STATE OF MICHIGAN

COURT OF APPEALS

ALEXANDER FIGURSKI, minor, by his conservator, HOWARD LINDEN,

Plaintiff-Appellant,

v

TRINITY HEALTH-MICHIGAN, d/b/a/ SAINT JOSEPH MERCY LIVINGSTON HOSPITAL, WILLIAM BRADFIELD, M.D., and CATHERINE McCAULEY HEALTH SERVICES CORPORATION, a/k/a SAINT JOSEPH MEDICINE FACULTY ASSOCIATES, a/k/a SAINT JOSEPH MERCY PRIMARY CARE,

Defendants-Appellees.

UNPUBLISHED
March 5, 2015

No. 318115
Livingston Circuit Court
LC No. 11-026466-NH

ALEXANDER FIGURSKI, minor, by his conservator, HOWARD LINDEN,

Plaintiff-Appellee,

v

TRINITY HEALTH-MICHIGAN, d/b/a/ SAINT JOSEPH MERCY LIVINGSTON HOSPITAL, WILLIAM BRADFIELD, M.D., and CATHERINE McCAULEY HEALTH SERVICES CORPORATION, a/k/a SAINT JOSEPH MEDICINE FACULTY ASSOCIATES, a/k/a SAINT JOSEPH MERCY PRIMARY CARE,

Defendants-Appellees.

No. 319086
Livingston Circuit Court
LC No. 11-026466-NH

Before: SAAD, P.J., and OWENS and K. F. KELLY, JJ.

PER CURIAM.

In this obstetrical medical malpractice action, plaintiff Alexander Figurski alleges that he sustained a hypoxic-ischemic injury and left middle cerebral arterial ischemic stroke (or PAIS) during labor and delivery.[1]

In Docket No. 318115, this Court granted plaintiff's application for leave to appeal an August 21, 2013 order of Livingston Circuit Judge Theresa M. Brennan,[2] which granted defendants' motion in limine to exclude plaintiff's causation expert concerning claims of perinatal malpractice and granting partial summary disposition as to those claims.[3] In that case, we hold that the trial court exceeded its role as gatekeeper and, instead, acted as the ultimate trier of fact. Because there was sufficient reliable scientific data in support of plaintiff's expert's opinion, we reverse and remand for further proceedings.

In Docket No. 319086, this Court granted defendants' application for leave to appeal an August 7, 2013 order of Judge Brennan denying their motion in limine to exclude plaintiff's economic expert.[4] In that case, we hold that the trial court properly exercised its gatekeeping function and affirm the order denying defendants' motion in limine.

I. BASIC FACTS

Mary Figurski was admitted to the hospital with ruptured membranes at approximately 11:30 p.m. on March 7, 2008. She had a lengthy labor – 29 hours – augmented by the drug Pitocin, beginning at 4:55 a.m. on March 8th. The baby failed to descend after two hours of pushing. Mary had developed chorioamnionitis (placental infection) and the doctors noticed the presence of meconium, which was an indication that the baby had a bowel movement in utero. The labor was terminated with a caesarian section at 4:45 a.m. on March 9, 2008. Records revealed "delivery of a healthy baby boy who weighed 9 lbs 13 oz." However, plaintiff began to show signs of seizing shortly after his birth and a CT scan performed on March 10th revealed an acute left middle cerebral artery ischemic stroke, which was "days to hours old."

Plaintiff's complaint was filed on December 28, 2011. The complaint alleged that "[t]he baby was at risk for, and did develop, brain injury from traumatic head compression and regional cerebral ischemia caused by failure to descend, macrosomia (large baby), excessive contractions in the presence of failure of descent as augmented with Pitocin, hypoxia-ischmeia (regional cerebral and/or systemic) caused by uteroplacental insufficiency and by cord compression and head compression." The complaint alleged that defendants were negligent in administering

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[1] Plaintiff also alleges that the injuries were caused by negligence after the delivery, but those injuries are not at issue in these appeals.

[2] Judge Brennan is a district court judge, sitting by assignment in the circuit court.

[3] *Figurski v Trinity Health-Michigan,* unpublished order of the Court of Appeals, entered April 8, 2014 (Docket No. 318115).

[4] *Figurski v Trinity Health-Michigan,* unpublished order of the Court of Appeals, entered April 8, 2014 (Docket No. 319086).

Pitocin, in failing to properly respond to fetal heart rate changes, and in failing to perform a timely c-section.

Plaintiff attached numerous affidavits of merit regarding breach of the standard of care for the various professionals involved, including that of Dr. Carolyn Crawford. Crawford opined that a number of factors led to plaintiff's injuries, including: mother in first time labor (primagravida), chorioamnionitis, prolonged rupture of membranes, prolonged second stage of labor, large child for the size of the mother's pelvis, fetal heart rate abnormalities, and birth trauma augmented by the use of Pitocin.

Defendants filed a motion to prevent plaintiff from pursuing a claim of global hypoxic ischemic injury secondary to trauma from the use of Pitocin and the forces of labor. Citing MCR 2.116(C)(10), MRE 702, and MCL 600.2955, defendants argued that plaintiff should not be able to pursue the claim where plaintiff had only been diagnosed with a left middle cerebral artery stroke; there was no hypoxic-ischemic injury other than the stroke. Defendant further argued that there was no scientific basis to support Crawford's opinion that the alleged hypoxic-ischemic injuries were caused by the mechanical trauma from Pitocin and the forces of labor and noted that this same theory of injury was rejected in *Craig v Oakwoood Hosp*, 471 Mich 67; 684 NW2d 296 (2004). Defendants argued that because there was no admissible expert testimony on proximate cause, plaintiff's claims regarding global hypoxic-ischemic injury should be dismissed.

Defendants filed a concurrent motion in limine or for summary disposition, seeking to preclude plaintiff from claiming that intrapartum care and treatment caused the left middle cerebral artery ischemic stroke. Defendants made many of the same arguments set forth in their motion to prevent plaintiff from pursuing a claim of global hypoxic ischemic injury, but this particular motion specifically addressed the perinatal arterial ischemic stroke – an injury that is not contested. While defendants acknowledged that certain *risk factors* for PAIS had been identified, the *cause* of the stroke continued to be unknown. Defendants argued that the medical literature upon which Crawford relied did not provide a reliable scientific basis for her ultimate opinion. Even if risk factors could show causation, the literature provided that ischemic stroke occurs in only 1 of every 200 live births with three or more known risk factors present, as opposed to 1 in 5,000. Defendants urged that because there was no admissible expert testimony on proximate cause, plaintiff's claims regarding the left middle cerebral artery stroke injury should be dismissed.

Plaintiff filed a combined response to both motions including as an exhibit, Crawford's May 4, 2013 affidavit wherein Crawford restates her opinion about the mechanism of plaintiff's injuries. Attached to her affidavit were 51 exhibits that Crawford utilized in reaching her opinion.[5]

[5] Plaintiff also included as exhibits, the affidavit and supporting papers for another expert – Dr. Barry S. Schifrin. However, Judge Brennan permitted plaintiff to present only one expert at the *Daubert* hearing and plaintiff chose to present Crawford.

Crawford averred that "the scientific literature establishes that the mechanism of trauma/head compression is injurious to the brain during parturition (labor/delivery) and more specifically that trauma/head compression can cause ischemic stroke." She concluded that defendants' negligence caused the stroke and that "[t]he PAIS would not have occurred but for the Defendants negligently allowing multiple known risk factors for PAIS to continue unabated and unresolved during such a prolonged and difficult labor" including "primagravida, chorioamnionitis, prolonged labor, prolonged second stage, and FHR [fetal heart rate] abnormalities."

Crawford also averred that defendants' conduct resulted in a global hypoxic-ischemic injury:

> I have reviewed the scientific literature. There is a sound scientific basis for the opinion that excessive cephalic pressure will and does cause ischemic injury in the fashion described herein. I have seen and/or reviewed in the literature a number of cases where this etiology is the reasonable and probable cause of injury. In my opinion, these forces were to a reasonable degree of medical probability the cause of Alexander Figurski's traumatic/ischemic/hypoxic brain damage at birth.

A hearing on defendants' motions took place on June 13, 2013. Both defense counsel and plaintiff's counsel made long and impassioned arguments regarding the admissibility of Crawford's testimony and whether more than one injury existed. The trial court indicated it would render its opinion at a later date. However, rather than issuing an opinion on the issue, the trial court entered an order indicating that, even after a searching inquiry of the articles presented, it was unable to determine whether plaintiff's experts should be able to testify as to causation. The order provided:

> When reviewing the medical literature supplied by the Plaintiff, the Court was cognizant of the opinion of Plaintiff's causation experts. The Court understands that the Plaintiff's causation experts opine that " . . .excessive compression of the fetal head caused by uterine tachysystole, hyperstimulation, uterine hypertonicity, prolonged labor, prolonged rupture of membranes and relative cephalopelvic disproportion . . ."
>
> The searching inquiry does not allow this Court to conclude that the Plaintiff's causation experts' opinions meet the requirements of MRE 702 and MCL 600.2955. A *Daubert*[6] hearing is necessary to determine if the opinions of Plaintiff's expert witnesses regarding proximate causation for Plaintiff's left middle cerebral artery stroke and global hypoxic-ischemic injury satisfies the requirements of MRE 702 and MCL 600.2955. Because the opinions of

[6] *Daubert v Merrell Dow Pharm, Inc*, 509 US 579; 113 S Ct 2786; 125 L Ed 2d 469 (1993).

> Plaintiff's causation experts do not differ, it is only necessary for one of Plaintiff's causation experts to testify at the *Daubert* hearing. The Plaintiff may select which doctor will testify.

After the *Daubert* hearing, where Dr. Crawford was the only witness to testify, the trial court issued an opinion and order granting defendants' motions in limine and for partial summary disposition. The trial court concluded that the basis for plaintiff's expert's opinions had not been subjected to scientific testing or peer review, and that there was no evidence that her theory of causation was generally accepted within the relevant medical community. The trial court found that Crawford did not present a reliable basis for her opinion, and there was no evidence her opinion had been relied on outside the context of litigation. Based on the exclusion of the expert testimony, the court found that there was no support for plaintiff's perinatal claims, and it granted partial summary disposition as to those claims.

At the same time that the parties battled over plaintiff's causation experts, defendants also filed a motion in limine to preclude plaintiff's economics expert, Dr. Anthony Gamboa, from testifying. Defendants argued that Gamboa's opinion was based on "junk science" and contrary to MRE 702 and MCL 600.2955. Defendants claimed that Gamboa, who did not have a degree in economics, was not qualified to offer an opinion. They argued that because he lacked a proper understanding of basic economic principles, his methodologies were flawed and were unreliable.

The issue was debated at a June 13, 2013 hearing. The trial court issued a written opinion and order on August 7, 2013, denying defendants' motion in limine to exclude plaintiff's economic expert.

II. STANDARDS OF REVIEW

> [A] court of appeals is to apply an abuse-of-discretion standard when it reviews a trial court's decision to admit or exclude expert testimony. That standard applies as much to the trial court's decisions about how to determine reliability as to its ultimate conclusion. Otherwise, the trial judge would lack the discretionary authority needed both to avoid unnecessary "reliability" proceedings in ordinary cases where the reliability of an expert's methods is properly taken for granted, and to require appropriate proceedings in the less usual or more complex cases where cause for questioning the expert's reliability arises. [*Kumho Tire Co, Ltd v Carmichael*, 526 US 137, 152; 119 S Ct 1167, 1176; 143 L Ed 2d 238 (1999) (internal quotation marks and citation omitted).]

However, our Court has recently explained:

> We review for an abuse of discretion a circuit court's evidentiary rulings. When our inquiry concerns whether the trial court correctly applied a rule of evidence, our review is de novo. Thus, we apply de novo review in assessing whether the trial court performed its gatekeeping role in conformity with the legal principles articulated in *Gilbert v DaimlerChrysler Corp*, 470 Mich 749; 685 NW2d 391 (2004), in which our Supreme Court adopted the *Daubert* framework. If the trial court correctly executed its gatekeeping role, we review its ultimate

> decision to admit or exclude scientific evidence for an abuse of discretion. When a trial court excludes evidence based on an erroneous interpretation or application of law, it necessarily abuses its discretion. [*Elher v Misra*, ___ Mich App ___; ___ NW2d ___ (Docket No. 316478, issued December 2, 2014) slip op, p 7 (internal citations and footnote omitted).]

III. GENERAL REVIEW OF THE LAW ON EXPERT TESTIMONY

> In order to establish a cause of action for medical malpractice, a plaintiff must establish four elements: (1) the appropriate standard of care governing the defendant's conduct at the time of the purported negligence, (2) that the defendant breached that standard of care, (3) that the plaintiff was injured, and (4) that the plaintiff's injuries were the proximate result of the defendant's breach of the applicable standard of care. [*Craig*, 471 Mich at 86.]

Proximate cause involves both the "cause in fact" and the "legal cause." *Skinner v Square D Co*, 445 Mich 153, 162-63; 516 NW2d 475 (1994). The first requires a showing that "but for" defendants' action, plaintiff would not have been injured whereas the latter focuses on foreseeability and whether a defendant should be held legally responsible for such consequences. *Id.* "A plaintiff must adequately establish cause in fact in order for legal cause or 'proximate cause' to become a relevant issue." *Id.*

There is no question that plaintiff suffered a perinatal arterial ischemic stroke, or PAIS. What is at issue is the connection between plaintiff's injury and defendants' conduct, both "but for" and "proximate" causation. "[A] plaintiff's prima facie case of medical malpractice must draw a causal connection between the defendant's breach of the applicable standard of care and the plaintiff's injuries." *Craig*, 471 Mich at 90. Crawford opines that "excessive compression of the fetal head caused by uterine tachysystole, hyperstimulation, uterine hypertonicity, prolonged labor, prolonged rupture of membranes, and relative cephalopelvic disproportion" caused the stroke. Defendants counter that there is nothing in the medical literature to support such a position and that the cause of PAIS remains largely unknown. While plaintiff contends that he also suffered a global hypoxic ischemic injury, defendants counter that no such injury was detected by plaintiff's treating physicians. These issues are clearly beyond the realm of the average lay person.

MRE 702 provides:

> If the court determines that scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education may testify thereto in the form of an opinion or otherwise if (1) the testimony is based on sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

"The admission of expert testimony requires that (1) the witness be an expert, (2) there are facts in evidence that require or are subject to examination and analysis by a competent expert, and (3)

the knowledge is in a particular area that belongs more to an expert than to the common man." *Surman v Surman*, 277 Mich App 287, 308; 745 NW2d 802 (2007). The so-called "trilogy of restrictions on expert testimony" includes a searching inquiry into "qualification, reliability, and fit." *Elher*, slip op, p 8. Defendants do not question Crawford's qualifications, but they take issue with reliability and fit. As the party offering the evidence, plaintiff bore the burden of persuading the trial court that the expert's opinion is based on a recognized field and methodology. *Craig*, 471 Mich at 80.

"MRE 702 requires the trial court to ensure that each aspect of an expert witness's proffered testimony—including the data underlying the expert's theories and the methodology by which the expert draws conclusions from that data—is reliable." *Gilbert v DaimlerChrysler Corp*, 470 Mich 749, 779; 685 NW2d 391 (2004), citing *Daubert*. Our Supreme Court has held:

> This gatekeeper role applies to all stages of expert analysis. MRE 702 mandates a searching inquiry, not just of the data underlying expert testimony, but also of the manner in which the expert interprets and extrapolates from those data. Thus, it is insufficient for the proponent of expert opinion merely to show that the opinion rests on data viewed as legitimate in the context of a particular area of expertise (such as medicine). The proponent must also show that any opinion based on those data expresses conclusions reached through reliable principles and methodology.
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> Careful vetting of all aspects of expert testimony is especially important when an expert provides testimony about causation. [*Gilbert*, 470 Mich at 782 (footnote omitted).]

In *Daubert*, the petitioners were minors who had suffered serious birth defects. Along with their parents, the petitioners sued respondent, a pharmaceutical company, alleging that the mothers' ingestion of Bendectin caused the birth defects. The respondent's expert averred that he had looked at a number of published studies and none had concluded that maternal use of Bendectin was a risk factor for birth defects. The petitioners responded with eight experts of their own, who pointed to test tube and live animal studies linking Bendectin to malformations. The petitioners pointed to "pharmacological studies of the chemical structure of Bendectin that purported to show similarities between the structure of the drug and that of other substances known to cause birth defects; and the 'reanalysis' of previously published epidemiological (human statistical) studies." The district court granted the respondent summary judgment because the petitioners' experts' opinions were not generally accepted. The federal appeals court affirmed, citing *Frye v United States*, 293 F 1013, 1014; 54 App DC 46 (1923). *Daubert*, 509 US at 582-584.

In vacating the decision, the United States Supreme Court did away with the "general acceptance" test previously relied upon in *Frye*, which required that before an expert could render an opinion on novel scientific evidence, the theory must have first gained general

acceptance. The Supreme Court concluded that *Frye* had been displaced by FRE 702.[7] *Daubert,* 509 US at 585-589. "That the *Frye* test was displaced by the Rules of Evidence does not mean, however, that the Rules themselves place no limits on the admissibility of purportedly scientific evidence. Nor is the trial judge disabled from screening such evidence. To the contrary, under the Rules the trial judge must ensure that any and all scientific testimony or evidence admitted is not only relevant, but reliable." *Id.* at 589.

Under FRE 702, "[t]he subject of an expert's testimony must be 'scientific knowledge.' The adjective 'scientific' implies a grounding in the methods and procedures of science. Similarly, the word 'knowledge' connotes more than subjective belief or unsupported speculation." *Id.* at 589-590. The Court cautioned that:

> Of course, it would be unreasonable to conclude that the subject of scientific testimony must be 'known' to a certainty; arguably, there are no certainties in science . . . But, in order to qualify as 'scientific knowledge,' an inference or assertion must be derived by the scientific method. Proposed testimony must be supported by appropriate validation—i.e., 'good grounds,' based on what is known. In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establishes a standard of evidentiary reliability. [*Id.* at 590.]

Moreover, the evidence must "fit" and connect to the "pertinent inquiry as a precondition to admissibility" in order to be deemed relevant. *Id.* at 591-592.

The *Daubert* Court explained that, unlike an ordinary witness, an expert was permitted to testify without any firsthand knowledge or observation and "[p]resumably, this relaxation of the usual requirement of firsthand knowledge . . . is premised on an assumption that the expert's opinion will have a reliable basis in the knowledge and experience of his discipline." *Id.* at 592. As a result, trial courts are charged with making "a preliminary assessment of whether the reasoning or methodology underlying the testimony is scientifically valid and of whether that reasoning or methodology properly can be applied to the facts in issue." *Id.* at 592-593. In so doing, "[m]any factors will bear on the inquiry, and we do not presume to set out a definitive checklist or test." *Id.* at 593. The Court then set forth a number of "general observations" that a trial court may consider, including: 1) whether a theory has been tested; 2) whether the theory has been subjected to peer review and publication; 3) the potential rate of error; and 4) whether the theory has gained general acceptance. *Id.* at 593-594. But "[t]he inquiry envisioned by Rule 702 is, we emphasize, a flexible one. Its overarching subject is the scientific validity and thus the evidentiary relevance and reliability—of the principles that underlie a proposed submission. *The focus, of course, must be solely on principles and methodology, not on the conclusions that they generate*." *Id.* at 594-595 (footnote omitted, emphasis added).

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[7] FRE 702 provided: "If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise."

The *Daubert* Court rejected the idea that its decision would result in a "free-for-all" and permit plaintiffs to present unsound evidence to the juries:

> Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence. . . . These conventional devices, rather than wholesale exclusion under an uncompromising 'general acceptance' test, are the appropriate safeguards where the basis of scientific testimony meets the standards of Rule 702. [*Id.* at 596.]

The United States Supreme Court revisited *Daubert* six years later in *Kumho Tire Co, Ltd v Carmichael*, 526 US 137; 119 S Ct 1167; 143 L Ed 2d 238 (1999), when it was called upon to determine how *Daubert* applies to experts who were not scientists. *Kumho* was a products liability case. The plaintiff sued a tire manufacturer after a tire blew and presented an engineering expert who opined that a defect in the tire caused the accident. *Id.* at 141-147. The manufacturer did not challenge the expert's qualifications, but nevertheless argued that the expert's methodology was unreliable. *Id.* at 153.

The Court first concluded that "*Daubert's* general holding—setting forth the trial judge's general 'gatekeeping' obligation—applies not only to testimony based on scientific knowledge, but also to testimony based on 'technical' and 'other specialized' knowledge." *Id.* at 141, 149. Because there are many different experts and various areas of expertise, the factors considered in determining whether to allow an expert to testify must be flexible: "we can neither rule out, nor rule in, for all cases and for all time the applicability of the factors mentioned in *Daubert*, nor can we now do so for subsets of cases categorized by category of expert or by kind of evidence. Too much depends upon the particular circumstances of the particular case at issue." *Id.* at 150. As *Daubert* made clear, "its list of factors was meant to be helpful, not definitive." *Id.* at 151. In performing its gatekeeping requirement, a trial court must "ensure the reliability and relevancy of expert testimony. It is to make certain that an expert, whether basing testimony upon professional studies or personal experience, employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field." *Id.* at 152.

The *Kumho* Court then went on to conclude that the district court did not abuse its discretion when it concluded that the expert's opinion was unreliable because it fell outside the range where experts might reasonably differ. *Id.* at 153. "[N]o one denies that an expert might draw a conclusion from a set of observations based on extensive and specialized experience," but the expert's novel method was not used by other experts in the industry despite the prevalence of testing. *Id.* at 156-157. Ultimately, the expert's testimony failed the four criteria set forth in *Daubert* and "any other set" of reasonable reliability criteria. *Id.* at 158. "In sum, Rule 702 grants the district judge the discretionary authority, reviewable for its abuse, to determine reliability in light of the particular facts and circumstances of the particular case." *Id.*

In response to *Daubert* and *Kumho*, Michigan's legislature enacted MCL 600.2955, which provides:

> (1) In an action for the death of a person or for injury to a person or property, a scientific opinion rendered by an otherwise qualified expert is not

admissible unless the court determines that the opinion is reliable and will assist the trier of fact. In making that determination, the court shall examine the opinion and the basis for the opinion, which basis includes the facts, technique, methodology, and reasoning relied on by the expert, and shall consider all of the following factors:

(a) Whether the opinion and its basis have been subjected to scientific testing and replication.

(b) Whether the opinion and its basis have been subjected to peer review publication.

(c) The existence and maintenance of generally accepted standards governing the application and interpretation of a methodology or technique and whether the opinion and its basis are consistent with those standards.

(d) The known or potential error rate of the opinion and its basis.

(e) The degree to which the opinion and its basis are generally accepted within the relevant expert community. As used in this subdivision, "relevant expert community" means individuals who are knowledgeable in the field of study and are gainfully employed applying that knowledge on the free market.

(f) Whether the basis for the opinion is reliable and whether experts in that field would rely on the same basis to reach the type of opinion being proffered.

(g) Whether the opinion or methodology is relied upon by experts outside of the context of litigation.

(2) A novel methodology or form of scientific evidence may be admitted into evidence only if its proponent establishes that it has achieved general scientific acceptance among impartial and disinterested experts in the field.

Our state courts have issued a number of opinions addressing the reliability of expert testimony. In *Gilbert*, the plaintiff sued her employer for sexual harassment, arguing that the harassment created a permanent change in her brain chemistry, which caused her to relapse into substance abuse and depression. *Gilbert*, 470 Mich at 753. She presented the expert opinion of a social worker who testified that the plaintiff would suffer an untimely and excruciating death. *Id*. The plaintiff's expert was the plaintiff's counselor and testified as both a fact witness and an expert witness. He testified that he received a master's degree in psychobiology and also received a prestigious award as an undergraduate, but neither of these claims was true. *Id.* at 759-760. The Court noted that "[t]his witness not only lacked any training, education, or experience in medicine, but also testified falsely about his credentials. Nevertheless, plaintiff asked the jury to treat this witness's testimony as a 'prognosis,' and to compensate plaintiff for the loss of her health and, eventually, her life." *Id.* at 753-754.

In a strongly-worded opinion, the Supreme Court took both the trial court and the appellate court to task for considering such "junk science":

> [P]roperly understood, the court's gatekeeper role is the same under *Davis–Frye* and *Daubert*. Regardless of which test the court applies, the court may admit evidence only once it ensures, pursuant to MRE 702, that expert testimony meets that rule's standard of reliability. In other words, both tests require courts to exclude junk science; *Daubert* simply allows courts to consider more than just "general acceptance" in determining whether expert testimony must be excluded.
>
> This gatekeeper role applies to all stages of expert analysis. MRE 702 mandates a searching inquiry, not just of the data underlying expert testimony, but also of the manner in which the expert interprets and extrapolates from those data. Thus, it is insufficient for the proponent of expert opinion merely to show that the opinion rests on data viewed as legitimate in the context of a particular area of expertise (such as medicine). The proponent must also show that any opinion based on those data expresses conclusions reached through reliable principles and methodology. [*Id.* at 782 (internal footnotes omitted).]

Noting the particular care that must be taken to vet expert testimony that touches on causation, the Court stated:

> When a court focuses its MRE 702 inquiry on the data underlying expert opinion and neglects to evaluate the extent to which an expert extrapolates from those data in a manner consistent with *Davis*[8]*–Frye* (or now *Daubert*), it runs the risk of overlooking a yawning "analytical gap" between that data and the opinion expressed by an expert. As a result, ostensibly legitimate data may serve as a Trojan horse that facilitates the surreptitious advance of junk science and spurious, unreliable opinions." [*Id.* at 783.]

The *Gilbert* court concluded that both the trial court and the Court of Appeals had failed to recognize such core gatekeeping principles. *Id.* at 783. The result was that a social worker who lacked any medical education, experience, training, skill or knowledge was permitted to interpret plaintiff's medical records and offer an "opinion that he was wholly unqualified to give." *Id.* at 784-785. The Court explained:

> Mr. Hnat unquestionably used the content of plaintiff's treatment records to render an opinion that required medical expertise. He speculated about plaintiff's impending physical inability to work, testified about the type of medical complications that plaintiff would soon experience, predicted the cause of her death, and gave testimony concerning plaintiff's life expectancy. Mr. Hnat expressed his "opinion" on physiological disease, cause of death, and plaintiff's lifespan. Yet there was no evidence or showing that Mr. Hnat was qualified by

[8] *People v Davis*, 343 Mich 348; 72 NW2d 269 (1955).

> training, experience, or knowledge to render such opinions or interpret medical records that would arguably support such a diagnosis or prognosis. There was, in other words, no evidence that Mr. Hnat was qualified to testify that defendant's actions concerning workplace harassment *caused* neurological and physiological changes in plaintiff and shortened her life. [*Id.* at 787-788.]

Thus, while the witness may have been an expert in social work and substance abuse, "[i]n order for Mr. Hnat to provide an admissible opinion interpreting medical records for purposes other than those related to the expertise of social workers, plaintiff bore the burden of showing that Mr. Hnat was qualified by knowledge, skill, experience, training, or education in *medicine*." *Id.* at 788. His qualification did not go merely to the weight of the evidence, but its admissibility in the first instance. *Id.* "Where the subject of the proffered testimony is far beyond the scope of an individual's expertise . . . that testimony is inadmissible under MRE 702. In such cases, it would be inaccurate to say that the expert's lack of expertise or experience merely relates to the weight of her testimony. An expert who lacks 'knowledge' in the field at issue cannot 'assist the trier of fact.'" *Id.* 789. The Court concluded that the witness's "prognosis" testimony that was based on his interpretation of the plaintiff's medical records was erroneous because the witness lacked medical training and, therefore, did not have the ability to interpret the records. *Id.* at 789-790. *Gilbert's* primary focus was on the fact that the witness was not qualified to offer an opinion. The Court's focus was on the witness's professional qualifications and whether his background permitted him to offer an interpretation of those records.

That same year, the Michigan Supreme Court decided the *Craig* case, which is very much in step with the case at bar, as it involved the same plaintiff's attorney and one of the same purported experts – Dr.Gabriel. The plaintiff in *Craig* suffered from cerebral palsy and mental retardation. The plaintiff argued at trial that the defendants (the hospital and treating physicians) negligently administered an excessive amount of Pitocin. The trial court denied the defendants' request to have a *Davis-Frye* hearing on the admissibility of the plaintiff's causation expert and ultimately a jury entered an award for the plaintiff. The Court of Appeals affirmed as to liability, but ordered remittutur. *Craig,* 471 Mich at 70-71. Our Supreme Court reversed. *Craig* was decided under the *Davis-Frye* framework and the Supreme Court noted that, under that framework,

> expert opinion based on novel scientific techniques is admissible only if the underlying methodology is generally accepted within the scientific community. Thus, in determining whether the proposed expert opinion was grounded in a 'recognized' field of scientific, technical, or other specialized knowledge as was required by MRE 702, a trial court was obligated to ensure that the expert opinion was based on accurate and generally accepted methodologies. [*Craig*, 471 Mich at 80 (internal footnotes omitted).]

The trial court in *Craig* did not rely on any of the literature submitted by the plaintiff in response to the defendants' motion in limine to exclude his testimony. "Instead of consulting plaintiff's proffered scientific and medical literature, the court erroneously assigned the burden of proof under *Davis-Frye* to defendant – the party *opposing* the admission of Dr. Gabriel's testimony – and held that defendant was not entitled to a hearing because it failed to prove that Dr. Gabriel's theory *lacked* 'general acceptance.'" *Id.* at 81. The Court noted that while the

plaintiff produced literature that Pitocin could cause brain damage, it did not connect to Dr. Gabriel's causal theory that the excessive contractions caused the plaintiff's head to be repeatedly ground against his mother's pelvis, resulting in head trauma and cerebral palsy. *Id.* at 83.

> Dr. Gabriel was unable to cite a single study supporting his traumatic injury theory during a voir dire conducted at trial. The only authorities he offered for the proposition that excessive amounts of Pitocin may cause cerebral palsy through the traumatic mechanism he described at trial were studies he cited in which Pitocin caused cerebral palsy in animals when given in excessive amounts. These studies did not involve the "bumping and grinding" mechanism on which Dr. Gabriel's expert testimony relied. In fact, Dr. Gabriel expressly distinguished the mechanism to which he attributed plaintiff's injuries from those at work in the animal studies. It would appear, then, that there was little evidence that Dr. Gabriel's theory was "recognized," much less generally accepted, within pediatric neurology. [*Id.* at 84.]

Dr. Gabriel could not identify what part of the mother's anatomy against which the child's head collided. *Id.* Moreover, "[a]t no point did Dr. Gabriel opine that the traumatic and vascular mechanisms he described could cause cerebral palsy, or that those mechanisms might produce the asymmetrical development shown in plaintiff's MRI. Thus, Dr. Gabriel's testimony supported plaintiff's medical malpractice claim only if the jury was permitted to assume, without supporting evidence, that a causal connection existed between these elements." *Id.* at 84-85. There was, therefore, a "yawning gap between Dr. Gabriel's testimony and the conclusions plaintiff hoped the jury would draw from it." *Id.* at 85.

The *Craig* Court held that the trial court erred in failing to grant the defendants' motion for judgment notwithstanding the verdict where the plaintiff failed to establish causation. "Even if plaintiff had shown that defendants breached the standard of care, the jury had no basis in the record to connect this breach to the cerebral palsy, mental retardation, and other injuries now presented by plaintiff." *Id.* at 90. The Court added that "[e]ven if we accept Dr. Gabriel's testimony in full, a fatal flaw remains in plaintiff's prima facie case: Dr. Gabriel never testified that the injuries stemming from this pounding and its accompanying vascular effects could cause cerebral palsy, mental retardation, or any of the other conditions now presented by plaintiff." *Id.* at 91. The *Craig* Court concluded:

> Dr. Gabriel began his testimony by explaining that an MRI image showed that plaintiff's brain tissue had developed asymmetrically. He failed, however, to trace this asymmetric development either back to the traumatic and vascular mechanisms he described or forward to the specific neurological conditions presently displayed by plaintiff. Thus, how exactly the mechanisms he described led to cerebral palsy (as opposed to any other neurological impairment) and how they were connected to the asymmetric brain development depicted in plaintiff's MRI was never explained.
>
> It is axiomatic in logic and in science that correlation is not causation. This adage counsels that it is error to infer that A causes B from the mere fact that

> A and B occur together. Given the absence of testimony on causation supplied by Dr. Gabriel, the jury could have found for plaintiff only if it indulged in this logical error-concluding, in effect, that evidence that plaintiff may have sustained a head injury, combined with evidence that plaintiff now has cerebral palsy, leads to the conclusion that the conduct that caused plaintiff's head injury also caused his cerebral palsy.
>
> Such indulgence is prohibited by our jurisprudence on causation. We have long required the plaintiff to show that but for the defendant's actions, the plaintiff's injury would not have occurred. Where the connection between the defendant's negligent conduct and the plaintiff's injuries is entirely speculative, the plaintiff cannot establish a prima facie case of negligence. [*Id.* at 93 (internal quotation marks and footnotes omitted).]

Three years later in *Chapin v A & L Parts, Inc*, 274 Mich App 122; 732 NW2d 578 (2007), our Court cautioned trial courts not to conduct "minitrials" when deciding whether an expert can testify at trial under MRE 702 and MCL 600.2955(1). In *Chapin*, plaintiff was diagnosed with mesothelioma, after having spent 45 years working as an automobile brake mechanic. "Part of his job involved grinding brake linings that contained chrysotile asbestos. At issue is whether plaintiffs' expert presented scientifically reliable, and therefore legally admissible, evidence drawing a causal connection between mesothelioma and inhalation of brake-lining dust." *Id.* at 125. Writing for the majority Judge Davis noted:

> [T]he trial court's role as gatekeeper does not require it to search for absolute truth, to admit only uncontested evidence, or to resolve genuine scientific disputes. The fact[] that an opinion held by a properly qualified expert is not shared by all others in the field or that there exists some conflicting evidence supporting and opposing the opinion do[es] not necessarily render the opinion "unreliable." A trial court does not abuse its discretion by nevertheless admitting the expert opinion, as long as the opinion is rationally derived from a sound foundation. [*Id.* at 127.]

Importantly, Judge Davis wrote:

> The fact that two scientists value the available research differently and ascribe different significance to that research does not necessarily make either of their conclusions unreliable. Indeed, science is, at its heart, itself an ongoing search for truth, with new discoveries occurring daily, and with regular disagreements between even the most respected members of any given field. A *Daubert*-type hearing of this kind is *not a judicial search for truth.* The courts are unlikely to be capable of achieving a degree of scientific knowledge that scientists cannot. An evidentiary hearing under MRE 702 and MCL 600.2955 is merely a threshold inquiry to ensure that the trier of fact is not called on to rely in whole or in part on an expert opinion that is only masquerading as science. *The courts are not in the business of resolving scientific disputes.* The only proper role of a trial court at a Daubert hearing is to filter out expert evidence that is unreliable, not to admit only evidence that is unassailable. The inquiry is not into whether an

> expert's opinion is necessarily correct or universally accepted. The inquiry is into whether the opinion is rationally derived from a sound foundation. [*Id.* at 139 (emphasis added).]

The Court concluded that, even in the face of contrary evidence, the trial court correctly permitted the plaintiff's expert to testify. "Although clearly not universally accepted, and although unsupported by epidemiological studies that may or may not be flawed, [the plaintiff's expert's] opinion is certainly objective, rational, and based on sound and trustworthy scientific literature." *Id.* at 140.

In *Edry v Adelman*, 486 Mich 634; 786 NW2d 567 (2010), the plaintiff brought an action against her doctor, alleging that his failure to follow-up on a bump under her arm delayed the diagnosis and treatment of breast cancer, impacting her survival rate. The Michigan Supreme Court affirmed the trial court's decision to not allow plaintiff's oncology expert to testify that the plaintiff's chances of surviving five years would have been 95 percent if she had been diagnosed earlier and that the delay in diagnosis reduced her five-year survival chance to 20 percent. *Id.* at 636-640. The *Edry* Court concluded:

> Here, [the plaintiff's expert's] testimony failed to meet the cornerstone requirements of MRE 702. Dr. Singer's opinion was not based on reliable principles or methods; his testimony was contradicted by both the defendant's oncology expert's opinion and the published literature on the subject that was admitted into evidence, which even Dr. Singer acknowledged as authoritative. Moreover, no literature was admitted into evidence that supported Dr. Singer's testimony. Although he made general references to textbooks and journals during his deposition, plaintiff failed to produce that literature, even after the court provided plaintiff a sufficient opportunity to do so. Plaintiff eventually provided some literature in support of Dr. Singer's opinion in her motion to set aside the trial court's order, but the material consisted only of printouts from publicly accessible websites that provided general statistics about survival rates of breast cancer patients. The fact that material is publicly available on the Internet is not, alone, an indication that it is unreliable, but these materials were not peer-reviewed and did not directly support Dr. Singer's testimony. Moreover, plaintiff never provided an affidavit explaining how Dr. Singer used the information from the websites to formulate his opinion or whether Dr. Singer ever even reviewed the articles. [*Id.* at 640-641 (internal footnote omitted).]

The Court emphasized that "[w]hile peer-reviewed, published literature is not always a necessary or sufficient method of meeting the requirements of MRE 702, in this case the lack of supporting literature, combined with the lack of any other form of support for Dr. Singer's opinion, renders his opinion unreliable and inadmissible under MRE 702." *Id.* at 641. It was not enough for a party to "point to an expert's experience and background to argue that the expert's opinion is reliable." *Id.* at 642.

Recently our Court reviewed the admissibility of expert opinion in *Elher v Misra*, ___ Mich App ___; ___ NW2d ___ (Docket No. 316478, issued December 2, 2014). In *Elher*, (unlike the case at bar) there was no dispute as to the injury or the mechanism of the injury. At

issue was whether the particular conduct (clipping both the bile and cystic ducts during gallbladder surgery) breached the standard of care. *Elher*, slip op, pp 1, 7. Nevertheless, Judge Gleicher cited many of the foregoing cases and noted that, in acting as gatekeeper, "'[t]he standard focuses on the scientific validity of the expert's methods rather than on the correctness or soundness of the expert's particular proposed testimony.'" *Id.* at slip op, p 8, quoting *People v Unger*, 278 Mich App 210, 217-218; 749 NW2d 272 (2008). Judge Gleicher wrote:

> We draw from *Kumho* and *Edry* several important lessons. A court screening scientific evidence must ensure that proposed scientific or technical testimony is reliable as well as relevant. But the algorithm for this analysis cannot be scripted in advance or applied in a vacuum. Rather, a court must determine which factors reasonably measure reliability given the specific factual context and contours of the testimony presented. [*Id.* at slip op, p 10.]

Because the focus must be on methodology and *not* the expert's ultimate conclusion:

> If an expert's reasoning is based on scientific principles, knowledge, experience and training, the testimony may fulfill the reliability standards even in the presence of conflicting conclusions predicated on precisely the same data, and an identical quantum of practical wisdom. This holds true even when a judge finds one side's approach more persuasive. The clashing standard of care opinions in this case are exactly the sort that "[v]igorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof" is designed to resolve. [*Id.* at slip op p, 14, quoting *Daubert*.]

Moreover, the courtroom door should not be closed on medical experts whose opinions are often supported by extensive relevant experience. *Elher*, slip op, p 18. In fact, the rule of evidence "expressly contemplates that an expert may be qualified on the basis of experience." *Id.* Judge Gleicher concluded:

> Gatekeeping courts are not empowered "to determine which of several competing scientific theories has the best provenance." *Ruiz–Troche v Pepsi Cola of Puerto Rico Bottling Co*, 161 F3d 77, 85 (CA 1, 1998). The test is whether the expert's *reasoning* is scientifically sound.
>
> ***
>
> Ultimately, the gatekeeping inquiry asks whether the expert has reached his or her conclusions in a sound manner, and not whether the expert's conclusions are correct. "Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence." *Daubert*, 590 U.S. at 597. Alternatively stated, the trial judge is "a gatekeeper, not a fact finder." *United States v Sandoval–Mendoza*, 472 F3d 645, 654 (CA 9, 2006). Here, application of immaterial *Daubert* factors led the trial court to exclude expert testimony possessing none of the hallmarks of "junk science." "[N]o one denies that an expert might draw a conclusion from a set of observations based on

extensive and specialized experience." *Kumho*, 526 US at 156. [*Elher*, slip op, pp 19-20.]

IV. PLAINTIFF'S CAUSATION EXPERT

A. JUNE 13, 2013 HEARING

At the June 13, 2013 hearing on defendants' various motions, defense counsel explained that he filed two separate motions for each of the alleged injuries to plaintiff – perinatal arterial ischemic stroke and hypoxic ischemic injury. He disputed that there was any evidence of the latter because no one other than plaintiff's experts diagnosed hypoxic ischemic injury. Furthermore, counsel argued that plaintiff could not show negligent perinatal care and treatment caused the stroke. He argued that, while there were known risk factors, there was simply no identified causal mechanism for PAIS. Defense counsel argued that these types of strokes could not be predicted or prevented.

Before he began his response, the trial court told plaintiff's counsel: "I want you to be able to show me in the articles exactly where it says what causes this, not just when it says what are the risk factors and associations." Plaintiff's counsel acknowledged the *Craig* case and differentiated it by saying that the plaintiff in *Craig* failed to offer a "scintilla" of support for Dr. Gabriel's opinion whereas the present case had "an avalanche" of support. Essentially, even if viewed as *just* risk factors, "this is an injury that can be prevented if they're careful."

The parties then went to address the motion regarding plaintiff's claim of global hypoxic ischemia injury secondary to alleged perinatal trauma from Pitocin and the forces of labor. Defense counsel argued that no literature supported a finding that excessive uterine contractions can cause brain injury and it was the exact theory that the *Craig* Court rejected. Uterine activity was just a risk factor, not a cause of ischemia. Defense counsel asked "where is the science that supports that long second stages of labor with Petocin [sic] cause a brain injury?"

Plaintiff's counsel denied that there were two separate injuries: "This baby suffered one injury. That injury included a stroke. That injury also included lack of oxygen to both sides of the baby's brain." Plaintiff's counsel then indicated that there were three injuries. The following exchange took place between plaintiff's attorney and the court:

> THE COURT: Tell me what caused the stroke in baby Figurski.
>
> MR. FIEGER: Ischemia. In this case, ischemia.
>
> THE COURT: And that's lack of blood?
>
> MR. FIEGER: No, not lack of blood.
>
> THE COURT: It's slow blood.
>
> MR. FIEGER: It's slow, it, it's less perfusion.

THE COURT: And what caused the slow blood?

MR. FIEGER: The forces of the Petocin [sic] being allowed to continue –

THE COURT: Okay.

MR. FIEGER: -- unabated for hours.

THE COURT: So the stroke was caused by ischemia. Ischemia is a slowing of the blood. The slowing of the blood was the result of the Petocin [sic].

MR. FIEGER: Yes.

THE COURT: So there's a stroke. That's an injury. There's ischemia, but ischemia you just said is what caused the stroke. So how is ischemia a different injury?

MR. FIEGER: Because it affects, the stroke is in the vessel and it's, affects a specific portion of the brain. The experts have also testified this child has injuries over different parts of his brain.

THE COURT: How is ischemia different than the stroke?

MR. FIEGER: The ischemia is a lack of perfusion where they call it a watershed event. That affects all the cells in the brain, not just the specific area of the brain that's affected by the stroke.

THE COURT: So the ischemia caused an, if I hear you, an injury different than the stroke. It was the part of the, a factor in the stroke but it also caused another injury.

MR. FIEGER: Yes. Exactly.

THE COURT: And what was it?

***

MR. FIEGER: He's got global brain damage.

***

THE COURT: So that's two things: stroke, ischemia which caused brain damage. What the third?

MR. FIEGER: Hypoxia.

THE COURT: Hypoxia is a lack of oxygen.

MR. FIEGER: That's, that's lack of oxygen, right, as opposed to perfusion.

THE COURT: And the lack of oxygen was caused by what?

MR. FIEGER: The Petocin [sic].

Defense counsel acknowledged that plaintiff claimed the same mechanism caused both injuries (the disputed global hypoxic ischemia and the undisputed stroke) and his response was that neither injury could have been caused by the squeezing of the child's brain. He argued that 1) there was no way of knowing what caused the stroke, and 2) there was no evidence of any injury other than the stroke; no one had diagnosed plaintiff with global hypoxic ischemia. Defense counsel explained his position:

> Mr. Fieger's experts, he has cited the Heuser article. He said excessive uterine contractions can lead to changes in the fetal heart rate, diminished placental perfusion. My motion doesn't address that. If his experts want to come in and say that excessive Petocin [sic] caused a decrease in placental perfusion in this case, my motion didn't address that. What my motion addresses is Plaintiff's experts coming in here and saying excessive uterine activity caused squeezing of the head which caused a decrease in perfusion pressure, which led to a global hypoxic ischemia, which caused the child's injury. . . .Your Honor, I'm not disputing that excessive uterine contractions can cause a problem with placental perfusion. What I am disputing and what my motion concerns is it doesn't cause head compression sufficient to cause a brain injury to a child.

B. DAUBERT HEARING

Dr. Crawford testified that for the past 40 years she specialized in neonatal perinatal medicine, or the care of sick newborns. She was board certified in pediatrics in 1976 and neonatal perinatal medicine in 1977. Crawford had been practicing part-time at Our Lady of Lourdes Medical Center in Camden, New Jersey, since 1999. It was a level three institution with a high risk perinatal center. Her practice involved "the understanding of the problems during pregnancy, labor, and delivery that impact on the fetus and newborn because that's the history of my patient." Part of her specialty was the diagnosis and treatment of newborns injured prior to, during, or after birth. Her other work included directing medical teams to provide care at medical clinics around the world. She routinely taught in Guatemala, Cuba and the United States. She also wrote a number of articles and chapters in medical textbooks. During cross-examination, Crawford acknowledged that she was not a pediatric neurologist. A pediatric neurologist – Dr. Woodhouse – had diagnosed plaintiff with a stroke in this case.

For the case at bar, Crawford reviewed plaintiff's medical records, which included prenatal records, neonatal postnatal records, and pediatric records. In so doing, she formed an opinion that plaintiff suffered a hypoxic ischemic injury. She explained that "hypoxia" was low oxygen "particularly to the tissues. It often includes hypoxemia which is low oxygen in the blood. Ischemia refers to a decrease in the flow of blood containing oxygen to the tissues." There was also a perinatal arterial ischemic stroke. She explained that "Arterial means it

involves an artery. And ischemic means it involves a disruption or decrease in the amount of blood flow through the particular artery, in this case the left middle cerebral artery which is a main branch off of the carotid that supplies a large portion of the cortex of the baby." It was not uncommon to find hypoxia ischemia in infants who suffered a perinatal arterial ischemic stroke.

A stroke could be either "wet," meaning a ruptured aneurism, or "dry," meaning that the blood did not flow properly because of increased intracranial pressure. As to the latter, Crawford explained:

> *Personally, I've had experience* with using blood pressure cuffs applied to children at the area of the biparietal diameter where the blood pressure cuff is pumped up to exceed systolic pressure. And *my experience* with doing that was with children who were receiving chemotherapy and we were trying to minimize the effect of the chemotherapy on the brain when they were being treated for things like leukemia. So it was a very effective mechanism. The literature also supports the efficacy, the effectiveness of applying a blood pressure cuff around the, uh, biperietal diameter of fetal animals, in particular sheep. And when the blood pressure cuff is inflated to 100, 150, 200 millimeters, you can get an isoelectric line for the EEG on the fetal animal. And you can get decrease in the flow of blood to the cortex of the brain, which is about 95% of the flow of the blood to the cortex gets disrupted when you have increased intracranial pressure during that type of experiment and study, where as the flow to the brain stem is actually increased. *So that, those type of animal experiments and my familiarity with using the blood pressure cuff to decrease cerebral blood flow has been part of the basis for my opinion* that when you have prolonged intense uterine contractions that you get an increase in the intracranial pressure that, or you get an increase in pressure on the head that may be such that the baby cannot increase its blood pressure to maintain circulation. So it can do it to a certain point, but there's a point of failure when the increased pressure on the head, particularly in the biparietal[9] diameter area which is where the middle cerebral artery is . . . and that's what, uh, leads to disruption of blood flow. [Emphasis added.]

Crawford testified that plaintiff did not just suffer a stroke, but also had diffused encephalopathy on the right side of his brain, which was abnormal brain function as manifested with changes in tone, seizures, and posturing. He also had apnea, which was the cessation of breathing. Plaintiff had several diffusely (not localized) abnormal EEG's. He had "multiple indications of having sustained a widespread brain injury." Plaintiff not only suffered a "left middle cerebral artery infarct" (stroke) but also a "diffuse hypoxia ischemia to the brain," meaning all over the brain.

Crawford explained that molding can lead to distortion of the head and blood vessels. In this case, plaintiff demonstrated significant molding and caput (swelling of the scalp edema fluid) as a result of trying to get the head through the pelvis. "And the reason the middle

[9] In the record, the term is spelled "biperietal" and "biparietal."

cerebrals are affected is that the area of maximum impact of the pressure and forces is around where you have the largest head circumference on the skill."

Crawford believed that misuse of the drug Pitocin increased the natural forces of labor by causing too many contractions:

> The use of Pitocin to the point where it resulted in too many contractions in a given period of time, something called tachysystole, results in increased intrauterine pressure. There was an IUPC utilized. That's an intrauterine pressure catheter. So the pressure within the uterus could be accurately measured. And it was set and calibrated to have a baseline of 20 millimeters. But during the labor, because of the increased number of contractions and the increased intensity of the contractions, the pressure within the uterus did not come down to resting levels on many occasions and in fact, was as high as 40 or double the normal amount.
>
> ***
>
> The impact of that, of too frequent contractions and pressure that is increased is that the blood doesn't come through the uterus to the placenta when the uterus is contracting and is strongly contracting and is frequently contracting, that results in interruption of blood flow and oxygen through the uterus to the placenta and to the baby. And the end result of that is you have a decrease in blood flow coming to the baby. So you have an ischemic component from that type of abnormal condition. You also have a hypoxic component because the blood carries the oxygen. As a result of not getting enough oxygen, the baby starts to use up its reserves and can become hypoxic and acidotic if this problem, if this condition continues.

The excessive contractions can also result in physical trauma where the head is essentially used as a "battering ram," especially when the pelvis has not been proven to accommodate a particular sized baby and especially in first-time labor, which is usually longer and more intense.

At this point, the trial court interrupted the testimony and asked why it was being presented with Crawford's actual opinion instead of hearing evidence that the medical literature supported the opinion. The court stated: "Essentially I am looking for something in the literature that says it caused, it can cause, not that it may, not that it could, not that there's a possibility. That's what I'm looking for."

Plaintiff's counsel then asked Crawford to review the package insert for Oxytocin, which warned that strong hypertonic or prolonged tectonic contractions could result in deceleration of heart rate and fetal hypoxia. Fetal hypoxia as a result of excessive uterine contractions was "a well understood phenomenon." Crawford testified that Pitocin was a high risk drug and the package insert specifically warns that if given inappropriately can cause hypoxia. This was well-known and every hospital that Crawford ever worked at had "Pitocin policy," setting forth how the drug should be administered to avoid tachysystole and fetal distress. Crawford acknowledged that the insert did not necessarily mention stroke as the specific result of misuse, but did refer to hypoxia, which was a mechanism by which a stroke can occur.

Crawford testified that a number of factors came into play in this case:

> [T]he membranes were ruptured for 29 hours before the baby was born. So the amount of amniotic fluid that the baby was in was very diminished. So the cord, the umbilical cord is not buffered, buffeted by the usual presence of amniotic fluid. So when the uterus contracts, there's pressure on the cord. And that pressure can lead to variable decelerations of the fetal heart. And there were numerous variable decelerations present. The impact of too frequent and too strong uterine contractions also led to what's called utero placental insufficiency in that there was a decrease in the amount of oxygen and blood flow going to the baby which results in hypoxia and ischemia. And as the uterus is being intensely affected by Pitocin and there are too frequent and too intense contractions, the baby's head is being compressed.

Therefore, three factors were at play: the cord, the function of the placenta and "the compressive effect of the uterine contractions on the baby itself." She explained:

> First of all, when you have a contraction, uterine contractions are looked upon as the apnea of the unborn. The placenta acts as the baby's lungs delivering oxygen to the baby. When there's [sic] contractions that are too frequent and too intense, there is a decrease in the amount of oxygen and blood flow that comes through the placenta because the uterus is being so constricted by the contractions. So the baby gets, has decreased oxygen, decreased blood flow. And that's hypoxia and ischemia. And when the cord gets compressed and you get variable decelerations, the heart rate goes down and there's a decrease in blood flow and oxygenation being delivered to the baby because of compression of the cord. And then finally, when you have in the first stage of labor the, the uterus is contracting around the fetus and in particular, the head because the head is the leading so-called battering ram that's coming through to get through to the birth canal. When you get into the second stage, then the compression on the baby's head is from the bones of the pelvis. And when the baby's head is large, the biparietal diameter is large, the baby's head gets jammed and doesn't progress. Then every contraction impacts on the baby's skull and impacts on the pressure that's being exerted on the baby's skull to the point where it increases to the point where, where it exceeds cerebral profusion pressure and you get disruption of blood flow in the brain, thus result in a stroke. [H II, pp 38-39.]

Crawford cited Kumar & Paes, *Contralateral Cerebral Infarction Following Vacuum Extraction*, 21 Am J of Perinatology 15-17 (2004) to support her conclusion that mechanical birth trauma has long been associated with intracranial stroke due to the skull being subject to abnormal forces, especially when there is a history of difficult labor. Crawford insisted that it was well recognized that subjecting a baby's skull to abnormal pressures can cause arterial stroke; it was not junk science. Upon questioning by the court, Crawford acknowledged that the Kumar article dealt with a vacuum extraction. The authors concluded that mechanical birth trauma is a direct cause of intracranial arterial injury that can then subsequently lead to stroke, including the natural forces of labor. Specifically: "'In the absence of identifiable cause, the infarction may be considered due to the skull being subjected to abnormal forces during the

birthing process especially when there's a difficult labor.'" Crawford explained that force did not necessarily mean instrumentation like forceps or vacuum; it included the force of labor itself intensified with Pitocin.

When defense counsel was questioning Crawford regarding the meaning of the Kumar article, the trial court interjected: "Just a minute. I know he disagrees with you and you disagree with him. **And I have to decide who is right**. . . . I'm going to tell you, I, I wrote all over this. This was a vacuum extraction and that's not what happened with baby Figurski. So that's really kind of where I'm at on this article." The following exchange took place regarding the article:

> THE COURT: This is a case report involving a vacuum extraction. So when I read this, I take it in its entirety from the beginning to the end. I'm told it's a vacuum extraction. So that's what mechanical birth trauma [][means] to me. You tell me where in this article it describes mechanical birth trauma as anything other than a vacuum extraction?
>
> THE WITNESS: The next paragraph, the next sentence where it says the trauma, mechanical birth trauma, they say, has been recognized as a direct cause of intracranial arterial injury leading to ischemic or hemorrhagic stroke in the newborn. The trauma could result from the process of natural birth or from assisted instrumentation with forceps or [vacuum]. So the authors are laying out the three types of mechanical birth trauma that can affect a baby and lead to –
>
> THE COURT: I just –
>
> THE WITNESS: --stroke.
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> THE COURT: It says from the natural birth process.
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> THE WITNESS: Yes. That's without –
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> THE COURT: That's one.
>
> THE WITNESS: Yes, that's without instruments.
>
> THE COURT: Or wait a minute. You said three.
>
> THE WITNESS: Right. Vacuum, forceps, and the natural forces of labor were the three.
>
> THE COURT: I don't know where you get three.
>
> MR. FIEGER: It says it in the last sentence, your Honor.
>
> THE COURT: Mr. Fieger, don't even. No.
>
> MR. FIEGER: The last sentence says it.

THE COURT: No. Mr. Fieger, you're not to testify. You, and you don't get to help your witness. I'm trying to understand. It says the trauma could result from the process of natural birth – to me, that's one – or from assisted instrumentation with forceps or – [vacuum] . . .

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THE COURT: So in other words, how I take this is it could happen naturally. It could happen to, God help us, any baby; not the Doctor's fault.

MR. FIEGER: Judge, is that what we're doing here?

THE COURT: Oh for heaven's sakes. Okay. Five minutes because you are not going to interrupt. Five minutes.

Crawford cited Amiel-Tison, *Cerebral Damage in Full-Term New-Born, Aetiological Factors, Neonatal Status and Long-Term Follow-Up*, 14 Biol Neonat 234-250 (1969), which concluded that inappropriate use of Pitocin caused problems in newborns, especially in babies who got stuck mid-pelvis (dystocia). The article also looked to fetal distress and the evidence of meconium stained amniotic fluid, which happened with plaintiff as well. Crawford explained that the purpose of the article was to describe the affected infants. The author was "looking at all affected infants and trying to describe the full spectrum of findings in that group. The controls would be all normal newborns. So the control group is perhaps unstated, but known." Although Crawford's affidavit says that too many contractions increase intracranial pressure and decrease in profusion of the brain, the article did not make that exact statement. Crawford testified that the Amiel-Tison article "sets the stage for the mechanism for intrapartum cerebral damage due to iatrogenic uterine hyperstimulation."

Defense counsel pressed that although the article suggested an injury, it did not "say if it's a decrease in the blood flow and oxygen through the placenta or whether or not it's an increase in intracranial pressure that causes a slowdown in cerebral profusion pressure." When defense counsel emphasized that the article only implied that injury *may* result, plaintiff's counsel objected:

MR. FIEGER: Judge, objection. What are we doing here? What, where is it supposed to say that 100% of the time it will? I thought we're here, they don't have a defense here in terms of this. This is not a legitimate Daubert hearing. This is something that they think they can get away [sic] and there's birth trauma. And this type of injury is so accepted in medicine that it's beyond belief that this is being questioned.

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THE COURT: You have said that to me so many times. . . . This is my response. I can't even count on two hands now how many times you've said that in writing, here, in chambers. You have, I don't know, I guess in some ways tried to make it sound like this is the most ignorant thing that I'm doing; that what a fool who could fall for the defense's argument. I find it somewhat insulting. I'm

> trying to do my job. And you wanted me to rely on an Oakland County Circuit Judge's opinion. That's not helpful. We're going to keep going. And stop saying that this is accepted all over the place, all over the country in courts all over the country because you haven't shown me that. And defense has never argued that it has to be 100% certainty. I certainly didn't say that in my opinion setting this hearing. And I asked her what may meant. . . . I don't want to hear you say that again. We're going to finish this.

After a break, defense counsel continued to question Crawford about the Amiel-Tison article. The article described seizures as a reflection of brain injury, but did not mention stroke. It discussed necrosis and death of the brain, however, which were not uncommon in stroke. Crawford stressed that the article was from the 1960's before there was advanced imaging. Upon questioning from the trial court, Crawford explained that the article set the stage for finding that too many contractions cause an unsafe increase in intracranial pressure, but acknowledged that the article did not specifically say that:

> THE COURT: Are you acknowledging that that's not what the Tison article says? I want to make sure my notes aren't wrong. I'm not asking you what she's saying in the article. I'm asking specifically are you acknowledging that's not what the Tison article says?
>
> THE WITNESS: I'm saying I should have had, as I said before, I should have had another footnote there which would be the Cushing.
>
> THE COURT: Boy, you're like some lawyers.
>
> THE WITNESS: Excuse me?
>
> THE COURT: Not wanting to answer the question. Is that what her article says?
>
> THE WITNESS: No.

Crawford further explained that "Cushing's response" is a protective mechanism for when there was an increase in the intracranial pressure. The natural response was to increase the blood pressure in the brain to maintain cerebral profusion. However, it works up to a point and if it fails then ischemic injury results.

Crawford also cited Towner & Ciotti, *Operative Vaginal Delivery: A Cause of Birth Injury or Is It?*, 50 Clinical Obstetrics & Gyn, 563-581 (2007), which dealt with a vacuum extraction. Crawford explained that the point of the article was that when excessive forces are used, there can be excessive molding (adjustment of the bones of the skull) and injury may result. When the baby is "stuck," especially a big baby in an unproven pelvis, and doctors simply increase the number and frequency of contractions, "it will get you nowhere." Generally the scalp will reveal evidence of dystocia and trauma – bruising, swelling, and indentation. Crawford explained that "what Towner is telling us is look, these babies have probably been in trouble and having head compression and problems before you even applied the vacuum or the forceps." Although Towner's article involved forceps and vacuums, "Towner's point is very

often the damage is already there." Crawford explained that global hypoxic injury is not caused *only* by forceps or vacuums. During cross-examination, Crawford acknowledged that the article was a "medical analysis" where the authors looked at various reported cases, which were primarily instrumented deliveries. Such operative vaginal deliveries would mean either forceps or vacuum. The article did not mention stroke, but mentioned cerebral palsy. The article did not state that fetal injuries that were associated with vaginal delivery could be explained by the use of force to overcome cephalopelvic disproportion.

Crawford referenced Volpe, *Neurology of the Newborn* (5th Ed) Saunders Elsevier (2008). Volpe was the chairman of pediatric neurology at Boston Children's Hospital. Volpe referred to a number of factors that increase the baby's chances of having a stroke, including fetal distress, prolonged labor, prolonged rupture of the membranes, infection, low amniotic fluid, and first time labor. The following exchange took place between plaintiff's attorney and Crawford regarding the Volpe text wherein Volpe set forth a number of risk factors for perinatal arterial ischemic stroke:

> BY MR. FIEGER:
>
> *Q*. Does risk factor mean a cause in your opinion, Doctor?
>
> *A*. The clustering of multiple risk factors gives you a high, a high risk situation for the development of stroke. In other words, the more risk factors that you have, the more likely it is that those risk factors were the cause of the stroke.
>
> ***
>
> For example, perinatal arterial ischemic stroke is uncommon, very uncommon in normal spontaneous vaginal deliveries or cesarean sections prior to labor. So the PAIS incidents with emergency [] cesarean sections and with evidence of meconium, evidence for fetal distress, uh, non-reassuring fetal strips, prolonged seconds stage, prolonged labor, dystocia, all of that put together indicates that those are the, when those factors are present, those are the causative factors for the stroke in the child. The more of those factors you have, the more likely it is that that's the, that those are the causes for the child's stroke.

The texts did not discuss asphyxia, although fetal heart rate abnormalities and prolonged second stage of labor were linked with asphyxia.

Crawford cited Lee et al, *Maternal and Infant Characteristics Associated With Perinatal Arterial Stroke in the Infant*, 293 J Am Med Ass'n 723-729 (2005). The article did not test whether head compression caused PAIS; rather, it was a retrospective controlled study. The article concluded that fetal heart rate abnormalities were no longer independently associated with perinatal arterial stroke. But Crawford pointed out that 46 percent of infants with stroke had fetal heart rate abnormalities compared with 14% that had no such abnormality, so fetal heart rate was still statistically significant. The article did not conclude that use of Pitocin was a risk factor for stroke, though Crawford opined that it was only the *inappropriate* use of Pitocin that was problematic. Crawford testified that "Oxytocin is widely used and most of the time it's used properly. So it wouldn't be surprising that you wouldn't see an effect. It's only when you have

improper use of the Pitocin that you get the increased pressure and increased compression." Ultimately, Lee concluded that it was impossible to know the causal pathway to perinatal stroke. However, with three or more risk factors, the odds of stroke was 1 in 200.

Crawford testified that, obviously, there could be no controlled studies on injuring children; instead, they had to rely on animal studies. There was no way to subject women in labor to excessive Pitocin to see what the result would be.

Crawford referenced articles that tested the effect of blood flow on fetal sheep. Harris et al., *Cerebral and Peripheral Circulatory Responses to Intracranial Hypertension in Fetal Sheep*, 64 Circulation Research 991-1000 (1989); Harris et al., *Efficacy of the Cushing Response in Maintaining Cerebral Blood Flow in Premature and Near-Term Fetal Sheep,* 43 Pediatric Research 50-56 (1998); O'Brien et al, *Effect of Cephalic Pressure on Fetal Cerebral Blood Flow*, 1 Am J Perinatology 223-226 (1984). Crawford explained that the articles revealed that "when you compress the head as the head is [] experiencing the impact of uterine contractions or being, acting as a battering ram coming through the pelvis, it can lead to or does lead to, and they determined that it leads to increased intracranial pressure and decreased cerebral profusion pressure" leading to "ischemic insult."

The Harris article explained that intracranial pressure resulted in the body shunting blood away from non-vital organs in order to increase blood flow to the brain. Crawford acknowledged that the article indicated that the Cushing's response was generally effective.

The O'Brien article was a study on the fetal lambs to observe the effect of pressure on the head. The following exchange took place:

> THE COURT: . . . This study, its conclusion wasn't that this pressure and the change in the, on the head resulted in a change in the blood such that ischemic hypoxia or stroke occurred.
>
> THE WITNESS: Not the point of the study. The point of the study is to show, was to show that increased pressure on the head causes a decrease in cerebral profusion. You have to take the next step –
>
> THE COURT: And cerebral profusion is?
>
> THE WITNESS: Is the difference between, cerebral profusion pressure is the difference between the increased pressure that's applied externally and the baby's main arterial pressure. So the Cushing effect is one whereby the baby increases its blood pressure to overcome this pressure –
>
> THE COURT: Pressure on the brain. Right, I get that.
>
> THE WITNESS: -- external pressure. Okay.
>
> THE COURT: And your point is –

THE WITNESS: And the baby can do that up to a certain point. And then as this pressure gets more and more, the baby can't respond. It can't increase its blood pressure any more. And what –

THE COURT: That's not, that's not what 14 goes to. Really, the only point.

THE WITNESS: Well –

THE COURT: It doesn't, the article doesn't go so far as to say exactly at what point this Cushing effect doesn't work anymore and there's going to be a stroke. That's all I'm trying to get at with 14.

THE WITNESS: Okay, you, what it says is that you interrupt, this kind of pressure can interrupt and does interrupt cerebral blood flow. Now if you do it long enough, which is not what they wanted to do – they just wanted to show that –

THE COURT: No, please don't –

THE WITNESS: -- this is an effect –

THE COURT: -- tell me you think if you do it long enough it's going to happen. All I'm trying to figure out is 14. It, it never concludes if you do it long enough there's going to be a stroke.

THE WITNESS: You have to go to –

THE COURT: All right, no. No.

THE WITNESS: -- other articles. You have to build –

THE COURT: Dr. Crawford, please. I'm just trying to understand each article.

Defense counsel questioned Crawford on Sorbe et al., *Some Important Factors in the Molding of the Fetal Head During Vaginal Delivery – A Photographic Study*, 21 Int'l J Gyn & Obstetrics 205-212 (1983), which dealt with molding. The article concluded that neither the frequency of the contractions nor the length of stage 1 labor were of importance. However, Crawford believed a prolonged *second* stage of labor – which was three and a half hours in this case – was very significant. The article did not specifically state that increased molding causes stroke; instead the focus of the article was the mechanical forces of labor leading to compression of the head. While molding can occur with natural labor, Crawford testified that "if you increase the number of contractions and have tachysystole and increased intrauterine pressure, you can have even greater mechanical forces of labor."

Crawford discussed Lapeer et al., *Fetal Head Molding: Finite Element Analysis of Fetal Skull Subjected to Uterine Pressures During the First Stage of Labor*, 34 J Biomechanics 1125-

1133 (2001), and how the size of the baby, first time birth, and use of Oxytocin are factors that increase the risk of molding, which may result in stroke. The following exchange took place regarding the article and the effect of molding on an infant:

> THE COURT: [Excessive molding may be a] causative factor, but it says may and that's what I'm struggling with. Will is different to me than may.
>
> THE WITNESS: Well medicine is not always 100%. In other words –
>
> THE COURT: I'm not –
>
> THE WITNESS: -- if you have a fever and I give you an aspirin –
>
> THE COURT: Trust me, I'm not, stop. Stop. I'm not looking for 100%, okay? Not 100%. I get that. Help me understand what this article is saying when it says may.
>
> THE WITNESS: I think what they're trying to say is that molding may be helpful to get the baby's head through, but if you have obstruction, if you have dystocia and you get, uh, so much force and compression on the head that you get significant molding. That is not just molding, that's significant molding. And that is not a good thing. Some babies may not be affected, but if you have a baby that has an intracranial injury where you've had that degree of molding which implies excessive forces, particularly during the first stage where the uterus is compressing the head, that's a cause.
>
> MR. FIEGER: Are you through, your Honor?
>
> THE COURT: Yes.

BY MR. FIEGER:

> *Q*. Doctor, in medicine is there anything 100%?
>
> *A*. Not, not that often.
>
> *Q*. Are there people who smoke that don't get lung cancer?
>
> *A*. Yes.
>
> *Q*. Does that mean smoking isn't –
>
> THE COURT: Don't waste my time with that, Mr. Fieger.
>
> MR. FIEGER: Well your Honor, you asked the question.
>
> THE COURT: I said I'm not asking for 100%.

BY MR. FIEGER:

Q. When medicine says may, does that mean in some instances if you subject a baby to those forces of labor, you can cause brain injury?

A. Yes.

Q. Okay. Are there some babies who it may not affect?

A. Yes.

Q. But does that mean that since it can happen sometimes, it may happen sometimes and sometimes that it may not, that it's okay to do it?

A. The risk is that you will injure the baby.

Crawford acknowledged that the Lapeer article was actually published by an engineering journal. The article concluded that excessive head molding may result in cranial birth injuries, but did not attempt to answer whether the same forces that caused fetal head molding caused stroke. Its focus was on the first stage of labor when the uterus was the "deforming agent."

Crawford testified as Clancy et al, *Focal Motor Seizures Heralding Stroke in Full-Term Neonates*, 1390 Am J Diseases in Children 601-606 (1985), which discussed strokes in full term babies. A seizure may be a sign of an infant having had a stroke. Plaintiff had a seizure after birth. The article says that hypoxia and ischemia can cause stroke.

Crawford rejected the notion that there was no recognition that hypoxia ischemic injury can cause stroke—"My knowledge is there's ample evidence that says that it is. It's not the only cause of a stroke, but it certainly is a well recognized cause of strokes . . . it certainly is a known causative factor." Crawford pointed to Govaert et al., *Diagnosis of Perinatal Stroke II: Mechanisms and Clinical Phenotypes*, 98 Acta Pediatrics 1720-1726 (2009) and Chabrier et al., *Obstetrical and Neonatal Characteristics Vary with Birthweight in a Cohort of 100 Term Newborns with Symptomatic and Arterial Ischemic Stroke*, 14 J Eur Paediatric Neurol Soc'y 206-213 (2010), for the notion that the bruising, swelling and molding of plaintiff's head indicated trauma, which was a risk factor. The trial court asked – "I'm trying to understand what the trauma is." The following exchange took place:

THE COURT: Wait just a minute. Wait a minute. Is there anywhere in here that I can understand what the trauma was that, that says was a cause of the perinatal stroke. That's all I wanted to know. And if there isn't there isn't.

THE WITNESS: I think the main, the point they made is the obvious stroke related to those kind of traumatic insults [vacuum and forceps] was not common.

Crawford explained that excessive uterine contractions was simply another form of trauma that the baby's head could experience. "So forceps, vacuum, excessive contractions impacting on the baby's head is another form of trauma. You don't need forceps. You don't need vacuum extraction to have trauma to a baby's head during labor." Likewise, hyperstimulated labor is a force of labor or "mechanical trauma."

Crawford acknowledged that the Govaert article was largely observational with no controls and therefore, no way to see what caused the strokes. The article did not define trauma. Crawford explained that the Chabrier article explored the different mechanisms for stroke in big and small babies. There was no control group, but Crawford believed that the risk factors were the same regardless of how the data was collected. Additionally, not all strokes were immediately diagnosed. If a newborn demonstrated signs of stroke, imaging was likely done quickly, whereas if there was a stroke in a different part of the brain, it may take months to see the effects. The article did not specifically say that increased intracranial pressure leads to decreased cerebral profusion and stroke, but Crawford noted that birth trauma and acute asphyxia are more common in big babies. Crawford believed that the article stood for the position that "bigger babies have more strokes because they get stuck. They have the prolonged compression of their head."

Crawford pointed to Filipiek et al, *Focal Cerebral Infarction in the Newborn: A Distinct Entity*, 3 Pediatric Neurol 141-147 (1987), which acknowledged that birth trauma was a cause of cerebral artery infarction even though it was not present in the patient for that particular case.

Crawford explained that Hamid et al., *Perinatal Arterial Ischemic Stroke: An Update with Literature Review*, 58 J Pak Med Ass'n 395-399 (2008), also pointed to the factors contributing to stroke, including: assisted deliveries, cesarean section, birth asphyxia, and intrapartum trauma. Crawford believed that the "more factors you have, the more likely it is that you'll have a stroke due to those factors." In this case, there was "excess uterine contractions, the effects of Pitocin leading to excessive molding and prolonged and ongoing compression of the fetal head." Crawford admitted the article was a literature review, not a scientific study. It referenced the Chalmers article regarding risk factors, but did not specifically conclude that increased intracranial pressure led to a decrease in cerebral profusion and a stroke. (H II, pp 226-228.)

Crawford testified that Cheong et al., *Neonatal Arterial Ischemic Stroke: Obstetric Issues*, 14 Seminars Fetal & Neonatology Med 267-271 (2009), indicated that chorioamnionitis and prolonged rupture of the membranes were risk factors for perinatal ischemic stroke and "widely associated with cerebral palsy." Crawford acknowledged that the Cheong article did not specifically relate intracranial pressure to decreased cerebral blood flow and stroke, but she explained that it talked of overdosing with Pitocin, leading to prolonged tachysystole, which can cut off a baby's oxygen. It was not a controlled study, but a review article looking at the obstetric factors in neonatal arterial ischemic stroke. It concluded that although perinatal risk factors have been identified, the "ideology" of stroke remained unclear and further studies were needed. While the article did not list increased intracranial pressure as a risk of stroke, Crawford opined that it could be implied. Meconium was not independently associated as a risk factor.

In concluding direct examination, plaintiff's counsel asked:

> *Q*. So a doctor such as yourself, if you see a, a, look at a medical record such as in this case and look at Alexander Figurski's course of labor and see the following: one, he has chorioamnionitis; two, a prolonged rupture of membranes; three, a prolonged second stage [of labor]; four, fetal distress; five, emergency c-

section and has a diagnosis of perinatal arterial ischemic stroke at birth, what does a physician do to make diagnosis [] in the real world?

A. You gather information from the history and the physical, the laboratory tests, the neuroradiological imaging tests and put it all together and make a diagnosis.

Q. And when you put all of that together here, what was your conclusion?

A. He had PAIS from, from an abnormal labor that was involved with excessive use of Pitocin and head compression . . .

Crawford explained that while not one particular individual article supported her theory, a combination of the articles and the information extracted therefrom supported her ultimate opinion.

Defense counsel asked Crawford to "forget about strokes" and point to literature that supported her opinion that increased intracranial pressure causes a decrease in cerebral profusion pressure, leading to global hypoxic ischemic injury or diffuse hypoxic ischemic injury. Crawford cited the Harris article, for the notion that compression of the fetal head during normal labor may produce transient cerebral ischemia or decreased cerebral blood flow. None of the fetal sheep showed evidence of cerebral ischemia, but Crawford pointed out that the article demonstrated that "intracranial pressure will increase or can increase more than the fetal arterial pressure. And this leads to cerebral or brain ischemia." Essentially, the Cushing method would fail because the external pressure was too great. Although the study was not conducted to determine whether increased cranial pressure would increase fetal arterial pressure and produce ischemia, the author references studies to that effect. And although none of the sheep suffered ischemia, "[t]heir study was just to show that in a short period of time you can get an interruption of cerebral blood flow with increased external pressure on the head."

Defense counsel then presented Crawford with Raju et. al, *Ischemic Perinatal Stroke: Summary of a Workshop Sponsored by the National Institute of Child Health and Human Development and the National Institute of Neurological Disorders and Stroke*, 120 Pediatrics 609-616 (2007), which concluded that "there are no reliable predictors of ischemic perinatal stroke on which to base prevention or treatment strategies." But Crawford pointed out that the author also concluded that perinatal stroke is usually multifactorial. Crawford believed that politics caused the article to conclude as it did.

C. TRIAL COURT'S OPINION

After taking the matter under advisement, the trial court issued a lengthy opinion and order on August 21, 2013. The court noted that defendants did not dispute Dr. Crawford's qualifications or whether expert testimony would assist the trier of fact. Rather, defendants challenged the scientific basis underlying plaintiff's causation theory.

The trial court first undertook a review of the literature in support of plaintiff's causation theory as it pertained to the alleged hypoxic-ischemic injury.

The court found that Dr. Crawford relied on the FDA-approved package insert for Pitocin, warning that overdosage could result in hyperstimulation of the uterus leading to fetal hypoxia, and that management of an overdose consists of immediate discontinuation of Pitocin. The trial court noted that

> while the package insert certainly states that fetal hypoxia may result from a Pitocin overdose and associated uterine hyperactivity, it does not indicate the mechanism by which such an injury may occur. In particular, it fails to lead to the conclusion that the hyperstimulation caused by the use of Pitocin will lead to increased intracranial pressure, decreased cerebral perfusion pressure and a hypoxic-ischemic injury. Consequently, without more, one cannot rationally extrapolate Plaintiff's causation theory from the warnings contained on the insert.

The court found that although it is indisputable that a baby's head can be likened to a battering ram, the literature cited by Dr. Crawford did not support a conclusion that hypoxic-ischemic injury of perinatal arterial ischemic stroke will occur as a result of that mechanism.

The trial court rejected Crawford's reliance on the Deaver, *Etiological Factors in Cerebral Palsy*, 28 Bull NY Acad Med 532-536 (1952), because it was an observation review and concluded that trauma, especially when a child was large and the pelvis was small, could cause injury. The article did not describe the mechanism of any injury. The trial court noted that "[a]lthough the [sic] Dr. Crawford has made reference to cephalopelvic disproportion, she referenced it only as [a] factor contributing to the mechanism of injury (increased intracranial pressure) and not as the mechanism of injury or trauma on its own."

The trial court rejected Crawford's use of the Towner article because it was an observatory review and not a scientific study. It did not deal with the causal mechanism alleged in this case, but only with operative vaginal deliveries. "This article does little to support Plaintiff's causation theory."

The trial court also rejected the Harris articles as well as Cushing, *Concerning a Definitive Regulatory Mechanism of the Vasomotor Centre Which Controls Blood Pressure During Cerebral Compression*, 12 Bull Johns Hopkins Hosp 290-292 (1901); and Paton et al., *Harvey Cushing and the Regulation of Blood Pressure in Giraffe, Rat and Man: Introducing "Cushing's Mechanism"*, 94 Exper Physiology 11-17 (2009), noting the "significant gap" between the scientific findings in those articles and Crawford's conclusions. As an example, the trial court pointed to Crawford's acknowledgement during cross-examination that the Cushing response was highly effective in sustaining cerebral blood flow. Additionally, the purpose of the study was the evaluation of the Cushing response in fetal sheep. The trial court noted that "[o]verall, the articles/studies [] lend essentially nothing to Dr. Crawford's conclusion/opinion that Cushing response may be overcome, opening the door for brain damage."

The trial court rejected the O'Brien article for Crawford's proposition that simulated uterine contractions in fetal sheep reduced cerebral perfusion. By Crawford's admission, none of the sheep suffered hypoxic-ischemic injury. The study indicated that redistribution of cerebral blood flow was unclear. "Consequently, without more, the O'Brien study/article fails to draw the causal link asserted in the Plaintiff's theory that forces of labor can lead to increased

intracranial pressure and a subsequent decrease in cerebral brain perfusion pressure resulting in a stoke or hypoxic-ischemic injury."

The trial court noted that while the Sorbe and Lapeer studies of molding showed that Pitocin-induced labors were more likely to result in molding, they did not conclude that excessive head molding could result in injuries such as hypoxia, ischemia, or stroke.

The trial court also rejected the Sato article and Schwarcz et al., *Pressure Exerted By Uterine Contractions on the Head of the Human Fetus During Labor in Perinatal Factors Affecting Human Development*, in Pan Am Hearlth Org, Sci Pub No 185 (1969), pp115-126, because the articles addressed pressures exerted on the fetal head during uterine transactions, but they did not conclude that such forces were likely to cause hypoxic-ischemic injuries. The trial court noted that the latter Schwarcz article only indicated "possible" damage to the brain and that further study was necessary.

The court noted that separate from Dr. Crawford's affidavit, plaintiff submitted additional articles: Hayes et al., *A Case-Control Study of Hypoxic-Ischemic Encepholopathy in Newborn Infants at > 36 Gestation*, 208 Am J Obstetrics & Gyn (2013); and Heuser et al., *Tachysystole in Term Labor: Incidence Risk Factors, Outcomes, and Effect on Fetal Heart Tracings,* 209 Am J Obstetrics and Gyn 1-6 (2013. The trial court noted that while Hayes and Heuser found that excessively frequent uterine contractions was a risk factor associated with hypoxic ischemic encephalopathy, and use of Pitocin was a strong predictor of excessively frequent uterine contractions during labor (or tachysystole), the articles did not link those contractions with hypoxic-ischemic injury.

Turning to the causation theory regarding perinatal arterial ischemic stroke (PAIS), the court found that Dr. Crawford and the cited articles only identified a number of risk factors associated with PAIS, and made no findings that would support plaintiff's theory of causation.

The trial court rejected the Clancy article because although it noted "that the etiology of stroke in three of the infants was intrapartum hypoxia-ischemia, it makes no finding or description as to the mechanism causing the hypoxia-ischemia or how global/diffuse cerebral injury could cause a focal injury."

The trial court also rejected the Kumar article for the proposition that natural childbirth could cause stroke as a result of the skull being subjected to abnormal forces. The use of a vacuum extraction was abnormal force and the study indicated that doctors should only implicate vacuum application "as a cause of stroke when they have ruled out other potential causes." Likewise, the trial court rejected Roessmann et al., *Thrombosis of the Middle Cerebral Artery Associated with Birth Trauma*, 30 Neurology 889-892 (1980), because it involved forceps (p 21 n 11).

The trial court rejected the Govaert article because "[a]lthough the article identifies 'asphyxia' as a cause of arterial ischemic stroke, it says nothing as to the mechanism causing the underlying asphyxia." Likewise, the trial court rejected Ment et al., *Perinatal Cerebral Infarction*, 16 Annals Neurol559-568 (1984), because "although cerebral infarction was noted to

be attributable to perinatal asphyxia, it makes no findings in relation to Plaintiff's causal mechanism."

The trial court rejected the Chabrier article because although the article concluded that "increased size is a risk factor associated with stroke, the article makes no mention of increased compression of the fetal head or hypoxic-ischemic injury caused by such pressure."

The trial court rejected the remaining articles and texts – including, Lee, Volpe, Cheong, because "they either 1) do not address, relate or study the Plaintiff's causation theory or 2) they identify only risk factors associated with PAIS rather than any type of determined cause."

The court found that plaintiff failed to specifically address the factors enumerated in MCL 600.2955(1).

Subsection (1)(a): The basis of plaintiff's experts' opinions had not been subject to scientific testing. "Instead, at most, the studies presented by the Plaintiff, when taken as a whole, stand for a belief that a logical assumption can be made that excessive uterine contractions could possibly increase intracranial pressure to a point and for a length of time which one could assume would potentially cause brain damage. However, an 'assumption' is not the equivalent to an actual cause and effect link."

Subsection (1)(b): The opinion was not subject to peer review. None of the articles discuss plaintiff's causal mechanism.

Subsections (1)(c) and (d): The literature did not establish a methodology or technique for testing or supporting the proffered opinion. "The inability to assess the Plaintiff's experts' opinion against generally accepted standards applicable to methodology or technique as well as a potential error rate further militate against the reliability of the opinion itself."

Subsections (1)(e): There was no evidence that the theory presented was generally accepted within the relevant medical community. Instead, the literature demonstrates that the "theory needs or warrants more study."

Subsection (1)(f): The trial court wrote:

> [I]t is clear the Plaintiff's experts have not presented a reliable basis for their opinions. Rather, the Plaintiff has relied on nothing more than the *ipse dixit* of the plaintiff's experts. In particular, as repeatedly noted in the Court's review of the literature, the articles and studies presented by the Plaintiff either completely fail to stand for the proposition they were cited for or their findings and discussion stop considerably short of the conclusion for which they were cited. Therefore, the gap between the data and the expert's opinion is only bridged by the expert's own statements.

Subsection (1)(g): Plaintiff's experts did not present a reliable basis for their opinions, and there was no evidence their opinions had been relied on outside the context of litigation.

The court found that plaintiff failed to meet his burden of demonstrating that his experts' causation opinions were reliable:

> Because the Plaintiff has failed to carry his burden to demonstrate that the opinion of his causation experts are reliable, this Court must exclude the testimony. In making this finding this Court is absolutely cognizant that its function is not to seek truth, weigh evidence or assess credibility. The Court fully recognizes that fulfilling its gatekeeper role under MRE 702 and MCL 600.2955 does not require it to hold a minitrial on causation or to determine what causal theory is definitely present in any given case. However, under MCL 600.2955 and MRE 702, it is incumbent on this Court to ensure that the Plaintiff's expert causation testimony meets the baseline threshold inquiry and that the opinion underlying the testimony is rationally derived from a sound foundation. That being said, because the literature and scientific evidence relied on by the Plaintiff fails to actually stand for the conclusions that Plaintiff's experts purportedly draw from it, this Court finds that the gap between Dr. Crawford's testimony/opinion and the scientific/medical evidence and literature from which she extrapolates it is too great. This gap not only makes the opinion unreliable it also indicates that it is irrelevant (not probative of a fact at issue) and will not assist the trier of fact to understand the evidence.

D. ANALYSIS

The trial court's opinion perhaps would have been appropriate had the trial court been sitting as the trier of fact. However, the trial court went well beyond her gatekeeping function and, instead of determining whether Crawford could *offer an opinion* on causation, the trial court actually *resolved* the issue of causation. No doubt the trial court was encouraged by defendants, who were also functioning under an erroneous view of plaintiff's burden and the trial court's gatekeeping function. In one of its motions in limine, defendants wrote that "a plaintiff has the burden of proof as to proximate causation and must present substantial evidence that excludes other hypotheses with a fair amount of certainty." That burden does not exist at the *Daubert* hearing; instead, plaintiff's burden at the *Daubert* hearing was to show that Crawford was qualified to render an opinion on causation and that her opinion was reliable and relevant. Such an inquiry must focus on principles and methodology, *not the conclusions they generate*." *Daubert,* 509 US at 594-595 (emphasis added.) The trial court failed to heed *Chapin's* admonishment that, as gatekeeper, the trial court's analysis must not hinge on discovering absolute truth or resolving genuine scientific disputes. *Chapin*, 274 Mich App at 139. Although the trial court repeatedly stated that it was aware of its role to not seek absolute truth behind the science, the record reveals that it simply failed to heed its own warning. The trial court undertook an examination of plaintiff's literature in an attempt to search for the "truth."

Particularly glaring is the trial court's failure to refer to Crawford's *Daubert* testimony in its opinion and order. Crawford explained that no single article supported her theory, but that the sum of all the articles supported her conclusion that injudicious use of Pitocin, compounded by

the other factors at birth, resulted in compression of plaintiff's head and lack of blood flow to the brain, or ischemia.[10] The trial court also completely ignored that Crawford's opinion was based, not only on the literature provided, but on her own extensive professional experience.

While the *Daubert* hearing was underway, the parties referred to an Oakland Circuit Court case that dealt with similar causation theories. That case – *VanSlembrouck v Halperin*, unpublished opinion of the Court of Appeals, issued October 28, 2014 (Docket No. 309680) – was decided while this appeal was pending. While an unpublished opinion of this Court lacks precedential value, the analysis therein is germane, helpful, instructive, and persuasive for the case at bar and we adopt its reasoning as our own. MCR 7.215(C)(1); *Paris Meadows, LLC, v Kentwood*, 287 Mich App 136, 145 n 3; 783 NW2d 133 (2010).

The child in *VanSlembrouck* had a host of neurological problems. The plaintiffs' experts opined that birth trauma caused the child's disabilities, while the defendants maintained that the child suffered from a genetic abnormality. The plaintiffs' experts acknowledged that the child's brain never fully developed, but urged that she would not have suffered significant deficits absent birth trauma. *VanSlembrouck*, slip op, pp 1-2. There was no debating that the child's birth was traumatic. While her head spontaneously delivered, her shoulders became stuck and doctors had to perform maneuvers to deliver her. She weighed 10.5 and had an Apgar score of one. She was limp, blue and unresponsive and had a fractured collarbone. *Id.* at slip op, p 2.

Like the case at bar:

> Plaintiffs' experts posited that Pitocin-induced hyperstimulation of Kimberly VanSlembrouck's uterus, combined with Markell's large size, compressed Markell's head during the last hour of Kimberly's labor. According to their theory, head compression resulted in cerebral ischemia (lack of adequate blood flow to the cerebrum), bleeding into the brain itself, and permanent brain damage attributable to the trauma. [*Id.* at slip op, p 2.]

The trial court conducted a four-day *Daubert* hearing on defendants' motion in limine to prevent the experts from offering such a theory of causation. The trial court found the plaintiffs' experts were qualified and their opinions were scientifically reliable. Ultimately, a jury found in favor of the plaintiffs. *Id.* at slip op, p 3.

On appeal, this Court looked at the evidence presented by the plaintiffs at the *Daubert* hearing. Like in the case at bar, the plaintiffs offered Dr. Crawford, Dr. Yitzchak Frank, Dr. Gabriel, and Dr. Barry Schifrin. *Id.* at slip op, pp 9-10.

Crawford testified that it was well known that trauma may occur when a baby's head acts as "a battering ram" against the mother's pelvis and that the trauma may be manifested as a brain bleed. This was especially true in large infants.

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[10] Even if there was no global injury, the uncontested fact remains that plaintiff suffered a perinatal arterial *ischemic* stroke.

> In her opinion, Markell's brain injury was attributable to "[l]ack of oxygen and lack of blood flow." She elaborated: "This baby was banged through the pelvis for a long period of time. The uterus was stimulated to contract excessively" by Pitocin. "[W]here you have so frequent contractions that you don't provide oxygenated blood to the baby's brain ... [y]ou cause increased pressure, the blood can't profuse the brain." [*Id.* at slip op, p 10.]

When confronted with the incongruence of her theory with the ACOG Task Force on Neonatal Encephalopathy and Cerebral Palsy, Crawford rejected the report and found that it was the medical community's self-serving attempt to cut down on lawsuits. *Id.* at slip op, p 11.

Dr. Gabriel similarly testified that the child's brain injury occurred as a result of lack of blood supply, or "ischemic abnormality to the brain." This was brought about by pressure on the child's skull during labor and delivery. He explained:

> "by virtue of reduced blood flow to the brain because the high pressure, the abnormal, the non-physiological [pressure] on the skull plates, what we call the calvari[um], during the delivery process increases the pressure in the brain which in turn reduces the ability of the arteries to supply the brain with blood. The artery pressure has to fight against the increased pressure in the brain. As a consequence blood flow diminishes and the cerebral blood flow diminishes to a point where ischemia can occur. It can occur global or [diffused] or focal or regional or multi-focal." [*Id.* at slip op, p 12.]

Like in the case at bar, Gabriel supported this theory with reference to Volpe, *Neurology of the Newborn*. Finding the actual passage in the text helpful for review, this Court bolded the text from Volpe: "'**when intracranial pressure increases, cerebral perfusion pressure decreases; if intracranial pressure increases markedly, cerebral perfusion pressure declines below the lower limit of autoregulation and CBF [cerebral blood flow] may be impaired severely**.'" *Id.* at slip op, p 13.

Dr. Schifrin testified that ischemia resulted in a decrease of blood flow, depriving the brain of oxygen.

> Maximum oxygen exchange between baby and mother occurs when the uterus is not contracting. "The greater the amount of uterine activity ... the greater the interference of oxygen availability." When the uterus contracts, Dr. Schifrin testified, the baby raises its blood pressure "slightly to overcome the rise in pressure in the uterus," thereby maintaining adequate blood flow to the brain. Usually, this mechanism allows a baby to preserve enough blood flow during contractions to protect the brain from injury. But the baby's ability to autoregulate flow in this manner may be overwhelmed "if the pressure is so high either because of the duration of the contractions" or when the "added effects of pushing" increase the amplitude of the contractions. Ischemia occurs when the duration or intensity of the uterine contractions overcomes the baby's ability to raise its blood pressure to compensate for the pressure being exerted by the uterus.

> In such circumstances, the baby may suffer an ischemic (rather than an hypoxic) injury. [*Id.* at slip op, p 14.]

As in the case at bar, the electronic fetal monitor strip indicated that the uterine activity was excessive "due to the administration of Pitocin." *Id.*

Unlike the case at bar, the defendant in *VanSlembrouck* offered a number of their own experts in an effort to challenge the plaintiffs' causation theory. *Id.* at slip op, pp 14-15.

In affirming the trial court's decision to allow the plaintiffs to present their causation theory, this Court first noted:

> that the following § 2955 factors are not germane to this case: "(a) Whether the opinion and its basis have been subjected to scientific testing and replication," and "(d) The known or potential error rate of the opinion and its basis." Defendants do not explain how plaintiffs' theories of fetal head compression could be subjected to scientific testing and replication in human children or evaluated regarding an "error rate." Nevertheless, *several medical articles submitted by plaintiffs describe scientific studies involving fetal sheep.* ***These studies lend support to plaintiffs' causation theory***. [*Id.* at slip op, p 22 (emphasis added).]

The Court then looked to the two factors in subsection 2955 which require the trial court to examine the scientific literature, particularly (b) which asks whether the opinion has been subject to peer review, and (g) whether the opinion has been relied upon by experts outside of litigation. The Court noted the volume of literature supplied in the case. *Id.* at slip op, p 22. Many of these same articles and treatises were presented in the case at bar:

> *Multiple peer-reviewed articles supplied to Judge Nichols lent credence to plaintiffs' experts' causation theory. Specifically, several articles and textbook excerpts substantiated that a traumatic birth process can cause fetal head compression, which in turn may result in brain bleeds and permanent neurological injury.* Dr. Crawford's thesis that in the presence of cephalopelvic disproportion the fetal head acts as a "battering ram" against the maternal pelvis emanates from a 2007 article published in a peer-reviewed obstetrical journal. This article corroborates that brain bleeding may result from head trauma:
>
> > Virtually all significant fetal head and neck injuries that are associated with vaginal (both spontaneous and operative) delivery can be explained by the use of force to overcome cephalopelvic disproportion. Cephalopelvic disproportion is a relative term as each specific maternal fetal pair is unique; unique fetal size and positioning in the maternal pelvis and unique pelvis size and shape. As the fetal head descends into the pelvis, it can be likened to a battering ram taking the brunt of the pelvic resistance leading to molding to allow passage. Molding of the fetal cranium eventually can overcome the disproportion, but potentially at a cost. Excessive molding leads to distortion of the relatively fixed

> tentorium and falx structures and subsequent tearing leading to subdural hemorrhages....
>
> The scalp is the fetal defense to the resistance of the birth canal tissues, both soft tissue and the bony pelvis. With significant resistance and repetitive pushing against this resistance, shear forces can be generated leading to scalp trauma and cephalohematomas. [Towner and Ciotti, *Operative Vaginal Delivery: A Cause of Birth Injury Or Is It?*, 50 Clinical Obstetrics & Gynecology 563, 571 (2007).][11]

A peer-reviewed medical journal article published in 1983 similarly explains that "[t]he mechanical forces of labor subject the infant's head to considerable compression, shearing, and molding. Intrapartum and neonatal death can occur from mechanical trauma to the brain during birth." Sorbe & Dahlgren, *Some Important Factors in the Molding of the Fetal Head During Vaginal Delivery—A Photographic Study*, 21 Int'l J Gynaecology & Obstetrics 205 (1983).[12]

***

The Volpe textbook also supports that mechanical trauma can damage a fetus's brain:

> In this discussion, ... "perinatal trauma" refers to those adverse effects on the fetus during labor or delivery and in the neonatal period that are caused *primarily by mechanical factors*. Thus specifically excluded are the disturbances of labor and delivery that lead principally to hypoxic-ischemic brain injury.... (Nevertheless, overlap between mechanical trauma and the occurrence of *hypoxic-ischemic* cerebral injury is important to recognize because perinatal mechanical insults may result in primarily hypoxic-ischemic cerebral injury, probably secondary to disturbances of placental or cerebral blood flow.) [Volpe, *Neurology of the Newborn* at 813 (italics in original, bold added).][13]

In a 1952 article, the author specifically identifies "trauma due to cephalopelvic disproportion" as a cause of cerebral palsy, elaborating:

> Most of the traumatic causes of brain injury at birth may be considered as physiologic. Just being born is a difficult hurdle to pass. In the birth process, the baby uses its head for a battering ram propelled by strong uterine contractions. When the child's head is large and the pelvis small,

[11] Exhibit 9 to Crawford's affidavit in this case.

[12] Exhibit 15 to Crawford's affidavit in this case.

[13] Exhibits 28 and 30 to Crawford's affidavit in this case.

> the natural safeguards which allow the skull to conform to the shape of the birth canal may be insufficient to protect the brain from injury. [Deaver, *Etiological Factors in Cerebral Palsy*, 28 The Bulletin: NY Acad Med 532, 536 (1952).][14]
>
> ***
>
> These articles generally validate that cephalopelvic disproportion and difficult, traumatic delivery can cause fetal distress, compression of the fetal skull, brain bleeds, and neurologic injury satisfying MCL 600.2955(b) and (g). [*VanSlembrouck*, slip op, pp 22-24 (some emphasis in original).]

The Court noted that the articles were primarily written by physicians other than the testifying experts. *Id.* at slip op, pp 24-25 n 17.

The remaining factors -- subsections § 2955(e) and (f) – dealt with whether the proffered theory was generally accepted. "Although defendants' experts claimed that plaintiffs' causation theories had been debunked or were no longer accepted as scientifically valid, defendants produced no literature supporting this argument. Given that plaintiffs' literature submissions corresponded to their causation theory, Judge Nichols did not abuse his discretion in finding the data 'legitimate.'" *Id.* at slip op, p 25 n 18.

In addressing the scientific reliability of the plaintiffs' proffered opinion under MRE 702, this Court noted that

> Trial courts must carefully evaluate whether adequate data supports an expert's opinion and whether the opinion qualifies as reliable in the relevant expert community. Part of this process involves consideration of alternate scientific explanations for a given result. . . . However, this does not mean that a trial court is empowered to decide which of two competing and adequately supported scientific theories should prevail. [*VanSlembrouck*, slip op, pp 26-27.]

The Court noted that *General Electric Co v Joiner*, 522 US 136, 142; 118 S Ct 512; 139 L Ed 2d 508 (1997) mandated that a trial court "close the evidentiary gate" only when "an expert's conclusions lack any genuine relationship to the science alleged to support them." *VanSlembrouck*, slip op, p 27. The Court then noted how the science and facts appeared to support both parties' causation theories. *Id.* "Faced with this conflict among the experts, the trial court did not abuse its discretion by deciding to admit both theories, finding both supported by peer-reviewed literature and credible expert opinion, thereby qualifying as reliable." *Id.* at 28.

The Court then rejected the defendants' attempt to impeach the plaintiffs' theory with evidence that Dr. Gabriel's causation testimony had been deemed inadmissible as unreliable by numerous other panels of the Court. The Court noted that "*Daubert* and *Craig* instruct that a

[14] Exhibit 8 to Crawford's affidavit in this case.

trial court's admissibility decision must flow from the record created during the reliability hearing." *Id.* at slip op, p 28. The Court also found unavailing the defendants' attempt to use *Craig* as res judicata of the issue of whether Pitocin caused birth trauma. The Court noted that in *Craig,* Dr. Gabriel's opinion lacked evidentiary support. "Unlike in *Craig*, the peer-reviewed literature in this case supports that head compression can cause brain injury" and the "plaintiffs' experts had no difficulty explaining the head compression mechanism." *Id.* at pp 29-30.

Looking to *VanSlembrouck*, we adopt its reasoning and conclude that plaintiff in this case presented sufficient scientifically reliable data to advance its causation theory. While defendants maintain that there is no known cause of PAIS and that further study is needed, they do not dispute, however, that there are many identified factors that are found in PAIS cases. And while it is plaintiff's burden to show that the experts' opinions are sound, it is notable that defendants failed to offer their own expert at the *Daubert* hearing to debunk Crawford's theory. Even if plaintiff's theory can be deemed "shaky," "[v]igorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence. . . . These conventional devices, rather than wholesale exclusion under an uncompromising 'general acceptance' test, are the appropriate safeguards where the basis of scientific testimony meets the standards of Rule 702." *Daubert,* 509 US at 596. Again, *Chapin* cautions:

> An evidentiary hearing under MRE 702 and MCL 600.2955 is merely a threshold inquiry to ensure that the trier of fact is not called on to rely in whole or in part on an expert opinion that is only masquerading as science. The courts are not in the business of resolving scientific disputes. The only proper role of a trial court at a Daubert hearing is to filter out expert evidence that is unreliable, not to admit only evidence that is unassailable. The inquiry is not into whether an expert's opinion is necessarily correct or universally accepted. The inquiry is into whether the opinion is rationally derived from a sound foundation. [*Chapin* 274 Mich App at 139.]

The trial court, in exceeding her role as gatekeeper, attempted to find absolute truth in the literature. Instead, the matter should have been presented to the trier of fact.

V. PLAINTIFF'S DAMAGES EXPERT

Defendants argue that the trial court abused its discretion when it determined that Gamboa could offer an opinion as to plaintiff's damages when he did not have sufficient knowledge, education, training, or experience in the field of economics. Because he lacked such basic knowledge, defendants claim that his methodology was unreliable. We disagree.

Defendants filed a motion in limine to preclude Gamboa from testifying because his opinion was based on "junk science" and contrary to MRE 702 and MCL 600.2955. Gamboa hoped to testify at trial regarding plaintiff's potential educational attainment, potential future earnings, and current and future cost of hiring a home health aide. Defendants however, claimed that Gamboa was not qualified to offer such an opinion. He did not have a degree in economics and thus lacked the knowledge of basic economic principles, including how to calculate educational attainment or wage growth rates. Gamboa did not know what a "Mincer equation"

was, which was the most widely accepted standard in analyzing a person's earning potential. He practiced self-proclaimed "vocational economics," but because he lacked a proper understanding, defendants maintained that Gamboa's methodologies were flawed and unreliable. Although he published and presented talks on these methods, these activities were limited to litigation. Gamboa's inflated growth factor provides for a 5.2% increase in wages regardless of education. He used the same rate to calculate the future cost of medical care by assuming the attendants will likewise experience a 5.2% growth rate in wages. To the extent that Gamboa relied upon legitimate sources, there was a "yawning analytical gap" between the data and his opinion.

The issue was debated at a June 13, 2013 hearing. The trial court noted: "I don't disagree that the argument shouldn't be, and I won't let it be, about whether [the growth rate] should be 3% or 5.2%. Absolutely not. It's whether the opinions he's given are based on reliable science and – and principles and peer review and not just part of litigation. I don't care if it were 10% if it could be backed up by all of that."

A. GAMBOA'S DEPOSITION TESTIMONY

Gamboa received a Bachelors of Science in Education, with a major in History and a minor in English in 1966. A year later in 1967 Gamboa completed a Masters Degree program in Guidance and Counseling, with an emphasis in Vocational Counseling. Gamboa went on to receive a Ph.D. in Guidance Counseling, with an emphasis on Vocational Counseling, as well as Research and Statistics. He had also taken post-doctoral work. Some of it was closely related to vocational counseling. His MBA had no relation to his Ph.D., but was very similar in terms of research. Gamboa completed a ten-month course with an econometrician (economist with strong statistical background), which focused on the economics of disability and the development of the first work-life expectancy tables for persons with disabilities.

Gamboa did not have a degree in economics. He acknowledged that the American Economic Association was the main association for economists. The following exchange took placed during Gamboa's deposition:

> *Q*. The American Economics Association defines different fields of research within economics.
>
> Correct?
>
> *A*. I believe that is correct.
>
> *Q*. And one of the research areas within the field of economics is something called, "labor economics"?
>
> *A*. Yes. That, I'm familiar with.
>
> *Q*. Okay. The term "vocational economics", is that a term of art for your company, or is that one of the areas or fields of research within economics recognized by the American Economic Association?

A. It's the former. Just as you have persons who identify themselves as "home economists", I don't believe the American Economics Association recognizes home economists or home economics as a subset of the field of labor economics or the subset of economics, per se.

But I can tell you that "vocational economics" is a term that we began using because we felt that it really described what we were doing.

When you look at an earning capacity loss, particularly in cases of partial disability, it's about 90 percent vocational and maybe 10 percent economics.

When you're looking at a case such as this, it's more vocational, really, than economics.

Gamboa prepared between 200 and 300 vocational economic assessments on an annual basis. Nearly all of the cases were litigation related. They prepared two to three Life Care Plans reduced to present day value.

Gamboa testified that in generating his report for this case he first interviewed Mary Figurski to determine the parents' level of education. The mother had an associate's degree and worked as a paralegal. The father had a high school diploma and worked as a sales consultant. Gamboa also asked about plaintiff's particular limitations. The mother reported that the four-year-old plaintiff had spastic hemiplegia and significant weakness in the right arm and leg. He walked with a brace around the foot and frequently fell. Plaintiff had an individualized educational program. He spoke in four to five word sentences and was in a special needs school where he received occupational therapy, speech therapy, and physical therapy. Plaintiff was eight to 10 months behind his peers developmentally. He was not yet potty trained. The mother reported that she did not believe plaintiff would ever live independently. Plaintiff could not dress himself and was limited in terms of daily living. After speaking with the mother and reviewing plaintiff's medical information, Gamboa determined that plaintiff was "catastrophically injured" and would "never live independently, and who will be dependent on others throughout the life expectancy." Gamboa explained that even though plaintiff was only 10 months behind his peers, that would likely change, "because what happens with youngsters who are as severely impaired as he is, they tend to grow into the disability, which means that the older they get and the more they're compared to their peer group, the further down the bell-shaped curve they fall."

Gamboa was asked to assess plaintiff's earning capacity loss and to perform a present value calculation on the Life Care Plan prepared by Dr. Yarkony. Michigan was unique in defining future value and present value using a simple interest discount factor. Gamboa explained that he used the approach but that the legislature may have recently changed that law so that interest could be calculated in a more "conventional manner," as understood in the financial community. Under new law, the deduction will be by compound interest and present value calculations will go down significantly. He explained that earning capacity varies greatly depending on the use of simple interest versus compound interest,. The conventional approach would show 2.1 to 2.8 million dollars and Michigan's approach would yield 6.2 to 8.3 million.

Gamboa explained that he used a five-step process in making a calculation. The first step was to define a base dollar figure that reasonably represented earning capacity without any injury. He could not use actual earnings because he was dealing with a four-year-old. Instead, he used a proxy or an approximation of plaintiff's earning capacity, or so-called "human capital," which typically involved examining the parents' education and the individual's intellect and physical ability. Plaintiff's pre-injury earning capacity was the average earnings of males with no disability who have either a high school diploma or an Associate's Degree. The range was $47,000 to $59,000. It was an age-earning cycle. At age 19, the earnings would be at $19,000 or the low $20,000's, but then those earnings would go up every year, reflecting an increase in human capital as a result of education and experience. Gamboa was not familiar with the "Mencer equation." He acknowledged that there might be other methods for calculating future earnings (i.e.. regressions), but he believed his method was valid and reliable, keying in on gender, capacity to complete formal education, age, and the absence of disability had there been no injury. There were certainly more variables to consider, but Gamboa believed that generic data was the preferred method. Gamboa would then take the wage and adjust it for future growth and then bring it down using the Michigan statute to reduce the present day value. Gamboa defended the use of growth factor rate at 5.2 percent.

Step two of the analysis was the pre-injury statistical work-life expectancy, which varies as a function of age, gender, and education. The work-life expectancy range was 36.6 years for high school graduates and 37.7 years for an Associate's Degree. The numbers took into account all of the variables that could cause an individual not to be employed, including, death, disability, recession, etc.

Step three would be the amount of wages somebody could earn with the current disability. Gamboa determined that there was no earning capacity and "that this is not an individual who will ever work competitively." Gamboa was basing this on information provided by doctors and was not coming to a medical opinion on whether plaintiff was disabled. The medical doctor had opined that plaintiff would need 24-hour care for the rest of his life.

Step four would be to calculate injury work-life expectancy, which would again be zero.

Step five was the present value calculation and, in Michigan, that involved a traditional present value calculation, a future calculation, using simple interest of five percent. For computing future value, the beginning cash flow had a growth factor of 5.2 percent attached. So the future cash value for when plaintiff turned 19 grows from $47,000 to $105,000. The 5.2 percent represented the increase in wage and fringe benefits for 60 years. Gamboa looked to 1951 to 2011. The growth rate was not specific to inflation, which would have been 3.7 percent. Wages typically came in a point higher than inflation and "wage growth is inappropriate to use. You really need to be using increases or growth specific to compensation, which takes into consideration both wages and fringe benefits." "I'm not using a wage growth factor. I'm using a compensation growth factor and that's 5.2 percent." Gamboa used the Consumer Price Index for the years 1951-2011 to predict inflation. It was not possible to accurately forecast future inflation.

Gamboa believed that his offset method of using "what today's dollars are" was appropriate. He assumed that the rate of growth and the discount on percentage was the same.

His 5.2 percent historical growth for 1951-2011 did not differentiate for educational levels. Gamboa did not know whether wages had grown from 1951-2011 at the same rate for people with just high school education versus people with a Bachelor's degree. He believed that wage growth for high school graduates has kept pace with inflation. "The basis for that is that if all workers experienced increases in compensation greater than inflation, I see no reason why I would expect high school graduates would not have." When asked whether he could point to any publications that forecasted a growth rate of 5.2 percent, Gamboa responded: "I can't think of any. That doesn't mean it doesn't exist. I don't know what the forecast has been, but I certainly have not seen anything." While there were some government reports, Gamboa was not familiar with them. The earning capacity loss was in the range of 2.2 million to 2.9 million dollars.

Gamboa also calculated the present day value of the Life Care Plan. Gamboa calculated the growth rate for home healthcare workers at 3.2 percent from plaintiff's age six to 12 and then 5.2 percent for when plaintiff was 12 to 25. One was considered short term and the other was long term. (G Dep, pp 100-105.) He was not aware of the specific hourly wage in Michigan for certified nursing assistants; the Life Plan used agency data (G Dep, pp 116-117).

B. TRIAL COURT'S OPINION

After taking the matter under advisement, the trial court issued a written opinion and order on August 7, 2013, denying defendants' motion in limine to exclude plaintiff's economic expert.

The court rejected defendants' argument that plaintiff was required to obtain an expert exclusively from the field of labor economics to meet the requirements of MRE 702 and MCL 600.2955. Dr. Gamboa's knowledge, skill, and experience were sufficient to meet the requirements of MRE 702. The trial court noted that Gamboa's method had been accepted by other courts, including the Oakland Circuit Court in the *VanSlembrouck* case. "Of particular relevance in this case is Gamboa's knowledge, skill and experience dealing with the economic aspects related to vocational issues." The trial court also noted that Gamboa had written a number of published articles directly on point.

The court found that Dr. Gamboa's opinion also met the requirements of MCL 600.2955. The court found that Gamboa's methods were subject to decades of research, had been subjected to peer review, and used generally accepted standards and methods. "The Court agrees with Plaintiff's argument that general acceptance is but one of the factors to be considered in the analysis and that disagreement is to be expected and encouraged." Any known or potential error was not relevant to the inquiry. The trial court also acknowledged that damage calculations were not necessary except in the context of litigation, so it was no wonder that Gamboa's methodology was created for use in litigation.

The trial court concluded:

> Gamboa's education and experience provides him with specialized knowledge pertaining to the immediate and ongoing needs of persons with

disabilities in finding and maintaining employment. His education and experience enable him to assess an individual's loss of lifetime earnings.

He does not have a degree in economics. A degree in economics is not a prerequisite to being qualified as an expert in the area of loss of earnings as a result of an injury. His vocational expertise qualifies him to testify as to the impact of the Plaintiff's loss of lifetime earnings and work-life expectancy. His expertise involves knowledge of statistics and how disabilities impact a person's ability to perform work and earn money. These are the skills Gamboa has used for over twenty years.

The Court agrees with Gamboa that forecasting future earnings is not an exact science. Furthermore, "general acceptance" does not require that 100% of the community accept this methodology. Or, that he have a degree in economics.

This Court is convinced that Gamboa's methodology is sufficiently reliable to satisfy MRE 702 and MCL 600.2955(1). While Defendants assert evidence as to why an individual should apply the methodologies of labor economics instead of vocational economics, it is not the Court's province to decide which is preferable. That is for the jury to decide. Instead, the Court must limit its review to its gatekeeping obligations and ensure that the testimony that will be introduced to the jury will assist it in understanding the evidence or determining a fact in issue. *Gilbert, supra*.

C. ANALYSIS

Unlike with Crawford where defendants conceded that she was qualified to offer an opinion, the primary issue here is Gamboa's qualification to offer his expert opinion on plaintiff's future wage loss. As previously stated, "[t]he admission of expert testimony requires that (1) the witness be an expert, (2) there are facts in evidence that require or are subject to examination and analysis by a competent expert, and (3) the knowledge is in a particular area that belongs more to an expert than to the common man." *Surman*, 277 Mich App at 308. The so-called "trilogy of restrictions on expert testimony" includes a searching inquiry into "qualification, reliability, and fit." *Elher*, slip op, p 8.

Unlike with the causation expert, the trial court clearly understood her role as gatekeeper. Gamboa was qualified as a vocational rehabilitation expert. He held a number of degrees, including a Master's in Vocational Counseling and Ph.D. in an area that included vocational counseling and education. Gamboa also received an MBA and testified that he liked to focus on statistics. Gamboa had been with Vocational Economics Inc. in one capacity or another since 1977. His work there necessarily included offering expert opinions on the cost of future care and compensation loss. He was a prolific writer in the area of earning capacity loss and work-life expectancy. As the trial court aptly noted:

In this case, the relevant community is a broad one; one that includes any discipline that might be able to accurately and reliably offer a calculation of Plaintiff's lost future earnings and medical costs in present day dollars. Neither

> MCL 600.2955(1) nor this Court is concerned with labels used to characterize a particular field of expertise. More importantly, the Court is concerned with ensuring that the proffered testimony "will assist the trier of fact to understand the evidence or to determine a fact in issue . . ." MRE 702. Therefore, the relevant community in this case includes both labor economics as asserted by Defendants and vocational economics as asserted by Plaintiff. It is not imperative that one have a degree in economics. The Court acknowledges that Dr. Gamboa's testimony will not be purely economic in nature. He will offer to the trier of fact his vocational expertise on the expected impact of the Plaintiff's disability on his annual earnings and work-life expectancy.

Moreover, although defendants did not like Gamboa's method, there was nothing unusual about his calculating the loss of lifetime earnings by looking to what plaintiff would have earned absent an injury, what he was capable of earning with the injury, and the present value of the difference. The trial court's detailed and thorough opinion clearly reveals that her goal was not to resolve whether Gamboa's method was the correct one, but whether he was entitled to offer his opinion at all. In rejecting defendants' claim that Gamboa's method was unreliable, the trial court noted:

> Defendants are highly critical of Gamboa's use of the 5.2% compensation growth factor and the method in which it was derived. Plaintiff responds that simply because Defendants disagreed with Gamboa's methodology is not a basis for precluding him from testifying. Rather, their disagreements should be the subject of cross examination. Plaintiff argues that the process of determining lost future compensation and reducing to present value dollars is nothing new and Defendants' complaints do not undermine the reliability of Gamboa's methodology. The Court agrees with Plaintiff's argument.

The trial court correctly concluded that the differing methods of calculating plaintiff's damages was best left to the ultimate trier of fact.

VI. CONCLUSION

In Docket No. 318115, we reverse the trial court's order that granted defendants' motion in limine to exclude plaintiffs' causation experts from testifying and granted partial summary disposition on plaintiff's perinatal claims.

In Docket No. 319086, we affirm the trial court's order denying defendants' motion in limine to exclude defendant's damages expert from testifying.

We remand for further proceedings not inconsistent with this opinion. As the prevailing party, plaintiff may tax costs. MCR 7.219. We do not retain jurisdiction.

/s/ Henry William Saad
/s/ Donald S. Owens
/s/ Kirsten Frank Kelly