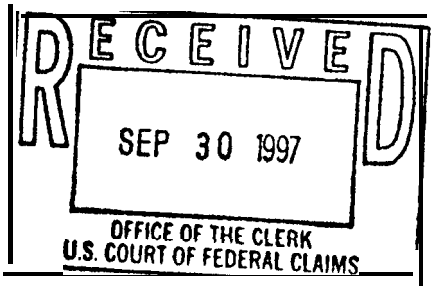
In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

(No. 90-3379V)

(Filed September 30, 1997)



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ADRIAN McCASKILL TYSON,

Petitioner,

v.

SECRETARY OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES,

Respondent.

* *

PUBLISHED

Peter F. Burns, Mobile, Alabama, for petitioner.

Kate Adam Coleman, United States Department of Justice, Washington, D.C., for respondent.

DECISION

WRIGHT, Special Master.

On October 1, 1990, petitioner, Adrian Tyson, filed a claim under the National Vaccine Injury Compensation Program (hereinafter "Vaccine Act" or the "Act").[1] Petitioner claims that as the direct result of the administration of a tetanus toxoid vaccination

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[1] The National Vaccine Injury Compensation Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, 42 U.S.C.A. § 300aa-1 et seq. (West 1991 and Supp. 1997), as amended **by Title II of the Health Information, Health Promotion and** Vaccine Injury Compensation Amendments of November 26, 1991 (105 Stat. 1102). References shall be to the relevant subsection of 42 U.S.C.A. § 300aa.

administered on January 19, 1988, he suffered Guillian-Barre Syndrome ("GBS").[2]

I.

PROCEDURAL BACKGROUND

On January 30, 1995, respondent filed a report in this matter recommending compensation be denied based on the absence of evidence to support petitioner's claim. An evidentiary hearing was held in this matter in Washington, D.C., on October 22, 1996, during which petitioner testified and presented the testimony of Dr. Richard deShazo, a clinical immunologist. Testifying for respondent was Dr. Barry Arnason, a neurologist. On December 9, 1996, the parties filed post-hearing briefs. After considering the entire record, and for the reasons discussed below, I find petitioner is not entitled to compensation.

II.

FACTUAL BACKGROUND

The following evidence is contained in the record in this matter:[3]

On Tuesday, January 19, 1988, in the late afternoon, petitioner, then 27 years old, accidentally lacerated his left palm. He was treated that evening, at about 5:00, at the office of Dr. Ben Freeman in Mobile, Alabama. Dr. Freeman cleaned the wound,

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[2] Dr. Barry Arnason, a medical expert herein, described GBS as follows: "a subacutely evolving inflammatory disease of the peripheral nerves ... [i]n which there is damage to the insulation that surrounds the nerves known as myelin[,][a]nd if the process is severe[,] to the nerve fibers or axons themselves." Tr. at 75. According to Dr. Arnason, the disease is characterized by loss of sensation and weakness to such a severe degree that paralysis may occur. Twenty to 25% of patients must be placed on a respirator. Ninety-five percent of patients gradually recover with 80% making a complete recovery. Tr. at 76.

[3] The evidence in the record consists primarily of exhibits submitted as part of the petition filed in this case ("P. Ex. "), respondent's exhibits filed in this matter ("R. Ex. "), plus evidence taken at the evidentiary hearing in this matter ("Tr. at ").

applied a steri-strip, prescribed Keflex, an antibiotic, and administered a tetanus toxoid booster shot. P. Ex. 4c at 330; Tr. at 12, 20.

Adrian testified that he was an athlete who worked out with weights regularly and ran an average of at least three miles a day. Tr. at 11. On Wednesday, the day after the shot, Adrian was feeling okay and ran about one mile.[4] Tr. at 16-17. Adrian testified that, when he awoke on Thursday morning, he could not take a deep breath and could not seem to get all the air he needed. He felt as though he had the flu. Tr. at 13, 17. He also testified his feet and toes felt like they were asleep but he was not concerned about that at the time because he was worried about his breathing difficulty. Tr. at 17. Adrian tried taking over-the-counter antihistamines to relieve his breathing problem. Tr. at 23.

Adrian did not run on Thursday or Friday because he was not feeling well. Tr. at 13. By Friday night, Adrian's breathing problems became more severe. He testified that early Saturday morning, at about 3:00 or 4:00 a.m., he awoke barely able to breathe. Tr. at 17-18. At that time, he also noticed that his legs and feet were tingling and numb and felt like they were cramping. He also had a severe headache. Tr. at 19, 22, 24. That day, he went to a football game with his friends and stayed for the entire game, although he felt fatigued when he stood up at the end of the game. Tr. at 26. He testified that he tried to run the next morning, Sunday. He stretched and began to run but, at about the end of the street, he collapsed and had to lay by the side of the road for about an hour before he could get up and make it back to the house. Tr. at 13, 14, 24. That night he went to his parents' house and told them he was not well. Tr. at 19.

The next day, Monday, January 25th, Adrian went to his family doctor, Dr. C. William Bodie, who noted complaints of numbness in Adrian's feet and hands and "parasthesias anterior ch[e]st." Dr. Bodie, who thought Adrian might be having an allergic reaction to the Keflex, discontinued the antibiotic and prescribed Prednisone. P. Ex. 4b at 321; P. Ex. 4a at 52; Stipulation of Facts filed Nov. 3, 1995. He instructed Adrian to return the next day at which time Adrian was referred to a neurologist, Dr. Chalhub. Tr. at 15; P. Ex. 4a at 52-55. Adrian was admitted to the Mobile Infirmary Medical Center ("Mobile Infirmary") on January 26, 1988, with an

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[4] Adrian testified that he only ran a mile that day because he had football practice on Wednesdays. Tr. at 20.

admitting diagnosis of "a progressive acute polyneuropathy probably consistent with Guillian-Barre Syndrome." P. Ex. 4a at 36, 38.

During his 21-day hospital stay, Adrian's weakness increased dramatically. He experienced headaches and muscle tenderness throughout his hospital course. He underwent plasmapheresis treatments beginning on. February 3rd.[5] When he was discharged on February 15th, Adrian's condition was improved and his prognosis was excellent, P. Ex. 4a at 52-53.

Following his discharge, Adrian's condition was monitored by Dr. Fritz A. LaCour, Dr. Chalhub's neurology associate. As of March 1, 1988, petitioner continued to manifest significant neurologic residua, including diminished facial function, a limp on the left side, headaches and jaw tenderness. He had total facial plegia and was unable to close his eyes. He had no reflexes and was still using a walker. P. Ex. 4d at 367; Stipulation of Facts at ¶7.

Adrian's condition improved. By June 13, 1988, he was noted by his doctor to be regaining his strength and was beginning to do a little work with weights. P. Ex. 4c at 331. A record of the Gulf Coast Therapy Services, dated November 20, 1989, notes "[p]ast medical history is significant for Guillian-Barre in February, 1988 which patient denies any residual effect." P. Ex. 4c at 351. On May 22, 1990, Dr. LaCour wrote the following in a letter to Vocational Rehabilitation Service:

> The patient has made a dramatic recovery from his Guillian-Barre Syndrome. His main difficulty now is episodes of nocturnal shortness of breath and an occasional tingling in his legs. He is a full-time student at the University of Alabama now living independently.
>
> His examination today is completely within normal limits except for minimal decreased pinprick and vibration sense distally. There are no reflexes in his upper extremities. His reflexes in his lower extremities are normal. His gait is perfectly normal. He has gained weight, appears the picture of health, and is well tanned. His chest is perfectly clear. His pulse is 80 and regular. He has difficulty with easy fatigability.

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[5] Plasmapheresis is "the removal of plasma from withdrawn blood, with retransfusion of the formed elements into the donor." Dorland's Illustrated Medical Dictionary at 1304 (27th ed. 1988).

He has no need for ongoing out-patient physical therapy but could benefit from vocational rehabilitation.

P. Ex. 4e at 372.

In a letter to petitioner's attorney, dated September 29, 1994, Dr. LaCour reported that since Adrian's discharge from the hospital on February 15, 1988, he has had spasms of the jaw, some numbness of the feet which has since resolved and recurrent headaches. He has had episodes at night where he awakens feeling as though his lungs are burning. He has also had some episodes of depression. Dr. LaCour concluded "[h]e appears to be the picture of health .... It is not my finding that he has significant clinical residua of his Guillain-Barre [sic]." P. Ex. 4e at 411; Stipulation of Facts at ¶13.

Expert testimonv.

Dr. deShazo

Petitioner presented the testimony of Dr. Richard deShazo, a board-certified clinical immunologist.[6] Dr. deShazo believes, to a reasonable degree of medical certainty, that Adrian's GBS was caused by the tetanus toxoid injection he received on January 19, 1988. Tr. at 31. He bases that opinion on the following.

First, according to Dr. deShazo, over 50% of GBS cases have an obvious or known trigger. Tr. at 33. He relies on the absence of any other obvious triggers for GBS in Adrian's case such as a viral or bacterial infection. Tr. at 32-33. He testified there was no clinical evidence that Adrian had any kind of infection at the time of his initial hospitalization, therefore, no serological testing was done. Tr. at 49.

Next, according to Dr. deShazo, the timing of onset of diagnosable symptoms of GBS fits within the appropriate time frame described in the medical literature. He distinguished between the "onset" of symptoms of GBS and the "diagnosability" of GBS, explaining that it is very difficult "in reading the literature to differentiate so far as timing is concerned the difference between" the two. Tr. at 36. Dr. deShazo appeared to be making the

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[6] Dr. deShazo is board-certified in four specialities. He is a professor of Medicine and Pediatrics at the University of South Alabama College of Medicine where he also serves as the chairman of the Department of Internal Medicine and director of the division of Allergy and Immunology. P Ex. 10d; Tr. at 28.

argument that the critical date is the time of diagnosis of GBS, not the time of the onset of symptoms. He believes that Adrian experienced the onset of GBS symptoms within two to three days of vaccination.[7] Tr. at 35-36, 57. However, Dr. deShazo does not believe Adrian's symptoms were "diagnosable" until about four or five days after vaccination and Adrian was not actually diagnosed until seven days after vaccination.* Tr. at 35-36, 58. In addition, Dr. deShazo reasoned that Adrian is a trained athlete who was a candidate for a professional football team and is very much in touch with the way he feels. With someone who has such sensitivity to his body as Adrian, Dr. deShazo believes it is reasonable that he would notice symptoms sooner than another who is not as physically fit. Tr. at 55-56. "I think he was so attuned to his level of fitness that he picked up his symptoms probably a lot earlier than most individuals ...." Tr. at 56.

Dr. deShazo also believes that the 13 tetanus immunizations Adrian received throughout his lifetime made it more probable that he was hyper-immunized to the vaccine and was predisposed to a rapid response such as he experienced.[9] Tr. at 42,44. In other words, this hyperimmunity, Dr. deShazo explained, contributed to the short onset period. Tr. at 35.

Finally, according to Dr. deShazo, those patients who experience a good recovery tend to be the ones who experience a rapid and early onset of symptoms with the full manifestation of the clinical disease occurring very quickly over a period of weeks. This theory, according to Dr. deShazo, is supported in the literature and is the case regardless of the trigger. Tr. at 32, 43,45-46 (citing P. Ex. C6; P. Ex. Cl at 918).

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[7] While most people with GBS experience the onset in their extremities or peripherally, it is possible, according to Dr. deShazo, to have the first symptoms appear in the respiratory muscles, as did Adrian. Tr. at 57.

[8] Dr. deShazo explained that he, himself, had GBS at one time from a swine flu vaccine. Tr. at 28. In his own particular case, he had symptoms within 24 hours of the vaccination but did not have neurological findings until two weeks later. Tr. at 36.

[9] Dr. deShazo used Penicillin anaphylaxis as an example of how a hypersensitivity reaction may occur. He reasoned that the more times one receives Penicillin, the more likely it is one will have a hypersensitivity reaction to it. Tr. at 42.

Dr. Barry Arnason

Dr. Arnason, a board-certified neurologist, testified on behalf of respondent.[10] He has treated and participated in the care of 200 to 300 GBS patients and, in his travels, has seen an additional 100 patients and reviewed the records of another 25. Tr. at 74. Dr. Arnason does not believe that Adrian's GBS was caused by the tetanus toxoid vaccination in question.

To begin, Dr. Arnason agrees with Dr. deShazo that Adrian's respiratory complaints comprised the first clinical manifestation of his GBS. Tr. at 79. It is Dr. Arnason's opinion that the timing of onset of GBS in Adrian's case makes it impossible to indict the tetanus vaccine as the trigger. Tr. at 98. Dr. Arnason is adamant that the earliest onset of GBS after a triggering event would be five days" while most cases of GBS occur beyond a week after the triggering event.[12] Tr. at 79-81, 82.

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[10] Dr. Arnason is a professor and chairman of the Department of Neurology at the University of Chicago. R. Ex. B. He has written 30 articles related to GBS. Tr. at 74.

[11] Dr. Arnason did admit he is familiar with one case reported in the literature in which the onset of seizures occurred within three days. In that case, the patient had a campylobacter infection. In the case of infection, however, Dr. Arnason testified, the bacteria is in the system for 24 to 48 hours before there is any clinical evidence of infection. Tr. at 79-80.

[12] The reason that onset is highly unlikely before five days, according to Dr. Arnason, is that it takes the body time to respond:

> [I]f one injects a vaccine, Tetanus Toxoid for example, contains protein. That ... protein has to make [its] way from site of injection to the lymph nodes where lymphocytes are. And that occurs either through the lymphatics or it's carried by cells from the site of infection to the lymph nodes. That takes time. When the protein gets to the lymph nodes, be presented to the lymphocytes, which then have to go through cycles of proliferation. And they go through several such cycles. And each of those cycles takes at least 12 and usually up to 24 hours. And that takes time. And the lymphocytes have to leave the lymph node and travel through the circulation. And there has to be at the same time an activation or a release of proteins that activate the

Dr. Arnason testified that, even assuming Adrian had the onset of GBS between five days and six weeks after a tetanus vaccine, a temporal relationship alone is not enough to establish that the vaccine caused the GBS. Tr. at 86. His opinion is that "once in a blue moon" GBS may result from a single tetanus toxoid vaccination. Tr. at 93-94. However, he suggested that the occurrence of multiple episodes following multiple vaccinations would be compelling and would lead him to accept a causal relationship in that particular case. Tr. at 88, 97.

III.

DISCUSSION

Causation in Vaccine Act cases can be established in one of two ways: either through the statutorily prescribed presumption of causation, or by proving causation-in-fact. Petitioner must prove one or the other in order to recover under the Act.[13] The Vaccine Injury Table lists certain injuries and conditions which, if found to occur within a prescribed time period, create a rebuttable presumption that the vaccine caused the injury or condition.[14] The presumption may be overcome by an affirmative showing that the

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> cells on the blood vessel walls so that they become sticky so that the lymphocytes can attach to them and get into the nerve. And that takes time. And then when the lymphocytes get into the nerve and that takes time. ... The lymphocytes appear in the nerve two days before there are any symptoms. And they had to get there before that. So that the entire process takes longer than two or three days.

Tr. at 81-82.

[13] Petitioners must prove their case by a preponderance of the evidence, which requires that the trier of fact "believe that the existence of a fact is more probable than its nonexistence before [the special master] may find in favor of the party who has the burden to persuade the [special master] of the fact's existence." *In re Winship, 397* U.S. 358, 372-73 (1970) (Harlan, J., concurring) *quoting* F. James, Civil Procedure 250-51 (1965). Mere conjecture or speculation will not establish a probability. *Snowbank Enter. v. United States, 6* Cl.Ct. 476, 486 (Cl. Ct. 1984).

[14] Section 14(a).

injury was caused by a factor unrelated to the administration of the vaccine.[15]

In order to demonstrate entitlement to compensation in an off-Table case, petitioner must affirmatively demonstrate by a preponderance of the evidence that the vaccination in question more likely than not caused the injury alleged. §§ 11(c)(1)(C)(ii)(I) and (II); *Grant v. Secretary of HHS, 956* F.2d 1144 (Fed. Cir. 1992); *Strother v. Secretary of* HHS, 21 Cl. Ct. 365, 369-70 (1990), *aff'd, 950* F.2d 731 (Fed. Cir. 1991). The Federal Circuit in *Grant* summarized the legal criteria required to prove causation-in-fact under the Vaccine Act. The court held that a petitioner must

> show a medical theory causally connecting the vaccination and the injury. Causation in fact requires proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury. A reputable medical or scientific explanation must support this logical sequence of cause and effect.

Grant, 956 F.2d at 1148 (citations omitted); see *also Strother, 21* Cl. Ct. at 370.

Petitioner does not meet this affirmative obligation by merely showing a temporal association between the vaccination and the injury. Rather, petitioner must explain *how* and *why* the injury occurred. *Strother, 21* Cl. Ct. at 370; see *also Hasler* v. United *States,* 718 F.2d 202, 205 (6th Cir. 1993), *cert. denied, 469* U.S. 817 (1984) (inoculation is not the cause of every event that occurs within a ten day period following it). If petitioner views the temporal relationship as "key," the claim must fail. *Thibaudeau v. Secretary of HHS,* 24 Cl. Ct. 400, 403 (1991). Nor may petitioner meet his burden by eliminating other potential causes of the injury. *Grant, 956* F.2d at 1149.

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[15] Section 13(a)(l)(B). Other prerequisites to compensation include: (1) that the injured person suffered the residual effects of a vaccine-related injury for more than six months after the administration of the vaccine. Section 11(c)(1)(D)(i); (2) that the petitioner incurred in excess of $1,000 in unreimbursable vaccine-related expenses. Section 11(c)(1)(D)(i); (3) that the vaccine was administered in the United States. Section 11(c)(1)(B)(i)(I); (4) that the petitioner did not previously collect a judgment or settlement in a prior civil action. Section 11(c)(c)(1)(E); and (5) that the action be brought by the injured person's legal representative. Section 11(b)(1)(A).

"[E]vidence in the form of scientific studies or expert medical testimony is necessary to demonstrate causation" for a petitioner seeking to prove causation in fact. H.R. Rep. No. 990908, 99th Cong. 2d Sess., pt. 1 at 15 (Sept. 26, 1986), *reprinted in* 1986 U.S. Code Cong. and Admin. News 8344, 8356. In this regard, the recent Supreme Court decision in *Daubert v. Merrell Dow Pharmaceuticals, Inc., 113 S.* Ct. *2786* (1993), is instructive. While that case dealt with the admissibility of scientific evidence and here we are assessing the scientific validity of evidence already presented, *Daubert* is helpful in providing a framework for evaluating the reliability of scientific evidence.[16] The Court in *Daubert* wrote:

> [I]n order to qualify as "scientific knowledge," an inference or assertion must be derived by the scientific method. Proposed testimony must be supported by appropriate validation -- *i.e.,* "good grounds," based on what is known. In short, the requirement that an expert's testimony pertain to "scientific knowledge" establishes a standard of evidentiary reliability.

Id. at 2795. The Court goes on to suggest a key criterion of scientific reliability is whether a theory has been tested and subjected to peer review and publication. *Id.* at 2796-97. While acknowledging that publication is not a *sine qua non* of admissibility, the Court finds the submission of a novel scientific theory to the scrutiny of publication is a component of "good science" and the fact of publication is a relevant, though not dispositive, consideration. *Id.* at 2797. Finally, the Court noted while not a precondition, the general acceptance of a theory within the scientific community of a scientific theory can have a bearing on the question of assessing reliability while a theory that has attracted only "minimal support" may be viewed with skepticism. *Id.*

Inasmuch as GBS is not an injury listed in the Vaccine Table, petitioner's claim that the tetanus toxoid vaccine caused GBS is one of causation-in-fact. The analysis in this case is two-fold: (1) *can* tetanus toxoid cause GBS? and (2) *did* tetanus toxoid cause GBS in this case? See Guy v. *Secretary of HHS, No.* 92-779V, 1995 WL 103348 (Fed. Cl. Spec. Mstr. Feb. 21, 1995) (two-step causation-

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[16] In *Daubert,* the Supreme Court held Federal Rule of Evidence 702 is binding on federal courts with respect to establishing the admissibility of scientific evidence. *Daubert, 113 S.* Ct. at 1795. It is noted that the Federal Rules of Evidence are not binding on this tribunal.

in-fact analysis used); *Alberding v. Secretary of HHS, No.* 90-3177V, *1994* WL 110736 (Fed. Cl. Spec. Mstr. March 18, 1994) (two-step causation-in-fact analysis used).

Significant'support exists for the notion that tetanus toxoid vaccine *can* cause GBS. The Institute of Medicine ("IOM") favors the existence of such a causal relationship. Adverse Events Associated with Childhood Vaccines, Evidence Bearing on Causality at 89 (National Academy Press 1994); J.D. Pollard and G. Selby, *Relapsing Neuropathy due to Tetanus Toxoid,* 37 JOURNAL OF NEUROLOGICAL SCIENCES 113 (1978); Norris Newton, Jr. and Abdorassol Janati, *Guillain-Barré Syndrome after Vaccination with Purified Tetanus Toxoid,* SOUTHERN MEDICAL JOURNAL 1053 (August 1987). In addition, there have been several cases under the Vaccine Program in which petitioners were successful in proving they had GBS caused by a Program-covered vaccination. See Guy v. *Secretary of HHS, No.* 92-779V (Fed. Cl. Spec. Mstr. Feb. 21, 1995); *Alberding v. Secretary of HHS, No.* 90-3177V (Fed. Cl. Spec. Mstr. March 18, 1994); *Robinson v. Secretary of HHS, No.* 91-01V (Fed. Cl. Spec. Mstr. Nov. 27, 1991). *See also, Housand v. Secretary of HHS, No.* 94-441V, 1996 WL 2822882 (Fed. Cl. Spec. Mstr. May 13, 1996) (special master accepted that Td can cause GBS, although petitioner not ultimately successful); *Coultas v. Secretary of HHS, No.* 93-0081V 1995 WL 605559 (Fed. Cl. Spec. Mstr. Sept. 29, 1995) (causal relationship accepted, although petitioner ultimately unsuccessful). Dr. Arnason conceded that "once in a blue moon" tetanus toxoid can cause GBS and was especially willing to recognize a relationship in the case of GBS following multiple tetanus toxoid vaccinations. I find there is sufficient support to recognize the existence of a causal relationship between tetanus toxoid and GBS.

As to the second question, however, petitioner has a more difficult task. Petitioner relies, essentially, on the temporal relationship between the tetanus toxoid and the onset of GBS and the absence of any other possible trigger for Adrian's GBS to show that tetanus vaccine caused GBS in his particular case. Respondent refutes that claim, arguing that the timing between vaccination and onset of GBS makes it impossible to implicate the tetanus toxoid vaccine.

The first step of the analysis, then, is to determine when was the onset of Adrian's GBS. The question is not easily resolved because there are inconsistencies between petitioner's account of the sequence of events following vaccination and the histories recorded in the contemporaneous medical records, as well as inconsistencies between the medical records themselves.

When questioned specifically about the timing of the onset of symptoms, Adrian was not altogether clear. He testified that he first noticed breathing difficulties on Thursday morning upon waking, although the breathing problems really became a concern when he awoke very early Saturday morning unable to get a breath. He also seemed to indicate that on Thursday his feet and toes felt as though they were asleep but he paid little attention to that until he awoke early Saturday with breathing difficulties. The following excerpt of testimony demonstrates Adrian's confusion regarding the timing of the onset of his symptoms.

> THE COURT: Okay. And Thursday is when you started feeling bad?
>
> MR. TYSON: Yes, Ma'am.
>
> THE COURT: Can you tell me exactly what your symptoms were on Thursday?
>
> MR. TYSON: I had problems breathing. It's like I couldn't take a deep breath. I was just struggling to get air, you know. It even woke me up at night the night before because I couldn't breathe. I just could not open my lungs to get all the air I needed it seemed.
>
> THE COURT: Did you have any other symptoms?
>
> MR. TYSON: My feet and toes. I don't know it was almost like my foot was asleep but I didn't pay it any attention at the time. Just the breathing part I was worried about . . . .
>
> THE COURT: And that was on Friday that your feet and toes felt numb?
>
> MR. TYSON: It started more on Saturday. But that Friday night, yes, that's when I had the first breathing problem really ....
>
> THE COURT: That was Friday night when you went to sleep?
>
> MR. TYSON: Yes. It woke me up in the morning, like Saturday morning early.
>
> THE COURT: Okay. So then Thursday -- okay. I'm a little confused about onset here. Just before you testified I thought that you started having the breathing

problems on Thursday_ And now you're saying that that was Friday night.

MR. TYSON: No. I had problems on Thursday but it wasn't as severe as it was on Friday. Thursday it was just like I said I thought I had the flu. ...

THE COURT: Now you testified earlier that Saturday was the first. day that your legs started tingling. But now you're saying it was Friday night that your feet and toes were numb? I'm just trying to pin down --

MR. TYSON: It was like early Saturday morning, you know. I woke up about 3:00 a.m. or 4:00 a.m. in the morning.

THE COURT: And that's the first time that you felt any tingling?

MR. TYSON: Yes. That's when my breathing really started bothering me.

THE COURT: Okay. And then when did your legs start to tingle.

MR. TYSON: All that next morning, that weekend.

THE COURT: Saturday and Sunday?

MR. TYSON: Yes.

Tr. at 17-19.

On cross-examination, petitioner was questioned further about the onset of his breathing difficulties:

Q But was it Wednesday night that you woke up in the middle of the night and started feeling like you couldn't breathe?

A It was more dramatic on that Friday. ... Thursday morning is when I really had problems breathing and I thought I had the flu or something.

Q Okay. Did you wake up on Wednesday night though having trouble breathing?

A No, that was on Friday night that I really had an awakening.

Tr. at 21-22.

The contemporaneous medical records do not entirely corroborate Adrian's version of the sequence of events. The records do not make note of breathing difficulties within the few days following vaccination. A consultation record of the Mobile Infirmary, dated January 26, 1988, relates the following history:

> After a day or two of therapy [(treatment for the laceration)] he began to notice a numbness in his lower extremities associated with some degree of weakness. The numbness also involved his fingertips. He attempted to exercise and stretch out his legs but felt that he could just not go as per his usual routine. He experienced some difficulty in breathing last night, felt that he could not get a deep enough breath to satisfy his respiratory need. These feelings have since passed. When he awakened today he noticed that he felt numb all the way up to his neck although he notes that he could still feel in these areas.

P. Ex. 4a at 54.

Another record, dated January 26, 1988, reports, since his tetanus toxoid shot last week, "noted numbness & weakness of legs over next few days." P. Ex. 4a at 39. The discharge summary of February 15, 1988, reports that "24 hours after receiving a tetanus injection began developing numbness and parasthesis of his feet and arms. This progressed until the patient was seen on 1/26/88 by Dr. Chalhub." P. Ex. 4a at 52. GBS was immediately suspected upon Adrian's admission. P. Ex. 4a at 38, 55, 222.

For the most part, I find petitioner's account of his breathing problems to be reliable. That is, petitioner convinced me that the breathing difficulties he experienced got his attention and concern before any other symptoms. Although petitioner's testimony seemed confused regarding when the numbness and tingling in his extremities first occurred, that is not important as both medical experts agreed that the respiratory problems represented the onset of Adrian's GBS. While the medical records are silent regarding the timing of the onset of respiratory difficulties, petitioner appeared confident and unequivocal that he first experienced them on Thursday morning upon awakening. I find petitioner's first GBS symptoms, then, occurred Thursday morning,

January 21, 1988, less than 48 hours after receiving his tetanus toxoid vaccination.

The timing of onset is a critical issue in this case. As an initial matter, if petitioner is unable to demonstrate that GBS can be caused by a tetanus toxoid vaccine administered less than 48 hours prior to the onset of GBS, petitioner's case will be significantly weakened. Petitioner's and respondent's respective medical experts ardently dispute this point.

Dr. deShazo referred to several medical articles to support his' assertion that tetanus toxoid can trigger the onset of GBS within the time frame alleged by petitioner. Tr. at 37. In one study cited by Dr. deShazo, a review of 19 cases of neurologic complication of tetanus toxoid, namely, polyneuropathy, reports that the onset of symptoms after vaccination occurred within the range of nine hours to 14 days. S. Lane Rutledge and Carl Snead III, *Neurological Complications of Immunizations,* 109 THE JOURNAL OF PEDIATRICS 917, 919 (1986); P. Ex. Cl; Tr. at 37, 123. Dr. deShazo conceded, however, that in that report there are no cases reported specifically with a diagnosis of GBS. Rather, there is a mixture of various forms of peripheral neuropathy, "probably some of which were Guillian-Barre but it's not clear." Dr. deShazo referred to a review of 14 cases of tetanus toxoid induced peripheral neuropathies in which onset ranged from nine hours to 14 days. However, Dr. deShazo conceded "this is not classical [GBS] but [GBS] is in this family of diseases." Leon Reinstein and Jeffrey M. Pargament, *Peripheral Neuropathy after Multiple Tetanus Toxoid Injections, 63* ARCH. PHYS. MED. REHABIL. 332, 334 (1982); P. Ex. *C3;* Tr. at 41. Next, Dr. deShazo relies upon the IOM Report which relates a case where an 11-year-old girl developed spastic paraparesis, bilateral papillitis and visual defects three days after receiving a tetanus toxoid booster. Adverse Events Associated with Childhood Vaccines, Evidence Bearing on Causality at 84 (National Academy Press 1994); P. Ex. 10c; Tr. at 38-39. Dr. deShazo also cited to a case report of a 36-year-old woman who developed polyneuropathy, myelopathy and encephalopathy five days after receiving a tetanus toxoid vaccination. Another report relied upon by Dr. deShazo documents a case in which a 23-year-old male developed a peripheral neuropathy five hours after receiving a tetanus toxoid injection. Dr. deShazo conceded that in that report as well the peripheral neuropathy was not GBS, although it was an acute neuropathy with acute onset. George I. Blumstein and Harold Kreithen, *Peripheral Neuropathy Following Tetanus Toxoid Administration,* 198 JAMA 166 (1966); P. Ex. C5; Tr. at 40. Finally, **Dr. deShazo relies upon an article that refers to the onset period for polyneuritis following tetanus vaccination in 10** cases as **ranging from** 3.1 to 10.3 days. Ute Quast, W. Hennessen

and R.M. Widmark, *Mono- and Polyneuritis after Tetanus Vaccination, 43* INTERNATIONAL SYMPOSIUM ON IMMUNIZATION *25 (1979);* P. Ex. *C6;* Tr. at 41.

Dr. Arnason remarked upon the literature relied upon by Dr. deShazo. The principal and most compelling point he made about those articles is the absence of specific references to *GBS.* Rather, the literature referred to disease categories that may or may not include GBS, or to diseases altogether different from GBS.

First, Dr. Arnason testified the patients in the first study referred to by Dr. deShazo did not necessarily have GBS. He explained that polyneuropathy is a "very global term" and sometimes it relates to GBS and sometimes it does not. Tr. at 101. With regard to the second article referred to by Dr. deShazo, Dr. Arnason testified that the term "induced peripheral neuropathies" also does not necessarily indicate GBS but could be a lumping of "various things together." Tr. at 104. Dr. Arnason pointed out that the IOM had that article available to them when they made their determinations regarding timing of onset. He noted "I would think they [the IOM] must have rejected such cases since they set the interval [between vaccination and onset] at five days." Tr. at 105. In the case of the three-year-old girl who developed spastic paraparesis, bilateral papillitis and visual defects three days after a tetanus toxoid vaccination, referred to on page 84 of the IOM, Dr. Arnason testified spastic paraparesis is not a polyneuritis, rather, it refers to a problem of the spinal cord and not the nerves, and bilateral papillitis involves the optic nerve which is part of the central nervous system and not the peripheral nervous system. Tr. at 106. Finally, the article that refers to a patient with brachial neuritis is irrelevant, according to Dr. Arnason; although brachial neuritis is a neurological reaction, it is a condition totally different from GBS. Tr. at 112-13.

Dr. deShazo recognizes that a peripheral neuropathy is not necessarily GBS but he argues that, regardless of whether the articles have lumped together GBS, polyneuropathy and mononeuropathy, his opinion does not change. "These are all neurological responses to tetanus toxoid which result in clinical manifestations which in one patient show up one way and in another another." Tr. at 119-120.

Dr. deShazo also relied on the relationship between rapid onset of GBS and the completeness of recovery. He testified that, according to the literature, Adrian's complete recovery is directly related to the rapidity of his onset of GBS after the vaccination. In the article, *Neurologic Complications of Immunizations,* the author reported with regard to polyneuropathy following tetanus

toxoid immunization "Recovery is usually complete, although the degree of recovery correlates with the, interval between injection and the onset of symptoms. Eight of 10 patients with onset before 14 days after injection recovered completely, but all three patients with onset after 14 days from injection had only partial recovery." Rutledge and Snead, *supra*, at 15; P. Ex. C1 at 918. Also, the authors of the article titled *Mono-and Polyneuritis after Tetanus Vaccination* report "The difference of incubation time in relation to the evolution of the disease is not statistically significant for cases involving only one nerve but it is significant in polyneuritis .... In the reported cases of neuropathy, the interval between vaccination and the initial symptoms was therefore longer in cases of delayed recovery." Quast et al., *supra,* at 15; P. Ex. C6 at 27.

Dr. Arnason disagrees with Dr. deShazo that the earlier the onset of GBS the better the recovery and insists that Dr. deShazo has it backwards. Tr. at 82. Rather, Dr. Arnason testified, the general rule, in terms of GBS, is "the shorter the incubation period the more severe the disease." Tr. at 83, 98, 114. He qualified that statement by adding that cases of GBS following campylobacter infection have a shorter incubation period than those following viral infections. Tr. at 83. Dr. Arnason explained that "it is possible that the interpretation that the earlier the onset the more severe the disease could be colored by the fact that certain infections are likely to cause more severe [GBS] than others." Tr. at 114. In a book chapter authored by Dr. Arnason, titled "Acute Inflammatory Demyelinating Polyradiculoneuropathies," Dr. Arnason stated, "Those cases of rabies vaccination-induced neuropathy with the shortest incubation period tend to be the most severe; those with incubation periods of four weeks or longer are often mild .... It is not known whether a short 'incubation' period is associated with a particularly severe form of this disease, but it may be." Peripheral Neurooathy at 2056 (Vol. II 1984); R. Ex. K.

It is clear from the medical literature and. the medical testimony that the *typical* range for onset of GBS symptoms following the triggering event is five days to six weeks. Even Dr. deShazo acknowledged this to be true. Tr. at 65; IOM Report, *supra,* p. 15 at *85;* Barry G.W. Arnason and Betty Soliven, *Acute Infalammatory Demyleinating Polyradiculoneuropathy,* PERIPHERAL NEUROPATHY VOLUME III at 1437, 1439 (1992) ("The interval between the prodromal infection and the onset of AIDP [GBS] symptoms varies; most frequently it is 1 to 3 weeks; occasionally it is as long as 6 weeks."); R Ex. L. Dr. deShazo testified that the usual timing is fourteen days which he described as "the magic number." He explained that Adrian's course is "definitely not the usual

course." Tr. at 62. Dr. deShazo was unable to cite to any literature that reports an onset of. 48 hours or less between tetanus toxoid and *GBS, specifically.* Rather, the references he cited to refer generally to polyneuropathies or other neurologic processes which may include GBS.

Numerous times throughout his testimony, Dr. deShazo recognized Dr. Arnason as a leading expert on GBS and testified he would defer to him regarding certain points. I, too, must credit Dr. Arnason as a renowned expert on the topic of GBS, with more expertise in that area than Dr. deShazo. Dr. Arnason was adamant that the onset of GBS may not occur within 48 hours of the triggering event. I find that based on Dr. Arnason's testimony and the medical literature, there is not enough evidence to find that it may.[17]

Establishing the second prong of the causation-in-fact analysis, *i.e.,* did the vaccine in question cause the injury in this particular case, almost always proves to be a difficult and formidable task for petitioners in Program cases. While I find tetanus toxoid *can* cause GBS, in this particular case, because the symptoms occurred less than 48 hours following vaccination, the evidence does not preponderate in favor of a finding that it did here. Unfortunately, in this case, petitioner has not demonstrated that GBS can be caused by a tetanus toxoid vaccine administered less than 48 hours prior to the onset of GBS. The literature simply does not support such a finding, and, further, I found compelling Dr. Arnason's testimony that such a short onset period would point to other causes.

Petitioner's only other support for a causal relationship in this case is the absence of other possible triggers for GBS. It is

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[17] I cannot credit Dr. deShazo's argument that the literature differentiates, as far as timing is concerned, between symptom onset and the diagnosability of GBS. Dr. Arnason testified that when a disease is diagnosable has nothing to do with when it began. Tr. at 95-96. He believes it is more common than not that the symptoms of GBS antedate the diagnosis of GBS by several days. Tr. at 96. In fact, I cannot find where in the literature a distinction is made between when GBS is diagnosable or diagnosed and the onset of symptoms. See, e.g., P. Ex. 10b at 45 ("[T]he first symptoms of GBS is mainly between 7 and 21 days."). Nor did Dr. deShazo satisfactorily point to any specific literature on that point. I believe, as Dr. Arnason testified, the critical reference point is the onset of symptoms, not the timing of the eventual diagnosis.

true that there was no documentation in the contemporaneous histories of a viral or bacterial infection. However, that one piece of evidence, alone, is insufficient to establish that the vaccine was in fact the culprit here. The absence of any other concrete evidence of a causal connection in this particular case, coupled with the absence of a general acceptance within the scientific community supporting the plausibility of a two-day onset of symptoms of GBS following a tetanus vaccination, compels me to find that petitioner has failed to demonstrate actual causation, and, therefore, does not qualify for a Program award.

IV.

FINDINGS OF FACT

1. Petitioner has not previously collected an award or settlement of a civil action in connection with any alleged injury sustained by petitioner due to the administration of the tetanus toxoid vaccine in question. Section 11(c)(1)(E); Stipulation of Fact at ¶15.

2. Petitioner was administered a vaccine listed in the Vaccine Injury Table, namely, a tetanus toxoid vaccination. Section 11(c)(1)(B)(i)(I); Stipulation of Fact at ¶2.

3. Said vaccine was administered in Mobile, Alabama. Section 11(c)(1)(B)(i)(I); P. Ex. 1 at 1.

4. There is not a preponderance of the evidence that the tetanus toxoid vaccination in question in fact caused petitioner's GBS.

5. There is not a preponderance of the evidence that petitioner expended in excess of $1,000 in unreimbursable medical expenses as a result of a vaccine-related injury.[18]

[18] Since I conclude that no vaccine-related injury occurred, I cannot conclude that any expenses incurred on petitioner's behalf were vaccine-related.

V.

CONCLUSION

Based on the foregoing, the undersigned finds, after considering the entire record in this case, that petitioner is not entitled to compensation in this case. In the absence of a motion for review filed pursuant to RCFC Appendix J, the clerk of the court is directed to enter judgment in accordance herewith.

IT IS SO ORDERED.

Elizabeth E. Wright

Elizabeth E. Wright
Special Master