who received [Propofol] was 9%, while that for patients who received standard sedative agents was 4%. While causality has not been established, [Propofol] is not indicated for sedation in pediatric patients until further studies have been performed to document its safety in that population.
> . . . .
> Dosage and rate of administration should be individualized and titrated to the desired effect, according to clinically relevant factors including preinduction and concomitant medications, age, ASA physical classification, and level of debilitation of the patient.
>
> [Propofol] should be individualized according to the patient's condition and response, blood lipid profile, and vital signs.
>
> Evaluation of level of sedation and assessment of CNS Function should be carried out daily throughout maintenance to determine the minimum dose . . . required for sedation.
>
> Safety and dosing requirements for induction of anesthesia in pediatric patients have only been established for children 3 years of age or older.

to rely on his opinion and weigh the conflicting evidence in the record. Based on the testimony outlined above, there was substantial and competent evidence to support the jury's verdict.

**D.**

Both doctors request attorney fees on appeal pursuant to Idaho Code section 12-121. Under Idaho Code section 12-121, a court may award attorney fees to the prevailing party in a civil action. I.C. § 12-121. Because neither of the doctors is a prevailing party on this appeal, they are not entitled to an award of attorney's fees under the statute.

**III.**

Accordingly, we vacate the j.n.o.v. and remand for entry of judgment pursuant to the jury verdict effective as of September 17, 2007.

Chief Justice EISMANN, and Justices BURDICK AND W. JONES CONCUR.

HORTON, J., specially concurring.

I join in the Court's decision in all respects, save for Part II (C). I join in the conclusion stated in that section of the opinion in which the Court holds that there was substantial evidence to support the jury's verdict. However, I am unable to join in the analysis contained in that section to the extent that it evaluates the admissibility of the evidence presented by Mrs. Coombs. In view of the conclusion that there was substantial evidence supporting the jury's verdict, the discussion of the admissibility of the evidence considered by the jury is merely dicta. Further, by engaging in the analysis of the admissibility of the evidence, I believe that the Court's decision repeats the error committed by the trial court and creates potential confusion.

By way of explanation, I start with the trial court's ruling on the doctors' motion for j.n.ov. The trial court's memorandum opinion began by stating the applicable legal standards governing a motion for j.n.o.v. The trial court correctly stated the standard of reviewing governing such motions. However, when addressing whether substantial evidence supported the jury's verdict, the trial court relied upon a statement found in *Daubert v. Merrell Dow Pharmaceuticals, Inc.,* 509 U.S. 579 (1993). In *Daubert,* the United States Supreme Court held that in the federal courts, there are "conventional

devices" available to the courts to address situations involving "shaky but admissible evidence." *Id.* at 596. The *Daubert* court identified directed judgments and summary judgment as such devices. *Id.* In Idaho, a motion for j.n.o.v. is simply treated as a delayed motion for a directed verdict, and the same standards are applied. *Leavitt v. Swain*, 133 Idaho 624, 628, 991 P.2d 349, 353 (1999); *Quick v. Crane*, 111 Idaho 759, 764, 727 P.2d 1187, 1192 (1986). It appears to me that the source of the trial court's error was applying standards applicable to federal courts in reaching its decision.

In this state, the appellate courts and the trial courts apply an identical standard in evaluating a motion for j.n.o.v. *Quick*, 111 Idaho at 764, 727 P.2d at 1192. As noted in Part II (B) of the decision, Idaho courts are "bound by the record" and may not retrospectively deem evidence presented to the jury to have been inadmissible and then, based upon a consideration of the remaining evidence, determine whether there was substantial evidence supporting the verdict. This stands in stark contrast to the federal approach, wherein the appellate courts are permitted to do otherwise. *Weisgram v. Marley Co.*, 528 U.S. 440, 457 (2000) (holding "that the authority of courts of appeals to direct the entry of judgment as a matter of law extends to cases in which, on excision of testimony erroneously admitted, there remains insufficient evidence to support the jury's verdict").

The trial court's reliance on the procedural principle articulated in *Daubert* explains why the trial court analyzed the sufficiency of the evidence presented to the jury in terms of the admissibility of that evidence, ultimately concluding that the evidence was not reliable and therefore did not constitute substantial evidence upon which the jury's verdict could rest. This was error. I believe that the portion of this Court's opinion that similarly addresses the admissibility of Dr. Hammer's opinion testimony replicates that error and potentially creates confusion as to the standard of review applicable to motions for a direct verdict or j.n.o.v.

By moving for j.n.o.v., the doctors "necessarily admitted the truth of all of the plaintiffs' evidence and every legitimate inference that could be drawn therefrom in the light most favorable to the plaintiff." *Quick*, 111 Idaho at 763, 727 P.2d at 1191 (citing *Stephens v. Stearns*, 106 Idaho 249, 252-53, 678 P.2d 41, 44-45 (1984)). In my view, the critical facts identified in Part II (C) that require this Court to uphold the jury's verdict

are simply these: (1) Dr. Hammer expressed and explained his opinion that the administration of Propofol was the cause of Michael's death; and (2) Dr. Hammer expressed and explained his opinion that the doctors breached the applicable standard of health care practice. This was substantial evidence; once presented to the jury, it was up to the jurors to accept or reject Dr. Hammer's opinions, as "[w]eighing the testimony of expert witnesses is uniquely within the competence of the trier of fact." *City of McCall v. Seubert*, 142 Idaho 580, 585, 130 P.3d 1118, 1123 (2006) (quoting *Rueth v. State*, 103 Idaho 74, 78, 644 P.2d 1333, 1337 (1982)).[25]

[25] Indeed, our standard jury instruction advises jurors: "The law does not require you to believe all of the evidence admitted in the course of the trial. As the sole judges of the facts, you must determine what evidence you believe and what weight you attach to it." IDJI 1.00.