IN THE HIGH COURT OF SOUTH AFRICA
(GAUTENG DIVISION, PRETORIA)
REPUBLIC OF SOUTH AFRICA

Case Number: **80670/2018**
(1) REPORTABLE: NO
(2) OF INTEREST TO OTHER JUDGES: NO
(3) REVISED: NO
DATE: 21 June 2023
SIGNATURE: **JANSE VAN NIEUWENHUIZEN J**

In the matter between:

JOHANNA MARTINA RAS	First Plaintiff
ADV BESTER N. O. obo JOHN JOSEPH CHINN	Second Plaintiff
and	
DR FRANCOIS GREEFF	First Defendant
DR F J JACOBS	Second Defendant
DR ERNA VAN VUUREN	Third Defendant
LIFE WILGERS HOSPITAL	Fourth Defendant

JUDGMENT

JANSE VAN NIEUWENHUIZEN J:

[1] This action emanates from surgical procedures that were performed on the second plaintiff at Wilgers hospital on 29 February 2016.

[2] The plaintiffs allege that the first to third defendants were negligent in the performance of their respective medical duties and that such negligence caused the second plaintiff to suffer a stroke with infraction to the right side of the brain.

[3] The trial only proceeded in respect of the merits of the claim.

Parties

[4] The first plaintiff, Ms Ras, sues in her capacity as the life partner of the second plaintiff for the damages she has suffered as a result of the surgical procedures performed on the second defendant.

[5] The second plaintiff, Mr Chinn, sues in his own capacity for the damages he has suffered as a result of the procedures.

[6] The first defendant, Dr Greeff, is sued in his capacity as a specialist surgeon who performed an inguinal hernia operation on the second plaintiff.

[7] The second defendant, Dr Jacobs, is sued in his capacity as a specialist urologist that performed a cystoscopy, pyelogram and transactional prostate biopsy ("biopsy") on the second defendant.

[8] The third defendant, Dr van Vuuren, is sued in her capacity as the specialist anaesthetist during the medical procedures. Dr van Vuuren passed away prior to the trial.

[9] The plaintiffs withdrew their claim against the fourth defendant, Life Wilgers hospital prior to the trial.

Common cause facts

[10] For purposes of the merits portion of the trial, the second plaintiff will hereinafter be referred to as "the plaintiff". On 21 January 2016 the plaintiff consulted with Dr Greeff in respect of the hernia operation and with Dr Jacobs in respect of the biopsy, that was to be performed on 29 February 2016.

[11] On 29 February 2019, the plaintiff was admitted at Wilgers hospital and the procedures were performed from approximately 8h00 to 9h30. Dr van Vuuren

consulted with the plaintiff in the pre-operative holding area prior to the procedures.

[12] The procedures were uneventful, and the plaintiff recuperated in a normal ward subsequent to the biopsy and the hernia operation. Both Dr Greeff and Dr Jacobs visited the plaintiff in the ward and were satisfied that the plaintiff's condition justified his discharge on the morning of 1 March 2016.

[13] Shortly after his discharge and whilst leaving the hospital premises, the plaintiff suffered a stroke and was immediately admitted to the Intensive Care Unit at the hospital.

Medical history

[14] In order to appreciate the alleged *nexus* between the medical procedures and the stroke suffered by the plaintiff, it is apposite to have regard to the plaintiff's medical history at the time of the procedures.

[15] The plaintiff was 69 years of age at the time and was classified as being obese. The plaintiff had hypertension, high cholesterol and suffered chronic venous statis in his lower legs. The plaintiff's surgical history indicates that he had a previous hip operation on 7 June 2006 during which a radiologist, Dr Maas, reported an enlarged heart and an unfolded aorta, possibly due to hypertension.

[16] The plaintiff took the following chronic medication for his medical conditions:

16.1 Pearinda, 8 mg;

16.2 Bayer Aspirin Cardio, 100 mg;

16.3 Simvacor, 20 mg;

16.4 Puresis 40 mg.

[17] At the time of admission, the plaintiff had two ulcers on his lower legs and his blood pressure reading was high, to wit 170/110 mmHg. A 'venous thrombo-embolism risk assessment document' was completed by the nursing staff

which assessment indicated that the plaintiff was an extremely high-risk candidate for developing a thrombose-embolism.

[18] Due to his high blood pressure reading, the plaintiff was given a Pearinda 8mg tablet (ACE inhibitor that reduces high blood pressure) before he was taken to the theatre.

Evidence

[19] Several witnesses testified on behalf of the parties, and I will refer to their evidence insofar as it is relevant to the determination of the disputes between the parties.

[20] Ms Ras testified and provided a general background in respect of the plaintiff's medical history as well as the circumstances pertaining to the procedures performed on the plaintiff. Her evidence was not seriously contested and confirmed to a large extent the common cause facts between the parties.

[21] In order to determine the cause of the plaintiff's stroke, the parties relied on the evidence of various experts. The evidence of the experts was presented to determine whether the stroke was caused by a post-operative embolic event or inter-operative hypotension.

[22] The evidence of the experts is based on medical notes taken prior to and inter-operative as well as examinations conducted by various medical specialists after the plaintiff suffered the stroke. The medical evidence is a matter of record and I do not propose to, safe were necessary, repeat same herein.

Embolic event

[23] Insofar as the embolic event is concerned, Professor Jacobson, a clinical haematologist and surgeon, testified on behalf of the plaintiffs and Dr Rosman, a neurologist, testified on behalf of the defendants.

[24] Professor Jacobson expressed the view that the hernia operation was a "major operation" and although the prostate biopsy in itself is considered a "minor operation", the combination of the two procedures is regarded as a

"major operation" in terms of the Venous Thromboembolism: Prophylactic and Therapeutic Practice Guideline published in 2013 ("the guideline"). Professor Jacobson was the primary author of the guideline.

[25] The guideline deals with the risk of deep-vein thrombosis (DVT) in medically ill patients. The guideline indicates that the prescription of prophylaxis to patients who are at risk of DVT prevents or at least reduces the development of venous thromboembolism (VTE).

[26] The distinction between minor and major operations determines whether prophylaxis should be prescribed and was the topic of much debate between the experts. Prophylaxis is, as a general rule and subject to patient-related risk factors, not prescribed for minor surgery.

[27] The patient-related risk factors applicable to the plaintiff were, according to Professor Jacobson, the plaintiff's age, obesity and underlying malignancies. It is not clear from Professor Johnson's evidence to which underlying malignities he referred.

[28] In respect of the cause of the embolic event, Professor Jacobson considers the most likely cause to be a paradoxical embolus via the patent foramen ovale (PFO). Professor Jacobson's aforesaid opinion is based on the absence of angiographic changes in the major arteries and the presence of the PFO.

[29] Dr Rosman opined that the stroke was caused by a cholesterol embolus.

[30] A PFO is an opening between the left and right atrium of the heart. The heart has four chambers, the top two are referred to as the atrium and pumps blood to the bottom part referred to as the ventricles. The ventricles pumps blood from either the right side of the heart to the lungs, or from the left side of the heart to the rest of the body. The pressure in the left side of the heart is normally higher than the pressure on the right side and the valve in the opening stays closed. If the pressure in the lungs increases, in this instance, according to Professor Jacobson, due to pulmonary hypertension, the pressure in the right side of the heart increases which could cause the valve to open.

[31] Once the valve opens it is possible for a clot to travel through the PFO. The clot travels to the brain, which causes the stroke. Professor Jacobson is of the view that a clot came from the plaintiff's leg or from his pelvis, went to his right atrium and then moved to the left atrium. Thereafter, it went to the left ventricle, into the aorta, from the aorta to the internal carotid and lastly to the right middle cerebral artery. I pause to mention, that the PFO was only discovered after the plaintiff had the stroke.

[32] Dr Rosman did not agree that a clot could have travelled through the PFO. Dr Rosman indicated that a medium-sized artery is considerably larger than the size of the PFO (the PFO is less than 2mm), and that a paradoxical embolus is unlikely as the thrombus could not pass through the PFO. Dr Rosman, furthermore, opined that, because the penetrating arteries which were involved in at least some of the lesions rose from the medium-sized arteries at 90 degrees and are small (100-220nm), it is very unusual to have an embolization into these vessels from a thrombus.

[33] Professor Jacobson opined that a stroke in patients that have a PFO and presents with hypertension is relatively common. Professor Jacobson stated that the finding that the plaintiff had pulmonary hypertension and that the stroke occurred post-operatively strengthens his view that the stroke was caused by paradoxical embolus via the PFO. It was pointed out to Professor Jacobson during cross-examination that the report indicating the pulmonary hypertension is dated 2023. Professor Jacobson responded that the stroke post-operatively combined with the finding in 2023 that the plaintiff suffered from pulmonary hypertension makes it is highly likely that the plaintiff already had pulmonary hypertension in 2016. In contrast to this evidence, Professor Jacobson and Dr Rosman agreed in a joint minute that there were no indications of pulmonary hypertension or lung problems at the time of the stroke in 2016.

[34] Professor Jacobson, however, testified that even without signs of pulmonary hypertension being present, a first clot close to the lungs will raise the pulmonary pressure, which opens the valve and allows a second clot to go across and to cause a stroke.

[35] It was put to Professor Jacobson that there was no evidence of a PFO, previous deep vein thrombosis or clots nor of a previous stroke prior to the performance of the medical procedures. In the result, there was no reason to prescribe prophylaxis. Professor Jacobson strongly disagreed and stated that other factors such as the plaintiff's age necessitated the prescription of prophylaxis.

[36] Professor Jacobson testified that a clot normally disengages when a patient gets active, which explains why the stroke only occurred after the plaintiff got up and started walking on the morning of his discharge.

[37] Professor Jacobson was of the opinion that it was the responsibility of both Dr Greeff and Dr van Vuuren to prescribe prophylaxis. According to Professor Jacobson, the biopsy performed by Dr de Jager would not have necessitated the prescription of prophylaxis. The failure to prescribe prophylaxis equates, in the opinion of Professor Jacobson, to substandard treatment.

[38] Insofar as Dr Rosman's opinion that the stroke was caused by a cholesterol embolus is concerned, Professor Jacobson stated that the absence of an ulcerated plaque on the angiogram of 1 March 2016, makes the probability of a cholesterol embolus highly unlikely.

[39] During his evidence in chief, Dr Rosman was referred to a finding in 2023 that the plaintiff had coronary arterial stenosis. Dr Rosman indicated that the aforesaid finding is evidence of more widespread arterial disease that has built up over a period of 30 to 40 years. The two major reasons for the disease are high cholesterol and high blood pressure and is indicative of a blood vessel disease somewhere else in the body.

[40] In support of his opinion that the stroke was caused by a cholesterol embolus, Dr Rosman testified that, although the event is not evident from the angiograph, an ulcerated plaque is very easily missed on an angiography, because the lesion is not raised. In Dr Rosman's view the probability is higher that the stroke was caused by a shower of platelet or a shower of cholesterol emboli from such plaque.

[41] Dr Rosman explained his view that the stroke was caused by a cholesterol embolus in more detail in the summary of his expert evidence, to wit:

> *"5.10 It is Dr Rosman's opinion that the most likely scenario would be an ulcerated plaque, probably in the region of the bifurcation of the carotid artery, although this could be more proximal or more distal to the bifurcation. An ulcerated plaque can easily be missed both on a Doppler examination as well as on an MR angiogram examination. If attention is only paid to stenotic lesions, then the ulceration will easily be overlooked. In a situation like this, the plaque breaks down and a shower of micro-emboli is released into the cerebral circulation, causing a number of small discrete strokes within the territory or territories of the involved arteries. In a situation like this the cerebral arteries will be normal, and the involved vessels are so small that they cannot be visualised on the MR angiogram."*

[42] Dr Rosman also stated that the aforesaid scenario explains the number of lesions demonstrated on the MRI scan and the radiological findings.

[43] Dr Rosman testified that there is nothing one can do to prevent a cholesterol stroke.

[44] Professor Jacobson disagreed with the notion that lesions will not be visible on an ultrasound and stated that the quality of ultrasounds has increased dramatically over the years.

[45] Professor Jacobson's opinion that a cholesterol embolus is not the likely cause for the stroke is based on an ultrasound performed on 2 March 2016 on the plaintiff, which shows that the embolus is clear. Professor Jacobson conceded that there is a possibility that a cholesterol embolus could still occur but stated that the more likely cause of the stroke is the presence of the PFO.

[46] During cross-examination Dr Rosman conceded that clots could form due to an enlarged heart and that it was preferable to refer a patient with an enlarged heart to a specialist physician.

Interoperative hypotension and postponement of the operation

[47] Professor Moshabi, a cardiac anaesthesiologist that testified on behalf of the plaintiff, was referred to the notes made by Dr van Vuuren prior to surgery and to the fact that Ms Ras informed Dr van Vuuren that the plaintiff's blood pressure was 170 in the ward. Professor Moshabi opined that the history of hypertension, enlarged heart, varicose veins and the blood pressure of 170 made the plaintiff a high-risk patient who should have been further investigated prior to the performance of any surgery.

[48] It was pointed out to Professor Moshabi that Dr van Vuuren wrote *"no signs of heart failure"* next to 'enlarged heart' and Professor Moshabi answered that it most probably indicates that Dr van Vuuren clinically examined the plaintiff. Professor Moshabi expressed the view that the surgery should have been postponed in order to obtain a proper diagnosis of the plaintiff's heart condition.

[49] It was pointed out to Professor Moshabi that the venous thromboembolism risk assessment indicating that the plaintiff is a high-risk patient was not brought to the attention of Dr van Vuuren. Professor Moshabi responded that it is safe practice to conduct such an assessment and that Dr van Vuuren should either have asked for the assessment or done it herself. The assessment would have enabled Dr Van Vuuren to prepare a plan to manage the anticoagulation pre- and post- operatively.

[50] Although Professor Moshabi is of the opinion that the surgery should have been postponed, she indicated that should a decision be taken to proceed with the surgery notwithstanding the risk factors, Dr van Vuuren should have managed the risks by prescribing anticoagulation medication.

[51] It appeared from the nurses' notes that Dr van Vuuren saw the plaintiff post-operatively in the ward at 10h45 and prescribed DF118 and Atarax. Professor Moshabi explained that Atarax is an antihistamine and DF118 is analgesic.

[52] In respect of intra-operative management of the plaintiff's blood pressure, Professor Moshabi was of the view that the arterial pressure was very low (hypotension) which results in poor cerebral perfusion. Under-perfused areas in the brain may cause direct tissue damage or clotting in the arteries that

could cause a stroke. This in turn causes declined cognitive functioning. Professor Moshabi could not find any evidence on the theatre notes kept by Dr van Vuuren that she gave a drug to maintain the diastolic blood pressure to defend the cerebral perfusion pressure. Professor Moshabi opined that this was unacceptable.

[53] Professor Moshabi testified that major surgery was surgery into a body cavity. Professor Moshabi deemed both the biopsy and the hernia repair operation when performed individually as minor surgery. The two together is, however according to her, major surgery.

[54] During cross-examination, Professor Moshabi was referred to the ward notes and it was put to her that the notes do not contain any evidence of a neurological fall out. Professor Moshabi agreed.

[55] I pause to mention, that Professor Jacobson and Dr Rosman agreed that a hypotensive event was most unlikely considering that the stroke was of sudden onset the day after the surgery.

[56] Professor Coetzee, a specialist anaesthesiologist, testified on behalf of the defendants. Professor Coetzee explained that a hypertensive patient has a narrowing of his/her blood vessels. This causes the increase of blood pressure in patients with hypertension when they get angry or anxious. As soon as the pressure decreases, hypertensive patients tend to present with a very low blood pressure. A single episode of severe low blood pressure or severe hypoxia is a relatively unimportant event. To cause brain injury, the low blood pressure needs to occur over a considerable amount of time.

[57] Professor Coetzee does not agree with Professor Moshabi's opinion that the plaintiff's blood pressure was low for an extended period and that this resulted in a brain injury. Professor Coetzee explained that once a brain cell is properly injured, it does not recover. It is therefore inconceivable, according to Professor Coetzee, that a patient who had hypotension during anaesthesia which injured the brain will function normally after waking up from anaesthesia. The plaintiff's functioning in the ward after surgery and the next morning on discharge does not indicate any abnormal functioning. The fact that the plaintiff functioned normally, therefore and according to Professor

Coetzee, dispels Professor Moshabi's opinion that the plaintiff suffered a brain injury during anaesthesia.

[58] Professor Coetzee was referred to the anaesthetic chart kept by Dr van Vuuren during the surgery and stated that the chart does not indicate any period of hypotension.

[59] Professor Coetzee did not agree that the plaintiff had heart failure pre-operatively. Professor Coetzee readily agreed that should the plaintiff have had heart failure the operation could not proceed. In support of his opinion, Professor Coetzee had regard to the two factors Professor Moshabi considered in concluding that the plaintiff might have had heart failure, to wit: his enlarged heart and swollen legs.

[60] Professor Coetzee considered the objective data contained in the anaesthetic chart and observed that the plaintiff's heartbeat was 80 beats per minute and his saturation was 94%. Professor Coetzee explained that a person that has heart failure cannot lie flat because their lungs are congested which means that the saturation will go down, one will be short of breath and will complain about lying flat.

[61] On a clinical examination one would hear palpitations in the lung basis. There is no indication on the anaesthetic chart of any such problems. The low heart rate is a further indication that the plaintiff did not have heart failure. The swelling of the legs was due to the varicose veins and is also not an indication of heart failure. Professor Coetzee added that the echocardiogram done on 1 March 2016 also confirms that the plaintiff did not have congestive heart failure.

[62] In the result, there was no reason to postpone the operation.

[63] The echocardiogram ("ECG"), according to Professor Coetzee, also dispels the notion of pulmonary hypertension as suggested by Professor Jacobson during his evidence. The right heart pathology on the echocardiogram does not show pulmonary artery hypertension. Professor Coetzee explained that the right side of the heart is the pump dealing with the pulmonary artery and if

there was pulmonary hypertension, it would have shown on the echocardiogram.

[64] The echocardiogram, furthermore, indicated that the plaintiff had mild aortic sclerosis, which indicates calcification of the aortic valve, with calcifications on the tri leaflet. According to Professor Coetzee, the calcifications can break loose and embolise to the brain. This is important because on the stroke information there seems to be embolization, which could be another cause for the stroke. The calcifications can break loose at any time and has nothing to do with the surgery.

[65] Professor Coetzee stated that a clot or embolus that is moving through a PFO would come from the deep venous system and not from superficial veins such as the varicose veins. According to Professor Coetzee, there is no suggestion in the evidence before court that there was a deep vein thrombosis.

[66] In respect of the question of whether the repair of a hernia is a major operation, Professor Coetzee explained that major surgery is performed when the surgeon enters body cavities or there is large extensive invasion of the body with lots of tissue damage and a long recovery period. Minor surgery is minimally evasive where the surgeon cuts the skin and a bit of subcutaneous tissue. The recovery period is short. Professor Coetzee indicated that he differs from Professor Jacobson that a hernia repair is major surgery. The plaintiff recovered overnight and was walking the next morning to the bathroom to shave.

[67] Professor Coetzee concluded his evidence in chief by stating that in his opinion the management of the plaintiff by Dr van Vuuren was not substandard.

[68] During cross-examination it was put to Professor Coetzee that Dr van Vuuren should, in view of the plaintiff's history of an enlarged heart have done an ECG. Professor Coetzee responded that the clinical examination done by Dr van Vuuren excluded heart failure, but to establish ischemia she would have had to do an ECG. Professor Coetzee conceded that there is no indication in the theatre records that an ECG was done.

[69] Dr Jacobs testified next and nothing much turned on his evidence. During cross-examination it was put to Dr Jacobs that either Dr Greeff or Dr van Vuuren should have made the decision to prescribe anti-coagulation medication, to which Dr Jacobs agreed.

[70] The last witness to testify was Dr Greeff. Dr Greeff testified that he saw the plaintiff on 21 January 2016 in his consulting rooms. The plaintiff had an inguinal hernia that needed to be repaired. Dr Greeff was referred to information provided by the plaintiff which indicated that the plaintiff had two previous operations, to wit, a hip replacement and retinal detachment. The medication was indicated as *"Disprin, Kuresys (?), Prexum and cholesterol medication".* At the time of the hip replacement in 2016, the plaintiff was diagnosed with an enlarged heart and an epidural was administered. The plaintiff had a chronic *"kuggie"* and problems with his nose.

[71] In respect of the notes made by Dr Greeff, he noted that the plaintiff had a previous hernia repair on his right side and residual hernia again. After a physical examination Dr Greeff confirmed that the plaintiff had "*residual hernia again, right."*. Dr Greeff testified that the only medication that bothered him was the disprin. The plaintiff discussed the possibility of an epidural and Dr Greeff indicated to the plaintiff that he would not prescribe an epidural because of the chances of bleeding when the needle is inserted.

[72] Dr Greeff explained that the other medication taken by the plaintiff was for high cholesterol and high blood pressure.

[73] It was put to Dr Greeff that he is criticised for not postponing the operation. Dr Greeff responded that there was nothing during the consultation that indicated that the operation should be postponed. Dr Greeff stated that a hernia repair is a minor operation and that he had performed a vast amount of such procedures over the course of his career.

[74] Insofar as the notes pertaining to the surgery pertains, Dr Greeff indicated a *"Standard Bassini repair with no bleeding, cauterised a few bleeders. Repair with nylon"*.

[75] Dr Greeff explained the operation entailed the following:

"A Bassini repair is for a hernia that is in the groin area and what you do is, you make a [indistinct] incision to go through the layers, then you get the external layer of the muscles on the abdominal wall, it is called the external [indistinct] which you then incise and right behind that is the spermatic cord and in the….., the spermatic cord is a cord that takes blood vessels and the seminal.., to the seminal vesicles of the testicle and the blood supply and that is where the hernia originates in that opening where it comes through the abdominal wall and it is a radiation of the internal lining of the abdominal wall that protrudes into that little defect and becomes bigger in time to a point where it allowed bowel to go into it and that is what, what we had here…"

[76] Upon a further question by Ms Munro, counsel for the defendants, Dr Greeff gave a more detailed explanation, to wit:

"Then you, you loosen the spermatic cord, there were a few adhesions because he had a previous operation but is not major, you just, …..finger or with…It is easy to do, you lift it up so that you can separate the, the hernia sack that comes from the opening, so you can loosen it, and from the spermatic cord and just tie it closed so it would go back into the abdominal cavity with the spermatic cord that intact still taking the blood and everything to the testicles. Then what we do is that where the [mechanical interruption] …comes in, you [mechanical interruption] tendon, that is the, the muscle layer on the upper part of the canal where the muscles join, it is called conjoint tendon and you stitch behind the spermatic cord to the inguinal ligament, that is the ligament in the groin. So that is how you obliterate the canal to prevent…...and sort of closes the, or makes the opening in the abdominal cavity where the spermatic cord comes through, makes it smaller so it has got to be so tight that you just can get a fingertip in there, not to strangulate it."

[77] Dr Greeff testified that one does not go into the abdominal cavity at all, and that the surgery takes approximately 20 minutes. Dr Greeff indicated that the

plaintiff's enlarged heart did not concern him because it is part of having hypertension.

[78] In respect of the prescription of a prophylactic, Dr Greeff stated that because it is a minor operation and the patient becomes mobile almost immediately after the surgery, he does not routinely prescribe a prophylactic. He only prescribes a prophylactic if there is a clear indication that it is necessary. In this instance there was no indication that the plaintiff had developed deep-vein thrombosis and therefore the prescription of a prophylactic was not necessary.

[79] During cross-examination, Dr Greeff stated that he did observe that the plaintiff had swollen legs and ulcers on the legs. It was put to Dr Greeff that Ms Ras testified that he gave an instruction that the disprin should be stopped. Dr Greeff strenuously denied this and added that disprin is, in any event, not going to prevent deep- vein thrombosis. Dr Greeff explained that disprin could increase the risk of bleeding, but that he was not concerned because it is open surgery and even if there is a bit of bleeding, he cauterises the bleeding.

[80] Dr Greeff testified that many of his patients are on disprin prior to the operation.

[81] It was put to Dr Greeff that the plaintiff was taking disprin for some cardiac reason. Dr Greeff stated that he did not know for which condition the plaintiff was taking disprin. Dr Greeff was reminded of his evidence in chief when he stated that the only medication that bothered him was the disprin. Dr Greeff could not provide a reasonable explanation for his evidence in chief in this respect.

[82] When it was put to Dr Greeff that it is strange that the plaintiff's swollen legs, ulcers on the legs, the taking of disprin and the enlarged heart did not bother him, Dr Greeff testified that it did concern him, but that the plaintiff was on sufficient treatment for a minor operation.

[83] It was pointed out to Dr Greeff that the plaintiff had risk factors, being his age and the fact that he was obese. Dr Greeff denied that the plaintiff was obese

and stated that he was merely overweight. Mr Joubert SC, counsel for the plaintiffs, pointed out that the plaintiff's BMI was 36 and that anything above 30 is considered obese. In respect of the enlarged heart being a risk factor, Dr Greeff testified that the plaintiff was on disprin to prevent blood clots in a heart that may not be functioning as well as it should.

[84] Mr Joubert put it to Dr Greeff that the factors he considered of little relevance are factors that should have been properly considered by the anaesthetist. Dr Greeff agreed and stated that the anaesthetist takes the final decision on whether the surgery will go ahead.

[85] Dr Greeff was referred to the guideline and he testified that he only had sight of the guideline shortly before trial. Dr Greeff, once again, reiterated that a hernia repair is a minor operation and that he does not prescribe prophylactics to his patients. It was put to Dr Greeff that the plaintiff had at least three of the patient-related risk factors mentioned in the guideline and therefore prophylactics should have been prescribed. Dr Greeff emphasised that the guideline refers to deep venous thrombosis and that the plaintiff did not present with such a condition.

Discussion

[86] The expert witnesses agree that the biopsy performed by Dr Jacobs did not contribute to or caused the stroke.

[87] In my view, the evidence of Professor Coetzee coupled with the joint opinion of Professor Jacobson and Dr Rosman, supports a finding that it is highly unlikely that the stroke was caused by a hypotensive injury suffered intra-operatively.

[88] The plaintiff did not present with any neurologic deficiencies after he recovered from anaesthesia and according to the ward records, he was alert, he was eating and wanted to shave the next morning prior to his discharge. In the result, I find that Dr van Vuuren's management of the plaintiff inter-operatively did not cause the stroke.

[89] The next question is therefore whether either Dr Greeff or Dr van Vuuren should have prescribed a prophylactic. Two reasons are advanced by the plaintiff's experts for the necessity to prescribe a prophylactic, to wit:

89.1 it was a major operation; and

89.2 the plaintiff had at least three patient-related risk factors.

[90] In the guideline, the following is stated in respect of procedure-related risk factors:

" *duration of the procedure*

* *degree of tissue damage (orthopaedic/trauma surgery carries a greater risk);*

* *degree of immobility following surgery*

* *nature of the surgical procedure (e.g. lower limb orthopaedic surgery, neurosurgery, etc.)."*

[91] In view of the evidence of Dr Greeff pertaining to the manner in which a hernia repair is performed, the degree of tissue damage and the length of immobility following surgery, I am of the view that a hernia repair is minor surgery. This finding corresponds with the evidence of Professor Moshabi and Professor Coetzee that a hernia operation is minor surgery.

[92] The only point of contention is whether the biopsy and hernia repair procedures performed one after the other qualifies the procedures as major surgery. If one has regard to the definition of major surgery provided by Professor Moshabi, to wit: major surgery is surgery into a body cavity, I fail to see the logic in considering two procedures that do not entail surgery into body cavities, as major surgery.

[93] In the result, I find that the surgery was minor.

[94] Insofar as the patient-related risk factors are concerned, I am satisfied that the plaintiff presented with two of the factors, to wit his age and the fact that he was obese. These factors indicate that a prophylactic should have been prescribed to the plaintiff insofar as the prevention of deep-vein thrombosis is concerned.

[95] The plaintiffs also aver that the surgery should have been postponed to properly investigate the plaintiff's underlying medical problems. Dr Rosman agreed that, in view of the plaintiff's enlarged heart, an examination by a specialist would have been preferrable.

[96] Professor Moshabi opined that the history of hypertension, enlarged heart, varicose veins and the blood pressure of 170 made the plaintiff a high-risk patient who should have been further investigated prior to the performance of any surgery. The opinion of Professor Moshabi seems reasonable in the circumstances.

[97] Notwithstanding the aforesaid concerns and clear indications that the plaintiff was a high-risk patient, Dr Greeff and Dr van Vuuren both neglected to prescribe prophylactics. Dr Greeff neglected to refer the plaintiff to a heart specialist and Dr van Vuuren neglected to, notwithstanding the risk factors, postpone the operation.

[98] Was their negligence the cause of the stroke suffered by the plaintiff, i.e., is there a causal link between their negligence and the stoke?

[99] To answer this question, one needs to determine which event, on the probabilities that emerge from the facts, caused the stroke. Was the stroke caused by a paradoxical embolus *via* the PFO as opined by Professor Jacobson or due to cholesterol embolus as suggested by Dr Rosman.

[100] Professor Johnson's initial opinion that a paradoxical embolus *via* the PFO caused the stroke, was dependent on pulmonary hypertension. The problem with this postulation is that Professor Jacobson and Dr Rosman agreed in their joint minute that there were no indications of pulmonary hypertension or lung problems at the time of the stroke in 2016. The evidence of Professor

Coetzee, who testified that the echocardiogram done on 1 March 2016 does not support the notion of pulmonary hypertension, supports their view.

[101] Professor Johnson's second postulation, which only emerged during cross-examination, presupposes that the paradoxical embolus via the PFO was caused by a first clot close to the lungs, which would have raised the pulmonary pressure and open the valve. Once the valve is open, a second clot could go across and cause the stroke. The second scenario was not canvassed during evidence in chief and only mentioned during cross-examination. Due to the limited evidence in respect of the second scenario, I am simply not in a position to properly consider the second scenario.

[102] Even if one, however, accepts that the second scenario is likely, a further problem, in my view is the size of the PFO. The size of the clot plays an important role in determining whether it was possible to pass through the PFO. Dr Rosman's evidence in this regard was not challenged and the suggestion that the clot was small enough to pass through the PFO, remains speculative.

[103] Dr Rosman's evidence of a cholesterol embolus fits in with the plaintiff's history of high cholesterol at the time of the surgery. The problem with Dr Rosman's opinion is the absence of an ulcerated plaque on the 1 March 2016 angiogram. Dr Rosman explained that an ulcerated plaque is easily missed on an angiography because the lesion is not raised. Professor Jacobson did not agree and stated that technology has improved to such an extent that an ulcerated plaque will be visible on an angiogram.

[104] In considering the different opinions and the facts underlying the opinions, I am unable to find which of the two events is the more likely cause of the stroke.

[105] On the evidence, the negligence of Dr Greeff and Dr van Vuuren could possibly have contributed to a paradoxical embolus *via* the PFO. Had the stroke, however, been caused by cholesterol embolus, Dr Rosman's evidence that neither Dr Greeff nor Dr van Vuuren could have done anything to prevent the stroke, stands uncontested.

Conclusion

[106] The stroke that Mr Chinn suffered on the 1st of March 2016 is most unfortunate and has had a devastating effect on his life and that of Ms Ras.

[107] The plaintiffs', however, bear the onus to prove that the negligent conduct of Dr Greeff and Dr van Vuuren caused, on a balance of probabilities, the stroke suffered by the plaintiff. In view of the finding that the probabilities are even, the plaintiffs have failed to proof their claim for delictual liability and their claim stands to be dismissed.

Costs

[108] No reasons have been advanced for a deviation from the normal cost order and costs will follow the cause.

ORDER

The following order is issued:

1. The plaintiffs' claim is dismissed with costs.

N. JANSE VAN NIEUWENHUIZEN
JUDGE OF THE HIGH COURT OF SOUTH AFRICA
GAUTENG DIVISION, PRETORIA

DATE HEARD:

13-17 & 20 March 2023

DATE DELIVERED:

21 June 2023

APPEARANCES

For the 1st and 2nd Plaintiff's: Advocate S. Joubert SC

Instructed by: Kriek Wassenaar & Venter Inc

For the 1st ,2nd and 3rd Defendants: Advocate W Munro

Instructed by: MacRobert Attorneys