THE SUPREME COURT OF APPEAL OF SOUTH AFRICA

JUDGMENT

Reportable

Case no: 331/2021

In the matter between:

M F **APPELLANT**

and

THE ROAD ACCIDENT FUND **RESPONDENT**

Neutral citation: *M F v Road Accident Fund* (Case no 331/2021) [2022] ZASCA 107 (15 July 2022)

Coram: ZONDI, CARELSE and MABINDLA-BOQWANA JJA and MEYER and PHATSHOANE AJJA

Heard: 20 May 2022

Delivered: 15 July 2022

Summary: Delict – personal injury claim – factual causation – cause and effect – whether soft tissue injury of neck and back sustained in motor vehicle accident caused involuntary muscle movement disorder – expert witness' reliance on medical literature – application of criteria from literature employed as a tool to establish a link between involuntary muscle

movement and trauma – logical and reasonable explanation by expert witness required.

ORDER

On appeal from: Gauteng Division of the High Court, Pretoria (Collis J, sitting as a court of first instance):

The appeal is dismissed with costs.

JUDGMENT

Mabindla-Boqwana JA (Zondi and Carelse JJA and Meyer and Phatshoane AJJA concurring)

Introduction

[1] The appellant, Mr M F[…], instituted a claim against the respondent, the Road Accident Fund, for the payment of compensation for damages resulting from bodily injury caused by a motor vehicle accident in which he was involved on 8 April 2007 (the 2007 accident). It is common cause that he sustained a soft tissue injury of the neck, commonly known as whiplash injury, in the 2007 accident. It is also common cause that the appellant suffers from an involuntary movement disorder, dystonia. The core issue between the parties is whether the dystonia was caused by the peripheral trauma to the appellant's neck, the whiplash injury. The Gauteng Division of the High Court, Pretoria (the high court) determined that question of factual causation first and separately from the other questions in the action. It found

that a causal link between the 2007 accident and the movement disorder had not been established, and granted the appellant leave to appeal to this Court.

Background facts

[2] The appellant was 34 years old at the time of the accident and practiced as an advocate until he was rendered incapacitated by continuous episodes of dystonia. Dystonia results from an abnormality or damage in the regions of the brain that control movement. This abnormality causes muscles in the affected parts of the body to move uncontrollably or involuntarily. The onset of the appellant's dystonia occurred approximately 10 months after the 2007 accident.

[3] Prior to the 2007 accident, the appellant had been involved in two other motor vehicle accidents. These occurred in 2003 and 2004/2005 respectively. He sustained whiplash neck injuries in those accidents as well. In the 2003 accident he also suffered a mid-back injury. The pain caused by those earlier whiplash injuries, however, resolved within a few weeks of each accident and he resumed his normal daily work without any difficulty. It is the 2007 accident which, it is alleged, presented dramatic changes to the appellant's life, which progressively led to his permanent incapacitation.

[4] The 2007 accident occurred while the vehicle which the appellant drove was stationary at a traffic light and another vehicle collided into its rear. The appellant alighted from the vehicle and exchanged information with the driver of the other vehicle. After about 30 minutes he started feeling nauseous and drove himself to the Eugene Marais Hospital (Eugene Marais). He also felt some pain in his neck, as a result of which x-rays were taken. He

was, however, not admitted but merely given medication. The following day he felt pain in his back (ie his shoulder blades in the mid-thoracic area) and went for physiotherapy. He also consulted his brother, an orthopaedic surgeon, who examined him. Because his back and neck pain got worse, his brother gave him two to three infiltrations. Each infiltration brought him pain relief for approximately half a day, after which the pain recurred. He returned to work after a few days but found that he was unable to work for more than two hours, as standing up activated the pain.

[5] He later went on holiday but spent most of the time in bed, as he could not move due to the neck and back pain. He stopped working for a while, during which time he attended physiotherapy sessions, which did not bring him much pain relief. His neck became intensely stiff and tight, which also worsened the thoracic pain. Stress also exacerbated his pain and discomfort. He, therefore, decided to stop the physiotherapy. He, however, resumed the physiotherapy sessions when he went back to work during the last three to four months of 2007, since the pain persisted. His brother referred him for x-rays and an MRI scan, from which he noted some ‘neck disc bulges’ which were not severe enough to justify surgery. His brother also gave him standard pain medication, which did not help him much.

[6] In January 2008, while he was sitting up and dictating notes, his left shoulder and left arm pulled up and he could not get them straight. His elbow became flexed. The problem lasted for about three hours. His brother gave him another infiltration in his back and prescribed further medication. Two days later, a similar incident occurred and his brother changed his

medication. According to Dr Johannnes Smuts, a neurologist who gave expert evidence on behalf of the appellant, this picture was suggestive of torticollis (a spasmodic contraction of the muscles of the neck) and the appellant's abnormal posture was very different from the muscle spasms that he had developed up to that point.

[7] A major attack happened on 17 February 2008,[1] while the appellant was sitting in a vehicle. He could not move, his body seemed not to be stiff or tight this time but everything felt 'loose', so that he could not move his arms or legs and he could not talk. He was effectively unable to control his voluntary movements and his father and wife had to carry him into the house. This episode took one and a half hours before he could voluntarily move again. Thereafter, he experienced residual stiffness for one and a half days, with a feeling of nausea and haziness. He had to be driven to work by other advocates as he was too stiff to drive.

[8] Yet another episode occurred while at a coffee shop with a colleague. It was so severe that the appellant fell over the table and collapsed onto his colleague. He had to be carried to his office and was taken to Eugene Marais where he was admitted for four to five days and referred for an MRI scan. Thereafter, he was referred to Little Company of Mary Hospital to consult a neurologist, Dr Duim. The MRI scan and angiogram which were done showed no abnormality. He had to be sedated in order to receive physiotherapy treatment. He also consulted a neurosurgeon, Dr du Plessis,

[1] The date is erroneously recorded as 17 January 2008 in Dr Smuts's first report. The 17 February 2008 date accords with the medical history as recorded by the respondent's expert witness, Dr Miller, with which the experts agreed, and with the narration by the appellant as put to Dr Smuts in evidence. The background facts are drawn from the joint minute prepared by the experts, the expert reports as well as the testimonies they gave in court.

who advised him that the pins and needles which he apparently experienced at that time and some of the spasms could be related to his neck injury but that there were pains and spasms which could not be explained by that injury. A decision was then taken to have him admitted to the DBC Training Centre in Centurion, for rehabilitation.

[9] During the period of his admission, the appellant became very ill. He could not open his eyes or talk but simply made groaning and moaning sounds. He remained fully conscious while all his muscles were tight with a constant pattern of movement changes when the neck and head would flex to the right and the right side of his body flexing in relation to the trunk and arm. During these attacks his speech slurred and his mouth pulled to one side.

[10] A lumbar puncture was performed to exclude multiple sclerosis. He was also placed on antipsychotic medication by a psychiatrist. Rehabilitation worsened the situation as with all exercises he would develop more spasms, attacks or contortions in the paravertebral part of his body. To continue with his exercises or rehabilitation, he was given more infiltrations into his back. He decided to stop the psychiatric medication, since he believed that that medication worsened his condition.

[11] The episodes would be triggered by someone touching him, bright light, loud sounds and vibrations. Similar attacks were also witnessed by the expert witnesses while examining him. All the doctors excluded epilepsy as a condition from which he suffered. Although he kept his practice open for a

year, he was not actually working due to the profusion of the episodes. His colleagues took over all of his work until 2009 when his practice could no longer continue. Since then he stayed at home looking after the birds that he bred and he involved himself in art collection, which has been his hobby. He could not play with his children; that also triggered his episodes of involuntary movements. He was referred to Professor C M van der Meyden, a neurologist, who diagnosed him as suffering from dystonia. Professor van der Meyden referred him to Dr Smuts, a neurologist and movement specialist at Wilgers Hospital. The appellant developed depression and at some stages experienced suicidal ideations.

Expert Evidence

[12] The appellant did not testify at the trial but called Dr Smuts as an expert witness. The respondent called Dr Percy Miller, a neurosurgeon, and Dr Donald Birrell, an orthopaedic surgeon, as its expert witnesses.

[13] Drs Smuts and Miller prepared a joint minute containing points of agreement and dispute. The experts agreed on many issues, including the fact that the appellant suffered from dystonia. The main difference between them related to the issue of factual causation. According to Dr Smuts, the appellant's clinical picture (on which the experts agreed) had a direct temporal relation to the 2007 accident and the neck injury, whilst Dr Miller was of the opinion that it did not. The experts agreed that the medical history could be drawn from Dr Miller's report which was more detailed and very precise in terms of specific time intervals.[2] It is important to deal with their evidence in some detail.

[2] Dr Smuts confirmed this in his evidence.

[14] I deal first with Dr Smuts's evidence. Dr Smuts prepared three reports dated 16 February 2009 (first report), 18 February 2016 (second report) and 28 January 2020 (third report) respectively. In relation to dystonia, he stated the following in his first report:

'The clinical picture of torticollis, blepharospasm and oromandibular movements can in combination only be described as cranio cervical dystonia.

There are difficult issues related to the dystonia; although dystonia has been described in association with head injury all cases were in severe head injury most often when there was haemorrhage in the basal ganglia. In this instance there has been no head injury of any significance. The second dilemma is that dystonia by its very nature is a sustained muscle contraction. In the case of this patient the dystonia comes in attacks. There are a number of dystonia syndromes that are episodic in nature [and] are well-known but very rare. I could not find any reference of any of these syndromes that can be related to injury of [the] brain or neck.

A second possible explanation is that he developed a movement disorder due to exposure to medication used for treatment of his cervical muscle spasms; this is known as tardive dyskinesia. In my experience tardive dyskinesia is usually also a sustained movement which can vary considerabl[y] depending on several factors.

A final possibility namely a psychogenic movement disorder should always be considered. In this regard he was consulted by 2 psychiatrists and in the case of Dr Steenkamp he explicitly stated to me that his opinion is that the movement disorder is of an organic rather than a psychogenic origin.

Video material of these attacks was also shown to a group of neurologists with a special interest in movement disorders and the opinion was no different from what has been stated above the opinion was divided between a very atypical dystonia or a psychogenic condition. With no objective tests remaining the opinion of the psychiatrist and the stereotypical pattern of the attacks is therefore considered dystonic in nature.'

Dr Smuts accordingly reached the following conclusion:

‘Based on the afore mentioned information the opinion is that the movement disorder is a form of tardive dyskinesia which resulted from medication used to treat the cervical problem that resulted from the accident.’

[15] In his second report, Dr Smuts referred to statements by two neurosurgeons, Drs du Plessis and Marus, whom the appellant had consulted. Dr Marus made the following remark:

‘Trauma has been associated with movement disorders. These usually relate to significant brain injuries associated with damage of the basal ganglia. It is clear that no head injury occurred and therefore it would not be a cause for his movement disorder. . .

The role of peripheral injury in the development of these form of movement disorders remains uncertain. . .

Injury of all sorts may result in development of abnormal movements that are secondary to psychological factors. In many situations it is extremely difficult to separate these abnormal movements from unusual or organic dyskinesias.’

[16] In relation to dystonia, Dr Smuts concluded that:

‘Post-traumatic dystonia as a diagnostic entity remains a subject of debate. In patients with cervical dystonia there is often significant illness or injury prior to the onset of their cervical dystonia.

This patient however presents with attacks or episodes of dystonia rather than a persistent movement. This type of presentation is seen in a condition known as paroxysmal kinesigenic dystonia. While dystonia occurring after trauma is well documented, paroxysmal exercise-induced dystonia occurring after trauma has only been described in one documented case I could find described. This is therefore a very rare presentation, but not impossible. An alternative possibility would be that this is a psychogenic disorder. Functional or psychogenic movement disorders are common and disabling, and often difficult to diagnose.

Given the long duration and persistent nature of the disorder in this patient it is my opinion that this is a post-traumatic dystonic disease that rendered the patient severely

impaired. Paying so much attention to the semantics regarding the type and precise cause of this condition serves little purpose and it is far more important to consider this a permanent condition. Since this is time related to the accident and trauma, this must be considered the precipitating cause of his disability.'

[17] In his third report, Dr Smuts simply described what paroxysmal movement disorders are and mentioned that they were rare. He further made an observation that '[d]ue to the rarity of these disorders, it is senseless to try and dig up literature references about the possible link of trauma to this particular case'. He concluded as follows:

'My opinion is that this happened in direct temporal relation to the accident, it persisted over many years, basically unaltered and this has had disastrous effects on this man and his career.

Proof of a link or not to the accident, is in my opinion more than the stated facts, it will remain pure speculation.'

[18] In evidence, Dr Smuts testified that his initial impression was that the appellant most likely suffered from tardive dyskinesia,[3] which was the only condition, in his clinical experience, that presented like the appellant's. What changed from the three possible diagnoses, as detailed in his three reports, which I have quoted above, was additional reading that he had done and new additional documentation that became available in the literature. This new information classified the criteria for the post-traumatic dystonia. This, he testified, was not available in 2009 (which is when he prepared his first report), but became available only in 2011 and the later publication in 2014,

[3] 'Tardive dyskinesia is a neurological disorder characterized by involuntary uncontrollable movements especially of the mouth, tongue, trunk, and limbs and occurring especially as a side effect of prolonged use of antipsychotic drugs (such as phenothiazine).' See Merriam-Webster online, available at *https://www.merriam-webster.com/dictionary/tardive%20dyskinesia*, accessed on 14 July 2022. It is described in the joint minute of the experts as a condition caused by exposure to a multitude of medications.

by an author known as Dr Joseph Jankovic, a very prominent person within the movement disorder societies in the world.

[19] The criteria used for diagnosis, as per the literature by Dr Jankovic (the Jankovic criteria) consisted of three requirements. Firstly, there must be trauma that is significant enough to warrant treatment within the period of at least two weeks; secondly, the dystonia must develop within one year from the period of trauma; and thirdly, the injury must be anatomically related to where the dystonia presents itself. Dr Smuts testified that he felt confident that the condition of the appellant conformed to this 'current' definition of post-traumatic dystonia and therefore decided that it was the most likely possibility for the cause of the appellant's medical condition.

[20] Dr Smuts further testified that he still could not exclude the possibility that 'the type that the appellant presented with was a type of dyskinesia'. The appellant, however, had not been exposed to any medication for many years. In the majority of cases when a patient stopped taking medication, while the dystonia did not go away, it would get better. As to the psychiatric aspect, the majority of the psychiatrists that the appellant consulted with came to the conclusion that the condition was not a primarily psychiatric disorder and he accepted that. He had not completely ruled out the genetic link because when he first saw the appellant, very few of the genetic tests were available at the time. Only the DYT test was available and it came back as negative. Over the years, however, more tests became available and locally there was a laboratory from which a batch of genetic tests could be ordered. However, those were quite expensive and very often unrevealing.

Another alternative would be to export blood samples to the United States of America, to get a more complete test. That still left another potential question which had been raised in the literature, that in many diseases one might carry a genetic defect that may develop a disease. However, for that to happen, there needed to be a trigger event. In this regard, a patient may present with a certain form of illness and upon testing, the rest of the family may be unaffected. The converse may also be true. Upon extensive questioning, no history of dystonia was found in the appellant's family.

[21] Turning to Dr Miller's evidence, in his report he excluded any direct psychiatric aspect to the appellant's dystonia. He also did not think that the appellant suffered from epilepsy. He observed that just about everyone (in the medical literature) agreed that dystonia would develop after a severe head injury which involved lesions to the thalamus or the basal ganglia. Even non-traumatic dystonia was related to some abnormality in the basal ganglia area of the brain. Dystonia very rarely may develop from neck pathology. While the appellant may have had neck and even thoracic pain for two to three days after the accident, any long-standing pain was most likely as a result of dystonia, not whiplash. It was unlikely that a soft tissue injury would still be present after so many years taking into consideration the following: that the appellant was, after the accident, not injured enough to go to the hospital; he alighted the vehicle and exchanged information with the other driver; and the dystonia first occurred 10 or 11 months later; whereas, with regard to the 2005 accident, the injury lasted only two weeks and then disappeared.

[22] The most compelling reason why there was no connection between the dystonia and the 2007 accident, according to Dr Miller, was simply that the appellant never had the clinical sign or picture of a significant head injury of any type at all. The MRI and angiogram were both normal. According to these, there was no evidence of any focal or chronic injury to the brain which could have produced or precipitated the problem. To develop dystonia on a post-traumatic basis, one would have had to have, at the very least, a moderate or most likely a severe to very severe head injury. The severe impairment of the appellant's life was an organic brain problem and not related to the neck at all, in Dr Miller's view.

[23] There were, however, cases recorded in the medical literature where cervical and shoulder injuries had been reported to produce dystonia either acutely, or in some cases on a chronic basis, after six to twelve months. The situation was controversial, because while all of the literature agreed that head injuries produced dystonia, 50 per cent of the literature did not mention neck or shoulder injuries producing dystonia at all. The other 50 per cent mentioned acute or chronic dystonia, following a single or a repetitive neck injury. It seemed that dystonia after a cervical or shoulder injury was exceedingly rare. On the other hand, if regard were to be had to the incidences of pre-existing trauma in patients with dystonia, approximately 5 to 20 per cent of cases would have had some form of trauma in the background, excluding those with obvious head injuries.

[24] Dr Miller further opined that given the common occurrence of minor to moderate neck trauma in the general population, it may be that the trauma

in cases of people who develop dystonia was merely incidental and not a causal finding. This was why 50 per cent of the literature did not even mention neck trauma as a pre-existing factor in some cases of dystonia; almost all of the literature mentioned head injuries and cranial trauma as a precursor of dystonia in some cases. Even in non-traumatic cases of dystonia, the disorder is classically one of brain dysfunction. Thus, after the exclusion of genetic dystonia, the most common causes of dystonia are tardive or idiopathic and non-genetic. If there were to be any association between cervical trauma or a whiplash and dystonia, it was a very rare phenomenon, and for that reason alone, the odds were that the appellant had developed secondary or idiopathic dystonia.

[25] According to Dr Miller, the neck injury of 2007 or the previous two neck injuries in 2003 and 2004/2005 were purely incidental phenomena, particularly since thousands and millions of whiplash injuries produced no overt or untoward effects of this type at all. In addition, almost all cases of dystonia began in middle age (which, according to him, was in the mid-thirties), which the appellant was approaching (at dystonia onset). Also, the appellant was given no psychiatric or antipsychotic medication until 2008 when his clinical situation was well advanced.

[26] Furthermore, post-whiplash dystonias were usually not supposed to be worse after exercise, while the appellant's dystonia was precipitated by exercise. Most importantly, post-whiplash, a post-traumatic cervical dystonia, involved only the neck, whereas the appellant's clinical picture was not only related to the neck, but was facial, ophthalmic, laryngeal,

truncal, brachial, or hemi- dystonia, thus making it exceedingly unlikely that this dystonia was related to the whiplash injury. The appellant's case was a more generalised type of dystonia, in Dr Miller's view.

[27] Dr Miller testified that he had experience with neck injuries and was to a certain extent acquainted with dystonia because from 2007 to 2014 he performed treatment called deep brain stimulation, which is one of the treatments for certain types of dystonia. In over 40 years' experience he had seen hundreds of thousands of cases with neck injuries and none had claimed that their neck injuries (whiplash) presented a dystonic picture. Furthermore, 15 to 40 per cent of those who had whiplash injuries experienced the problem for two to three weeks, which would make it easy for them to fit in with the first requirement of the abovementioned Jankovic criteria. Much of what is in the criteria commonly happened in whiplash cases.

[28] The appellant did not only have focal or cervical dystonia, he actually collapsed during the episodes. This was because he had dystonia that involved the trunk where it would flex very badly. He also had a laryngeal dystonia. So, it was not only the neck, the face and eyes that were involved. Dr Miller had also observed the appellant during his examination presenting with these movements. The appellant could not breathe properly, he could not sit upright and had to be lifted from the floor after he fell.

[29] Finally, Dr Miller testified that the appellant did not tell him about the 2003 accident. The appellant only informed him about the 2005 and 2007

accidents. The possibility of the dystonia being caused by the cumulative effect of injuries resulting from the three different accidents would not be a strange phenomenon, according to Dr Miller. Dr Birrell's evidence did not take the matter any further. And so, nothing further needs to be said in this regard.

[30] Having analysed the evidence, the high court found:

'In applying the [Jankovic] criteria and the whiplash injury sustained by the plaintiff; post-accident the plaintiff would have presented with only post-whiplash dystonia, whereas in the case of the plaintiff he however suffers from a more generalized type of dystonia. Thus, on the criteria formulated by Dr Jankovic, it does not appear as if the onset of movement disorder is related to the site of the injury, i.e. his neck.'

Factual causation

[31] In answering the question of factual causation it must be shown that 'but for' the 2007 accident the appellant would not have suffered from dystonia.[4] The enquiry is whether it was more probable than not that the involuntary movements suffered by the appellant were caused by the accident.[5] This question need not be answered with absolute certainty but must be established on a balance of probabilities.

Approach to expert evidence

[4] *Life Healthcare Group (Pty) Ltd v Dr Suliman* [2018] ZASCA 118; 2019 (2) SA 185 (SCA) para 12.
[5] Ibid para 16.

[32] The correct approach in evaluating expert evidence was laid down in *Michael and Another v Linksfield Park Clinic (Pty) Ltd and Another* (*Linksfield*),[6] where this Court held:

'. . . [W]hat is required in the evaluation of such evidence is to determine whether and to what extent their opinions advanced are founded on logical reasoning. That is the thrust of the decision in the medical negligence case of Bolitho v City and Hackney Health Authority [1998] AC 232 (H.L.E)). With the relevant *dicta* in the speech of Lord Browne-Wilkinson we respectfully agree. Summarised, they are to the following effect.

The court is not bound to absolve a defendant from liability for allegedly negligent medical treatment or diagnosis just because evidence of expert opinion, albeit genuinely held, is that the treatment or diagnosis in issue accorded with sound medical practice. The court must be satisfied that such opinion has a logical basis, in other words that the expert has considered comparative risks and benefits and has reached "a defensible conclusion" (at 241G-242B).'

[33] The fact that a body of professional opinion is almost universally held would not make the opinion reasonable, if it disregarded an obvious risk that could have been prevented. In this regard, this Court further stated in *Linksfield*:[7]

'A defendant can properly be held liable, despite the support of a body of professional opinion sanctioning the conduct in issue, if that body of opinion is not capable of withstanding logical analysis and is therefore not reasonable. However, it will seldom be right to conclude that views genuinely held by a competent expert are unreasonable. The assessment of medical risks and benefits is a matter of clinical judgment which the court would not normally be able to make without expert evidence and *it would be wrong to decide a case by simple preference where there are conflicting views on either side, both capable of logical support*. Only where expert opinion cannot be logically supported at

[6] *Michael and Another v Linksfield Park Clinic (Pty) Ltd and Another* [2001] ZASCA 12; [2002] 1 All SA 384 (A) paras 36-37.
[7] Ibid para 39.

all will it fail to provide "the benchmark by reference to which the defendant's conduct falls to be assessed" (at 243A-E).' (My emphasis.)

[34] Further, there is a difference between scientific and judicial measures of proof. This difference was highlighted in the Scottish case of *Dingley v The Chief Constable, Strathclyde Police*,[8] as follows:[9]

'One cannot entirely discount the risk that, by immersing himself in every detail and by looking deeply into the minds of the experts, a judge may be seduced into a position where he applies to the expert evidence the standards which the expert himself will apply to the question whether a particular thesis has been proved or disproved - *instead of assessing, as a judge must do, where the balance of probabilities lies on a review of the whole of the evidence*.' (My emphasis.)

[35] Expert evidence must be weighed as a whole and it is the exclusive duty of the court to make the final decision on the evaluation of expert opinion. Isolated statements made by experts should not too readily be accepted, 'especially when dealing with a field where medical certainty is virtually impossible'.[10] With these principles in mind, I now turn to the evaluation of the evidence.

Assessment of the evidence

[36] In advancing his opinion on the accident being the cause of the appellant's dystonia, Dr Smuts relied on an article titled, *Movement disorders induced by peripheral trauma*[11] authored by José Cláudio Nobrega

[8] *Dingley v The Chief Constable, Strathclyde Police* [2000] UKHL 14; 2000 SC (HL) 77 at 89D-E.
[9] See also *Maqubela v S* [2017] ZASCA 137; 2017 (2) SACR 690 (SCA) para 5, where this Court held that '[t]he scientific measure of proof is the ascertainment of scientific certainty, whereas the judicial measure of proof is the assessment of probability'.
[10] *Life Healthcare Group* fn 4 para 15.
[11] J C Nobrega, C R Campos, J C Limongi, M J Teixeira, and T Y Lin 'Movement disorders induced by peripheral trauma' (2002) *Arq Neuropsiquiatr* 60(1):17-20 (*Nobrega*).

and others (*Nobrega*), who adopted the criteria devised by Dr Jankovic. This was sourced from Dr Jankovic's earlier article, *Post-traumatic movement disorders: central and peripheral mechanisms*[12] (*Jankovic 1*), which advanced a case that peripheral trauma may cause dystonia and proposed a criteria of classifying cases in establishing the cause and effect relationship between the two.

[37] Before I deal with Dr Smuts's evidence in relation to this article, I need to say a word about what the law says in relation to an expert's reliance on literature. It is perfectly acceptable for an expert to rely on medical literature, including a published article. The expert must, however, by reason of their own training, affirm the correctness of the statements made in the article, at least in principle, and such work relied upon must be written by a person of established repute or proved experience in that field.[13]

[38] It is apparent from the reading of the high court's judgment that it considered *Jankovic 1*. This article and another, *Can peripheral trauma induce dystonia and other movement disorders? Yes!*[14] (*Jankovic 2*), also authored by Dr Jankovic, were attached as annexures to the notice of appeal. Both articles are cited in *Nobrega*. From the reading of the record, the two Dr Jankovic articles were not canvassed in evidence. This is important, because they are the source of the hypothesis postulated by *Nobrega* upon

[12] J Jankovic 'Post-traumatic movement disorders: central and peripheral mechanisms' (1994) *Neurology* 44 (11): 2006-2014 (*Jankovic 1*).
[13] *Menday v Protea Assurance Co Ltd* [1976] 1 All SA 535 (E); 1976 (1) SA 565 (E) at 569G, endorsed by the Constitutional Court in *Van der Walt v S* [2020] ZACC 19; 2020 (2) SACR 371 (CC); 2020 (11) BCLR 1337 (CC) para 31.
[14] J Jankovic 'Can peripheral trauma induce dystonia and other movement disorders? Yes!' (2001) *Mov Disord* 16(1): 7-12 (*Jankovic 2*).

which Dr Smuts relied, and they express the criteria in terms more nuanced than *Nobrega*.

[39] We were informed that the judge in the high court was furnished with a copy of *Jankovic 1* after the completion of the evidence, but before delivery of the judgment. Dr Smuts testified that Dr Jankovic is well known in the movement disorder professional community. This is evident from the three articles placed before us.

[40] Dr Jankovic's expertise in the field as well as the weight to be attached to the article(s) before the high court were not seriously challenged by the respondent. Apart from the issues being tamely put to the expert witnesses in evidence, the issue does not seem to have been contentious in the high court. Furthermore, while Dr Miller questioned the Jankovic criteria, he did not seem to dispute his reputation, as Dr Jankovic was not known to him. I have no difficulty in accepting that Dr Jankovic is a well-known scientist in regard to movement disorders, the bigger issue is around the controversy of his hypothesis, which he acknowledged was not universally embraced. In *Jankovic 2*, he observes:

'A cause-and-effect relationship between brain injury and subsequent movement disorder is well established, but the existence of such a relationship following peripheral injury has not yet been universally accepted. Because movement disorders usually occur without any history of prior trauma, and as trauma is usually not associated with movement disorders, some skeptics argue that the relationship between trauma and the subsequent movement disorder is purely coincidental.'[15]

[15] Ibid at 7.

[41] As appears from *Nobrega*, *Jankovic 2*'s postulation was apparently disputed. In this regard, it cites an article, *Can peripheral trauma induce dystonia? No!*, authored by W J Weiner.[16] What is contained in this article, or other views critiquing the Jankovic criteria, were regrettably not placed in evidence as a way of balancing the opinions and in helping to assess the logical basis and reasonableness of the hypotheses.

[42] Furthermore, while *Nobrega* endorses the notion that peripheral trauma may induce movement disorders, they conclude that 'additional experimental studies [were] needed to further clarify the mechanisms possibly involved in abnormal movement production and the ways in which a peripheral lesion could affect basal ganglia activity'. It is not clear whether any further experimental studies were done, or whether Dr Jankovic's views and criteria had become universally accepted, since the last of the articles presented to us was published in 2002. It has been held, however, that the lack of general acceptance of a scientific theory may not be the basis to reject it, without more.[17]

[43] Dr Smuts's testimony that the information was not available in 2009 (when he compiled his fist report) is puzzling. His testimony was that this information was only published in 2011 and later in 2014. He repeated this a few times in his evidence. This is undoubtedly incorrect, as the earliest article was published in 1994 and the one he relied on in 2002.

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[16] W J Weiner 'Can peripheral trauma induce dystonia? No!' (2001) *Mov Disord* 16(1): 13-22, cited in footnote 18 of *Nobrega*.

[17] *Oppelt v Head: Health, Department of Health Provincial Administration: Western Cape* [2015] ZACC 33; 2016 (1) SA 325 (CC); 2015 (12) BCLR 1471 (CC) para 40.

[44] It is also perplexing that Dr Smuts, being a specialist in movement disorders, would only discover in 2020 the phenomenon described by Dr Jankovic, shortly before the trial, when, from the reading of the articles, the concept had been the subject of debate for many years before. I say so, because in his last report, dated 28 January 2020, Dr Smuts concluded that proof of a link or not to the accident would remain a pure speculation. In his evidence he disavowed some of his previous findings and conceded that he had changed his mind many times over the years. He even made a concession that he was wrong and did not understand paroxysmal non-kinesigenic dyskinesia, which is a condition he had concluded, in one of his reports, that the appellant suffered from. This may lend credence to the assertion made by Dr Miller in the joint minute that Dr Smuts, like he (Dr Miller), did not have clinical experience of the causal relationship between peripheral trauma and a motor vehicle accident; an occurrence which both experts agree was very rare. That is not to say that he was not an expert in movement disorders in general.

[45] Nonetheless, what is to be tested is the logical basis and reasonableness of Dr Smuts's latest opinion, in which he embraced the Jankovic criteria. In this context, Dr Smuts's evidence must be viewed as a whole. This, together with Dr Miller's counter-opinion. Dr Miller conceded that he was not an expert in movement disorders, although he had experience in neck injuries and performed deep brain stimulation in some dystonia patients. He also conducted research on the current subject. The relevant expertise of both experts in relation to their evidence is kept in mind.

[46] Dr Jankovic's view that trauma to the central nervous system can cause tremors and dystonia is well established.[18] He further suggests that movement disorders can also be produced by peripheral trauma. In his view '[a]lthough initially challenged, the concept of peripherally induced movement disorders is becoming more accepted'. He also argues that this hypothesis is growing in support.

[47] As already indicated, Dr Jankovic admits that the cause-and-effect relationship in cases of movement disorders following peripheral trauma is less apparent, but that some clinicians have raised the possibility that injury to the peripheral nervous system can also produce the movement disorders.[19] To minimise the possibility of coincidence, he and others proposed the following criteria for diagnosis, stated in *Jankovic 1*:[20]

'(1) Injury must have been severe enough to cause local symptoms persisting for at least 2 weeks or requiring medical evaluation within 2 weeks after the peripheral injury, (2) the onset of movement disorder must have occurred within a few days or months (up to 1 year) after the injury, and (3) the *onset* of movement disorder must have been anatomically related to the site of injury.' (My emphasis.)

[48] In *Jankovic 2*, the third criterion is expressed in the following terms: 'the *initial manifestation* of the movement disorder is anatomically related to the site of injury'. The articulation of this third criterion is couched in slightly different terms by *Nobrega*, which is the article the experts, and in particular Dr Smuts, relied on in their evidence. It articulates this requirement as follows:

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[18] *Jankovic 1* fn 12.
[19] Ibid.
[20] Ibid.

‘3. The abnormal movements should be *anatomically related to the site of the injury*. Moreover, the causal relationship should be supported by the absence of other causes capable of producing the same symptoms, presence of reflex sympathetic dystrophy and poor response to conventional treatment.’ (My emphasis.)

[49] From this passage, it will be observed that the expressions ‘onset’ or ‘initially’ are absent from the description of the criterion. This is significant, because, according to *Jankovic 2*:

‘In many cases, the movement disorder *starts locally* in the injured region *but may later* spread to involve adjacent and ipsilateral body parts, eventually crossing over to the contralateral side. When a movement disorder occurs within a few days after injury, the cause-and-effect relationship is relatively easy to appreciate, but such an association becomes less obvious as the latency between injury and onset of the movement disorder increases. Although most studies insist on a relatively short (< 1 year) delay between trauma and the initial appearance of dystonia, some investigators accept the diagnosis of post-traumatic, peripherally induced dystonia after a latency as long as several years.’[21] (My emphasis.)

Significantly, Dr Smuts made no mention of this distinction, and in particular the stage of the dystonia, in which the criterion relates. This is because he relied on an article that used different wording to that which is contained in the Jankovic articles.

[50] Dr Jankovic accepts that there are limitations to the criteria he proposes, in that not all patients who satisfy the criteria can be confirmed with absolute certainty to have a peripherally induced movement disorder, alternatively it is possible that some patients whose movement disorders are causally related to peripheral injury do not fulfil all of these criteria.[22]

[21] *Jankovic 2* at 8.
[22] *Jankovic* 2 at 7.

[51] *Nobrega* argues that there are reasons to suggest that a direct mechanical effect upon the peripheral nervous system may not itself cause abnormal movements. Instead, a traumatic injury may exhibit an indirect effect precipitating or aggravating a pre-existing subclinical dysfunction. This article suggests that the low incidence of peripherally induced movement disorders compared to the large incidence of traumatic events in the general population suggests that some predisposing factor may be present before the trauma. Commonly associated factors are: family history of essential tremor and/or dystonia; premature birth; perinatal hypoxia; delayed psychomotor development; and use of neuroleptic drugs.

[52] In the end, *Nobrega* suggests that although in some instances the association between trauma and movement disorders might be coincidental, the close temporal and anatomical relationships frequently observed by them and others suggest a cause-and-effect phenomenon. Suggesting further that the phenomenon that peripheral trauma can alter sensory input and induce central cortical and sub-cortical reorganisation to generate abnormal movements has gained scientific support.

Evidence relating to the application of the Jankovic criteria

[53] The application of the first two ground rules of the criteria to the appellant's dystonic picture present no difficulties. As to the first requirement, his trauma appears to have caused pain in the neck and back and required medical evaluation within two weeks after the accident. He, therefore, fulfils that criterion. As Dr Miller testified, however, most people

who sustain whiplash after a motor vehicle accident would easily fit into this rule.

[54] As regards the second criterion, both experts agree that the onset of the dystonia was approximately 10 months after the 2007 accident. Again, there is no argument about the fulfilment of this rule, as Dr Jankovic gives the delayed onset a period of up to a year.

[55] Complications arise with the application of the third criterion. It will be recalled that Dr Jankovic suggests that the onset or the initial manifestation of the movement disorder must have been anatomically related to the site of the injury. In terms of the expert reports, on 17 February 2008, the appellant experienced a major attack while sitting in a motor vehicle. He could not move, everything felt 'loose', so that he could not move his arms and legs and he could not talk. He had to be carried by his father and wife into the house. This lasted for one and a half hours. He later experienced residual stiffness and felt nauseous. This lasted for a couple of days. He was too stiff to drive and had to be driven to work by colleagues.

[56] The next episode happened at a coffee shop when he fell over the table and collapsed over his colleague. He had to be carried to the office and was also taken to hospital. At the hospital he had episodes of stiffness, pain in the neck, and he would be bunched up and tight in all of the muscles, the neck, head, arm and trunk would flex and his speech would be slurred.

[57] It appears from Dr Miller's report that the appellant was told by Dr du Plessis, a neurosurgeon he consulted before he was referred to Dr Smuts, that the pins and needles in his arms and some spasms may be related to the neck, but 'there were problems with the pain and the spasms which could not be explained by the neck', and the decision at that stage was to refer him for rehabilitation.

[58] Unfortunately, Dr Smuts did not separate the timelines relating to the onset of the dystonia and the progression of the condition after the initial manifestation, when applying the Jankovic criteria, as he ought to have done. This is more so, because the appellant's condition became more severe and spread out as the years progressed, as Dr Jankovic says often happens.[23]

[59] Applying the third requirement indiscriminately, Dr Smuts simply stated in his examination-in-chief that '[y]ou cannot bump your toe and develop a measure of blepharospasm and say that that is related to one another because your eyes and your toes are not linked in such a manner. But, if you develop the dystonia in a region where the trauma took place that is important'.

[60] Unfortunately, Dr Smuts was not asked to explain why the appellant's entire body, including the legs and the trunk, was affected, and how those body parts were anatomically related to the region of the injury that was caused by the accident, ie the neck and back. In cross-examination he was asked about the appellant's dystonic condition at onset, which indicated that he could not move and that he froze. Dr Smuts simply responded:

[23] *Jankovic 2* at 8.

‘That is what [the appellant] stated there, yes. However, . . . there is something that is maybe very important to state here that in the initial phases when the patient presented with these things (a) he was very frightened, (b) nobody really understood what was going on.’

[61] I am of the view, however, that this answer does not adequately account for these symptoms. This is because the next episode at the coffee shop presented itself in a similar manner when the attack occurred. Counsel for the respondent read from the medical history narrated by the appellant, which added that the appellant was dizzy, had the facial muscles pulled downwards, was nauseous, had a speech slur, and became completely limp. Dr Smuts’s reply was that that was not how whiplash injuries normally presented; that those symptoms looked like a movement disorder; and also what would not normally be seen in classical idiopathic dystonia.

[62] Counsel for the respondent took Dr Smuts through the narration of the medical history by the appellant over a couple of months since the first episode in February 2008. In all of those the appellant had stated that he had muscle spasms and twisting over the entire body which led to complete limpness and included heavy breathing and slurring speech.

[63] Dr Smuts did not directly answer the question about the different types of abnormal muscle movements that the appellant experienced (which occurred in 2008), which were pointed out to him by the respondent’s counsel. He simply referred to different impressions he had and clinical findings he had made over a period of time. He did not explain how

abnormal movements in different body parts were related to the neck injury encountered in the accident.

[64] In cross-examination, Dr Smuts appeared to suggest that the appellant's dystonia was generalised and atypical, which was his first concern. He testified that a classical presentation of idiopathic dystonia was focal in form. *Jankovic 2*, however, states that the post-traumatic cervical dystonia, which usually occurs between three to 12 months, is similar to the phenotype of non-traumatic, idiopathic, cervical dystonia. Dr Smuts's second concern was that the appellant was a completely healthy person who had an accident and after medical intervention ended up the way he did. To him, that was more than a coincidence.

[65] Dr Smuts appears to have simply moved from the position that because there was a neck injury sustained as a result of the 2007 accident by a person (the appellant) who was previously healthy, it was highly probable that that caused the dystonia. He, however, did not explain how the generalised abnormal movements were anatomically connected to the neck injury sustained in the accident. This is an omission and was important, because, as the Jankovic articles explain, the cause and effect in movement disorders, where the onset is delayed, becomes less obvious than when the movement disorder occurs a few hours or days after the injury. That is the reason why the criteria were developed. It was to minimise the possibility that the peripheral injury and subsequent movement disorder were linked by coincidence. This is especially so, because the whiplash- induced dystonia is rare. I accept that, rarity is not the basis to reject Dr Smuts's opinion.

However, compliance with the Jankovic safeguards is the issue. It has been accepted that even in circumstances like these, a spontaneous onset, excluding the genetics and other probable causes, is not a strange phenomenon as qualifiedly conceded by Dr Smuts in cross-examination.

[66] In all these circumstances, as tragic as the appellant's condition is, I am impelled to find that the *Linksfield*[24] test was not met. Consequently, it has not been shown, on a balance of probabilities, that the soft tissue injury of the neck and back that the appellant sustained in the 2007 accident was causally connected to the involuntary movement disorder that manifested 10 months later. With other probable causes, ie use of medication, genetics and psychogenic origin being excluded, it is more probable than not that the dystonia was idiopathic and the whiplash sustained in the accident was simply a coincidence. The judgment of the high court therefore should stand.

[67] What remains to be determined is the issue of costs. There is, in my view, no reason to depart from the general rule that costs should follow the event and that the successful party is awarded costs as between party and party. However, even though the respondent employed the services of two counsel, it is not entitled to such a costs order, in my view. This Court lamentably derived little benefit from the engagement of two counsel in this matter.

[68] In the circumstances, the following order is made:
The appeal is dismissed with costs.

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[24] See fn 6.

N P MABINDLA-BOQWANA
JUDGE OF APPEAL

Appearances

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