IN THE HIGH COURT OF SOUTH AFRICA

(GAUTENG LOCAL DIVISION,JOHANNESBURG)

REPUBLIC OF SOUTH AFRICA

Case number: 2658/2014

DELETE WHICHEVER IS NOT APPLICABLE

(1) REPORTABLE: NO

(2) OF INTEREST TO OTHER JUDGES: NO

(3) REVISED

DATE: ……SIGNATURE:……

In the matter between:

P, L. F. Plaintiff

and

DR B. M BLOY Defendant

JUDGMENT

BHOOLA AJ:

Introduction

[1] The plaintiff claims damages from the defendant arising from the standard of care and treatment rendered to her from February 2011 to September 2012, which she alleges resulted in her suffering various *sequelae*. At the commencement of the trial an order was granted in terms of Rule 33(4) separating the issue of liability from the determination of quantum, and postponing the determination of quantum *sine die.* The matter proceeded on the issue of liability only.

The plaintiff's pleaded case

[2] The plaintiff, who was 43 years old at the time and employed as a credit controller at Servest Landscaping, pleads that she was admitted to the Flora Clinic under the care of Dr Becker on 4 February 2011 after having presented with atypical chest pain. This was investigated and she underwent a coronary angiogram, the results of which were normal. She was discharged on 24 February 2011.

[3] The plaintiff alleges that she was under the care of the defendant during this period. It is common cause that she was diagnosed with Addison's disease ("Addisons*")* by the defendant during this time. Addisons is an adrenocortical insufficiency due to dysfunction of the adrenal gland, which does not produce enough of the hormones cortisol and aldosterone, and is a life threatening condition. She alleges that the diagnosis was made without proper investigation, and purportedly on the results of two blood tests performed on her at the defendant's request. After the defendant diagnosed her with Addisons he placed her on corticosteroidal medication (commonly referred to as "steroids") called Covocort (a hydrocortisone) and Florinef (a fludrocortisone). This medication will be referred to herein as "steroid medication" or "steroid therapy". The defendant explained to the plaintiff that Addisons is a life threatening condition and that she will require lifelong steroid replacement therapy.

[4] The plaintiff alleges that after she commenced the steroid therapy her condition in general gradually deteriorated. She was medically boarded in May 2012 following the defendant's declaration of her as medically unfit to continue in

her employment. She alleges that she was on the prescribed steroid medication for approximately 18 to 19 months.

[5] The plaintiff alleges in her particulars of claim that she complied with the medication regime prescribed by the defendant until September 2012. She further alleges that the steroid medication interfered with her immune system thus disposing her, over the prolonged period that she took the medication, to the development of certain infections, with tuberculosis ("TB") being one of them.

[6] On 6 September 2012 the plaintiff attended the Chris Hani Baragwanath Hospital ("CHB") as a result of elevated blood pressure. At this stage she was no longer on a medical aid scheme and could not go to a private hospital. She had stopped taking the steroid medication prescribed by the defendant about approximately a month before her visit to CHB. At CHB, under the care of Professor Huddle, a Long Synacthen Stimulation Test ("Synacthen test") was performed on her. This test is known as the gold standard test for purposes of diagnosing Addisons. The results were negative. Professor Huddle directed that the plaintiff should gradually be withdrawn from the steroid medication.

[7] According to the plaintiff, she was then referred to the Rheumatology Clinic where all her symptoms were diagnosed as being secondary to the steroid usage and not to any other underlying rheumatological condition. The plaintiff was given Zolmeta infusion for osteoporosis. A magnetic resonance imaging ("MRI") scan of her brain was performed and was found to be normal apart from periventricular changes but an MRI scan of her cervical spine revealed degenerative changes.

[8] In November 2013 the plaintiff attended the Helen Joseph Hospital. She was found to have cervical lymphadenopathy on the right hand side that on biopsy proved to be secondary TB. She alleges that she was also diagnosed with neurological symptoms in her lower limbs and with TB of the spine. She was placed on anti-TB treatment on 4 November 2013. The plaintiff alleges that the diagnosis of TB was most likely a direct result of her having been on the steroid medication prescribed by the defendant for 19 months.

[9] The plaintiff alleges that the diagnosis of Addisons in February 2011 was incorrect and negligent, and that the defendant was negligent in that:

9.1 He relied solely on two blood tests and failed to do any other appropriate tests;

9.2 He failed to avoid the misdiagnosis when by the exercise of reasonable care he should have done so; and

9.3 He failed to perform the long Synacthen stimulation test to confirm the diagnosis when he could and should have done so.

[10] The plaintiff pleads that as a result of the misdiagnosis by the defendant she received steroid medication for the period February 2011 to September 2012 when same was not indicated. As a result she suffered the following *sequelae:*

10.1 She developed symptoms and complications associated with long term steroid therapy;

10.2 Her immune system was suppressed resulting in her acquiring infectious diseases and making her more susceptible to infectious disease;

10.3 She developed tuberculosis, alternatively aggravation of any pre-existing tuberculosis;

10.4 She developed tuberculosis in the spine, alternatively aggravation of any pre-existing tuberculosis of the spine;

10.5 She developed osteoporosis, alternatively aggravation of any pre-existing osteoporosis;

10.6 She developed aggravation of any pre-existing hypertension;

10.7 She developed depression, alternatively aggravation of any pre-existing psychological problem;

10.8 She suffered increased risk from factors associated with coronary artery disease;

10.9 She developed significant coronary artery disease, alternatively aggravation of any coronary artery disease, resulting in cardiac surgical procedures;

10.10 She developed increased risk for developing left ventricular hypertrophy; and

10.11 She developed increased risk for cataract.

[11] The plaintiff pleaded that she currently suffers from a range of problems inclusive of cervical lymph node and spinal TB, upper and lower limb dysfunction, brain damage and psychological problems. Before her admission to the Flora Clinic on 4 February 2011 and before her treatment under the care of the defendant commenced, her medical history presented only with hypertension and periodic headaches, and hence her current medical problems are due to her being placed on steroid therapy by the defendant.

The Defendant's pleaded case

[12] The defendant admitted the following facts:

12.1 That the plaintiff was under his care during the period 4 to 24 February 2011 but that she was not continuously at Flora Clinic during this period.

12.2 That he diagnosed the plaintiff with Addisons based on two blood tests and that he prescribed Covocort and Florinef.

12.3 That the diagnosis of Addisons was incorrect but he denies that he was negligent in making it as it was based on her low cortisol levels.

[13] The defendant pleaded that the plaintiff was diagnosed with a compression fracture at level T12, reactive depression and hypertension during her stay at the Flora Clinic between 4 and 24 February 2011.

[14] The defendant denied that the plaintiff suffered from the *sequelae* as alleged, alternatively denied that the alleged *sequelae* were a result of the medication prescribed by him on the basis of his incorrect diagnosis.

[15] The defendant pleaded that he had no knowledge of the allegations of the damages suffered by the plaintiff and accordingly denied same.

The issues

[16] It is common cause that the defendant's diagnosis of Addisons was incorrect and that the defendant did not perform the Synacthen test at any stage prior to making the diagnosis. Hence, wrongfulness is not in issue and the first issue is whether the defendant was negligent in making the diagnosis of Addisons. If he was negligent, I am required to determine whether the 20 mg of Covocort he prescribed following his diagnosis is causally linked to the plaintiff's alleged *sequelae.*

[17] It also became common cause, as a result of a concession by plaintiff's counsel in his closing submissions, that the evidence of the plaintiff's expert that the combined dosage of corticoids prescribed by the defendant was supraphysiological or a high dose was incorrect. The plaintiff therefore accepts that the dosage was physiological, in other words the normal dose produced by the body, which is also referred to as a low dose.

Expert evidence

Plaintiff's expert: Dr Promnitz

[18] The plaintiff relied on five reports prepared by Dr Promnitz a specialist physician, in terms of Rule 36(9) (a) and (b). In his evidence Dr Promnitz explained that Addisons is a rare condition in which the adrenal glands fail for a variety of reasons to produce cortisone and another hormone called aldosterone. The clinical presentations are that the patient is often fatigued, has poor appetite, experiences weight loss, muscle aches and joint pains and often presents with low blood pressure. The plaintiff however had normal cortisone secretion from her adrenal glands. He was informed that the defendant prescribed Covocort and Florinef for 19 months after diagnosing the plaintiff with Addisons. He agreed with the defendant's expert, Dr Kok that the defendant prescribed 20mg of Covocort, which is equivalent to 5mg of prednisone.

First report of Dr Promnitz: 24 December 2013

[19] Dr Promnitz prepared his first report prior to the plaintiff instituting action in 2014. He noted that the plaintiff had underlying medical problems and suspected that she had long standing depression that may account for her many problems. He noted that she was given Protos for treatment of osteoporosis. According to him exogenous cortisone would have aggravated her existing osteoporosis despite her being on Protos and she would have to undergo treatment to reverse the osteoporosis as she is at greater risk for fractures. He stated that the use of exogenous cortisone would also have aggravated her pre-existing depression and pre-existing hypertension. The report noted further that the use of cortisone for a period of 19 months when it was not indicated would have aggravated her hypertension and it appears that when she was admitted to CHB her blood pressure was not well controlled. This would predispose her to the development of left ventricular hypertrophy and an increased risk for cerebral vascular incidents. He also noted that the clinical notes from CHB indicate that the doctor who attended to her considered that she might have proximal myopathy, and according to him this is a complication of long-term steroid therapy that would result in muscle weakness. Furthermore, she would have been at a higher risk of developing infections. Importantly, he stated that it is not apparent from the clinical notes whether the plaintiff developed any infections while she was on steroids, but in his view the suppression of her immune system would continue for some time despite her being off steroids. His view was that the steroid therapy would not have interfered with her ability to continue to perform her occupation and that her inability to work at the time was probably related to her depression. However, while she was on the high dose of steroids, her existing depression would have been aggravated.

Second report of Dr Promnitz: 27 December 2013

[20] Dr Promnitz deals in this report with an email about the plaintiff's admission to Helen Joseph Hospital in October and November 2013. He records that the plaintiff was found to have cervical lymphadenopathy on the right hand side, which on biopsy proved to be secondary TB. She was investigated for neurological symptoms in her lower limbs and a diagnosis of TB of the spine was made. This,

he concludes, is most likely a direct result of her having been on corticosteroids for 19 months. He stated that corticosteroids interfere with the immune system and predispose the patient receiving glucocorticoids for prolonged periods to certain infections, with TB being one of them. In his view this might have been secondary reactivation of latent TB, which is common in South Africa where people are exposed to TB at a young age. The primary infection remains dormant until reactivation occurs under certain circumstances. He concluded that it was well accepted that patients on long-term steroids are at a greater risk of developing primary or secondary TB. He did not refer to the dosage actually prescribed by the defendant.

Third report of Dr Promnitz: 12 August 2015

[21] Dr Promnitz deals, for this first time, with the dosage prescribed by the defendant, and confirms that she was placed on Covocort 10mg morning, 5mg noon and 5 mg night as well as on Florinef. He states that when the plaintiff was informed in October 2013 that she had TB of the cervical node, she was also told that she had TB of the thoracic spine. He also confirmed that in October 2014 she presented with chest pains at the Helen Joseph Hospital and was diagnosed with a myocardial infarct, had an angioplasty performed and went back for insertion of a stent on 18 November 2014. He further confirmed that in April 2015 she collapsed and was referred to Helen Joseph Hospital for further investigations. She was told that she has ongoing ischaemic heart disease and required a coronary bypass graft. He records the plaintiff's current complaints as being the following: generalised body pain; headaches and dizziness; weakness in both limbs with the left being more pronounced; paraesthesia in both limbs with the left being worse than the right; depression; episodes of chest pain; ischaemic heart disease; calcification of her brain; and blackouts that had not been adequately investigated at the time and might present as epilepsy since she was already on low dosages of Epilim. Dr Promnitz also records that she informed him that prior to her treatment at the Flora Clinic in February 2011 she had only suffered from hypertension and periodic headaches (this was subsequently shown in evidence to be incorrect). He also recorded that on examination the plaintiff had pigmentation on her face in the malar distribution, which she explained resulted from her treatment for TB. Dr Promnitz further noted evidence of left ventricular

myopathy, and importantly that she had a T12 compression fracture. He stated that this was subsequently diagnosed as secondary TB for which she was still on treatment. He again classified the dosage prescribed by the defendant as a high dose of Covocort, which in his opinion would have aggravated the TB of the spine. The steroids would also have resulted in development of osteoporosis, which would in turn aggravate her spinal problem. These two factors, he explained, have undoubtedly resulted in the plaintiff having a permanent neurological deficit in her lower limbs.

[22] In relation to ischaemic heart disease, Dr Promnitz stated that he had sight of the angiogram Dr Becker performed in February 2011 when there was no evidence of coronary artery disease, but four years later the plaintiff required a coronary artery bypass graft. This suggests to him that she developed ischaemic heart disease since 2011. He stated that it is documented that long-term steroid use might influence the risk factors associated with coronary artery disease such as lipids and hypertension adversely and this might be contributing factor. He went further to state that in his opinion the steroid therapy she was incorrectly placed on had predisposed the plaintiff to developing significant coronary artery disease requiring a coronary artery bypass graft. She had also been left with what appears to be impaired left ventricular function following her development of ischaemic heart disease and this will not improve over time.

Fourth report of Dr Promnitz: 31 January 2016

[23] Dr Promnitz responds in this report to the report prepared by Prof Richards on behalf of the defendant. He agreed that in the literature 20mg of Covocort is equivalent to 5mg prednisone. However, he states that in replacement therapy for Addisons one uses hydrocortisone rather than prednisone as the latter has an 8 to 12 hour duration and has a mineralocorticoid effect as well. He confirmed that the plaintiff was also on a fludrocortisone (Florinef), which is classified as a steroid with a mineralocorticoid effect, but he does not state the dosage that the defendant prescribed. Despite this he is of the opinion that the plaintiff received a higher dosage of steroids than that referred to by Prof. Richards. He questioned Prof Richards's opinion that the plaintiff's use of Covocort of 20mg for approximately 19 months would not have predisposed her to the development of

TB as this is alluded to in most articles with reference to steroid usage and the risk of TB. He referred to an article dealing with meta-analysis of controlled trials in which glucocorticoids or placebo were given to patients, and which reported that infection occurred more significantly with steroid therapy. He stated that "the infection rates were significantly increased only in patients given an average dose of prednisone of more than 10mg per day or an accumulative dose per day of 700mg". According to him, the plaintiff received a cumulative dose of approximately 2800g (he corrected this in cross examination to milligrams) of prednisone over the period that she was taking Covocort. This in his view placed her at greater risk of reactivation of TB, and is evident from the fact that she was diagnosed with TB of the cervical gland. He referred to the existence of considerable literature on the immune effect of prolonged steroid usage and in his opinion the fact that the plaintiff received steroids for a long period of 19 months predisposed her to reactivation of TB. In regard to TB of the spine he did not have sight of the x-rays from Helen Joseph Hospital but assumed that it was fair that the orthopaedic surgeon would have found some or other evidence to suggest TB involvement of the spine.

Fifth report of Dr Promnitz: 15 February 2016

[24] Dr Promnitz noted from an x-ray report dated March 2011 that the plaintiff had a compression fracture at T12; that there was gibbus formation in the area and the comment on the report was that this has the appearance of post-traumatic compression fracture. It was also recorded that this was an old injury and there is no mention of osteopenia or osteoporosis. He noted that the right hip showed a T-score of minus 1.4 which represents some osteopenia and the x-ray report notes some osteopenic changes in the right hip and right femur but no other areas. Dr Promnitz notes that he would not have treated the plaintiff for osteoporosis (as the defendant did) because it is only indicated where there is a T-score of minus 2. There was in his opinion no indication for prescribing Protos on these results. He also noted that the bone density scan of 30 January 2012 indicated no evidence of osteoporosis, which implies that her condition had improved on the Protos, but he still stated there was no reason for prescribing Protos. He confirmed that the plaintiff undoubtedly does not have osteoporosis based on the information supplied to him.

Defendant's expert: Professor Richards

[25] Prof. Richards, a specialist physician, agreed that the diagnosis of Addisons was incorrect and inappropriate. He confirmed that a T11 and T12 disc protrusion would cause pain the chest region, in other words, in the lower chest and upper abdomen area. He confirmed his agreement with Dr Promnitz that the plaintiff's diagnosis of cervical lymph node TB in November 2013 was probably a reactivation of pre-existing exposure to TB. He testified that most people have been exposed to TB at some time, but that it could remain latent for long periods of time without it ever manifesting as infection, or one could develop overt infection, which is most frequent in the lungs. He confirmed that the use of corticoids could re-activate latent TB but that this could only occur when supraphysiological doses were administered.

[26] Prof. Richards confirmed that it is well known that the dose of Covocort given to the plaintiff was less than the physiological dose, and this dose does not result in suppression of the immune system. He repeatedly stated that side effects only occur when supraphysiological doses are administered, and that if the plaintiff had been given an immuno-suppressive dose, the TB would have manifested during the period she was still on steroid therapy. In cross-examination he confirmed, when referred to the medical literature, that the risks from steroid medication occur during use and the immune system reconstitution occurs rapidly thereafter. In the plaintiff's case, she developed TB long after the cessation of the steroid use and hence in his opinion there was no association with the steroid use.

Defendant's expert: Professor Greeff

[27] Prof. Greeff is a registered pharmacologist. He explained that corticosteroids are synthetic analogues of the natural hormones produced by the body's adrenal cortex. There are two types of corticosteroids: mineralocorticoids, which are primarily involved in the regulation of electrolytes and water balance and can cause the retention of sodium and water in the body; and glucocorticoids, which are predominantly involved in carbohydrate, fat and protein metabolism. Glucocorticoids have an anti-inflammatory, immunosuppressive and vasoconstrictive effect. In other words, they have an ability to suppress the body's

immune system. According to him the defendant prescribed Florinef in a dosage of 0.1mg and this is a mineralocorticoid with no appreciative glucocorticoid effect at usual daily dosages of 0.05mg to 0.2mg. This reference was made relying on the Goodwin and Gilman textbook. He therefore confirmed that the defendant prescribed a physiological dose of steroids to the plaintiff, in combining Covocort and Florinef. This has since been conceded by plaintiff's counsel.

[28] Prof Greeff explained the difference between a physiological and supra-physiological dose as follows:

"Supra-physiological means higher concentration that the body normally has in its own right, so if you add to that, more than what the body needs and this is why with hydrocortisone the 20mg is a physiological dosage. It's less than what the body normally produces on a daily basis and it's not too much. If you administer too much the HPA axis will be suppressed and you will stop secretion and if you do that for a long time, you will suppress the adrenal gland and when you stop using, you won't be able to produce the hormones anymore and that's a danger of this and that's why you have to stay within your dosages to make sure that you don't get total suppression of the adrenal glands so that when you discontinue treatment the [Indistinct] can re… If you will re-secrete its own hormones in the normal way. That's why we are cautious not to give high dosages or supra-physiological doses."

[29] Prof Greeff's evidence was that due to the different pharmacological actions of the Florinef and the Covocort, Florinef at the dosage given to the plaintiff could not have had any appreciable additive glucocorticoid effect on the plaintiff and hence could not have contributed to the plaintiff's alleged *sequelae.* He explained that Florinef could not increase the serum levels of cortisone in the body. Cortisone is relatively available in the body at a concentration of 60 nanomole per millilitre. Once the body needs cortisol, the cortisone is converted to cortisol by an enzyme in the kidney, and then it can have a pharmacological action or an action in the body. Thus, there is a fine balance and there is enough cortisone if the body needs it to convert into cortisol. His evidence was that if there is too much cortisol in the body its negative feedback mechanism will kick in and the pituitary gland will not secret any more steroid hormones, so there will be less glucocorticoids

secreted by the body. The body therefore keeps the balance of what it needs, depending on circumstances. Hence the ideal glucocorticoid replacement therapy would mirror the normal physiological state of the patient as closely as possible. He confirmed that the defendant prescribed 20 mg of hydrocortisone to the plaintiff and this is the acceptable daily dose in two or three divided doses. According to him the long-term effects of all drugs are dose dependent and evidence of long-term side effects must be evaluated in the context of the doses of the glucocorticoid preparations administered.

[30] Prof. Greeff explained that a physiological dose is the normal acceptable dosage in replacement therapy, and it will not suppress the adrenal glands. However, it was put to him that the plaintiff was not on a replacement dose because her steroid hormone concentration levels were normal. He replied: *" [t]he tests that … [the defendant] has done, showed low concentrations, which clinically in his judgment was inadequate and that's why he started replacement therapy. So we cannot say that she's had enough, so we've added the two and now she's had too much. The body will secrete less if you add more to the body."*

[31] With regard to the suppression of the immune system, which the plaintiff alleges contributed to the plaintiff acquiring or having aggravated infectious diseases such as TB, he testified that where patients receive more than 15 mg of prednisone (equivalent to 80 mg of cortisone) daily, there was an increased risk of contracting TB whilst on long term glucocorticoid therapy. This does not apply to the plaintiff. The defendant's prescription of 20mg of hydrocortisone daily is equivalent to 5mg of prednisone, and this dosage did not put the plaintiff at a higher risk of developing TB especially 14 months after the cortisone was discontinued. In relation to the academic article by Jick *et al* which calculated the ratio for people not exposed to glucocorticoids and those on recent and past glucocorticoid therapy to be a ratio of 1 and 1.4 respectively, he commented that although a trend of increased risk was observed even with a physiological dose, the association did not reach statistical significance and it therefore cannot be said that the plaintiff was at a higher risk of contracting TB or any other infectious disease. The article also confirmed that there was no clear effect of duration or cumulative dose on the risk of TB.

[32] Prof. Greeff confirmed that he saw the entry in the CHB records dated 4 November 2013 that the plaintiff experienced severe back pain radiating to the whole back from the cervical spine. He also saw the entries dated 26 and 27 March 2012 indicating that the plaintiff received physiotherapy for her neck and back.

[33] In regard to osteoporosis, his evidence was that the plaintiff's bone mineral density ("BMD") improved on glucocorticoid therapy prescribed by the defendant over one year (from 16 February 2011 to 30 January 2012) as seen on the radiology reports.

[34] In relation to ischaemic heart disease, Prof Greeff referred to a report by Liu *et al* on a population base study comparing 68781 glucocorticoid users and 82282 non-users which found the rate of cardiovascular events to be significantly higher in patients prescribed high glucocorticoid dosages (greater than 7.5mg per day of prednisone or greater than 30 mg of cortisol). According to him cardiovascular risk was not increased in patients using less than 7.5mg per day of prednisone or less than 30 mg of cortisol, which was the dosage applicable to the plaintiff. Prof. Greeff also referred to another large retrospective case control study which found that current glucocorticoid use was associated with significantly increased risk and cardiovascular risk was found to be greater with higher glucocorticoid doses and with current use.

[35] In regard to depression, Prof. Greeff stated that the plaintiff was diagnosed with reactive depression on her first admission to hospital (in 2011) and before she received cortisone treatment. He stated further that most patients with psychiatric reactions to corticosteroids usually recover from their symptoms with dose reduction or upon cessation of therapy.

[36] In concluding his testimony Prof. Greeff stated that given the lower dose of cortisol that was prescribed and the time span between treatment and the development of clinical conditions, it was highly unlikely that any of the alleged *sequelae* were caused by the cortisol therapy. In support of his opinions and reasoning, Prof. Greeff referenced (in addition to the literature referred to by Dr Promnitz), further articles and studies on the impact of glucocorticoids.

[37] When it was suggested to Prof. Greeff in cross-examination that he could not exclude the possibility that if a low dose of cortisol is administered to a patient that it could cause the *sequelae* exhibited by the plaintiff, he replied that *"anything is possible in medicine"*. He added that in his opinion the probability is very low and unlikely albeit *"not improbable to really happen"* and that "*different patients react differently to drugs".*

Defendant's expert: Dr Kok

[38] Dr Kok is a specialist physician. She testified that although the defendant found two borderline levels of cortisol, there were no other features of Addisons clinically and she confirmed that the diagnosis was incorrect. She stated that the defendant could have confirmed the diagnosis with a more robust test and assessed the plaintiff's response to the proposed treatment. However, given the plaintiff's initial presentation at the Flora Clinic, including fatigue, low blood pressure in normally hypertensive patient and borderline levels of cortisol it would have been reasonable for the defendant to treat the plaintiff as an Addison's disease patient.

[39] In her view the dosage prescribed by the defendant was not of such magnitude that any metabolic or bony *sequelae* would follow. There was evidence of a T12 fracture, which had occurred in 2007, but this was four years before the defendant saw the plaintiff. Her view was also that the subsequent lymphadenitis due to TB was remote from any possible influence by the defendant's treatment. Dr Kok confirmed that she could not find any biopsy of the spine that confirmed the diagnosis of TB of the spine or that showed that TB of the spine was presumed after the lymphadenitis was diagnosed. What is important is that she stated that the reports of the chest radiographs did not reveal any evidence of TB or Ghon focus (lesions in the lung) indicating a risk of TB or past exposure to TB. Her evidence was that the plaintiff had a normal full blood count on several occasions and there was no anaemia or chronic disorder, which if present, would be in keeping with TB. Furthermore the plaintiff's erythrocyte sedimentation levels ("ESL") were never elevated, which would have indicated inflammation.

[40] Dr Kok also stated that the likely cause of the spinal insufficiency fracture would have been osteoporosis and that this preceded any treatment by the defendant. She further stated that the plaintiff received treatment for pain neuropathic systems and muscle weakness throughout the entire period and this was not due to the defendant's treatment. It is also significant to note her evidence that the defendant attempted to improve the plaintiff's symptoms with appropriate treatment when he saw her and these symptoms are unrelated to the Covocort and Florinef subsequently prescribed by him. The defendant's treatment of her with Protos as well as vitamin D and calcium supplementation was appropriate as reflected by a subsequent bone density scan in 2012 which confirmed an improvement in her bone mineral density.

[41] In relation to the plaintiff's low cortisol levels, Dr Kok confirmed that on both July 2012 and August 2012 when the plaintiff was at the Garden City Clinic her blood levels for cortisol showed *"not detectable".*

[42] In regard to heart disease, Dr Kok stated that she could find any link or association between the plaintiff's ischaemic heart disease and the defendant's treatment. In her opinion the defendant's treatment did not cause or contribute to the plaintiff's alleged diagnosis of heart disease.

The plaintiff's evidence

[43] The plaintiff testified that prior to February 2011, she was relatively healthy and could walk about 3km a day to work, and was only chronic medication for high blood pressure. Her general practitioner, Dr Bodhania, had arranged an appointment with the defendant for 7 February 2011 for syncope (collapsing and fainting). She initially denied that that was the reason she had been referred to the defendant, but conceded this in cross-examination. Prior to the appointment and on 4 February 2011 she had to be admitted to the Flora Clinic with complaints of chest pain and high blood pressure. She was admitted under the care of Dr Becker, a cardiologist, who did an angiogram on 7 February 2011, the results of which were normal.

[44] The defendant also attended to her during this time and he ran some tests and diagnosed her with Addisons. The diagnosis of Addisons came as a shock to

her and she was unable to cope because the defendant advised her that it was a lifelong disease and she would have to take chronic medication. The defendant called in a psychiatrist to consult with her at the Flora Clinic in order to cope with the diagnosis. It was put to her in cross-examination that she consulted with a psychiatrist, Dr Magnus, at the clinic prior to the diagnosis of Addisons, but she could not recall this. The plaintiff had sadly had many traumatic life experiences including the murder of her husband, relocation from one province to another, as well as being a single parent of three children. She could not recall being involved in a car accident in 2007 or having x-rays taken as a result.

[45] Before her discharge from the Flora Clinic on 24 February 2011 after this first admission, and after the steroid treatment commenced, she experienced symptoms of weakness in her knees and fatigue. She was treated by a physiotherapist for neck and back pain at the clinic but denied that she complained about back pain prior to her admission to the clinic. She also received acupuncture treatment in the hospital. She said she was "*just getting more and more sick*" and she left the hospital in a wheelchair although she was fully ambulant when she was admitted. The defendant told her that this was the nature of the illness and informed her to take the cortisone medication daily as she had a life-threatening illness. He prescribed 20 mg Covocort which she was required to take three times a day (in three divided doses of 10 mg, 5 mg and 5 mg) and Florinef 0.1 mg, which she was instructed by the defendant to take each morning.

[46] She was also given a prescription to be filled at the pharmacy for the same medication for a further six months. Later that year, in November 2011, the defendant gave her a second prescription for another six months of repeat medication. She complied with the treatment regime and took the medication daily because of the warning she had received from the defendant about the seriousness of her condition.

[47] After her first hospital admission she had episodes of collapsing at home and her children often had to rush her to the hospital. She experienced severe back pain, tiredness and her legs were weak. Her children gave her a walking frame to assist her with walking. After yet another episode of passing out her children took her to the Garden City clinic instead of Flora Clinic. She was placed

in a psychiatric ward where a physician told her she does not have Addisons because it is not associated with high blood pressure. She insisted on returning to the defendant for treatment and she informed him of the advice given to her at Garden City to the effect that she does not have Addisons.

[48] She confirmed that the defendant completed a form for the purpose of her claiming disability benefits in March 2011. The form indicated that the diagnosis had been made on 5 February 2011. She also confirmed that she suffered from all the symptoms that the defendant listed on the form including: impossible to perform seated/sedentary tasks; impossible to climb, walk on uneven terrain; difficulty with bending, lifting, pushing and pulling, operate light and heavy machinery, and working with weights. The defendant had indicated on the form that these symptoms had appeared a few months prior to that date. She confirmed this in cross-examination. She confirmed that he also completed a certificate for her employer, at her request, booking her off from work "*till further notice*" and as a result she was medically boarded from her employment in May 2012. Her last visit to the defendant was in April 2012 when he told her to continue with her medication. She was fully compliant with taking her medication until 15 April 2012 but by August 2012 she was very sick. She stopped taking the steroid medication and her daughter in fact threw them away.

[49] She attended the endocrine clinic at CHB in September 2012 where Professor Huddle informed her that she does not have Addisons. She testified that he was very upset about her being diagnosed with Addisons. He told her she had to be weaned off the medication as a result of which she was kept in hospital for approximately six weeks for that purpose.

[50] In May 2013 she developed painful lumps in her neck and was referred by her clinic to the Helen Joseph hospital where a biopsy was performed. She started to feel better but still had to receive physiotherapy as her legs were still weak. She returned to Helen Joseph hospital in October 2014 where she was informed of her diagnosis with TB. In October 2014 she was admitted to the Helen Joseph Hospital presenting with chest pain and was diagnosed with a myocardial infarct. She was transferred to the Charlotte Maxeke Hospital where a coronary angiogram and angioplasty was performed on her and a stent was inserted on 18

November 2014. In April 2015 she collapsed and was taken to the Helen Joseph Hospital where a coronary artery bypass was performed on her on 14 May 2015. In July 2015 she again had a syncopal (fainting) attack.

The plaintiff's medication regime

[51] On the dispute as to whether the defendant prescribed steroid therapy for 12 or 19 months, the plaintiff testified that she was given a third prescription by the defendant on 13 April 2012 for a further six months' steroid medication. It was put to her in cross examination that this was not a prescription but rather an admission order because she was being admitted to the Flora Clinic that day and the defendant was placing an order for the medication to be administered to her during her stay as well as for various tests to be conducted. She accepted that this was not a prescription for medication for her to obtain from the pharmacy and take at home.

[52] The plaintiff then suggested that she would still have had repeats for medication remaining from the November 2011 prescription, because during her hospital admissions she was given medication from the hospital and upon discharge she would be given medication to take at home. Under cross examination, however, she was shown that none of the discharge forms after the first six months script in March 2011 recorded any take home Covocort or Florinef and the plaintiff then confirmed that she did not receive any Covocort or Florinef to administer at home upon discharge.

[53] The plaintiff's evidence was that but for the periods when she was admitted to hospital from 10 February 2011 until 15 April 2012 (when she was discharged from Flora Clinic in respect of her last admission on 13 April 2012) when Covocort and Florinef was administered to her during her hospital stay, she only received and used the two six month prescriptions that were issued by the defendant.

[54] For the period 24 February 2011 (when the plaintiff was discharged from the Flora Clinic) until 18 March 2011, the defendant placed on record before the Court that he accepts that during this period the plaintiff took 20 mg of Covocort and 0.1 mg Florinef per day. This would comprise a total of 23 day. From 18 March 2011 (the first prescription) and 9 May 2012 (when the last prescription

would have finished) the plaintiff was in hospital for 34 days when she received hospital medication. This means that the steroid medication prescribed by the defendant would have ended in mid-June 2012. Defendant's counsel submitted that this inference can be drawn from the undisputed and admissible evidence of the plaintiff that she took her medication every day; that during the periods she was in hospital she was not taking her own medication and she would in addition get "take home" medication prescriptions; and her concession that during 18 March 2011 to April 2012 she did not receive any additional Covocort or Florinef from the hospitals as "take home" medication. It appears therefore on the probabilities that the plaintiff stopped taking the Covocort and Florinef prescribed by the defendant in June 2012 and had not been on the steroids prescribed by the defendant for 19 months as was pleaded.

[55] Plaintiff's counsel informed the court during his opening address that her evidence would be that she stopped taking the prescribed medication a month before her admission to CHB in September 2012 because she could no longer afford the medication. He however stated that he subsequently consulted with her and her evidence will be that she stopped taking the medication because her daughter told her to stop because her condition was not improving but that she still had some cortisone medication. Counsel took responsibility for the discrepancy. In her evidence the plaintiff indeed confirmed that she stopped the medication in August 2012, but this had been based on her incorrect version of the so-called third prescription issued by the defendant on 13 April 2012.

[56] Defendant's counsel submitted that the only reason why the plaintiff would still have steroid medication left after August 2012 would be that she was not fully compliant with taking her medication daily, as she had testified. As a matter of logic if she was fully compliant she would not have had any leftover medication after June 2012. This is confirmed by the two *"not detectable"* cortisol levels taken at Garden City Clinic in July 2012 and August 2012 as testified by Dr Kok. The plaintiff would not have had such negligible cortisol levels if she was still taking Covocort and more so if Dr Promnitz's additive theory was correct.

[57] It must also be accepted, as was submitted by defendant's counsel, that the plaintiff took 20 mg Covocort and 0.1 mg Florinef daily during this period. The

plaintiff testified that she took the dosages prescribed on the bottle, which was 20 mg. There were three instances when it appears in hospital records that 25 mg was administered to her, but this would seem to be an error.

Events post treatment

[58] The plaintiff disputed that she took any other form of steroid therapy other than that prescribed by the defendant. However, on her admission to CHB the admission form reflects that she was on "Meticorton". She testified that she did not know what this referred to. Dr Promnitz (who testified before she did) said that at the time of her admission to CHB she was on Meticorton according to the hospital records, and that this was a high dose of prednisone. This was based on his incorrect understanding that the defendant had issued a third prescription for Covocort and Florinef on 13 April 2012. When it was put to him that the defendant's version would be that the 13 April 2012 document was not a prescription, Dr Promnitz's evidence about the Meticorton changed. He then no longer relied on it being a high dose of prednisone, but said it was a less potent version of Covocort sold when a pharmacy does not have stock of Covocort. This evidence was not substantiated.

[59] The plaintiff testified that after she was informed at CHB that the Addisons diagnosis was incorrect, she took a decision not to go to any doctors or hospitals again. However, in May 2013 she developed lumps on her neck and was diagnosed with TB of the cervical glands at the Helen Joseph hospital. This version was not put to Dr Promnitz since the plaintiff gave evidence after he did and this was the first time this version was given by the plaintiff. In 2015 she had continuous angina pains and had to have heart surgery.

Dr Bodhania

[60] Dr Bodhania, the plaintiff's general practitioner, confirmed in his testimony (based on the notes in his patient file), that the plaintiff did not complain of or receive treatment at his practice prior to 4 February 2011 for TB, myopathy, general weakness, cardiovascular problems or psychiatric difficulties. She was treated for syncope as she had symptoms of collapsing and fainting, as well as for headaches and back pain in 2010 (the plaintiff initially denied consulting him for

back pain but in cross examination indicated that she would defer to him). He confirmed that in July 2012 she received an injection for pain. She was also treated for insomnia (a sleeping tablet was prescribed for anxiety and not sleeping well at night), myalgia (muscle or soft tissue pain), non-specific muscle pain (28 February 2011) which led to a referral to a rheumatologist, a type of infection (3 March 2011), abdominal pain (January 2012 with symptomatic treatment for ulcers and cramps), and vomiting and fever (2 July 2012). The plaintiff submits that this confirms she was only treated after her steroid treatment commenced for some the new or steroid related complications.

Defendant's evidence

[61] The defendant is a specialist physician and nephrologist. He testified that he received a call from the plaintiff's general practitioner, Dr Bodhania, prior to the plaintiff's admission to the Flora Clinic on 4 February 2011. Dr Bodhania informed him that the plaintiff had pre-existing hypertension and had a syncope (fainting) episode, and that he required a specialist to examine her. An appointment was made for the plaintiff to see him but a week before the scheduled appointment she was admitted to the Flora Clinic with chest pain. She was under the care of Dr Becker but the defendant was asked to consult with her when she told staff she had a scheduled appointment with him.

[62] The defendant testified that although the plaintiff complained of pain, she was unable to localise the pain. The medical team at some point considered whether the plaintiff was exaggerating her pain since they were unable to find the cause. Two x-rays were conducted to establish the cause because Dr Becker's investigations did not reveal a cause for the pain. The tests revealed that the plaintiff had a fracture on the T12 vertebra of her thoracic spine and a significant wedge compression. According to the defendant this was one of the worst fractures he had seen and the medical team could not understand why she had not been in severe pain from this fracture prior to her admission. They provided the plaintiff with a back brace on the understanding that the T12 fracture was a recent fracture and that it would take eight weeks to heal. The plaintiff could not recall how she had sustained the fracture. Under cross examination the plaintiff agreed that she had subsequently been informed by a pain management clinic

that what she thought was chest pain was actually pain radiating from her back. The defendant also confirmed that when he saw the plaintiff in court she was using crutches and a back brace.

[63] The defendant was concerned about her history of syncope (collapsing), hyperpigmentation on her face, low blood pressure, chronic fatigue, depression, pain, weakness and chest pain and he ordered blood tests to establish her cortisol levels. Her symptoms suggested underlying adrenal gland issues. The first test yielded a low result (her cortisol level was 96 when it should have been between 101 and 535). He wanted to identify if she had Addisons or another underlying disease.

[64] Once the low cortisol results came back he tried to obtain Synacthen to perform a test to confirm the diagnosis but Synacthen was not available.[1] As an alternative, on 10 February 2011 he ordered a second cortisol test to confirm whether her cortisol levels were consistently low and also ordered a course of Covocort to be administered subject to the second test results. When he found out that the nurses had already administered a dose of Covocort before obtaining the second test results, he instructed the nursing staff to *"omit"* the administration of Covocort for the next three days. The results indicated that her cortisol level was just above normal (in other words it was borderline), but together with the plaintiff's other symptoms he considered a clinical diagnosis of Addisons to be appropriate. He prescribed the lowest possible dose of steroids, being 20mg Covocort and 0.1mg Florinef. He could not recall the discussion he had with the plaintiff when he informed her of the diagnosis but he would have informed her that she had to take the medication daily and strictly since it is a chronic condition.

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[1] The defendant does not rely on the alleged stock outage of Synacthen as part of its defence. When it was put to the defendant in cross examination that his reliance on the stock outage was a concoction and would have been pleaded as a defence had he disclosed it to his legal representatives, his counsel placed it on record that their case was that he did not do the test, and that they do not intend raising his inability to obtain the Synacthen as a defence.

[65] He testified that the plaintiff requested him to complete more than one form for disability claims from her insurance and he did so. He got a sense that all she wanted was to submit a claim for disability benefits.

[66] He confirmed that he only issued two six month repeat prescriptions to the plaintiff and that the so called third prescription of 13 April 2012 was in fact an admission note which contained all the tests (including blood tests) which had to be done as well as well as the medication to be administered during the plaintiff's hospital admission.

[67] The defendant testified that during the plaintiff's repeated admissions to hospital she would arrive with elevated blood pressure but as soon as she was given pain medication her blood pressure would normalise as depicted by the various readings taken during her admissions.

[68] His evidence was that the plaintiff's complaints during the various hospital admissions were always related to her back or chest pain. Her chest pain was later identified as back pain radiating from the T12 fracture. He said that despite having placed the plaintiff on treatment for the fracture, including a brace, Protos, Vitamin D, calcium and multiple analgesics for months he was unable to determine why she still complained of severe pain. Plaintiff's counsel submitted that this made it imperative to obtain certainty about the Addisons diagnosis, which he had failed to do.

[69] He conceded that his diagnosis of Addisons was incorrect, but testified that her symptoms at the time were in his opinion consistent with Addisons. The defendant was referred to the agreements between Dr Promnitz and Dr Kok and Dr Promnitz and Prof Richards to the effect that the diagnosis of Addisons was incorrect and the appropriate tests to confirm the diagnosis were not done, and he conceded this. His testimony was that the gold standard for testing for Addisons was the Synacthen test as well as the insulin induced hypoglycaemia test. He confirmed in cross-examination that although he could not obtain Synacthen from the hospital pharmacy he did not even perform the insulin induced hypoglycaemia test. He testified that he continued to treat the plaintiff with corticosteroids as he

was *"fairly convinced*" that his diagnosis was correct. He accepted that this was his *"best inconclusive shot at diagnosing Addison's disease".*

Evaluation of expert evidence

[70] The approach to be taken by a court in evaluating expert opinion has been set out by the Supreme Court of Appeals in *Bee v Road Accident Fund* [2] as follows:

> *"[22] It is trite that an expert witness is required to assist the court and not to usurp the function of the court. Expert witnesses are required to lay a factual basis for their conclusions and explain their reasoning to the court. The court must satisfy itself as to the correctness of the expert's reasoning. In Masstores (Pty) Ltd v Pick 'n Pay Retailers (Pty) Ltd [2015] ZASCA 164; 2016 (2) SA 586 (SCA) para 15, this court said '[l]astly, the expert evidence lacked any reasoning. An expert's opinion must be underpinned by proper reasoning in order for a court to assess the cogency of that opinion. Absent any reasoning the opinion is inadmissible'. In Road Accident Appeal Tribunal & others v Gouws & another [2017] ZASCA 188; [2018] 1 ALL SA 701 (SCA) para 33, this court said '[c]ourts are not bound by the view of any expert. They make the ultimate decision on issues on which experts provide an opinion'. (See also Michael & another v Linksfield Park Clinic (Pty) Ltd & another [2002] 1 All SA 384 (A) para 34.)*
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> *[23] The facts on which the expert witness expresses an opinion must be capable of being reconciled with all other evidence in the case. For an opinion to be underpinned by proper reasoning, it must be based on correct facts. Incorrect facts militates against proper reasoning and the correct analysis of the facts is paramount for proper reasoning, failing which the court will not be able to properly assess the cogency of that opinion. An expert opinion which lacks proper reasoning is not helpful to the court. (See*

[2] *Bee v Road Accident Fund* (093/2017) [2018] ZASCA 52; 2018 (4) SA 366 (SCA) (29 March 2018)

also Jacobs v Transnet Ltd t/a Metrorail [2014] ZASCA 113; 2015 (1) SA (SCA) paras 15 and 16; see also Coopers (South Africa) (Pty) Ltd v Deutsche Gesellschaft Für Schädlingsbekämpfung mbH 1976 (3) SA 352 (A) at 371F.

[71] In *Michael and Another v Linksfield Park Clinic (Pty) Limited and Another*[3] the court noted " *....it is perhaps as well to re-emphasise that the question of reasonableness and negligence is one for the court itself to determine on the basis of the various, and often conflicting, expert opinions presented. As a rule that determination will not involve considerations of credibility but rather the examination of the opinions and the analysis of their essential reasoning, preparatory to the court's reaching its own conclusion on the issues raised."*

[72] The court held (at [36]) that what a court is required to do is evaluate the evidence of experts to determine *"whether and to what extent their opinions advanced are founded on logical reasoning. That is the thrust of the decision of the House of Lords 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.*

> *[37] 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" (at241 G - 242 B)....*
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> *[39] 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*

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[3] *Michael and Another v Linksfield Park Clinic (Pty) Limited and Another* 2001 (3) SA 1188 (SCA) (at [34])

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

[40] Finally, it must be borne in mind that expert scientific witnesses do tend to assess likelihood in terms of scientific certainty. Some of the witnesses in this case had to be diverted from doing so and were invited to express the prospects of an event's occurrence, as far as they possibly could, in terms of more practical assistance to the forensic assessment of probability, for example, as a greater or lesser than fifty per cent chance and so on. This essential difference between the scientific and the judicial measure of proof was aptly highlighted by the House of Lords in the Scottish case of Dingley v The Chief Constable, Strathclyde Police, 200 SC (HL) 77 and the warning given at 89 D-E that:

"(o)ne 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."

[73] In evaluating the expert evidence, regard must also be had to the medical literature Dr Promnitz provided which he asserted supported his opinion that the Covocort and Florinef prescribed by the defendant as a result of his incorrect diagnosis of Addisons was the cause of the plaintiff's alleged *sequelae*.

The high dosage theory

[74] Dr Promnitz testified with reference to an academic article that the Florinef prescribed, (which he incorrectly stated as being 1 mg instead of 0.1mg), had a glucocorticoid potency of 5 times that of the 20mg Covocort prescribed and the two together had a high or supraphysiological potency. In his opinion, in addition to this exogenous cortisol the plaintiff's body was still producing its natural levels of physiological cortisol. Thus, there was an additive effect. He persisted in this view despite it being put to him in cross examination that the hypothalamic pituitary adrenal ("HPA") axis meant that the body's negative feedback loop would kick in and the hypothalamus would ensure that the adrenal gland stops releasing cortisol when cortisol levels in the blood get high. Both Dr Kok and Prof. Greeff disputed Dr Promnitz's assertion, which they stated was biologically and scientifically incorrect. The plaintiff has now accepted this by way of the concession made in closing argument and no longer relies upon the evidence of Dr Promnitz that the plaintiff received supraphysiological doses of corticosteroids. This was a concession Dr Promnitz refused to make when it was put to him in cross-examination, i.e. that not only that he had made a mathematical error in calculating the dosage of the prescribed steroids and that his opinion of a supraphysiological dosage was not supported by the literature.

[75] Dr Promnitz testified however that even though the plaintiff might have been given physiological or low doses of corticoids, the drugs would still have had an influence on her pituitary adrenal axis resulting in suppression and side effects from the combination of the Covocort and Florinef she was prescribed. In his opinion, a patient who is given steroids for no indication (a normal patient, apart from hypertension in the plaintiff's case), is subjected to the additive effect of the *“two drugs”* on suppressing the adrenal gland. In his opinion the combined use of these agents exhibit glucocorticoid activity, and hence it was recorded at CHB that the plaintiff had a cushingoid appearance and steroid induced proximal myopathy. He testified that although a dosage of 5mg to 10mg a day of steroids was considered a low dose, there are trials that have concluded that even low doses cause many adverse events such as osteoporosis, myopathy, cardiovascular disease, cataracts and increased risk of infections. His counsel, to the extent that it resulted in supraphysiological doses, correctly rejected his additive effect theory, and his generalised assumption that all steroids have adverse effects added no

probative value to proving a link between the treatment administered to the plaintiff and her alleged *sequelae.*

[76] I agree with defendant's counsel that without the high dosage theory none of the findings of Dr Promnitz are supported. Hence, when this was in fact put to him in cross-examination he refused to make the concession because it would have been an error fatal to the plaintiff's case. He eventually conceded that he was wrong, but persisted without providing a basis for his reasoning that Florinef 0.1mg had a glucocorticoid potency level of more than 20mg of Covocort. This was despite the fact that the table he had himself provided and the extract from the Goodman and Gillman textbook indicated that at the dosage prescribed by the defendant there was no appreciable glucocorticoid effect. The table in fact is supportive of Prof Greeff's evidence that that Florinef (as a fludrocortisone) has no appreciable glucocorticoid potency at the dosage prescribed by the defendant.

Tuberculosis

[77] In his second report Dr Promnitz stated the lymphadenopathy on the right-hand side proved was secondary TB and the diagnosis of TB of the spine were most likely the direct result of the plaintiff having been on corticosteroids for 19 months. This is because corticosteroids interfere with the immune system thus predisposing the patient to the development of certain infections, with TB being one of them. His opinion was that the plaintiff's TB might have been a reactivation of latent TB and went further to say that it is well accepted that patients on long-term steroids are at a greater risk of reactivation of TB or primary TB infection. This evidence was again based on the incorrect assumption of a high dosage and, very significantly, he did not deal with the actual and correct dosage of the steroids prescribed by the defendant.

[78] TB is the main *sequelae* the plaintiff relies on, and in this regard the academic article relied on by Dr Promnitz (the Nicolaides article) fails to support Dr Promnitz's conclusions of the link between steroids and the plaintiff's *sequelae* when regard is had to the sources the article he relied upon. The source articles, which Prof Greeff testified about, confirm that Dr Promnitz's conclusions are scientifically unsupported. As one example, the article Dr Promnitz refers to a

study by Dickson as stating that patients receiving 5mg prednisolone continuously for the last three months, six months or three years had a 30%, 46% or 100% increased risk of serious infection respectively. However, it excludes reference to the fact that this increased risk applies to current users of prednisolone. The plaintiff was not in the category of a current user as she had been diagnosed with TB of the cervical gland in November 2013, more than a year after she stopped taking the medication prescribed by the defendant. Hence, the conclusions would not apply to her, and accordingly Dr Promnitz, in relying on the article to support his proposition that the plaintiff was more susceptible to TB because of the cortisol prescribed by the defendant, would have been relying on misleading information. Also, the article Dr Promnitz relied upon does not state (which the source Dickson article does), that discontinuing a two-year course of 10mg prednisolone, six months ago halved the risk compared to ongoing use. It should also be noted that 10mg prednisolone is double the dosage of cortisol prescribed by the defendant and a two-year course is longer than the period the plaintiff was on the steroid medication prescribed by the defendant. Prof. Greeff alerted the court to the fact that he had considered the Dickson article (which was not provided by the plaintiff), and the Dickson article does not contain the information and figures that are attributed to it. The article relied upon by Dr Promnitz clearly contained incorrect information upon which he relied.

[79] Dr Promnitz's opinion that the steroid therapy led to the plaintiff developing TB more than a year after she stopped the steroid therapy, was moreover not supported with reference to any evidence. In the joint minute prepared by Dr Promnitz and Dr Kok both agreed that the plaintiff was diagnosed with possible spinal tuberculosis at Helen Joseph Hospital and that they did not have sight of the x-rays on which the diagnosis was based. Notwithstanding the absence of a bone biopsy (Dr Promnitz held the view that it was inappropriate to perform a bone biopsy), Dr Promnitz attempted to elevate the possible spinal TB to that of a definitive diagnosis with reference to a handwritten note questioning the presence of spinal TB. The TB diagnosis was made by way of a needle biopsy on 29 August 2013. On the plaintiff's version in evidence in chief and without the concessions made in cross-examination, she stopped using the Covocort and Florinef prescribed by the defendant a year before her lymph node TB diagnosis. Furthermore, Dr Promnitz's evidence on the existence of spinal TB was that the

Helen Joseph Hospital records have an entry referring to the plaintiff's T11 *"end plate eroded"* being highly suggestive of TB of the spine or what is called *"POT spine,"* which in his view is consistent with TB of the spine. Previously there was no such erosion only the T12 fracture diagnosed in 2011.

[80] Dr Promnitz continued to rely on literature which is not relevant to the facts in this matter because it applies to high dosage and also, in respect of the causal criteria, states that the effects were not present for past users, such as the plaintiff. Another misleading or incorrect reference to the academic article relied upon by Dr Promnitz arose in regard to the conclusion that an epidemiological study of patients with TB showed they were nearly five times more likely to have been using glucocorticoids at the time of their diagnosis. When the source article is consulted it qualifies the five-fold reference by stating that patients who were currently exposed to a glucocorticoids had an approximately five fold increased risk of developing new TB. The plaintiff had subclinical or dormant TB not new TB and hence these conclusions are not applicable to her. Furthermore, the article states that the magnitude of association was larger with the prednisone equivalent dose than with a physiological dosage (i.e. 7.5 mg daily) and was larger with more than one prescription for a glucocorticoid. When this was put to Dr Promnitz in cross-examination his response was that the articles were only provided for guidance purposes.

[81] Dr Promnitz's reliance on a second academic article in his report of 31 January 2016 was also shown in cross-examination to be wholly incorrect and misleading. According to him the article refers to infection rates from a meta-analysis of control trials in which glucocorticoids were given, and this supported his opinion that the plaintiff was at a greater risk of reactivation of TB as a result of the Covocort prescribed by the defendant. The reference in his report that infection rates were significantly increased only in patients given an average dose of prednisone of more than 10mg/day (which is not applicable to the plaintiff) or an accumulative dose of 700mg (which would be applicable to the plaintiff if it was correct) is the converse of what was actually stated in the article. It said : "*The rate was not increased in patients given a daily dose of less than 10mg [which includes the plaintiff] or accumulative doses of less than 700mg of prednisone".* This contradicts Dr Promnitz's testimony. He interpreted the article to mean that the

converse would apply i.e. if the rate is not increased in patients given a daily dose of less than 10mg a day then it must mean that it does increase with a daily dose of more than 10mg a day. As matter of logic this reasoning is flawed. The plaintiff was given a dose of 5mg prednisone daily and hence her rate of risk was not increased because her dosage was below 10mg prednisone daily. The use of the word "or" makes it clear that the article finds that in either category i.e. a daily dose of 10mg daily or accumulative doses of less than 700mg, the risk is not increased. Since the plaintiff fell into the first category i.e. her daily dose was less than 10mg, she could not as a matter of logic have also fallen into the second category as well. Dr Promnitz interpreted this to mean that she would have been in both categories and in relation to the second category she would be at higher risk of infection. Reference to the article makes it clear that this cannot be the correct interpretation. The article also goes further than this and provides a graph which makes it clear that even at a duration of 1000 days (i.e. more than double the duration for which the plaintiff was prescribed the medication by the defendant) provided the prednisone daily dosage is less than 10 mg, there was no increased risk of infection. This was confirmed by Prof Greeff in his evidence.

[82] The experts for the defendant thus testified that, unlike the articles referred to by Dr Promnitz, the literature clearly shows that the increased risk of developing TB associated with glucocorticoid therapy is increased for those who are current users and on a high daily dose, but not for past users such as the plaintiff.

[83] There is no support in the literature for Dr Promnitz's opinion, particularly when she was already off steroid therapy at the time of her diagnosis of TB and even earlier when she noticed the lump in her neck. Thus, defendant's counsel submitted, plaintiff cannot advance a case that she contracted TB whilst on the treatment prescribed by the defendant. This would be in direct contradiction of the proven facts and the plaintiff's own version that she developed the lumps on her neck in May 2013, which is 11 months after she stopped the steroid therapy. In order for her allegation to be true, this court would have to find that throughout the time she was on steroid therapy from February 2011 until June 2012 and she was in and out of hospitals and examined by various doctors, they all ignored signs of TB which would have been present had she developed the infection whilst on the treatment, which was the testimony of Dr Kok. This also disposes of the

submission by plaintiff's counsel that the lymphadenitis must have started developing or reactivating during the period that she was on the steroid therapy. This is not an inference that can correctly be drawn from the facts.

Ischaemic heart disease

[84] The academic article that Dr Promnitz relied upon stated that the relative risk for cardiovascular events in patients receiving high dose glucocorticoids (more than 7.5 mg of prednisolone) was 2.5.6 after adjustment for covariates. The article also states that current use of glucocorticoids was associated with increased risk of heart failure and a smaller risk of ischaemic heart disease. In the joint minute of Dr Promnitz and Dr Kok, Dr Promnitz admits there is no direct link of steroid usage causing coronary artery disease but stated that there are indirect effects on lipids and blood pressure, which may contribute to its development. He again relied on the high dose theory, which has since been rejected by his legal representatives. This confirms there is no scientific or reasonable basis for the statement that the steroid therapy contributed to the plaintiff's ischaemic heart disease.

[85] In his third report Dr Promnitz states that the plaintiff appears to have been left with impaired left ventricular function following her development of ischaemic heart disease. However, under cross examination, when he was referred to the report of Dr Kalk prepared after the plaintiff's 2015 procedure which indicated that the plaintiff had no left ventricular hypertrophy, Dr Promnitz conceded that he did not actually conduct any tests to establish whether the plaintiff in fact had impaired left ventricular function. This once again confirms, as was submitted by defendant's counsel, that Dr Promnitz did not properly apply his mind to whether or not glucocorticoid therapy had any causal link to the plaintiff's ischaemic heart disease and which occurred three years after she stopped the steroid treatment.

[86] In relation to the acute myocardial infarct suffered by the plaintiff in October 2014, Dr Promnitz stated that the fact that in 2011 she had normal coronary

arteries, rendered it very unusual [4] that a person her age would suddenly develop significant coronary artery disease. He confirmed however that there was no direct link between steroid therapy and ischaemic heart disease. He said that the plaintiff had been left with what appeared to be impaired left ventricular function following her development of ischemic heart disease. However, he confirmed that when he examined the plaintiff subsequently, he did not find any evidence of left ventricular hypertrophy. Hence his only conclusion was that he found it difficult to account for someone who had normal coronary arteries in 2011 to have required a bypass three to four years later.

Osteoporosis

[87] In the academic article that Dr Promnitz relied upon, a study showed that therapy with high doses of oral glucocorticoids caused significant decease in bone mineral density ("BMD") even in the first two months of therapy. As a result, there is an increased risk of osteoporotic fractures and it has been estimated that fractures may occur in up to 30 to 50% of patients on glucocorticoid therapy but fortunately there is a rapid decrease on the risk on cessation of therapy. Similar findings are observed in a more recent study showing that low daily dose prednisone (less or equal to 7.5 mg per day) with high accumulative doses increases the risk for fractures. The article continues to say that the risk declines rapidly, with the decrease beginning three months after cessation of therapy.

[88] Dr Promnitz conceded that the plaintiff does not have osteoporosis. He confirmed that he and Dr Kok compiled a joint minute in which they agreed that the plaintiff had osteopenia not osteoporosis. This was based on the bone density scan done on 16 February 2011. The plaintiff produced no contemporaneous bone density scores to establish that she has osteoporosis, and despite having examined the plaintiff and having stated in an earlier report that the plaintiff is at high risk of developing osteoporosis, Dr Promnitz did not himself perform the necessary bone density tests.

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[4] The transcription suggests that Dr Promnitz used the word *"usual"* in the context, which is incorrect.

[89] Dr Promnitz criticised the fact that the defendant prescribed Protos for the plaintiff, even though the plaintiff's bone density scores increased considerably while she was on the glucocorticoid therapy together with the Protos and vitamin supplements prescribed by the defendant. In fact, the academic article that the plaintiff relies upon indicates that the plaintiff, at the time she saw the defendant and he diagnosed her with osteoporosis, was in the higher risk category for osteoporosis, as she is over 40, has a previous osteoporotic fracture and a T-score of minus 2.5. The article recommends that adults with a moderate to high risk should be treated with calcium and Vitamin D as well as an oral bisphosphonate. This was the treatment the defendant prescribed upon making the diagnosis of osteoporosis in 2011.

[90] Dr Promnitz indicated in his report that a diagnosis of osteoporosis could only be made where there is a T-score of minus 2. The Helen Joseph Hospital diagnosed the plaintiff with osteoporosis in September 2012 based on T-scores of minus 1.2 and minus 1.5, and based on Dr Promnitz's evidence this is also incorrect. Therefore, other than criticising the defendant for treating the plaintiff for osteoporosis when in his opinion there was no basis for doing so, Dr Promnitz was unable to support the allegation that there is a link between the steroid therapy prescribed by the defendant and any possible osteoporosis diagnosis.

Other relevant symptoms and complications associated with long-term steroid use: cushingoid features and hypertension

[91] The plaintiff does not strongly rely on the other symptoms and complications associated with long-term steroids except for cushingoid features and hypertension. These are dealt with below.

Cushingoid features

[92] The only inference that can be drawn from the fact that the plaintiff was recorded as being on Meticorton is that she had been taking Meticorton without a prescription after she stopped the treatment prescribed by the defendant, and this caused the cushingoid features. This is consistent with the literature. Dr Promnitz conceded that the plaintiff did not have Cushing’s or Cushing Syndrome and Dr Kok was in agreement. Dr Promnitz testified that when plaintiff was admitted to

CHB it was recorded upon her admission that she was on Meticorton 10mg three times a day, and that this is 30mg of prednisone daily (i.e. a high dose). This was at the point where the plaintiff relied upon the April 2012 admission note as the third prescription, the implication being that the defendant had prescribed the Meticorton. Dr Promnitz's version changed in cross examination when it was put to him that the document was not a prescription but an admission sheet and he then downplayed Meticorton as simply a generic of Covocort, which he said is administered when a pharmacy cannot supply Covocort. However, Dr Kok's evidence was that Meticorton is a powerful glucocorticoid far more potent than the hydrocortisone prescribed by the defendant. Dr Kok testified that if the plaintiff was taking 10mg Meticorton prior to her admission to CHB and after her last repeat script from the defendant finished, even for a short period of time, this would account for her cushingoid features. This was consistent with Dr Promnitz's evidence in chief. The cushingoid features therefore cannot on the probabilities be attributed to the treatment prescribed by the defendant.

Aggravation of pre-existing hypertension

[93] The Nicolaides article relied upon by Dr Promnitz states that increased glaucoma, depression and increased blood pressure were observed in patients receiving dosages of more than 7.5mg per day of prednisone. It is common cause that the plaintiff's dosage was less than this threshold. Dr Promnitz's reliance on this article was based on his mistaken belief that the plaintiff was on a higher dose than that which the defendant had actually prescribed.

[94] In his evidence in chief Dr Promnitz relied upon a package insert for Florinef, which was confirmed by Prof Greeff to list every possible adverse reaction that could occur and to have no probative value in this matter. It is accordingly disregarded.

[95] In the absence of any material evidence that establishes that the plaintiff's risk of hypertension increased after glucocorticoid therapy ceased, there is no causal link established pre the defendant's misdiagnosis of Addisons and post the steroid therapy prescribed by defendant. In order to establish that the steroid therapy aggravated plaintiff's pre-existing hypertension, the plaintiff was required

to show that prior to the commencement of the therapy the plaintiff's hypertension was under control, but no evidence to this effect was led. Secondly, the plaintiff would have had to show that post her cessation of the steroid therapy the hypertension was under control. Thirdly, the plaintiff would have had to show that while on the steroid therapy her hypertension was more elevated than it was before she was put on the steroid therapy. The plaintiff was unable to do so.

Has negligence been established?

[99] It is trite that he who asserts must prove and it is therefore the plaintiff who bears the onus of proving that the defendant was negligent, and that his negligence caused the damage suffered. Negligence involves an enquiry into whether the conduct of the defendant measured up to the standard expected of a reasonable person in the particular circumstances: *Kruger v Coetzee.*[5] This has been elevated in medical negligence cases to the reasonable medical practitioner. The court held that for the purposes of liability *culpa* arises if - (a) a *diligens pater*familias in the position of the defendant - (i) would foresee the reasonable possibility of his conduct injuring another in his person or property and causing him patrimonial loss; and (ii) would take reasonable steps to guard against such occurrence; and (b) the defendant failed to take such steps.

[100] In *Meyers v MEC Department of Health, Eastern Cape*[6] Plasket JA reminded us that more than a 100 years ago, in *Mitchell v Dixon*[7] the then Appellate Division held in relation to the standard of care expected of medical practitioners that : *"a medical practitioner is not expected to bring to bear upon the case entrusted to him the highest possible degree of professional skill, but he is bound to employ reasonable skill and care; and he is liable for the consequences if he does not."*

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[5] *Kruger v Coetzee* 1966 (2) SA 428 (A) at 430 E.
[6] *Meyers v MEC Department of Health, Eastern Cape* (1010/2018) [2020] ZASCA 3 (4 March 2020) at [1].
[7] *Mitchell v Dixon* 1914 AD 519 at 525.

[101] Thus the approach taken by the courts[8] is that the standard that is required is not based on what can be expected of the exceptionally able doctor, but on what can be expected of the ordinary or average doctor in view of the general level of knowledge, ability, experience, skill and diligence possessed and exercised by the profession, bearing in mind that a doctor is a human being and not a machine and that no human being is infallible. Furthermore, an error of clinical judgment will not constitute negligence if the doctor has adhered to the requisite standard of reasonable care. Defendant's counsel referred the court to *Pringle v Administrator Transvaal* [9] where the court considered whether a surgeon committed an error of clinical judgment and referred with approval to *Whitehouse v Jordan*[10] and stated the following:
"The true position is that an error of judgment may, or may not, be negligent; it depends on the nature of the error. If it is not one that would have been made by a reasonable professional professing to have the standard and type of skill that the defendant held himself out as having, and acting with ordinary care, then it is negligent. If on the other hand, it is an error that a man, acting with ordinary care might have made, then it is not negligence. (Counsel's emphasis).

[102] The plaintiff pleaded three bases for alleging that the defendant was negligent in making the diagnosis of Addisons: he relied solely on two blood tests and failed to do any other appropriate tests; he failed to avoid the misdiagnosis when by the exercise of reasonable care he should have done so; and he failed to perform the long Synacthen stimulation test to confirm the diagnosis when he could and should have done so.

[103] In determining whether the defendant's conduct in incorrectly diagnosing Addisons involved the application of reasonable skill and care of a physician in the circumstances, I accept the defendant's evidence that the low cortisol levels and clinical presentation of the plaintiff in February 2011 led to him to suspect she had

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[8] *Castell v De Greef* 1993 (3) SA 501 (C), 1994 (4) SA 408 (C). *Michael v Linksfield Park Clinic* (Pty) Ltd 2001 (3) SA 1188 (SCA) at 1192. *Goliath v MEC for Health Eastern Cape 2*015 (2) SA 97 (SCA) at para 8. *Mitchell v Dixon* 1914 AD 519 at 525.
[9] *Pringle v Administrator Transvaal* 1990 (2) SA 379 (W).
[10] *Whitehouse v Jordan* 1981 (1) All ER 267 at 276H.

Addisons. She reported symptoms that he recorded on the disability claim form he completed on her behalf. Notwithstanding his exercise of clinical judgment, in my view he ought to have foreseen, as reasonable physician, that without following up with appropriate tests (the insulin hypoglaecemic or the Synacthen test) to confirm the Addisons diagnosis, there was a reasonable possibility that the diagnosis was wrong. The link between his diagnosis and her high blood pressure also indicated that he was incorrect. A reasonable physician would in my view have taken steps to guard against an incorrect diagnosis in circumstances where a test is available to confirm the diagnosis. Insofar as the defendant's counsel does not rely on Synacthen being unavailable at the time the diagnosis was made, there was no explanation why he did not even administer the insulin hypoglaecemic test to confirm his diagnosis. The defendant conceded that he relied on two tests indicating normal (the first albeit just under normal) cortisol levels. He conceded that his diagnosis was incorrect and that he did not do the necessary tests.

[104] The experts called by the defendant were understandably careful to avoid reference to negligence, instead referring to the treatment and/or diagnosis without a follow up test being "*inappropriate*". The expert consensus in relation to the issue of negligence includes the joint minute of Dr Promnitz and Prof Richards in which they agree that the diagnosis of Addisons was inappropriate and the appropriate tests were not performed by the defendant to make the diagnosis, as a result of which the plaintiff was placed on Covocort and Florinef medication for a period of approximately 19 months. In the joint minute of Dr Promnitz and Dr Kok they agree that the diagnosis of Addisons by the defendant was incorrect and it was imperative that a Synacthen test should have been performed before placing the plaintiff on lifelong replacement therapy (although it became common cause he had only issued prescriptions for one year).

[105] I am therefore in agreement with the submission by plaintiff's counsel that the common cause facts and the expert consensus in relation to the issue of negligence in making the Addisons diagnosis based purely on his clinical judgment and the two blood tests, limits the dispute between the parties to that of causation. In other words, in failing to conduct follow up tests to confirm his incorrect diagnosis of Addisons and in prescribing steroid therapy as a result, the defendant did not employ the reasonable skill and care of a physician in the

circumstances. He ought to have foreseen that the plaintiff might suffer adverse consequences if his diagnosis was incorrect and failed to take adequate steps to prevent this.

Has causation been established?

[106] It follows that the issue to be determined is whether the negligent diagnosis of Addisons, and the consequent administration of low doses of steroid therapy for a period of 12 months, caused the series of *sequelae* the plaintiff allegedly suffered.

[107] It is trite that *res ipsa loquitur* (the facts speak for themselves) is not applicable in medical negligence matters both in respect of negligence and causation. This has been confirmed in the *locus classicus* of medical negligence cases, *Van Wyk v Lewis.*[11] In *Goliath v MEC for Health, Eastern Cape* [12] the Supreme Court of Appeal reconsidered the relevance of the doctrine and referred to the reluctance of the courts to apply the maxim as being *"....because, as Lord Denning MR observed in Hucks v Cole [1968] 118 New LJ 469 ([1993] 4 Med LR 393) 'with the best will in the world things sometimes went amiss in surgical operations or medical treatment. A doctor was not to be held negligent simply because something went wrong'. For to hold a doctor negligent simply because something had gone wrong, would be to impermissibly reason backwards from effect to cause (Medi-Clinic Limited v Vermeulen (504/13) [2014] ZASCA 150 (26 September 2014) para 27).* The court confirmed however that (at [12]) , "*in every case, including one where the maxim res ipsa loquitur is applicable, the enquiry at the end of the case is whether the plaintiff has discharged the onus of resting upon her in connection with the issue of negligence ..."*

[108] The plaintiff is thus required to prove on a balance of probabilities that a causal link was established between her alleged *sequelae* and the negligent diagnosis of Addisons in February 2011 and subsequent 20mg cortisol prescribed

[11] *Van Wyk v Lewis* 1924 AD 438.
[12] *Goliath v MEC for Health, Eastern Cape* 2015 (2) SA 97 (SCA) at [9].

which ceased (at the latest on her own version) in August 2012 (but on the probabilities on June 2012).

[109] Causation comprises of two components to establish liability. The first is factual causation, which requires the question of whether a negligent act or omission caused the harm giving rise to the claim. The second is legal causation, which requires a consideration of whether the negligent act or omission linked to the harm giving rise to the claim is sufficiently close or directly connected for legal liability to occur. If the link on the facts between the negligent act and the consequences is at best tenuous then the plaintiff fails at proving factual causation and legal causation does not even become relevant.

[110] The test for factual causation as set out in *Lee v Minister for Correctional Services*[13] is still the "*but-for*" test, as counsel for the defendant submitted, since the plaintiff is not relying on an omission but an act by the defendant, being the misdiagnosis of Addisons and the consequent prescription of steroids. This requires a plaintiff to establish that the wrongful and negligent conduct was probably a cause of the loss, which calls for a sensible retrospective analysis of what would probably have occurred, based upon the evidence and what can be expected to occur in the ordinary course of human affairs: see *Mashongwa v Passenger Rail Agency of South Africa (PRASA) t/a Metrorail.* [14] This court is thus required to determine whether, but for the defendant's wrongful conduct in diagnosing Addisons and prescribing steroids, the plaintiff's alleged *sequelae* would not have occurred. The plaintiff must therefore prove that it is more likely than not, but for the defendant's wrongful and negligent conduct, the harm she suffered would not have occurred. In other words, but for the low dose steroids prescribed by the defendant following his negligent diagnosis of Addisons she would not have suffered further problems resulting in her being medically boarded in 2012 and had further *sequelae* that manifested in 2013 and 2014, most

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[13] *Lee v Minister for Correctional Services* 2013 (2) SA 144 CC.
[14] *Mashongwa v Passenger Rail Agency of South Africa (PRASA) t/a Metrorail* 2016 (3) SA 528 (CC).

importantly cervical gland TB (which plaintiff suggests must have started in 2011 - 2012), and cardiac disease.

[111] Plaintiff's counsel submitted in regard to causation, that neither the defendant nor anyone else forwarded any other possible cause for the conglomerate of complications that the plaintiff experienced and which are common to steroid use. Furthermore, counsel submitted that it was not helpful that defendant's case is that the complications only arise with supraphysiological doses of steroids, because the literature does refer to adverse effects in consequence of low, normal or high doses. Counsel accepts that the risk is higher with higher dosages but submits that the adverse effects can manifest even at low or normal dosages. In this regard, he relied on the key concession made by Prof Greeff that the effect of steroids is not the same on all individuals, and that different patients respond differently to steroid use. The plaintiff thus submits that she fell into a category of those who are more susceptible even though the dosages prescribed for her were not high but normal. The balance of probabilities therefore strongly favour her on causation in that the impact of the steroids on her showed up very shortly after the administration of the steroid therapy in February 2011. She was discharged on 8 February and the next day collapsed and had to be wheeled back into hospital and she spent the next year in and out of hospital with the defendant not knowing what was causing her symptoms. Thus, it is clear that her complications are linked to the steroid therapy, and the evidence that stands firm is that post February 2011 the plaintiff presented with a conglomeration of complications which are common to steroid use. Plaintiff's counsel accepts that her chest pain in February 2011 was in all probability caused by the old T12 compression fracture. However, in his submission the fracture was a red herring and hence the plaintiff does not rely on anything related to the old fracture.

[112] Plaintiff's counsel also submitted the defendant did not contest the lymph node TB in cross examination but merely suggested that if the plaintiff first noticed the lump in her neck in 2013 and her steroid therapy ceased in 2012, there could be no link. However, this misses the point because both Dr Promnitz and Prof Richards suggest that it was reactivation of latent TB, and the court is bound by their opinion as the Supreme Court of Appeal made it clear in *Bee v Road*

Accident Fund[15] that when experts agree on facts or opinions it becomes common cause unless a party repudiates the agreement. Hence, plaintiff's counsel submitted that it was cast in stone that firstly, plaintiff presented with lymph node TB in 2013 and tested positive for it. It is not clear, counsel submitted, when the latent TB began to reactivate but it is common sense that it would not appear overnight. Also, her heart was normal in February 2011 but within two to three years she started to develop serious problems, which led to a heart attack and a stent being inserted. Thus, it was submitted that there can be only one cause for heart disease and the TB and that is the steroids, and the defendant has proffered no other cause. This is with respect not an inference I can correctly draw on the facts in evidence. It calls for a conclusion, that it was necessary to *"individualise"* patients and the plaintiff was more susceptible to steroids than other patients might be. There were no facts established in this regard. Hence we have only Dr Promnitz's opinion that since corticosteroids have different effects on different patients, one can never predict what effect it will have on a patient. He was unable to provide the court with any other possible causes for the series of complications the plaintiff presented with, except to suggest that the medical literature reveals a definite association with corticosteroids and an increased risk of certain diseases and conditions, because steroids work on the immune system and this affects the inflammatory process. However, the evidence led by defendant's experts, and based on the literature, was that only high doses have an effect on suppressing the immune system and hence Dr Promnitz's evidence on this issue falls to be rejected.

[113] It is also clear from the literature and documentary evidence discussed above that the literature relied upon by Dr Promnitz in preparing his reports and in his testimony does not support his conclusions. In this regard, I am in agreement with the defendant's counsel that a careful consideration of the literature, as was done in cross examination, shows that he in most instances did not consider the literature in its entirety but rather cherry picked aspects that supported his theories

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[15] *Bee v Road Accident Fund* (093/2017) [2018] ZASCA 52; 2018 (4) SA 366 (SCA) (29 March 2018)

and opinion and in some instances as indicated above, often misinterpreted the literature to support his opinion. This is why Prof Greeff's evidence demonstrated that the very literature Dr Promnitz relied upon did not support his opinion. It is clear that because Dr Promnitz had made the fatal error of classifying the plaintiff's dosage as high dose, he was inevitably going to fall into the trap of referring to the aspects of the literature that related to high doses and not the low dose the plaintiff was on that became common cause at the end of the trial. For the first time in his third report in August 2015 he referred to the dosage actually prescribed by defendant. Up to this point his generalisations regarding the link between Covocort and the plaintiff's alleged complaints could be excused on the basis that he did not know what dosage was prescribed. In this report he noted that the dosage was Covocort 10mg, 5mg at be administered at noon and 5 mg at night. He stated further that the plaintiff was also prescribed Florinef. He then explained that this still led to a supraphysiological dose because this exogenous dose of synthetic steroids would have had an additive effect given her body's own physiological dose. This theory came crumbling down in cross examination and he refused to concede a basic issue of maths and logic and it was left to his legal team to refuse to deal with his evidence in their heads of argument. Hence I agree with the submission made by defendant's counsel that having regard to the circumstances of the matter, and the proven and common cause facts between the parties, it is evident that there is no support in the scientific and medical literature or on the facts for the contention that the dosage prescribed by the defendant placed the plaintiff at higher risk of infection and caused her symptoms.

[114] The experts for the defendant, Prof Greeff, Prof Richards and Dr Kok, based their evidence on the facts and the scientific and medical literature. Their opinions were logical and reasoned. Prof Greeff's extensive knowledge based on his experience as a clinical pharmacologist of was immense benefit to the court despite the plaintiff's suggestion that his evidence should be disregarded as he was not involved in the development of steroids and did not have experience with treating patients. His evidence was based on the facts, literature and logic and when an issue fell outside his area of expertise he elected not to comment and deferred to the relevant experts. In contrast, when Dr Promnitz was faced with the absurdity of his suggestion that Florinef at the dosage prescribed by the defendant had a glucocorticoid effect, he refused to make the concession despite the fact

that there was no support for this view. He persisted with his outlier view until his legal representatives were forced to make the concession during closing argument.

[115] Prof Greeff was of great assistance to the court in explaining how medical trials are conducted and the importance of determining the risk to benefit ratio in this process. He explained that glucocorticoids are dangerous drugs but at the dosage prescribed by the defendant they were not dangerous. His evidence was that dosage is a material factor determining at what stage a drug is considered to be dangerous and thus not worth the benefits it may yield. His evidence was also helpful in explaining that the pamphlets inserted by manufacturers into drugs packing has to list all possible side effects and cannot be understood to be a concession as to the probabilities of such side effects manifesting when the drug is administered. Dr Kok's evidence was similarly of assistance to the court and despite the plaintiff's submissions as to her subjectivity, her inexperience and somewhat naive approach as a first-time witness in court lent to her objectivity. She made the necessary concessions about the inaccuracy of certain facts she relied upon for her conclusions and adequately explained why she had asked for a second joint minute to be signed.

Conclusion

[116] In my view therefore, if one has regard to the fact that the plaintiff's osteopenia was improved while on the steroid therapy with Protos, vitamin D and calcium supplements prescribed by the defendant; the plaintiff was diagnosed with TB of the cervical glands a year after she ceased the steroid therapy; and she experienced the cardiac event three years after she ceased the steroid therapy, the probabilities do not favour a causal link. Nor is there a close enough connection, as was required by the Constitutional Court in *Mashongwa v Passenger Rail Agency of South Africa (PRASA) t/a Metrorail.*[16] The Court

[16] *Mashongwa v Passenger Rail Agency of South Africa (PRASA) t/a Metrorail* 2016 (3) SA 528 (CC) at [64].

cautioned against the conflation of wrongfulness and causation, and held that the wrongfulness element - the normative consideration based on social and policy considerations - should not be used to contaminate the factual dimension of the causation enquiry. If this approach were to be accepted then the net of liability would be cast too wide. The Court stated[17]: "*no legal system permits liability without bounds. It is universally accepted that a way must be found to impose limitations on the wrongdoer's liability. The imputation of liability to the wrongdoer depends on whether the harmful conduct is too remotely connected to the harm caused or closely connected to it. When proximity has been established, then liability ought to be imputed to the wrongdoer, provided public policy considerations based on the norms and values of our Constitution and justice also point to the reasonableness of imputing liability to the defendant".*

[117] In applying this approach to the facts in evidence it is clear there is no causal link between the low dose Covocort and Florinef and the plaintiff's alleged *sequelae*. It was not established that the steroid therapy predisposed the plaintiff to developing significant coronary artery disease. All that was left of the plaintiff's version in the end was Dr Promnitz's opinion that he found it difficult to account for her coronary surgery in 2015 after her coronary arteries were found to be normal in 2011, and the need for an *"individualised"* approach. Dr Promnitz also conceded that she did not have osteoporosis and hence the link between the steroid therapy prescribed by the defendant and any osteoporosis diagnosis was not proven. It was also not shown on a balance of probabilities that the degenerative changes in the plaintiff's spine were due to the steroid therapy. Dr Promnitz testified that the T12 fracture could not be disregarded but then simply dismissed it as a red herring. It was in addition not established that TB of the spine was a definitive diagnosis. The evidence of the defendant's experts established that the reactivation of latent TB could only occur when supraphysiological doses were administered, as such doses result in suppression of the immune system. Given that it became common cause that the plaintiff was not placed on an immuno-suppressant dose prescribed by the defendant, any proximate cause between the

[17] At [64].

steroid therapy the defendant prescribed (the Meticorton issue and her evidence on leftover medication indicates the plaintiff may have been on medication other than that prescribed by the defendant) and her alleged *sequelae* were not proven.

[118] It must follow then that the negligent act committed by the defendant in failing to perform any test to confirm his diagnosis of Addisons, as a result of which he placed the plaintiff on the low dose steroid therapy for a year, is not causally linked to the plaintiff's alleged *sequelae*. Insofar as it was submitted on behalf of the plaintiff that the defendant failed to point to another cause of the medical consequences, it is not for the defendant to do so but for the plaintiff, as a matter of logic and reasoning, to prove factual and/or legal causation. The high dosage theory contended for by the plaintiff was proven to be a fallacy and the concession from the plaintiff at the end of the trial was fatal to the plaintiff's case. In any event, having considered the evidence on each of the alleged *sequelae*, it was clearly established that none of the *sequelae* were suffered while the plaintiff was on the steroid treatment and the evidence does not support a conclusion that *sequelae* suffered one to three years after steroid treatment ceased is causally linked to the treatment.

[119] The task of this court is to decide, at the conclusion of the trial, whether, on all the evidence and the probabilities and the inferences to be drawn, the plaintiff has discharged the onus of proof resting upon her on a preponderance of probabilities. Despite my immense sympathy for the plaintiff, I must find, for the reasons set out above, that she has not done so.

Order

[120] In the result, I make the following order:

The plaintiff's claim is dismissed with costs.

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U. BHOOLA

Acting Judge of the High Court of South Africa

Gauteng Local Division, Johannesburg

Appearances:

For the Plaintiff: Adv G. J. Strydom SC

For the Defendant: Adv L. Segeels - Ncube