Bentley v. Highlands Hospital Corporation et al Doc. 564

UNITED STATES DISTRICT COURT
EASTERN DISTRICT OF KENTUCKY
SOUTHERN DIVISION
PIKEVILLE

ROBYN BENTLEY, Plaintiff, v. HIGHLANDS HOSPITAL CORP., et al., Defendants.	)	Civil No. 15-97-ART **MEMORANDUM OPINION AND ORDER**

*** *** *** ***

Science, like life, knows few certainties. So doctors who wish to offer expert testimony at trial "need not be purveyors of ultimate truth in order to be allowed on the stand." Jahn v. Equine Servs., PSC, 233 F.3d 382, 393 (6th Cir. 2000). But before they may share their opinions with a jury, the Court must be convinced that the doctors' qualifications are in order and that their testimony is relevant and reliable. Fed. R. Evid. 702. In aid of her medical-malpractice suit, Robyn Bentley offers the testimony of several expert witnesses; among them, Drs. Carlos Pardo-Villamizar, Robert DeLorenzo, and Christopher Betz. The defendants, though, insist these three doctors cannot reliably opine about how a certain treatment or particular medications might have affected Bentley. So the Court must now determine for itself whether their testimony has a reliable foundation, reliably applied to the facts at hand.

I.

A.

In the early morning hours of July 29, 2013, Robyn Bentley checked into the emergency room at Paul B. Hall Regional Medical Center ("PBH"). Bentley was nauseated, suffering

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from back pain, and having difficulty urinating. Pain and a tingling sensation, too, radiated throughout her legs, which felt sapped of their usual strength. Concerned that Bentley's spinal column might have been compromised, emergency physician Dr. Thomas Styer sent her for a CT scan. But that scan showed no problems. So Dr. Styer diagnosed Bentley with acute back pain, prescribed pain medication, and discharged her around 5:00 a.m. with instructions to follow up with her family doctor. See R. 110 at 6–8; R. 356-2 at 7–8, 11–15.

Sadly, Bentley's condition deteriorated rather quickly after that. By 9:30 a.m., she had lost control of her left foot and her reflexes were severely diminished, so her family physician, Dr. Burchett, sent her for an MRI at Highlands Regional Medical Center ("Highlands"). When Highlands performed the scan early that afternoon, its radiologist reported no signs of trouble. Unfortunately, there was a shadow in the images of Bentley's spinal cord—the radiologist just missed it. Meanwhile, Bentley's symptoms continued unabated, the loss of motor control and sensation ascending into to her abdomen. Troubled by Bentley's worsening symptoms, Dr. Burchett decided to send her to Central Baptist Hospital, a couple hours away in Lexington, Kentucky. See R. 110 at 8–9; R. 356-2 at 15–21; R. 352-3 at 13–14; R. 356-3 at 2–6.

By the time she arrived at Central Baptist, Bentley had lost motor control in both legs. She now felt, too, that her symptoms were moving into her diaphragm, making it harder for her to breathe. Doctors at Central Baptist decided to run another MRI. This time, they spotted inflammation in Bentley's spinal cord, surmised it might be Devic's disease,[1] and started her on intravenous steroids. By morning, Bentley's symptoms had stopped progressing, her ease

[1] Devic's disease, or neuromyelitis optica, is an autoimmune disorder in which the body's immune system attacks the optic nerves and/or spinal cord. *See Devic's Disease (Neuromyelitis)*, Cleveland Clinic, http://my.clevelandclinic.org/health/articles/devics-disease (last visited Dec. 20, 2016).

of breath had returned, and her pain had abated. But sadly, neither steroids, plasmapheresis, immuno-suppressants, nor months of therapy ever returned the motor control that Bentley had already lost by the time she reached Central Baptist. She remains paralyzed from the chest down. See R. 110 at 9; R. 356-3 at 7–12; R. 356-6 at 2–3; R. 356-7.

B.

A little over two years later, Bentley filed suit against PBH, Dr. Styer, and Whitaker National Corporation, the company that hired Dr. Styer out to the hospital. R. 1-2.[2] Bentley alleges that PBH and Dr. Styer negligently failed to diagnose and treat her emerging neurological condition. According to Bentley, her range of symptoms suggested that the culprit was not her spinal column, but her spinal cord. If Dr. Styer had recognized as much, she says he would have ordered an MRI instead of a CT scan. And that MRI, read properly, would have revealed her spinal cord inflammation and prompted Dr. Styer to initiate steroids at a time when the treatment could have halted her paralysis in its tracks. See R. 110 at 6–11.

The defendants have offered several defenses to Bentley's claims. For one, they argue that there is no evidence that steroids are effective in combatting the type of spinal cord inflammation from which Bentley was suffering.[3] So, the defendants say, Bentley cannot prove that PBH could have stopped the progression of her paralysis. See, e.g., R. 259 at 2.

[2] Bentley also sued Highlands and its radiologist, Dr. Terry Hall, but both recently settled. See R. 477; R. 484.

[3] Described most generally, Bentley's suffered from a bout of "transverse myelitis," a condition involving inflammation of the spinal cord that is "characterized by symptoms and signs of neurologic dysfunction in motor and sensory tracts on both sides of the spinal cord." See What is Transverse Myelitis?, Johns Hopkins Medicine, http://www.hopkinsmedicine.org/neurology_neurosurgery/centers_clinics/transverse_myelitis/about-tm/what-is-transverse-myelitis.html (last visited Dec. 20, 2016). Transverse myelitis has many possible causes, and in Bentley's case, doctors initially attributed the inflammation to Devic's disease, an autoimmune disorder. Now, though, the parties seem to agree that Bentley's condition was instead caused by a bout of strep throat a week earlier. As a result, Bentley's condition might be more precisely labeled "acute, post-infectious inflammatory myelopathy"—meaning a form of transverse myelitis involving short-term inflammation of the spinal cord brought on by a prior infection.

Second, PBH says that Bentley previously signed a release waiving her claims against the hospital in exchange for PBH canceling her medical bill. See, e.g., R. 257 at 1–2; R. 266 at 2.

Anticipating these defenses, Bentley has retained the services of three experts. Dr. Carlos Pardo-Villamizar and Dr. Robert DeLorenzo, both neurologists, are prepared to testify that PBH could have stopped (or even reversed) the progression of Bentley's paralysis if it had administered steroids when she still had motor control and sensation in her legs. See R. 389; R. 238-1. Dr. DeLorenzo and Dr. Christopher Betz, meanwhile, have each opined that Bentley was cognitively impaired when she signed PBH's release because she was on central nervous system ("CNS") depressants and an opioid painkiller at the time. See R. 442-3; R. 442-2.

PBH has now moved to exclude each of these opinions pursuant to Federal Rule of Evidence 702 and Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993). See R. 441; R. 443; R. 444. PBH argues that the doctors' opinions are unreliable because there is insufficient proof that either the steroids or Bentley's prescription medications would have had the effects the doctors now claim. As the party seeking to admit the doctors' opinions at trial, Bentley bears the burden of proving, by a preponderance of the evidence, that their testimony is admissible. See Nelson v. Tenn. Gas Pipeline Co., 243 F.3d 244, 251 (6th Cir. 2001).

II.

First, a housekeeping matter. Technically, no defendant remaining in this case has yet moved to exclude Dr. Pardo's steroid/causation testimony. Only Highlands challenged its admissibility initially. See R. 377. But Highlands recently reached a settlement with Bentley and notified the Court that it wished to withdraw its summary judgment and Daubert motions. See R. 477. Upon learning of this settlement, PBH, Dr. Styer, and Whitaker moved to join in Highlands's Daubert challenge to Dr. Pardo. R. 478. Bentley swiftly objected. R. 481.

Bentley is correct that the defendants' request comes rather late. The scheduling order in this case instructed the parties to notify the Court in advance of the Pre-Motion Post-Discovery Conference of any anticipated Daubert challenges. R. 107 at 4–5. And as Bentley notes, Highlands filed notice of its intent to challenge the causation testimony of both Dr. Pardo and Dr. DeLorenzo, see R. 275; R. 276, but PBH mentioned only the latter, see R. 259.

Nevertheless, the defendants may join in Highlands's motion. Dr. Pardo and Dr. DeLorenzo's causation opinions, and the grounds for excluding them, overlap each other. See R. 438 at 4–5. Bentley has had a full opportunity to brief the admissibility of both. See R. 447. And for its part, PBH has actively participated in the briefing and hearings concerning the doctors' shared theory that earlier steroid intervention would have minimized Bentley's paralysis. See, e.g., R. 374; R. 404; R. 422; R. 438; R. 443; R. 450. So Bentley has been on notice that the defendants (including PBH) were challenging the reliability of her experts' opinions. And with no risk of unfair surprise, economy and consistency suggest the better course is to address the admissibility both opinions now, rather than wait until trial for the defendants to object anew on Rule 702/Daubert grounds.

III.

The Federal Rules of Evidence take a liberal approach toward the admissibility of opinion testimony—including from expert witnesses. Glaser v. Thompson Med. Co., 32 F.3d 969, 971–72 (6th Cir. 1994). But that does not relieve the Court of the difficult task of drawing "the often-elusive line between admissible opinion and inadmissible speculation." Tamraz v. Lincoln Elec. Co., 620 F.3d 665, 667 (6th Cir. 2010). Not all "experts" are truly expert, and not all "expert opinions" are really worth the label. So when a party challenges another's expert witness, the Court assumes a gatekeeping role to ensure that the jury is not exposed to

"junk science" masquerading as expert testimony. See Daubert, 509 U.S. at 597; Best v. *Lowe's Home Ctrs., Inc.*, 563 F.3d 171, 176–77 (6th Cir. 2009). Only if an expert opinion is relevant, reliable, and rendered by a genuine expert will it be admissible at trial. See Fed. R. Evid. 702; In re Scrap Metal Antitrust Litig., 527 F.3d 517, 529 (6th Cir. 2008).

In this case, expertise is hardly an issue. In fact, the defendants concede that Drs. Pardo, DeLorenzo, and Betz are qualified to offer their respective opinions. See R. 377 at 8; 374 at 7. And it is easy to see why. If anyone is in a position to discuss the cause and treatment of Bentley's condition, it is Dr. Pardo, a renowned neurologist and the head of Johns Hopkins's Transverse Myelitis Center. See R. 388-3. Dr. Betz is a registered pharmacist and a doctor and professor of pharmacology. See R. 392-1. And remarkably, Dr. DeLorenzo has expertise spanning both fields: he has a medical degree as well as a neuropharmacology Ph.D., and he is a tenured professor of both neurology and pharmacology at Virginia Commonwealth University. See R. 383-1; R. 438 at 2. All three doctors, meanwhile, have extensive clinical experience treating spinal cord disorders (in the case of Drs. Pardo and DeLorenzo) and monitoring prescription drug interactions (in the case of Drs. DeLorenzo and Betz).

The doctors' opinions will also help the jury "understand the evidence or determine a fact in issue." Fed. R. Evid. 702(a). Dr. Pardo and Dr. DeLorenzo's testimony regarding the palliative effects of steroids on spinal cord inflammation is a key link in Bentley's claim that the defendants contributed to her paralysis. And Bentley will rely heavily on Dr. DeLorenzo and Dr. Betz when she argues that her medication significantly impaired her cognitive function at the time she signed PBH's release in February 2014. If true, the release likely has no legal effect and thus cannot bar Bentley's suit. The doctors' opinions are thus central to Bentley's affirmative and rebuttal cases. Not only that, but how these medications interact with the body

and combat or cause certain symptoms is a technical subject outside the ordinary understanding of a lay juror. So, a bit of expert testimony on the subject will do the jury some good.

That leaves reliability. To be reliable within the meaning of Rule 702, expert testimony must be grounded in "scientific knowledge." Daubert, 509 U.S. at 589–90. In other words, a witness may testify in the form of an expert opinion only if his testimony is "ground[ed] in the methods and procedures of science," and more than just "subjective belief or unsupported speculation." Id. at 590. But the subject of an expert's testimony does not have to be "'known' to a certainty"—"arguably, there are no certainties in science." Id. Instead, an expert may testify as to an "inference" he has drawn provided it is "derived by the scientific method"—i.e. "supported by appropriate validation" or "'good grounds,' based on what is known." Id.

The reliability inquiry is flexible one. See id. at 594–95. The Supreme Court has provided a list of questions to aid district courts when assessing the reliability of expert testimony under Rule 702, including whether the expert's theory has been tested, peer-reviewed, or generally accepted. See id. at 591–95; Glaser, 32 F.3d at 972. But that list is not exhaustive, nor any one factor dispositive. In re Scrap Metal, 527 F.3d at 528–29. District courts are permitted "considerable leeway in deciding in a particular case how to go about determining whether particular expert testimony is reliable." Kumho Tire Co. v. Carmichael, 526 U.S. 137, 152 (1999). The goal is simply to ensure that a witness's expert testimony has a "reliable basis in the knowledge and experience of [his] discipline." Jahn, 233 F.3d at 388; see also Rosen v. Ciba-Geigy Corp., 78 F.3d 316, 318 (7th Cir. 1996) ("[A] district judge asked to admit scientific evidence must determine whether the evidence is genuinely scientific, as distinct from being unscientific speculation offered by a genuine scientist.").

IV.

A.

Whether Drs. Pardo and DeLorenzo can reliably opine that steroids improve outcomes for patients like Bentley is a point of contention. Both sides (and their experts) agree that Bentley's inflammation came with a recommended treatment: the high-dose intravenous corticosteroids administered by Central Baptist. See R. 450 at 2; R. 404 at 3 (explaining that PBH does not dispute that "the administration of steroids [is] the standard of care for transverse myelitis patients"). But, PBH says, doctors administer steroids to transverse myelitis patients not in the knowledge that they will work, but in the hope that they might. R. 450 at 2; R. 451 at 2. According to PBH, steroids are, for lack of a better option, the "first treatment offered to hasten recovery, reduce disease activity, and restore neurological function." R. 450-3 at 4–5. But there is an "absence of evidence" that they actually work. Id. As a result, PBH says, Drs. Pardo and DeLorenzo can only offer the type of "useful but untested hypothesis" that the law "generally treat[s] as inadmissible speculation." *Tamraz*, 620 F.3d at 677.

Despite PBH's protests, Dr. Pardo and Dr. DeLorenzo's steroid opinions are not mere conjecture. Rather, both doctors' testimony represents an informed inference that each has drawn by applying three types of knowledge to Bentley's case. Consider each in turn.

First, the doctors begin with several general (and generally accepted) propositions. Inflammation is a byproduct of the immune system's attempt to combat harmful stimuli.[4] It is a sign, in other words, of the body's attempt to defend itself. Sometimes, though, the immune system takes aim at the wrong target, mistaking the body's own cells for foreign dangers and

[4] See generally What is Inflammation?, U.S. Nat'l Library of Medicine: PubMed Health, https://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0072482/ (last visited Dec. 20, 2016).

attacking healthy body tissue as a result. That is what happened in Bentley's case. The parties agree that Bentley was suffering from post-infectious inflammatory myelopathy. See R. 351-4 at 2; R. 447 at 5. A recent bout of strep throat had tricked Bentley's immune system into attacking nerve cells in her spinal cord. The resulting inflammation climbed up both sides of her lower spinal cord, destroying the myelin sheathing on her nerve cell fibers, and disrupting communication between her spinal cord and the remainder of her central nervous system.[5]

Doctors, meanwhile, have long turned to corticosteroids to treat a host of inflammatory conditions. Corticosteroids like methylprednisolone—which Central Baptist used to treat Bentley—mimic the effects of cortisol, a natural anti-inflammatory hormone produced by the adrenal glands.[6] These cortisol-like chemicals suppress the body's immune response on multiple fronts, inhibiting the production of pro-inflammatory chemicals and disrupting the operation of the body's white blood cells.[7] And it is these same properties that have led doctors to use corticosteroids to treat spinal cord inflammation in transverse myelitis patients.[8]

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[5] See generally Transverse Myelitis Fact Sheet, Nat'l Institute of Neurological Disorders and Stroke, https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Fact-Sheets/Transverse-Myelitis-Fact-Sheet (last visited Dec. 20, 2016) (explaining that, in transverse myelitis patients, "[a]ttacks of inflammation can damage or destroy myelin, the fatty insulating substance that covers nerve cell fibers" resulting in "nervous system scars that interrupt communications between the nerves in the spinal cord and the rest of the body").

[6] See, e.g., Steroids, The Johns Hopkins Lupus Center, https://www.hopkinslupus.org/lupus-treatment/lupus-medications/steroids/ (last visited Dec. 20, 2016).

[7] See, e.g., Peter J. Barnes, How Corticosteroids Control Inflammation: Quintiles Prize Lectures 2005, British J. Pharmacol., 148(3), 245-254 (June 2006); Livertox: Corticosteroids, U.S. Nat'l Library of Medicine, https://livertox.nih.gov/Corticosteroids (last visited Dec. 20, 2016); Corticosteroids, Cleveland Clinic, http://myclevelandclinic.org/health/articles/corticosteroids (last visited Dec. 20, 2016).

[8] See, e.g., Amer Awad & Olaf Stüve, Idiopathic Transverse Myelitis and Neuromyelitis Optica: Clinical Profiles, Pathophysiology and Therapeutic Choices, Current Neuropharmacology, 9(3), at 419 (Sept. 2011) ("The rationale of using steroids in [transverse myelitis] is based on its numerous effects on the immune system leading to a global immunosuppression. Some of these effects include but not limited to: inhibition of lymphocyte proliferation and differentiation, redistribution of lymphocytes, alteration of lymphokine function of especially tumor necrosis factor (TNF), IL-1 and IL-2, and inhibition of macrophage function, in particular antigen presentation and processing."); Transverse Myelitis Fact Sheet, supra ("Physicians often prescribe anti-inflammatory corticosteroid therapy soon after the diagnosis is made in order to decrease inflammation and hopefully improve the chances and speed of neurological

According to Dr. Pardo and Dr. DeLorenzo, the idea that steroids would benefit patients like Bentley is thus simply an application of a generally accepted treatment to a particular manifestation of the condition it treats. Steroids combat inflammation by suppressing the body's immune response. Inflammation is responsible for the loss of neurological function in patients with inflammatory myelopathies. Ergo, steroids are capable of counteracting the neurological decline in transverse myelitis patients. If administered, too, before the immune system has pushed nerve fibers beyond the point of no return, steroids can preserve what function remains and provide cover for the body to rebuild the damaged myelin.[9] See generally R. 434 at 7–10 (Dr. DeLorenzo); R. 440 at 16–19 (Dr. Pardo). A "causal chain" that the defendants even concede is plausible—and perhaps even right. R. 451 at 2.

Dr. Pardo and Dr. DeLorenzo find further support for this syllogism in a second source of knowledge: the evolving literature on inflammatory conditions of the central nervous system. That literature is far more supportive of Dr. Pardo and Dr. DeLorenzo's opinions than the defendants let on. See R. 447 at 11–14, 25–26. For instance, studies published in 2004 and 2006 suggested that "high-dose steroids improved time to ambulation and ultimate motor recovery" in transverse myelitis patients,[10] and that steroids can be "somewhat effective if

recovery. Although no clinical trials have investigated whether corticosteroids alter the course of transverse myelitis, these drugs often are prescribed to reduce immune system activity because of the suspected autoimmune mechanisms involved in the disorder.").

[9] See generally Monika Bradl & Hans Lassman, Oligodendrocytes: Biology and Pathology, Acta Neuropathol, vol. 119, at 46–47 (2010), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2799635/pdf/401_2009_Article_601.pdf (discussing remyelination or "the restoration of new myelin sheaths to demyelinated axons").

[10] Chitra Krishnan et al., Demyelinating Disorders: Update on Transverse Myelitis, 6 Current Neurology and Neuroscience Reports, no. 3, 236, 238 (2006).

given in the acute phase."[11] A recent peer-reviewed article concluded with a high degree of statistical confidence that earlier onboarding of steroids correlated with improved neurological outcomes among juvenile transverse myelitis patients in an Indian hospital.[12] And finally, several other studies have shown that the palliative effects of corticosteroids extend to other demyelinating conditions of the central nervous system: reducing inflammatory lesions in patients suffering from multiple sclerosis[13] and fighting exacerbations of neuromyelitis optica.[14] At the end of the day, then, several observational studies—of patients with transverse myelitis or other inflammatory disorders of the spinal cord—suggest that earlier steroid intervention does in fact improve neurological outcomes in patients with inflammatory conditions of the spinal cord.

Importantly, this pattern has also borne out in the doctors' practices. This brings us to the third pillar on which the doctors rest their opinions: clinical experience. See Messick v. Novartis Pharm. Corp., 747 F.3d 1193, 1198 (9th Cir. 2014) ("Medicine partakes of art as well as science, and there is nothing wrong with a doctor relying on extensive clinical experience[.]"). Between them, Drs. Pardo and DeLorenzo have treated, reviewed, or consulted on hundreds of cases of transverse myelitis, generally, and inflammatory myelopathy, specifically. See R. 440 at 14–15; R. 434 at 18. They report that those

[11] Chitra Krishnan et al., Transverse Myelitis: Pathogenesis, Diagnosis, and Treatment, 9 Frontiers in Bioscience, 1483, 1492 (2004).

[12] See Renu Suthar et al., Acute Transverse Myelitis in Childhood: A Single Centre Experience from North India, 20 J. European Pediatric Neurology Soc'y, no. 3, 352 (May 2016).

[13] See, e.g., Florian Then Bergh et al., Monthly Intravenous Methylprednisolone in Relapsing-Remitting Multiple Sclerosis—Reduction of Enhancing Lesions, T2 Lesion Volume and Plasma Prolactin Concentrations, BMC Neurology, 6:19 at 6 (May 2016).

[14] See, e.g., Kleiter, I. et al., Neuromyelitis Optica: Evaluation of 871 Attacks and 1,153 Treatment Courses, Annals of Neurology, 79:2, 206–17, at 207 (2016).

experiences have shown the power of steroids to combat neurological decline in patients suffering from acute inflammatory myelopathies. Specifically, they have discovered that patients treated with IV corticosteroids while they still have motor control and/or sensation overwhelmingly have fair or good outcomes. At the least, the steroids halt the loss of neurological function, and in many cases they reverse the deficits all together. And the faster the patient is treated the more function left to preserve. Because if a patient still has motor control or sensory function when doctors administer steroids, then the corresponding nerves have not yet been fully destroyed. See R. 434 at 10, 17, 26; R. 440 at 17–18, 33–38.[15]

With that knowledge in hand, Drs. Pardo and DeLorenzo examined Bentley and reviewed both her medical records and her deposition testimony. See R. 389 at 1; R. 238-1 at 1. They noted that Bentley was still able to walk with assistance when she left PBH on the morning of July 29. See R. 440 at 36; R. 356-2 at 15. In fact, it was not until Bentley visited her family doctor four hours later that she first lost control of an appendage (her left foot). See R. 440 at 49; R. 356-2 at 15–16. So Drs. Pardo and DeLorenzo surmised that Bentley still had substantial neurological function in her lower extremities when she left PBH—meaning steroids might have done her some good. Crucially, too, once Central Baptist administered steroids, the upward progression of Bentley's paralysis apparently stopped and her shortness of breath ceased.[16] See R. 440 at 94; R. 434 at 26–27; R. 356-3 at 11. So the doctors found

[15] The defendants make much of the idea that neither doctor can reliably determine when Bentley's inflammation has resulted in permanent cell death, which the defendants suggest marks the moment of no return for neurological function. See, e.g., R. 443 at 8–9. However, this criticism does not appear to undermine the reliability of the doctors' opinions. After all, the doctors' testimony appears to define the "therapeutic window" not based on cell death (something they cannot reliably determine) but based on outward signals of motor control and sensory perception. And either way, because Bentley still had both when she left PBH, presumably her nerve cells were still hanging on.

[16] The defendants argue that the doctors have misread Bentley's medical records, see, e.g., R. 443 at 7–8, but this type of factual dispute is best left for the jury. See, e.g., In re Scrap Metal, 527 F.3d at 530.

indications in the record that steroids actually did help Bentley—even if they came too late to save much of her neurological function. Finally, there was an explanation for this success in Bentley's lab work from July 30, 2013: her spinal fluid held a high concentration of lymphocytes, white blood cells particularly susceptible to steroid therapy. See R. 447 at 14.

Drs. Pardo and DeLorenzo have thus laid a foundation for each step they have taken from the general proposition with which they began (steroids combat inflammation) to the specific inference with which they end (steroids would have helped Bentley if given earlier). True, that steroids have anti-inflammatory properties is not, standing alone, enough to justify the doctors in concluding that steroids would have helped Bentley in this case. But Drs. Pardo and DeLorenzo offer more than that. They have explained both the physiological process by which steroids combat inflammation, and why patients who have sensation or motor control when steroids are administered are likely to at least retain that function. They have pointed to scientific literature that, though not definitive, supports the general principle that steroids improve neurological outcomes for inflammatory myelopathy patients. They thoroughly reviewed Bentley's medical records, assessing her symptomology to determine that she still had significant function in all extremities when she left PBH. And perhaps most importantly, they have found support that steroids actually worked in this case.

Rule 702 permits experts to draw conclusions "from a set of observations based on extensive and specialized experience." Kumho Tire, 526 U.S. at 156. The experts need only explain how they have reliably applied their experience to the facts at hand. See Thomas v. City of Chattanooga, 398 F.3d 426, 432 (6th Cir. 2005). Under the circumstances, Drs. Pardo and DeLorenzo have done at least that much, so their steroid opinions are sufficiently reliable to be admitted at trial. See, e.g., Jahn, 233 F.3d at 391 (holding doctors' methodology was

reliable where they drew conclusions based on clinical indicia in the patient's incomplete medical records); Heller v. Shaw Indus., Inc., 167 F.3d 146, 155 (3d Cir. 1999) ("[E]xperience with hundreds of patients, discussions with peers, attendance at conferences and seminars . . . are tools of the trade, and should suffice for the making of a differential diagnosis even in those cases in which peer-reviewed studies do not exist to confirm the diagnosis[.]").

B.

The defendants base much of their opposition to Dr. Pardo and Dr. Lorenzo on the argument that their steroid-intervention testimony is not supported by sufficient scientific literature. The American Academy of Neurology ("AAN"), PBH notes, concluded in 2011 that there "is insufficient evidence to determine the utility of corticosteroids in alleviating [transverse myelitis] attacks." R. 450-3 at 4. Indeed, the defendants point out, no study has conclusively established that early steroid treatments reduce neurological deficits in cases like Bentley's. See R. 443 at 5–7; R. 444 at 5–9. On this much, the defendants are right. No double-blind or epidemiological study has firmly proven that steroids are effective in treating acute inflammatory myelopathy. And the type of retrospective studies that might fill the gap are only now underway—including some under Dr. Pardo's supervision. See R. 440 at 88.

Nevertheless, the defendants' protests are overblown. For starters, a medical expert does not need to cite published studies for his opinion to be reliable within the meaning of Rule 702. See, e.g., Dickerson v. Cardiac & Thoracic Surgery of E. Tenn., P.C., 388 F.3d 976, 980 (6th Cir. 2004). Submission of a medical theory "to the scrutiny of the scientific community" is no doubt "a component of 'good science.'" Daubert, 509 U.S. at 593. But some propositions are simply "too particular, too new, or of too limited interest to be published." Id. So experts may sometimes testify that some act caused a patient's medical

harm—even if no published literature has yet reached that same conclusion. See, e.g., Best, 563 F.3d at 180–81. And the same is true when the causation is flipped and an expert wants to testify that some act might have prevented a patient's medical harm.

Dr. Pardo and Dr. DeLorenzo, of course, have cited literature that supports their testimony. As discussed, there have been an array of small (and peer-reviewed) studies that suggest both that steroids improve neurological outcomes in patients with spinal cord inflammation and that faster treatment is correlated with better medical outcomes. Even the AAN acknowledges as much—referring to this simply as "Class IV" evidence, i.e. evidence other than from controlled trials, including "consensus or expert opinion." See R. 443-2 at 4 ("Only Class IV evidence exists concerning the utility of steroids.").[17] What's more, Drs. Pardo and DeLorenzo have explained why those early studies were so tentative in their conclusions: Until recently, researchers failed to differentiate between various forms of transverse myelitis, only some of which involve inflammation and so are treatable by steroids. See, e.g., R. 440 at 13; R. 434 at 6. Sure, at the end of the day none of these studies definitively answer the steroid-intervention question on which the two doctors will opine. But Rule 702 "does not require anything approaching absolute certainty" in an expert's opinion. Tamraz, 620 F.3d at 671. And given that the doctors were neither required to cite studies nor reach their conclusions with absolute certainty, it would seem more than a bit incongruous to fault them for drawing support from studies that "suggest" their clinical observations are correct.

The defendants also argue that Dr. Pardo and Dr. DeLorenzo cannot extrapolate from these various studies. For example, because they involved different neurological disorders (for

[17] The AAN defines "Class IV" evidence in an appendix to its evidence based guidelines, which is available at http://www.neurology.org/content/77/24/2128/suppl/DC1.

example, multiple sclerosis or neuromyelitis optica) or a different class of patients (children). See R. 443 at 5–6; R. 444 at 6–8. But Drs. Pardo and DeLorenzo have explained that the inflammatory process is the same across these cases: The disorder provokes the immune system into attacking nerve cells throughout the spinal cord, causing inflammation that damages nerve tissue and short circuits neural signals. Steroids counteract that inflammatory process in several ways, including by inhibiting the production and operation of certain white blood cells.[18] See R. 434 at 6–9, 13–14; R. 440 at 17–18. And introduced early enough, steroids can accomplish that task before the immune system pushes those nerve cells beyond the point of repair. Under the circumstances, Dr. Pardo and Dr. DeLorenzo may reliably extrapolate from these studies to Bentley's analogous condition, especially when the studies merely confirm what their clinical practice has already borne out. See, e.g., Kennedy v. Collagen Corp., 161 F.3d 1226, 1230 (9th Cir. 1998); R. 409-3 at 7 ("UpToDate Article") ("Even without placebo-controlled trials evaluating glucocorticoids specifically in TM, there is good evidence that intravenous glucocorticoids are effective in acute inflammatory central nervous system diseases like TM, such as multiple sclerosis.").

Nor do the defendants' remaining arguments undermine the reliability of the doctors' opinions. Dr. DeLorenzo did not, as PBH off-handedly suggests, form his opinion solely for purposes of this case, see R. 443 at 10—he has been treating transverse myelitis patients for years. Nor is this a case where the extant scientific literature contradicts the experts' testimony. Contra Tamraz, 620 F.3d at 670–71; Conde v. Velsicol Chem. Corp., 24 F.3d 809, 813–14 (6th

[18] See Awad & Stüve, supra, at 419 ("The rationale of using steroids in TM is based on its numerous effects on the immune system leading to a global immunosuppression. Some of these effects include but are not limited to: inhibition of lymphocyte proliferation and differentiation, redistribution, of lymphocytes, alteration of lymphokine function of especially tumor necrosis factor (TNF), IL-1 and IL-2, and inhibition of macrophage function, in particular antigen presentation and processing.").

Cir. 1994). More importantly, though, while other courts have occasionally rejected generic medical testimony that "earlier treatment is better," those cases share flaws not present in this one: Drs. Pardo and DeLorenzo have reliably explained the mechanism by which steroids work, why earlier intervention is crucial in inflammatory myelopathies, and why they believe Bentley could have benefitted from it. Contra McDowell v. Brown, 392 F.3d 1283, 1299–1301 (11th Cir. 2004); Jones v. Pramstaller, 874 F. Supp. 2d 713, 724–25 (W.D. Mich. 2012); Tomlinson v. Collins, No. 2:09-cv-0125, 2010 WL 4317030 (S.D. Ohio Oct. 25, 2010).

C.

None of this is to say that Dr. Pardo and Dr. DeLorenzo are correct. Perhaps the jury will view their opinions skeptically. But whether their conclusions are accurate is not for the Court to decide. See Daubert, 509 U.S. at 594–95. At this stage, the question is simply whether their testimony is "the product of reliable principles and methods" applied reliably to the facts of this case. Fed. R. Evid. 702(c)-(d). In other words, do the doctors have "good grounds" for concluding that it was more likely than not that PBH could have minimized Bentley's paralysis by starting her on IV corticosteroids earlier on July 29? See Pride v. BIC Corp., 218 F.3d 566, 577 (6th Cir. 2000) (quoting Daubert, 509 U.S. at 590).

The answer to those questions is yes. The doctors began with a generally accepted principle: Steroids combat inflammation and reduce tissue damage by suppressing the body's immune response. From there they journey to a specific conclusion: Steroids could have minimized the inflammatory damage to Bentley's spinal cord. But the doctors do not simply leap from A to Z. They connect their specific inference to the general premise through their clinical experience, their knowledge of the physiological processes of inflammation, and analogies to related disorders (and the studies about them). And perhaps most important of

all, the doctors point out that their theory finds support in the facts of this very case: Bentley's symptoms stopped progressing after Central Baptist administered IV steroids. Under the circumstances, Drs. Pardo and DeLorenzo have exhibited the degree of intellectual rigor expected of someone in their field. See, e.g., Kennedy, 161 F.3d at 1229–30. And any remaining concerns regarding their methodology or conclusions can be handled through vigorous cross examination. See Daubert, 509 U.S. at 596; Best, 563 F.3d at 180.

V.

Along with pharmacologist Dr. Christopher Betz, Dr. DeLorenzo has also opined that Bentley lacked the mental capacity necessary to make independent business or legal decisions when she signed PBH's liability release in February 2014. See R. 442-2; 442-3. Drs. Betz and DeLorenzo attribute Bentley's impaired state to the "synergistic effects" of three medications that she began taking after a February 11 surgery. R. 442 at 9 (discussing the doctors' reports). According to the doctors, two of those medications were CNS depressants (gabapentin and lamotrigine), which inhibit the transmission of neural signals and have sedative effects. See id. at 9–11. A third (hydrocodone) was an opioid that blocks pain receptors in the brain and can cause "clouding of consciousness, drowsiness, mental deficiency, sedation, and stupor." See id. at 11. Drs. Betz and DeLorenzo have testified that, in their clinical experience, these medications can cause "mental fogginess and fatigue" when used in combination. See id. at 12 (citing R. 432 at 19–20); see also R. 434 at 111–12. And between Bentley's dosage and her testimony that her memory of this period is significantly impaired, the doctors conclude that Bentley's three-drug regimen must in fact have impaired her cognitive function.

As support, Drs. Betz and DeLorenzo cite entries in two databases used by pharmacists. The Lexi-Drugs database entries for gabapentin and lamotrigine, they note, both warn that

these drugs may enhance the CNS-depressant effect of hydrocodone. See R. 442-6 at 9; R. 442-8 at 13. And a Lexicomp Interaction Analysis for hydrocodone and CNS depressants recommends that doctors start patients "with a 20% to 30% lower hydrocodone dose when using [it] with any other depressant." R. 442-11 at 2. The reason? "This warning is due to the potential for additive or synergistic CNS depression, leading to an increased risk of adverse effects such as respiratory depression, hypotension, excessive sedation, and coma." Id.

Surveying the whole, half of the doctors' testimony appears admissible: Dr. Betz and Dr. DeLorenzo can reliably testify regarding the possible side effects of these medications. Both have extensive experience in pharmacology—Dr. DeLorenzo has forty-one years as a neuropharmacologist under his belt, see R. 442-3 at 1, and Dr. Betz has spent years managing and monitoring medication regimens in a clinical setting, see R. 432 at 4–5. Both doctors have also grounded their account of the side effects in their experience, in an assessment of Bentley's medical records, and in industry warnings regarding these medications. See R. 442-2; R. 442-3. From this foundation, the doctors can reliably say that these medications, individually or in tandem, can sometimes impair cognitive function. See, e.g., Best, 563 F.3d at 180–81; Jahn, 233 F.3d at 391–92; Dickerson, 388 F.3d at 980–81.

Go beyond this opinion, though, and the ice grows too thin to support the doctors' testimony. Dr. Betz and Dr. DeLorenzo also want to testify that the medications had such an effect on Bentley. The doctors' journey to this conclusion takes them down the following path: (1) In their experience, and according to warnings accompanying these medications, these drugs can impair cognitive function. (2) Bentley reports impaired memory of the period when she was taking them. (3) Therefore the drugs must have impaired her cognitive function. Standing alone, though, this reasoning is more logical fallacy than scientific knowledge.

Simply put, Drs. Betz and DeLorenzo have not reliably connected the various links in their chain of inferences. The doctors admit that not all patients experience the side effects they describe—indeed, only a minority appear to. See, e.g., R. 432 at 42; see also R. 442-6 at 7 (indicating that immediate release formulations of gabapentin may cause dizziness in 17-28% of patients and drowsiness in 19-21% of patients); R. 442-8 at 11 (reporting that fewer than 10% of lamotrigine patients experience drowsiness and fatigue). Nor can Dr. Betz or Dr. DeLorenzo predict reliably which patients will fall in that minority. What's more, Drs. Betz and DeLorenzo cannot say when or how often these drugs have synergistic effects. The best they offer is the Lexicomp notation that these drugs "may" have synergistic effects, see, e.g., R. 442-8 at 13, since they are not aware of any study of the drugs' interaction, see R. 432 at 45; R. 434 at 112–14. But even these warnings, the doctors admit, are just drug companies' attempts to insulate themselves from liability through disclosures made in an abundance of caution. See R. 441-1 at 10–11; R. 441-2 at 2; cf. *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1248–50 (11th Cir. 2005) (rejecting an expert's reliance on FDA recommendations because public health guidelines took a protective "cost-benefit approach" to disclosure that is less demanding than that required of an expert wishing to testify that a supplement actually caused medical harm). And finally, even assuming the medications caused Bentley fatigue or drowsiness, Dr. Betz and Dr. DeLorenzo do not explain how they know that these effects would have been so severe as to deprive Bentley of the mental capacity necessary to exercise independent and informed judgment. They just say they must have been. But then again, the "'ipse dixit of the expert' alone is not sufficient" to make his opinion reliable. Tamraz, 620 F.3d at 671 (quoting Gen. Elec. Co. v. Joiner, 522 U.S. 136, 146 (1997)).

In the end, Drs. Betz and DeLorenzo cannot connect their general observation to the specifics of this case. Instead, they end where they started: with the idea that the Bentley's prescription drug regimen must be responsible for the loss of memory she reported in her deposition. See, e.g., R. 441-1 at 2–3. But this type of post hoc ergo propter hoc reasoning is insufficient to render the second (case-specific) half of their opinion reliable under Rule 702. See, e.g., Rolen v. Hansen Beverage Co., 193 F. App'x 468, 473 (6th Cir. 2006). The Court will therefore grant PBH's motion insofar as it seeks to exclude this particular component of Dr. Betz and Dr. DeLorenzo's pharmacological opinions.

VI.

Why the divergent treatment of the two sets of opinions considered today? After all, neither is the paradigmatic example of reliability, backed by double-blind, peer-reviewed clinical studies affirming the specific conclusions that the experts now draw. And absent such lodestars, both are guided largely by the doctors' clinical experience and "suggestive" secondary literature. So one might ask whether the Court can reliably draw a line between the two. The answer, though, is yes, because these similarities are largely superficial.

Dr. Pardo and Dr. DeLorenzo have advanced a model for predicting when patients are capable of neurological recovery: If a patient still has motor control or sensation, then their nerve fibers have not yet been irreparably damaged. They have seen this model play out in clinical practice, and they applied it to the evolution of Bentley's symptoms, noting that she did not lose motor control of an appendage until hours after she left PBH. They also explain the mechanics of it all, drawing on the anti-inflammatory properties of steroids and a range of studies discussing the effectiveness of steroids in combating both transverse myelitis in children and a range of other inflammatory neurological conditions. And lastly, the doctors

circle back to the fact that doctors appeared to have documented that steroids actually stopped the progression of Bentley's symptoms once they were administered at Central Baptist. Under the circumstances, Bentley has proven by a preponderance that the doctors' steroid-intervention conclusions are reliably based in the methods and principles of their discipline—they are not, in other words, pure conjecture or "junk science." Dickerson, 388 F.3d at 982.

Dr. Betz and Dr. DeLorenzo's capacity opinions, by contrast, rely not on a drug's primary purpose, but its side effects. Side effects only a small minority of patients suffer, and ones no doctor actually observed in this case. The best these experts can say with confidence is that Bentley's medication might cause symptoms such as fatigue and drowsiness. Their clinical experience and the drug database reports bear that much out, so they can testify reliably to this point. But Drs. Betz and DeLorenzo cannot say why or when certain patients fall into the small minority who actually suffer these side effects. Or when these drugs will have "synergistic" effects, let alone effects substantial enough to deprive someone of independent judgment. And most importantly, they cannot reliably count Bentley among the apparent minority of side-effect sufferers with no clinical indications other than her own self-serving testimony. Dr. Betz and Dr. DeLorenzo therefore cannot reliably opine that Bentley's medications actually caused her to lack the mental capacity necessary to sign PBH's release.

Accordingly, it is **ORDERED** as follows:

(1) PBH, Dr. Styer, and Whitaker's motion to join in Highlands's Daubert challenge to Dr. Pardo's causation opinion, R. 478, is **GRANTED.**

(2) PBH, Dr. Styer, and Whitaker's motion to exclude Dr. Pardo's causation opinion, R. 444, is **DENIED**.

(3) PBH's motion to exclude Dr. DeLorenzo's causation opinion, R. 443, is **DENIED**.

(4) PBH's motion to exclude Dr. DeLorenzo and Dr. Betz's capacity opinions, R. 441, is **GRANTED IN PART** and **DENIED IN PART**. The doctors may testify regarding the side effects of the three prescriptions medications as a general matter, but they may not testify that Bentley's medication regimen actually rendered her unfit to sign the release in February 2014.

This the 27th day of December, 2016.

Signed By:
Amul R. Thapar AT
United States District Judge