2012 WL 7985252 (Mich.App.) (Appellate Brief)
Court of Appeals of Michigan.

Leo TONDREAU, Personal Representative of the Estate of Sandra Peetz, Deceased, Plaintiff/Appellee,
v.
Sachinder S. HANS, M.D., P.C., Sachinder S. Li Zhang, M.D., Defendants/Appellants,
and
HENRY FORD MACOMB HOSPITAL-CLINTON TOWNSHIP CAMPUS, a foreign nonprofit corporation, and Macomb Anesthesia, P.C., a domestic professional service corporation, Defendants.

No. 300026.
March 21, 2012.

Oral Argument Requested
Macomb County Circuit Court No. 09-002913-NH

**Appellants Sachinder S. Hans, M.D. and Sachinder S. Hans, M.D., P.C.'s Brief On Appeal**

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## *v STATEMENT OF JURISDICTION

In this medical malpractice case, defendants Sachinder S. Hans, M.D. and his P.C. have been granted leave to appeal from an order entered on August 16, 2010, by the Honorable Matthew S. Switalski of the Macomb County Circuit Court, allowing plaintiff's causation experts to testify about a novel theory of “brain retraction.” See Tab A This was despite the fact that the phenomenon has never been seen or experienced by either expert, and has never been studied or reported in any medical authority. Appellants' initial application for leave to this Court was denied by order of May 26, 2011 (Tab B), but the Supreme Court subsequently decided that the issue should be reviewed prior to trial and on January 25, 2012, remanded the case under “MCR 7.302(H)(1), in lieu of granting leave to appeal... for consideration as on leave granted” (Tab C). This Court thus has jurisdiction to hear this case interlocutorily under MCR 7.205(A) and MCL 600.308(2)(e).

## *vi STATEMENT OF THE QUESTION PRESENTED

**Expert causation opinions in medical malpractice cases must be derived from recognized scientific or medical knowledge and methodology. Novel and untested theories do not qualify as admissible evidence. Plaintiffs causation experts offer a hypothesis of brain retraction never documented in the medical literature, never reported by the medical community, and never professionally encountered by these experts, despite decades of experience in the field. Should their brain retraction testimony be excluded at trial?**

The trial court answered *this question* "No."

Plaintiff-*Appellee will answer* this question "No."

Defendants-Appellants Dr. Hans and his P.C. contend that the correct *answer is "Yes."*

## *1 STATEMENT OF MATERIAL FACTS AND PROCEEDINGS [1]

### A. Introduction.

This medical malpractice action arises out of the death of 67-year-old Sandra Peetz following a procedure to remove a blockage in her carotid artery. The procedure, known as a carotid endarterectomy (CEA), was performed by Dr. Sachinder S. Hans, M.D., at Henry Ford Macomb Hospital. Mrs. Peetz died after the surgery from a chronic subdural hematoma, which is a collection of blood outside the brain and under the dura, a membrane that covers the brain and spinal cord. The pressure of the hematoma caused the brain to shift and fold into the brain stem.

Plaintiff has offered two experts willing to venture a novel theory of causation. Their hypothesis is that a reduction in blood flow to the brain during the CEA procedure caused the brain to shrink and retract from the skull. This retraction purportedly stretched and then sheared the blood vessels connecting the brain to the dura. Plaintiff's experts theorize that when blood flow was fully restored to the brain after the procedure, blood leaked through the connecting veins now sheared away from the dura and filled up the space outside the brain, forming the subdural hematoma.

The problem is, no one has ever heard of this theory of brain retraction. Neither expert could point to any peer-reviewed medical literature or case studies establishing that this phenomenon had ever occurred. And neither expert had ever personally seen or heard of a brain shrinking so rapidly as to result in a shearing away of the connecting vessels during this type of surgery. Nor has either expert personally seen or heard of a subdural hematoma (usually the ***2** result of a blunt force trauma) forming in connection with a surgical procedure. This is despite decades of practice in the field.

Because plaintiff's specific causation theory lacks the clear indicia of reliability required by MCL 600.2955(1) and MRE 702, the trial court erred in deciding that these experts could present their novel and unsubstantiated theory of causation at trial. Dr. Hans thus asks this Court to reverse the ruling of the trial court and bar plaintiff's experts from offering such an unreliable theory to the jury.

### B. Background: an introduction to carotid arteries and carotid endarterectomies.

Carotid arteries are the vessels that branch off from the aorta and migrate or travel upward on each side of the body through the neck. They carry blood and therefore oxygen to the brain and to other structures in that part of the body. [2] Like any other artery, a carotid artery can become clogged over time with plaque, lesions, etc., which can interfere with blood flow. Clogged carotid arteries can and do cause strokes. Carotid endarterectomy (CEA) is the procedure used to clear these arteries of blockage. It can be performed in one of two ways. The safest and most common method is to place the patient under local anesthesia, after which the surgeon enters the neck and clamps off the artery just below the blockage. After the blockage is cleared, the surgeon

closes the incision and unclamps the artery, allowing the blood to again pump up to the brain. While the clamp is in place and while the surgeon is clearing away the blockage, blood and oxygen are pumped through the carotid artery on the other side of the body and then through what is known as the "Circle of Willis," a network of redundant arteries circling the brain and servicing both hemispheres. [3]

***3** During every such "clamp" procedure, brain function is monitored through continued communications with the patient, who remains awake. Patients may be asked to respond verbally to questions or squeeze an object or move a limb, etc. The entire procedure usually takes anywhere from 1 to 21/2 hours, depending on the extent of the blockage. [4]

The second method of performing a CEA is to place a shunt in the affected artery to allow it to continue carrying the blood around the blockage and up into that hemisphere of the brain. The patient must be fully anesthetized for this procedure and brain activity is usually monitored with an EEG. Plaintiff and defendants in this case agree that the use of a shunt creates additional risks, such as clotting and embolisms. [5] For that reason, the preferred method is the clamp method.

**C. What happened in this case.**

On December 6, 2007, Dr. Hans performed surgery on Mrs. Peetz using the clamp CEA method. Plaintiff's standard of care expert agrees that Mrs. Peetz was an appropriate candidate for this procedure because she had a somewhat high degree of stenosis (narrowing of the artery), and because testing revealed the blockage was increasing. She was also deemed to be in good enough health to withstand the procedure. [6]

Dr. Hans began Mrs. Peetz's procedure sometime after 7:00 a.m. Anesthesia was administered at about 7:30 a.m. and the clamp was applied at 8:08 a.m. [7] As Dr. Hans worked on the artery, he realized that the blockage extended higher up than anticipated. To ensure that Mrs. Peetz remained perfectly still and suffered no pain, he increased the dosage of her medication at ***4** 9:12 a.m. [8] The CEA procedure was concluded without incident and in a timely fashion, with the clamp being removed at 10:15 a.m. and the incision then closed. Mrs. Peetz was in the recovery room by 11:00 a.m. [9]

At 12:15 p.m., Dr. Hans saw Mrs. Peetz in the recovery room and became concerned about certain neurological deficits. He suspected a clot or dissection in the carotid artery and promptly ordered her back to the operating room for exploratory surgery. [10] Again, plaintiff's standard of care expert agrees this was the appropriate course of action. [11] Dr. Hans found no clotting or lesion or embolism during this second procedure but to be safe, he inserted a stent. Plaintiff's standard of care expert still has no criticism of Dr. Hans' actions. [12] Dr. Hans then concluded his exploratory procedure and returned Mrs. Peetz to the recovery room by 3:45 p.m. After further observation following her emergence from the second round of anesthesia, Dr. Hans ordered a CT scan at 4:57. It was administered at 6:30 p.m. The scan revealed the subdural hematoma - it was a high density chronic bleed [13] outside the brain, in the space between the brain and the dura. [14] As the hematoma expanded, it compressed the brain and ultimately caused the brain to shift until it herniated into the brain stem. [15] Mrs. Peetz was declared brain dead soon after the CT scan. Life support was withdrawn the following morning.

***D. Plaintiffs theory of brain retraction as the cause of the subdural hematoma.***

Plaintiff wants to blame Dr. Hans for the hematoma. To that end, he has offered two experts: Dr. Wayne Flye, a vascular surgeon from Missouri, and Dr. Donald C. Austin, a ***5** neurosurgeon from Michigan, who last performed surgery in 1998. Dr. Flye is offered on both standard of care and causation; Dr. Austin is a causation expert only. There is no dispute that the cause of death was the high density subdural hematoma, which placed so much pressure on the brain that it caused the brain to shift to the right and herniate into the brain stem. The question is whether the subdural hematoma can be blamed on Dr. Hans due to the manner in which he performed the CEA. To make a case against Dr. Hans, plaintiff's experts came up with their novel "brain retraction" theory.

The theory begins with the proposition that when the left carotid artery is clamped during the CEA procedure, the left hemisphere of the brain becomes hypoperfused, which means it receives less than the usual flow of blood. Plaintiff's experts then deduce that in this one case, and no other, the hypoperfusion caused the patient's brain, within the span of two hours, to shrink so rapidly in volume that it pulled away from the skull and stretched and tore the "bridging veins" between the brain and the dura. [16] The theory then goes on to hypothesize that when the clamp was removed, blood pulsed through the arteries, into the brain, and escaped through the veins that had been sheared away when the brain retracted. That blood then escaped outside the brain and formed this high density hematoma.

Plaintiff's attorneys have at times tried to downplay this idea of a retracting or shrinking brain. But it is the basic premise of the causation testimony. When defense counsel asked Dr. Flye to describe cause of death, he explained that during the second hour of the operation, when Mrs. Peetz was fully anesthetized, her hypoperfused brain was "a little less turgid," "a little less ***6** engorged" than the right hemisphere, which caused the brain to "sag" away from the skull and in turn tore the "bridging veins" underneath the dura, which "initiated the bleed." [17]

Q. So it's your position that for a two-hour period of time the left hemisphere is being inadequately fed, for want of a better expression?

A. Yes. We know in the first hour the neurons are getting enough blood and oxygen to function in a gross fashion, and then the second hour we don't know.

Q. Okay. Okay. Now, how did that cause her to have the bleed?

A. **I think during the time that the brain was not as turgid, it was sort of sagging away from the skull.** Later when it begins to swell, it's swelling again, it's now - - **when the blood pressure is not as high in the brain, it sort of decreases in volume just *a little and it's pulling against these bridging veins* that cross the dural space, subdural space.**

Q. And that caused the bleed?

A. Yes, that initiated the bleed.

Mr. Jaklanen: I'm sorry?

A. Initiated the bleed. So venous bleed. This is small veins bleeding.

Q. All right. So again, so that we can make sure that I understand you, this left ventricle, this left hemisphere is not being perfused. As a result of the lack of perfusion or the absence of adequate perfusion - -

A. Yeah. I would say under-perfusion. It's being perfused some.

***7** Q. Okay. But as a result of the adequate perfusion, the - - I'm sorry. As a result of the presence of inadequate perfusion or as a result of the lack of adequate perfusion, the brain decreases in volume for the simple reason that the blood that would normally keep it at that same - - bulked up, is not there?

A. Correct.

Q. In sufficient amount and so it shrinks, and in shrinking, it tears away the venous structure that's attached to the skull?

A. To the dura, that's right. [18]

Dr. Austin testified similarly. He described "cerebral hypoperfusion" as an inadequate flow of blood through the veins. [19] In his opinion, hypoperfusion caused Mrs. Peetz's brain to retract from the inner surface of the dura and begin to bleed. [20] "What happened was that the brain began to retract and the bridging veins from the brain to the superior sagittal sinus were torn." [21]

Q.... what I'm understanding you to say is that this was sort of a one, two, three event, the brain tears away causing the initial bleed, the brain then is subject to or the vessels in the brain are then subject to the hypertension producing more blood through those vessels that have been disrupted and they don't clot off because she's on Heparin?

A. Right.

Q. Do I have it correctly?

A. Yes. Put very well. [22]

***8** Plaintiff's theory of liability clearly hinges on a causation scenario that involves a claimed or alleged shrinkage and retraction of the brain during the two hours in which the brain was, in fact, being adequately perfused by the Circle of Willis.

E. Neither expert has ever seen or heard of a brain retracting during hypoperfusion.

Both of plaintiff's experts have spent decades practicing and or teaching in their respective fields. Yet neither has ever experienced or heard of a retracting brain caused by hypoperfusion. Nor has either expert seen or heard of a subdural hematoma forming or developing following a CEA. [23] Both experts agree that this was a one-of-a-kind incident. "The dynamics of it is what's unique here." [24] When asked: "you've never seen anything like this before, though, in your practice, have you," Dr. Flye answered: "No." [25]

Nor has Dr. Austin ever had any experience with "patients who had undergone this retraction experience as a result of a carotid endarterectomy." "Not that I can recall, specifically, no." [26] As a neurosurgeon with four decades of experience, he has "no personal experience" with "a patient who suffered retraction to the point that it led to a subdural hematoma as a result of the carotid endarterectomy." [27]

Q. Have you ever seen such a case?

A. No.

Q. Have you ever read of such a case?

A. I've read of subdural hematomas.

Q. No, no, a case like this?

***9** A. No, I've not read of any, but that is irrelevant. [28]

This lack of personal experience is despite the fact that he has treated all types of problems, including subdural hematomas from other causes, and including embolisms, tumors, strokes, infection, etc.

**F. There are known risks associated with the CEA clamp procedure, but brain retraction and subdural hematomas are not among them.**

Plaintiff's attorney has argued that subdural hematomas are a known complication of uncontrolled high blood pressure during CEAs. But that argument is unsupported by any expert testimony and is the result of an imprecise use of medical terminology and an overly broad summary of the research. When the evidence is closely examined, one finds there is no evidence that Mrs. Peetz's subdural hematoma was caused by the manner in which the procedure was performed.

As is true of any medical procedure, carotid endarterectomies do present some risks, even when performed in full compliance with the applicable standard of care, a point expressly acknowledged by Dr. Flye. [29] The medical consensus, however, is that the risks are far outweighed by the benefits for most patients.

Plaintiff's attorney provided the trial court with eleven articles describing the risks of CEA. [30] Not one identified brain shrinkage/retraction and/or subdural hematomas as a complication that can result from the hypoperfusion of the brain during CEA. Known risks are:

- Wound hematomas
- Recurrent stenosis (narrowing of the artery)
- Hypertension (high blood pressure)

***10** ● Hypotension (low blood pressure)

● Cerebral hyperperfusion syndrome (headaches, cerebral edema, and on rare occasions, bleeding in the brain tissue, but not in the subdura)

1. *Wound hematomas*

Wound hematomas and blood clots are fairly common; they are caused by post-operative bleeding into the neck after the surgery has concluded.[31] A hematoma might compress against nerves and cause problems such as a lateral weakness in an arm or a leg, or a drooping of one side of the face.[32] A hematoma might compress against the artery, and cause "diminished blood flow on the operative side of the brain," "or it can be clot-forming within the artery that you've operated on. And the clot could be either blood clot or dissection of a plaque within the arterial wall itself."[33] The remedy for a wound hematoma is to "act as quickly as possible to ... decompressing; that is, opening the wound and evacuating the clot... "[34] This was exactly what Dr. Hans set out to do when he observed possible neurological deficits in Mrs. Peetz the first hour after her surgery. Dr. Flye had no criticism of that effort.

2. Hypertension and hypotension

High or low blood pressure is also a fairly common post-operative risk of CEA.[35] The reason is unclear, though the data suggests that blood pressure problems occur less often with regional anesthesia and the clamp method applied here. Hypertension and hypotension can, over time, lead to additional complications, such as cerebral edema (swelling in the brain tissue) and cerebral hemorrhaging (bleeding in the brain tissue, rather than outside the brain). Usually, the ***11** patient's blood pressure stabilizes after surgery, sometimes with the help of medicine. The goal of the surgeon is to try and control blood pressure during the post-operative period.

3. *Cerebral hyperfusion syndrome*

This is the rarest post-operative risk of CEA,[36] occurring in less than 1% of patients.[37] It is also the most devastating.[38] The syndrome refers to a set of symptoms that occur after blood flow is increased to the brain once the clamp is removed. "[L]ittle is known about the etiology and management of the syndrome."[39] It is believed, however, that the brain fails to successfully autoregulate the abrupt increase in blood flow after the clamp is removed. Another theory is that the brain has suffered some prior injury, either an infarction or some blunt force trauma. Whatever the cause, the patient experiences headaches, seizures, and in the worst cases, brain edema (swelling of the brain tissue itself) and even intracerebral hemorrhage (bleeding in the brain tissue), usually developing over the course of several days. At least four of the authorities relied on by plaintiff suggest that hemorrhaging in the brain is almost always fatal.[40]

None of the articles produced by plaintiffs identify shrinking or retracting of the hypoperfused brain and/or subdural hematomas as a known risk of the CEA clamping procedure. There is no suggestion that the brain can lose so much volume in the span of two hours as to cause it to rapidly shrink and retract from the skull, shearing blood vessel from the dura.

***12 G. Neither expert could point to any recognized medical authority in support of their brain retraction theory.**

At deposition, plaintiff's experts were questioned extensively about the basis for their brain retraction theory. They were unable to point to any medical authority or case study documenting such a phenomenon. Defense counsel expressly asked Dr. Flye "[c]ould you tell me where I could find literature to support this concept?" [41] Dr. Flye responded that it is very hard to document the phenomenon because everything happens inside of the head, out of sight, and there are no tests that can really prove what is occurring to brain volume during hypoperfusion: "in terms of clinical literature, it would be very hard to do that because you don't look at this brain normally I mean, you're not seeing the brain at these states, it's still in a closed box. And even with PET scanning where we can look at flow and transcranial Doppler studies, it won't tell you about the volume of the brain per se during this period [of underperfusion]." [42] "Well, because the brain is in a closed box, I don't know that I can find a paper for that...." [43] "I don't know that we'll find a specific paper treating exactly this condition...." [44]

When pressed about whether he could even "point to a scenario where a patient who underwent left carotid endarecteromy died within 12 hours as a result of brain herniation caused by a subdural hematoma," or to any "literature where a subdural hematoma is a component in a patient who either died or had significant injury following a... carotid endarterectomy," Dr. Flye answered "I cannot at this point, but I have not surveyed that to any degree of ***13** extensiveness." [45] He was sure the literature was out there but it would be "the neurosurgical literature," and as a vascular surgeon, he could not give any specifics. [46]

When Dr. Austin, a neurosurgeon, was asked to name the literature or studies that supported his theory of a hypoperfused brain shrinking and retracting from the skull so severely and rapidly as to shear the veins connecting to the dura, he referred to the Youmans treatise, particularly the section on endarterectomy in chapter 97, which is discussed below. [47] Otherwise, "that's just something that neurosurgeons, neurologists, vascular surgeons would know just from their training and experience," [48] even though in all his years of practice, he had never encountered such an event.

**H. The literature later produced by plaintiff's attorney likewise fails to document brain shrinkage and retraction during hypoperfusion.**

After taking the depositions of plaintiff's experts and hearing about this novel theory of brain shrinkage and retraction, Dr. Hans moved to preclude such insupportable opinion testimony at trial. In response, plaintiff's attorney produced 11 different medical treatises or journals or excerpts therefrom. A list of these authorities is provided at Tab H; copies of each article or excerpt are attached as Tabs I through S. Not one of these authorities even hints at the phenomenon of a retracting brain caused by hypoperfusion during a CEA clamp procedure. Only four articles mention subdural hematomas at all (Morrish, Yokote, Doherty and Ellis) and none in connection with a retracting brain during hypoperfusion. There is simply no suggestion in any one of these articles that a hypoperfusion of the brain during a clamping procedure can cause a rapid decline in brain volume and a retraction from the skull so as to disrupt the venous ***14** system underneath the dura and cause death in just a matter of hours. Most of the articles discuss the known risks of CEA as summarized in Section E of this fact statement.

Dr. Hans will address five articles in particular because they mention subdural hematomas or have otherwise been singled out by plaintiff.

1) *Morrish*

In 2000, William Morrish published a report on an alternative procedure used to treat severe blockages in carotid arteries between 1996 and 1999. Rather than the CEA clamping or shunting methods, these cases utilized stenting and angioplasty. Morrish's retrospective review looked at the incidence of intracranial bleeding. He reported that of the 104 carotid arteries

treated in that manner, there were four “intraparenchymal hemorrhages,” or “intracerebral hemorrhages” which refer to a bleed in the brain tissue itself. [49] This was a significantly higher percentage of bleeds than associated with CEA. [50] Plaintiff makes much of the fact that one of these four patients also had a subdural hematoma, in addition to the cerebral hemorrhage. But nothing in the Morrish report suggests that the subdural hematoma was caused by a hypoperfusion of the brain rather than a prior injury, which is how subdural hematomas are usually inflicted. And given the stenting procedure used in that study, there is no reason to even infer that hypoperfusion occurred.

2) *Ellis*

George Ellis published an article in 1990 reporting that **elderly** patients are more prone to subdural hematomas because their veins are less elastic. “The **elderly** are at increased risk of ***15** bridging vein disruption because of the increased fragility of veins that occurs with age.” [51] Ellis describes how: “most subdural hematomas result from bleeding originating from bridging veins.... lacking tortuosity and being firmly attached to the right dura at one end and the moveable hemisphere at the other, these veins are intolerant of movement of the brain.” [52] The movement Ellis is referring to, however, is described as “forces that result in high rates of acceleration onset, such as falls and assaults.” [53] He also referred to subdural hematomas associated with the atrophy of a brain over time as occurs with the **elderly**. [54] Plaintiff's attorney argues that this article is evidence that the “concepts” relied on by his experts in this case are supported by the medical literature. But the “concept” that needs support is the phenomenon of a rapidly shrinking and retracting brain during an hour or two of hypoperfusion and as to that concept, Ellis says nothing.

3) *Doherty*

Deborah Doherty reports on a 24-year-old man who was diagnosed with a subdural hematoma some 8 months after he sustained a closed head injury. Doherty's case study is cited by plaintiff because of its description of how subdural hematomas are formed through breakage of the bridging veins, a fact not in dispute. “In the presence of cerebral atrophy from any cause, the bridging veins are stretched and traverse a greater distance in the subdural space. They are thus more susceptible to tearing with minor trauma.” [55] Doherty describes how the atrophying process can also cause the brain to be “shrunken away from the inner table of the skull.” [56] Once ***16** that occurs, a “minor unnoticed trauma is sufficient to cause tearing of a bridging vein.” [57] Doherty in no way suggests that a brain can atrophy over the course of a two-hour hypoperfusion in connection with a CEA (recall that the brain is still being perfused by the Circle of Willis).

**4) Yokote**

This article is a 1985 report on three cases of chronic subdural hematomas after open heart surgery. Yokote points out that “most chronic subdural hematomas result from the rupture of the bridging veins due to minor trauma to the head.” [58] But on the basis of this study of three patients who underwent open heart surgery and reported severe headaches four to seven days later, Yokote concluded that the combination of hemodialysis and the administration of mannitol (a diuretic) might change the brain volume with sufficient speed as to lead to a subdural hematoma over the course of a few days. [59] Mrs. Peetz did not undergo open heart surgery or receive hemodialysis or mannitol. And the claim is that she developed a subdural hematoma over the course of two hours.

5) *Youmans*

Dr. Austin testified that Youmans was the authority on which he relied for his causation theory. But Youmans says nothing at all about subdural hematomas in connection with CEAs. In the section on intracerebral hemorrhages (again, bleeding in the brain itself), Youman notes that most patients with intracerebral hemorrhages, which tend to occur several days after the procedure, "have had a recent antecedent brain infarct and preoperative hypertension." [60] Youmans offers no discussion of subdural hematomas caused by retracted brains during short-term hypoperfusion.

***17 I. The Trial Court Opinion.**

The trial court understood that plaintiff's causation theory required recognition that the brain can rapidly shrink and retract during CEA clamping/hypoperfusion. [61] And the court understood that Dr. Hans was challenging the reliability of that theory. But in attempting to perform its gatekeeping role, the court focused primarily on the type of procedure and provided a thorough summary of the opinions and credentials of each expert. When it came time to discuss the medical support for this brain retraction theory, however, the court merely stated:

The literature produced by plaintiff "address[es] the concepts of preoperative hypertension, post-operative hypertension and hyperperfusion syndrome, cerebral hemorrhage, subdural hematoma, heparin, anticoagulation, change in size of the brain, rapid change of brain volume, swelling brain, brain atrophy, bridging veins, stretching and tearing bridging veins, all resulting in a common theme that the brain gets smaller through atrophy or 'rapid change of brain volume. [62]

The trial court offered no other analysis of how the literature supported the idea of rapid brain shrinkage through CEA hypoperfusion. Other relevant comments by the trial court include the following:

● There are no studies of what happens to brain volume during hypoperfusion; [63]

● Carotid hyperperfusion syndrome (or re-perfusion syndrome) is well documented but the incidence of subdural hematomas is "not as clear cut;" [64]

● Neither expert has ever seen such a rare occurrence; [65]

● Failure to control hypertension places the patient at risk for re-perfusion syndrome [i.e., high blood pressure, which is not what caused Mrs. Peetz's death]; [66]

***18** ● Dr. Austin's opinion is based on his training, his 44 years of practice [during which he never encountered this scenario], his review of the literature over the years [no study of this problem has ever surfaced], and "his attendance at major meetin's over the years having to do with neurosurgical issues and topics;" [67]

● Dr. Austin was able to point to Youmans as an authority [as noted, Youmans says nothing about brain retraction or subdural hematomas]; [68]

● "[T]he occurrence of brain retracting is just something that neurosurgeons, neurologists, and vascular surgeons would know;" [69]

● It is irrelevant that Dr. Austin has "not encountered in his own personal experience of the condition as described concerning the decedent, nor had he read of such a case;" [70]

● "[B]oth experts expressed rational bases for their conclusions. [71]

Dr. Hans and his P.C. sought leave to appeal from this ruling by timely filing an application for leave to this Court on September 10, 2011. On May 26, 2011, the Court denied the application, with Judge Saad voting to grant (Tab B). Dr. Hans and his P.C. then timely filed an application for leave to appeal with the Supreme Court, which on January 25, 2012, remanded the case under "MCR 7.302(H)(1), in lieu of granting leave to appeal... for consideration as on leave granted" (Tab C).

## *19 ARGUMENT

**Expert causation opinions in medical malpractice cases must be derived from recognized scientific or medical knowledge and methodology. Novel and untested hypotheses do not qualify as admissible evidence. Plaintiff's causation experts offer a hypothesis of brain retraction never documented in the medical literature, never reported by the medical community, and never professionally encountered by either expert, despite decades of experience in the field. This is the quintessential "Daubert" problem and the trial court erred in deciding that such testimony can be produced at trial.**

### A. Standard of Review.

Evidentiary rulings are reviewed for an **abuse** of discretion, which occurs where the trial court issues a decision outside the range of principled outcomes. Edry v Adelman, 486 Mich 634, 639; 786 NW2d 567 (2010); People v Babcock, 469 Mich 247, 269; 666 NW2d 231 (2003). The **abuse** of discretion standard has some extra "bite" when it comes to the admission of expert testimony, because the law requires trial courts to act as gatekeepers to ensure that only relevant and reliable expert testimony gets to the jury. See Krohn v Home-Owners Ins Co, 490 Mich 145, 167; 802 NWd (2011) and Daubert v Merrell Dow Pharm, Inc., 509 US 579 (1993). "A trial judge may neither 'abandon' this obligation nor 'perform the function inadequately."' Gilbert v DaimlerChrysler Corp, 470 Mich 749, 780; 685 NW2d 391 (2004), quoting Kumho Tire Co Ltd v Carmichael, 526 US 137, 158-159 (1999) (Scalia, J., concurring). And the burden is always on the proponent of the evidence to show that the evidence passes muster. Edry, supra.

**B. Expert causation testimony must be derived from proven, reliable, and accepted methods and principles.**

This is a dispute over the reliability of a truly novel theory of causation. MRE 702 requires a searching inquiry of the expert opinions advancing such novel theories. Before being admitted at trial, the opinion testimony must meet the following three conditions: (1) the expert ***20** must be qualified, (2) the testimony must be relevant to assist the trier of fact to understand the evidence or determine a fact in issue, and (3) the testimony must be "derived from recognized scientific, technical, or other specialized knowledge." Clerc v Chippewa County War Mem Hosp, 267 Mich App 597, 602; 705 NW2d 703 (2005), affirmed on relevant grounds, 477 Mich 1967; 729 NW2d 221 (2007).

> 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. [MRE 702.]

It is plaintiff, who as the proponent of the evidence, "bears the burden of establishing relevance and admissibility" under MRE 702. Gilbert v DaimlerChrysler Corp, supra, at 781, quoting People v Crawford, 458 Mich 376, 388 n 6; 582 NW2d 785 (1998). It is not Dr. Hans' burden to disprove the theory advanced by these experts. Dr. Hans did just that, however, in challenging this opinion testimony, first by showing the lack of reliability in the opinions from plaintiff's experts and then by producing the affidavits of his own expert to confirm what the literature shows: there is no known linkage between subdural hematomas and the hypoperfusion that occurs as a result of the clamping during carotid endarterectomies. [72]

MRE 702 effectively imposes a "gatekeeping role" on trial courts confronted with a challenge like this one from Dr. Hans. In Edry v Adelman, supra, the Supreme Court affirmed the dismissal of a medical malpractice case because the causation expert did no more than offer a personal opinion. In that case, Dr. Barry Singer testified that cancer survival rates could be ***21** determined from the number of lymph nodes infected when treatment started. He ignored the American Joint Cancer Commission manual, which he acknowledged as authoritative, in favor of his own personal theory. At deposition, he claimed there were textbooks and journal articles that supported his approach. But "plaintiff never produced those authorities to support his testimony" when given the opportunity. Id at 640. Instead, plaintiff's attorney performed some internet research afterward and came up with "general statistics about survival rates of breast cancer patients." Id. But, as this Court emphasized, none were "peer-reviewed published literature" supportive of the expert's opinion. Id. at 641.

Relying on Craig v Oakwood Hospital, 471 Mich 67; 684 NW2d 296 (2004), Gilbert v DaimlerChrysler Corp, supra, and Daubert v Merrell Dow Pharm, Inc, supra, the Court agreed that Dr. Singer's testimony "failed to meet the cornerstone requirements of MRE 702," Edry, id., at 640, because an expert's causation testimony must be grounded in facts, principles or methods of analysis that are demonstrably reliable:

> Plaintiff failed to provide any support for Dr. Singer's opinion that would demonstrate that it has some basis in fact, that it is the result of reliable principles or methods, or that Dr. Singer applied his methods to the facts of the case in a reliable manner, as required by MRE 702. While 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. See generally, Craig, 471 Mich at 83-84; Daubert, 509 US at 593-594. [Edry, id., at 641].

Even before Edry, Michigan appellate courts stressed that the careful vetting of **all aspects of expert testimony** is especially important when an expert **provides testimony about** causation. Gilbert, supra, at 782.

***22** This gatekeeping responsibility has even been codified by the Michigan Legislature following the lead of Daubert. MCL 600.2955(1) now requires the trial court to "examine the opinion and the basis for the opinion" in light of the following:
(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...

(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.** [MCL 600.2955(1) (emphasis added).]

(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. [MCL 600.2955(2).]

While the trial court in this case correctly cited the applicable law, it fell far short of applying the stated principles.

**C. There is no demonstrated basis for the theory that brains rapidly shrink and retract from the skull while being hypoperfused. Nor is there any further demonstrated basis for the theory that such retraction creates a risk of subdural hematomas.**

Dr. Hans looked to the trial court to perform its gatekeeping role under MRE 702 and MCL 600.2955 regarding the theory that brains rapidly shrink and retract from the skull, ***23** shearing the connecting blood vessels, as the result of the hypoperfusion that occurs during a CEA clamp procedure. Dr. Hans sought to exclude testimony that hypoperfusion causes brain retraction and subdural hematomas *and* that it is a known risk with the CEA procedure to be avoided by the surgeon. He filed a motion challenging plaintiff to come up with reliable medical support for such a causation story. Plaintiff had to meet that motion with proof that that his expert opinions were derived from their professional experience, or reported clinical studies, or other recognized medical authorities.

Neither causation witness had any personal experience to support the theory that brains shrink and retract from the skull during the temporary hypoperfusion required for a CEA clamp procedure. And both acknowledged that there were no clinical studies of what happens to the brain during this procedure. Still, the trial court was impressed with the experts' "vast experience" in connection with other medical conditions and procedures and concluded that their explanation for this unique event was rationally based, even in the absence of reported incidents of this kind, and even in the absence of personal experience or clinical studies.

Given the expert's lack of experience and knowledge concerning actual brain retractions during hypoperfusion, plaintiff met Dr. Hans' motion in limine with a series of articles. These articles generally describe the CEA procedure and the known risks,

none of which is retraction of the brain and a subdural hematoma. Some of the articles talked about subdural hematomas in other contexts, but not in connection with temporary hypoperfusion during CEA And most of the articles talked about intracerebral bleeds (bleeding in the brain tissue) as being caused by factors such as prior injuries. If anything, plaintiff's literature suggests that Mrs. Peetz had an unknown chronic bleed caused by some prior event or injury, which led to her wholly ***24** unexpected hematoma and death after undergoing the CEA. The literature produced by plaintiff confirms that in the face of such an intracranial bleed, the morbidity rate is very high.

Plaintiff managed to convince the trial court of the reliability of her expert opinions without ever discerning among the subjects reported in the literature. In a perfunctory fashion, the trial court followed suit and simply lumped all of the articles together and laundry-listed the "concepts" discussed overall. From this list, the court concluded that the literature supports the idea that brains can rapidly lose volume during the CEA clamp procedure, retract from the skull, shearing away vessels, creating the stage for a subdural hematoma. [73]

Something is clearly missing. The carotid endarterectomy is not a new procedure; it has been the established treatment for clogged carotid arteries for some decades. Much has been written and reported on it. If the clamping required for CEA can truly create a risk of rapid brain shrinkage and retraction from the skull - something would have been published on it. The fact that plaintiff and her experts can produce nothing speaks volumes.

The trial court failed to address this gaping hole in the literature. But as difficult as it may be for the non-medical layperson to digest the science - and this writer appreciates only too well how difficult that process is - that is what is required of the courts if they are to conduct a searching inquiry into the reliability of novel expert theories such as the one asserted in this case. It would be a grave error to allow this case to go to trial on such an unsubstantiated and unreliable theory.

A case decided several years ago provides a good example of how the analysis in this case ought to proceed. In Brabant v St John River Dist Hosp, unpublished opinion per curiam of the Court of Appeals, issued December 20, 2005 (Docket No. 263168)(Tab U), plaintiff alleged ***25** that defendants' delay in performing a C-section caused her twins to develop neurological problems and mental retardation. She presented the testimony of a pediatric neurologist who opined that the twin's condition was caused by reduced oxygen delivery to brain. The witness admitted, however, that there had already been neurological damage due to the mother's risk factors, which included obesity and hypertension. And his opinion concerning reduced oxygen was based solely on his own experience as a pediatric neurologist and his own unfinished research article (far more than what plaintiff offers here). S1 Op, p 6. The trial court excluded the witness and this Court affirmed. Plaintiff did not show that the expert's theory "has been tested, subjected to peer review and publication, or is generally accepted within a relevant scientific community." S1 Op, p 7.

The same result is called for here. Plaintiff's experts have set forth a simple proposition. They contend that Dr. Hans' alleged negligent performance of this CEA caused Mrs. Peetz's brain to become so underperfused that it rapidly shrunk in size and pulled away from the skull, tearing the veins to the dura. This created the path for the blood to form the subdural hematoma when the clamp was removed and blood flow was restored to the brain through the carotid artery. Dr. Hans has never heard of such a shrinking brain phenomenon and so he challenged plaintiff to support the theory with reliable medical authority. Plaintiff did not meet the challenge. She failed to produce a single report, case study or article evidencing a brain retraction during CEA. And the possibility of this phenomenon has never even been discussed in any medical literature. If ever there was a case to apply MRE 702, the statute and the Daubert line of cases, this is it.

**D. Plaintiff has never addressed the specific challenge to these causation experts.**

Dr. Hans's argument is a very focused "Daubert" challenge. The question is whether plaintiff's experts should be permitted to tell the jury that hypoperfusion of the brain during a ***26** carotid endarterectomy can cause it to shrink or retract so rapidly as to shear away the connecting blood vessels, allowing blood to flow outside the brain and form a subdural hematoma. Dr. Hans contends that this is a novel and unique theory, an untested hypothetical. He has shown that: (1) despite decades of experience as practitioners, neither of plaintiff's experts has ever seen or heard of a shrinking brain/sheared vessel event, (2) neither of

plaintiff's experts could point to any scientific studies or cases reporting such a phenomenon, and (3) the attorney-researched literature failed to uncover any support for the theory. To adequately meet this "Daubert" challenge under MRE 702 and MCL 600.2955, plaintiff must point this Court to a reliable, medical basis for submitting this hypothetical to the jury.

In responding to the motion in limine and to the appellate applications, plaintiff has spent most of his time justifying his experts' qualifications and competency to testify in this field of medicine. Dr. Hans is not questioning the qualifications of plaintiff's experts. Nor is he challenging the admissibility of Dr. Flye's opinions on standard of care, or on the known risks of this CEA procedure, or on the methods for reducing those risks. Nor is Dr. Hans challenging the admissibility of Dr. Austin's opinions on the known risks of this procedure or on the fact of a subdural hematoma, or the manner in which the hematoma caused this patient's death. To the extent plaintiff presumes otherwise in his appellee's brief, those arguments can be ignored.

What this Court must find from plaintiff's response to this appeal is some reliable basis for telling the jury that a brain hypoperfused for two hours (again, recall that blood is still being pumped to that area of the brain through the venous network known as the "Circle of Willis") can shrink so rapidly that it will retract from the skull, which causes the shearing of the connecting blood vessels and allows blood to flow outside the brain and form a subdural hematoma between the brain and dura. This Court will likely see plaintiff shying away from the ***27** shrinking brain theory of his experts. But it is clear from the expert testimony that brain retraction is the basic premise of the causation picture.[74] As much as plaintiff would like to downplay that theory, he cannot because it is the foundation of his experts' opinion testimony. Dr. Hans is entitled to a careful assessment of the reliability of this theory of retraction and its admissibility under MRE 702 and MCL 600.2955.

If plaintiff responds to this appeal as he has previously, he will concede his experts' lack of personal knowledge and experience with retracting brains and sheared blood vessels during the hypoperfusion that occurs during a CEA. Plaintiff will instead emphasize a point made by Dr. Flye regarding his experience with livers. Dr. Flye testified at deposition that when one side of the liver becomes less engorged than the other side, it becomes softer: "I would expect... [the brain] to behave much in the same fashion as the liver lobe."[75] Dr. Flye, however, provided no discussion of the similarities and dissimilarities between the two organs. And he said nothing to suggest that this "softening" of the liver was in fact a rapid shrinking and shearing away of connecting blood vessels so as to cause hemorrhaging.

To the extent plaintiff relies alternatively on the literature, this Court will see that he is referring to other medical principles in published, peer-reviewed medical studies that say nothing about his brain retraction hypothetical. Plaintiff's attorneys contend that these articles substantiate relevant "concepts" that, in turn, justify the hypothetical. But the "concepts" in these articles are, in plaintiff's own words, "traditional mainstream medicine,"[76] i.e., known risks of the procedure, such as wound hematomas, clotting, hypertension, hypotension, and cerebral ***28** bleeds (bleeding in not on the brain, and even that is a rare event that is almost always fatal unrelated to any negligence on the part of the physician). Traditional mainstream medicine does not acknowledge rapidly shrinking brains and sheared vessels during hypoperfusion.

Plaintiff has also argued that peer-reviewed publications are not a prerequisite to the admission of expert opinion testimony. This is true. See Edry, supra. But in the absence of any peer-reviewed literature, there must be some other verifiable basis for determining that the opinion is derived from good science. Usually, the alternative to published studies is personal experience, which is not present here. Plaintiff offers nothing else. His argument concerning a "differential diagnosis" begs the question, because differential diagnosis simply refers to the process of identifying all **known** diseases and conditions consistent with the presenting symptoms. At issue here is whether a shrunken hypoperfused brain is a medically recognized condition associated with the CEA procedure.

**E. How this case should look on remand.**

Dr. Flye is critical of Dr. Hans for failing to control the patient' s hypertension (high blood pressure), which he says put her at risk for a reperfusion injury after the clamp was removed.[77] In his opinion, Dr. Hans should have tried harder to bring down

her blood pressure,[78] particularly right after the initial surgery.[79] Hypertension is one of the known risks of this procedure, about which Dr. Flye can testify. Managing blood pressure is part of the standard of care, about which Dr. Flye can testify.

***29** Dr. Flye is also critical of Dr. Hans for having failed to obtain a CT scan immediately after the 2nd surgery.[80] According to Dr. Flye, once Dr. Hans learned that a clot was not the cause of this patient's sneurological deficits, he should have started looking for another answer and ordered the CT scan. Follow up care is a matter on which Dr. Flye can testify.

Plaintiff will then be left with trying to show that the high blood pressure or the failure to order a CT scan earlier caused Mrs. Peetz' s death from a chronic subdural hematoma. If he cannot do so through reliable medical testimony, defendants will be entitled to summary disposition or a directed verdict.

## *30 CONCLUSION AND RELIEF REQUESTED

For the reasons stated, Dr. Hans asks this Court to bar plaintiff's experts from testifying that hypoperfusion of the brain during this CEA caused the brain to rapidly retract and tear the blood vessels connecting the brain to the dura, thus allowing the formation of a subdural hematoma.

Footnotes

1 This statement of facts relies heavily on the plaintiff's expert testimony and medical literature and requires no fact-finding by the Court.
2 See Tab D, depiction of the carotid artery (Plaintiff's summary disposition Exhibit 1).
3 Flye Deposition, Tab E, p 90.
4 Flye Deposition, Tab E, p 47.
5 Flye Deposition, Tab E, pp 45-46.
6 Flye Deposition, Tab E, pp 36, 38, 41.
7 Flye Deposition, Tab E, p 49.
8 Flye Deposition, Tab E, p 49.
9 Flye Deposition, Tab E, p 49.
10 Flye Deposition, Tab E, pp, p 49.
11 Flye Deposition, Tab E, pp 49-50, 53.
12 Flye Deposition, Tab E, p 53.
13 Austin Deposition, Tab F, pp 45-46.
14 Austin Deposition, Tab F, p 34. 15
15 Austin Deposition, Tab F, pp 34-35.
16 Flye Deposition, Tab E, p 48.
17 Flye Deposition, Tab E, pp 58-59.
18 Flye Deposition, Tab E, pp 59-60.
19 Austin Deposition, Tab F, p 23.
20 Austin Deposition, Tab F, pp 46, 48.
21 Austin Deposition, Tab F, p 48.
22 Austin Deposition, Tab F, p 49.
23 Dr. Flye has been practicing since 1977. Dr. Austin practiced from 1963 through 1998.
24 Flye Deposition, Tab E, p 63.
25 Flye Deposition, Tab E, p 67.
26 Austin Deposition, Tab F, p 56.
27 Austin Deposition, Tab F, p 58.
28 Austin Deposition, Tab F, p 59.
29 Flye Deposition, Tab E, p 87.

30 The 11 articles are listed at Tab H, and are reproduced at Tabs I through S.
31 lye Deposition, Tab E, p 50.
32 Flye Deposition, Tab E, p 51.
33 Flye Deposition, Tab E, p 52.
34 Flye Deposition, Tab E, p 52.
35 Barash, Tab K, p 953.
36 Youmans, Tab L; Piepgras, Tab R; Adhiyaman, Tab O.
37 Morrish, Tab M p 1911.
38 Morrish, Tab M, p 1911.
39 Miller, Tab J, p 2.
40 Youmans, Tab L, describes "significant morbidity and mortality," p 1644; Doherty, Tab Q, says "[m]orbidity and mortality remain high," p 543; Piepgras, Tab R, observes that "[o]nce hemorrhage occurs, it is usually devastating and even when evacuation is performed early the outcome is poor for most patients," p 535.
41 Flye Deposition, Tab E, p 61.
42 Flye Deposition, Tab E, p 61.
43 Flye Deposition, Tab E, p 63.
44 Flye Deposition, Tab E, p 63.
45 Flye Deposition, Tab E, p 66.
46 Flye Deposition, Tab E, p 89.
47 Austin Deposition, Tab F, pp 55, 61.
48 Austin Deposition, Tab F, p 56.
49 Morrish, Tab M, p 1911, Results, and p 1912. See also Wikipedia, for a definition of intraparenchymal as "cerebral hemorrhage," accessed 9/3/10
50 Morrish, Tab M, p 1911, Conclusion.
51 Ellis, Tab S, p 285.
52 Id.
53 Id.
54 Id.
55 Doherty, Tab Q, p 543.
56 Doherty, Tab Q, p 543.
57 Doherty, Tab Q, p 543.
58 Yokote, Tab P, p 520,
59 Yokote, Tab P, p 523.
60 Youmans, Tab L, p 1644.
61 Trial Court Opinion and Order, Tab A, p 2.
62 Trial Court Opinion and Order, Tab A, p 15.
63 Trial Court Opinion and Order, Tab A, p 6.
64 Trial Court Opinion and Order, Tab A, p 6.
65 Trial Court Opinion and Order, Tab A, pp 7, 10.
66 Trial Court Opinion and Order, Tab A, p 7.
67 Trial Court Opinion and Order, Tab A, p 10.
68 Trial Court Opinion and Order, Tab A, p 13.
69 Trial Court Opinion and Order, Tab A, p 13.
70 Trial Court Opinion and Order, Tab A, p 13.
71 Trial Court Opinion and Order, Tab A, p 15.
72 See Tab T, affidavits of defense experts Chris D. Kazmierczak, M.D., Jonathan W. Hopkins, M.D., and Robert E. Lee, M.D.
73 See Trial Court Opinion and Order, Tab A, p 15.
74 See Plaintiff's Response in Opposition to Court of Appeals Application for Leave, pp 16-21, in which plaintiff cites Dr. Flye's testimony verbatim.
75 Plaintiff's Response in Opposition to Court of Appeals Application for Leave, p 18, citing Flye Deposition, p 66.

76 Plaintiff's Response in Opposition to Court of Appeals Application for Leave, p 22.
77 Flye Deposition, Exhibit E, pp 67-68.
78 Flye Deposition Exhibit E p 69.
79 Plaintiff's Response in Opposition to Court of Appeals Application for Leave, p 8; Flye Deposition, Exhibit E, pp 77-79
80 Flye Deposition, Exhibit E, p 76.

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