IN THE HIGH COURT OF SOUTH AFRICA
WESTERN CAPE HIGH COURT, CAPE TOWN

Case No.: 5450/2005

In the matter between:

SMD TELECOMMUNICATIONS CC Plaintiff

and

MUTUAL AND FEDERAL INSURANCE COMPANY LIMITED Defendant

JUDGMENT: 18 May 2009

DAVIS J:

Introduction

[1] Plaintiff, a close corporation, entered into a contract of insurance with defendant in terms whereof defendant, inter alia, agreed to pay compensation to plaintiff in the event of the death of a member of its managerial staff through bodily injury caused solely by violent, accidental, external and visable means which injury, shall independently of any other cause be the sole cause of death.

[2] On 10 October 2002, Mr Dennis Compton-James ('the deceased'), who held a 35% member's interest in plaintiff was involved in a motor accident. At the time he was the chief executive officer of plaintiff. He subsequently died on the 18 May 2003, pursuant to which death, plaintiff claimed in terms of the contract of insurance.

[3] The quantum of plaintiff's claim in the amount of R 611 693.24 has been admitted and is no longer an issue in dispute. The case turned on a question of causation; that is whether the death of the deceased fell within the scope of the contract of insurance.

[4] In turn, that question is dependent upon the meaning and scope of the contract of insurance, the critical part of which is to be found in the following clause:

> "*Conditional upon the prior payment of premium by the insured and the receipt thereof by or on behalf of Mutual & Federal Insurance Company Limited (hereinafter called "the Company") the Company shall insure and agrees to indemnify the insured by payment or at its option by reinstatement or repair or to pay compensation in respect of the Defined Events as provided hereinafter in the Sections forming part of this Policy occurring during the Period of Insurance Limit of indemnity amount of Compensation or other amounts specified.*
>
> *These General Exceptions and Conditions shall apply in all respect to the Insurance granted by this Policy except as they may be varied by any specified Exception or Condition in any Section Specification or Annexure to any Section Endorsement which are to be read together and any word or expression to which a specific*

meaning has been given in any part thereof shall hear such meaning wherever it may appear.

This insurance contract is conditional upon and will come into effect only following payment of the Premium by the insured and the receipt thereof by or on behalf of the Company.

PERSONAL ACCIDENT (GROUP) SECTION

DEFINED EVENTS

The events insured by this Section are as follows

If any Occurrence described hereunder shall happen to the Insured person during the Period of Insurance and the insured Person shall thereby suffer any Result described hereunder the Company will pay to the Insured the Compensation specified hereunder.

OCCURRENCE

Bodily injury caused solely by violent accidental external and visible means which injury shall independently of any other cause be the sole cause of any of the Results.

RESULTS	*COMPENSATION*
A. Death	*A. A sum equal to the number of years remuneration or the Amount state in the Schedule*

[5] In order to ascertain the exact contours of the dispute between the parties, it is now necessary to set out a brief chronology of the medical history of the deceased.

Dennis Compton-James: a history of coronary problems?

[6] On 6 December 1999, the deceased first consulted Dr Joseph Tyrell, a cardiologist. Dr Tyrell determined that the deceased suffered from high blood pressure, a cystolic murmur and had previously been on treatment to lower his blood pressure. Dr Tyrell also noted that the deceased smoked cigars, consumed alcohol and that he presented clinical features of aortic stenosis, indications of a previous myocardial infraction together with a slightly leaking mitral valve and evidence of left ventricular hyptrophy.

[7] On the 19 December 2001, the deceased again consulted Dr Tyrell. It appeared that he had developed mild claudication, a narrowing of the arterial supply to the muscles of the legs. He also suffered from moderate aortic stenosis and there was some ECG evidence of cardier disease. He was again advised to stop smoking and drinking.

[8] On the 1 July 2002 the parties renewed the contract of insurance, which was known as 'asset care'.

[9] On the 10 October 2002, the deceased was involved in a motor accident in Bothasig, Cape Town in which he sustained serious injuries. He was admitted to Milnerton Medi Clinic for trauma care and treatment. His injuries included an intratrochanteric fracture of the right femur, a comminuted supra-condylar fracture of the femur, a comminuted fracture left of the patella and an injury to the right chest and abdomen.

[10] Pursuant thereto, on the 11 October 2002, he was taken to theatre at the Medi Clinic where Dr Sander performed an operation, in terms of which he carried out an open reduction and internal fixation of the right femur fracture and an open reduction and internal fixation of the left femur and a patellectomy of the left knee.

[11] On 25 October 2002, the deceased was transferred to the Panorama Rehabilitation Unit for after care. On 29 November 2002 it appeared that his left knee flared up and was painful. On the same day, 29 November 2002, Dr Sander carried out an arthroscopy and a wash out of turbid fluid of the left knee. Again he was taken back to the Panorama Rehabilitation Unit.

[12] On the 13 December 2002, he was discharged to his home. He walked with the aid of a walking frame and required the use of a wheelchair. While at home, it appeared that there was a wound on the left heel and

toe. A nurse was required to change the dressing. He was then referred to Dr Henk Lourens, a vascular surgeon, who, on 15 January 2003, carried out a femero-popliteal bypass of the leg. He was again discharged 23 January 2003. On 27 January 2003 a process of physiotherapy commenced at the deceased home, conducted by Ms Wanda van der Merwe.

[13] On 30 January 2003, he was again readmitted to Panorama Clinic as it appeared that he had taken a turn for the worse. He could now not get out of bed, his speech was slurred and he looked pale and drawn. Dr Rossouw, a general practitioner, was then consulted. Dr Rossouw referred the deceased, by way of a letter on the 5 February 2003, to Dr Neil Du Toit, a physician. After setting out the deceased's condition and his prescribed medication, he said; '*Ek mis iets hier ek hoop jy kan help'.* Dr Rossouw's description of the deceased's condition was that he was anaemic, he stammered, his CRP was 226 which had reduced rapidly to 195, after five days of anti-biotic treatment. His sodium level was 120 CRP stands for C – reactive protein, is found in blood in small quantities and the normal level is less than 5mg per litre. It is an important inflammatory marker.

[14] After a series of test, Dr Du Toit determined that the deceased had developed a methacillen resistant stafaureus infection. He placed the

deceased on vancomissien and rifampesien, which are two powerful anti-biotics.

[15] On 18 February 2003 an orthopaedic surgeon, Dr Lategan, removed hardware from the right hip because it was infected.

[16] The deceased consulted Dr Du Toit again on 7 March 2003. Dr du Toit indicated that the deceased's condition had improved. He could walk with a frame, his CRP had dropped to 40, his sodium level has improved and his blood pressure had now been controlled because he was taking norvasc.

[17] On the 14 April 2003, Dr Du Toit noted that the left knee was now sore that the right hip was improved, the CRP level had improved to 30 his haemoglobin had improved from 9.1 to 11.4, but the sodium level was still low at 114 mol per liter.

[18] According to Ms van der Merwe, physiotherapy ceased on the 15 April 2003 as no further improvement appeared to be possible. The deceased at this stage walked approximately 20 meters with a zimmer frame he attended work on a daily basis but only for a couple of hours. He determinedly continued to attend office for a few hours a day on a relatively regular basis until the 18 May 2003 when he was found dead.

The essence of the dispute

[19] Plaintiff's case therefore is that his death, on 18 May 2003, flowed from the collision of 10 October 2002 or as stated in the pleadings, the death 'resulted directly and solely from the bodily injuries sustained in the said motor vehicle accident (which) occurred independently of all other causes and was the sole cause of death.'

[20] Defendant did not dispute that the deceased died on 18 May 2003, probably of a myocardial infarction precipitated by a plaque rupture. Defendant contended however that plaintiff was obliged to prove that the injuries suffered by the deceased in the accident, independently of any other cause, was the sole cause of the deceased death in order to justify its claim in term of terms of the policy.

Plaintiff's Case

[21] The plaintiff relied, to a considerable extent on the evidence of Dr Tyrell who had been the cardiologist whom the deceased had consulted on a regular basis. Dr Tyrell postulated the cause of death as myocardial infarction caused by coronary thrombosis. He informed the court:

> *"So the mechanism of myocardial infarction, which is the – in the heart, is this process of plaque rupture, clot formation and so-called coronary thrombosis. And that leads to the condition where the*

heart muscle is deprived of its blood supply and becomes damaged or in fact infracted."

In this case, Dr Tyrell explained that the patient had suffered major trauma after a number of operations and still had chronic inflammation only days before his death:

"I think it is highly likely that he died of myocardial infarct, precipitated by, caused by those multiple traumas that he – to which he had been exposed."

[22] Dr Tyrell then continued:

"... its well recognised that's (sic) various natural and unnatural triggers can lead to myocardial infarction...

And then various unnatural triggers ay also provoke myocardial infarction and the way they do that is because they cause release of pro-inflammatory chemicals and they tend to make blood more likely to clot. So they increase the likelihood that these plaques, which are dormant in the coronary and other vascular circulation, may rupture ... So both by increasing the probability of plaque rupture and by increasing the tendency of blood to clot, they create an environment in which myocardial infarction is more likely to occur".

In essence, Dr Tyrell took the view that, while the deceased could hardly be described as a fit or a completely well person prior to the accident.

However at the time immediately before the collision, his peripheral vascular disease may have been chronic but it was stable. At that stage his condition in no way affected his day to day functioning. In short, according to Dr Tyrell, a stable chronic condition had been rendered lethal by the overall impact of the various insults to the deceased body which had taken place as a result of the accident and the medical treatment that the accident had so necessitated.

[23] Dr Tyrell was asked whether he was surprised that the deceased had died some seven months after the accident. To this question he replied:

> *"Not really. For example, My Lord, post-operative heart attack or myocardial infarction is a common problem in everyday practice, and my colleagues see many patients, who were sent specifically to try and assess what the risk of patients who have a myocardial infarction or heart attack after some none-cardiac surgery. One tries to assess the risk and then one of course tries to minimise the risk of this sort of thing happening, but it is a common and well recognised and feared complication of surgery. ... And then the same applies also to conditions of chronic inflammation, for example bronchiectasis, which is a chronic inflammation of the lungs, or rheumatoid arthritis which is a chronic joint conditions. So these are chronically – chronic inflammatory conditions which also increase the risk of myocardial infarction."*

He then told the court:

> *"We mentioned also that myocardial infarction is more common after surgical procedure and after trauma and also that myocardial infarction is more common with infections. So all these factors were in operation in the case of the deceased. So my opinion as to the cause of death is that he suffered a myocardial infarction which was a consequence of all the trauma and surgery and infection to which he had been subjected as a result of the accident."*

[24] Under cross examination by Mr Dane, who appeared on behalf of the defendant, Dr Tyrell was asked in particular as to how surgery and trauma was instrumental in causing the plaque to rupture which then precipitated the myocardial infarction:

> "*Now what is the process whereby surgery causes a plaque to rupture? Because you haven't explained that. You've made the allegation, the bald allegation that surgery and trauma was instrumental in causing this myocardial infarction and I'd like to know how does surgery cause this plaque rupture?"*

Dr Tyrell replied:

> "*My Lord, we can deal with this in two ways. One is the epidemiological evidence, and that is that after none-cardiac surgery the risk of myocardial infarction is increased. Obviously it*

depends on what kind of surgery it is, and it depends on the patient to who you are referring. But the epidemiological evidence is that the risk of myocardial infarction is increased after surgery of various kinds. The other way to look at it, if one wants a mechanism, is to deal with this problem of the process of plaque rupture, and the mechanism is simply that some plaques are vulnerable, and some plaques are stable and what surgery tends to do and what trauma tend to do, it tends to induce and inflammatory state, and that is the body's response to trauma or to surgery. That initiates the healing process. So, for example white blood cells. Heart rate increases. Blood pressure goes up. The platelets in the blood become more active. They are more likely to initiate proteins in the blood, the levels of clotting proteins in the blood increased. The surgery and trauma induce a pro-inflammatory state and they induce a pro-coagulant state. These two combined increase the possibility of plaque rupture and thrombosis…".

[25] Plaintiff also called Mr Mower to testify on its behalf. His evidence was of limited importance. He was employed by plaintiff from 1974 as the company driver. He became friendly with the deceased before the accident. He assisted the deceased both before and after the accident with his purchasers. He testified that prior to and subsequent to the accident deceased drank between 1 and 2 glasses of wine per day and

smoked at least one cigar. After the deceased was discharged from hospital, he could only move with the Zimmer frame and a wheelchair. Mr Mower took the deceased both to work and home. Although there was considerable debate about the extent to which the deceased employed a treadmill for exercise which was located at his home, (and hence was relatively active prior to the accident) Mr Mower never saw him actually use the treadmill.

[26] Ms van der Merwe, a physiotherapist visited the deceased home on 27 May 2003 at which stage he was mobilised in a wheelchair and employed the Zimmer frame. When she saw him again on 19 March 2003 his left knee was particularly stiff. On 26 March 2003 she reported that his left knee could not extend and that he complained that he was dizzy he stretched his quads. On 31 March 2003 she reported that his condition was slightly improved. The quad lag was 30 degrees. He walked in an improved fashion from the couch to the kitchen. She saw him again on 4 April 2003, on 07 April 2003, on 22 April 2003 at which stage he walked approximately 20 meters with the Zimmer frame. The quad lag was still 30 degrees. She decided that, at this stage, as there was no improvement in his condition, she should not continue and thus ceased treatment.

[27] After the plaintiff had closed its case it sought by way of an application to reopen its case in order to lead the testimony of a vascular surgeon Dr

Jeffery. The basis upon which Dr Jeffery's opinion was sought was due to a suggestion from defendant that the infected heal and toe could have been the result of the natural progression of the deceased's peripheral vascular disease (PVD) and was thus unrelated to the injuries sustained in the collision. Before Dr Jeffery were called, defendant indicated that it no longer persisted with any basis of causation on the grounds of the heel and toe; that is that the injury to the heel and toe may have interrupted the chain of causation. Plaintiff however continued with its presentation of the evidence of Dr Jeffery because it contended that this was material to the general theory advanced by Dr Tyrell regarding plaque rupture. The question as to whether this part of Dr Jeffery's evidence is relevant and indeed admissible can only be determined after an examination of the evidence presented by defendant.

Defendant's case

[28] In essence, defendant's case was that there was no empirical basis to support the theory of Dr Tyrell, that surgery, trauma and inflammation precipitated rapture of the plaque resulting in the myocardial infarction seven months after the initial accident and three months after the last procedure to which the deceased had been subjected on 18 February 2003.

[29] The thrust of the evidence of Dr Neil du Toit and Dr Thomas Mabin, both of whom were called by the defendant, was to contend that neither clinical practice nor the available medical literature supported the approach adopted by Dr Tyrell.

[30] I turn first to deal with evidence of Dr du Toit. Dr du Toit who practiced as a specialist physician at the Panorama Medi Clinic until June 2008, testified that on 6 February 2003 he was approached by Dr Rossouw to assist in the treatment of the deceased's condition. Subsequent to a popliteal bypass on 15 January 2003, the deceased's condition had suddenly deteriorated. Pursuant thereto, Dr du Toit conducted numerous investigations. His emphasis indicated that the deceased presented with a low sodium, a CRP of 196, a haemoglobin count of 9.1 and a methacillen resistant staf auraeus infection cultivated from the left heal. An ECG test indicated that the deceased suffered from aortic stenosis. After the operation by Dr Lategan, in which the infected hardware had been removed from the right on 18 February 2003, some form of improvement in the deceased condition took place.

[31] When Dr du Toit saw him on 7 March 2003, the CRP had declined significantly to 40. There remained a course of treatment of three weeks of anti biotics. The sodium level had also improved. Further improvements took place when Dr du Toit saw the deceased again on 14

April 2003. The left knee at this stage appeared to be warm and therefore the issue arose to whether it had been infected. According to Dr du Toit, he did not know from the deceased of a previous myocardial infarction. He only become aware of this condition some months later. For this reason and, given reports by Dr Murray and Dr Bodenstein which had suggested severe aortic stenosis, Dr du Toit testified that, when he completed the death certificate, he had written that aortic stenosis had been the cause of death. It was only after obtaining the further information that he accepted that the deceased's death was as a result of myocardial infarction.

[32] Dr du Toit resisted Dr Tyrell's evidence that the deceased's death had been precipitated by plaque rupture which was the result of surgery trauma and inflammation. He also rejected the suggestion by Dr Tyrell that inflammation is the same in all conditions and that Dr Tyrell's conclusion was supported by the condition of rheumatoid arthritis which had been linked to acute myocardial infarction.

[33] In this connection the thrust of Dr du Toit's evidence was reflected in the following passage:

> *"But this concept that inflammation is a stereotype response to injury, infection or to immune diseases, is not true, because they are unique. They have unique patterns of, I'll call this term again*

cytokines, because, really, that drives the inflammatory response. They are different in every disease process. If it was the same, the results would have been all the same. That is why you find such unique differences in various diseases which is coupled with inflammation. His references to certain auto immune diseases like rheumatoid arthritis and I will throw in there for the argument's sake, systemic lupus erythemotosus SLE, are the classic examples of auto immune illnesses where your body's own immune system attack some of your own tissue. And it leads to a specific form of inflammation of which one of those manifestations is accelerated blood vessel deterioration."

It was for this reason that Dr du Toit testified that an inflammatory process in an artery which leads to vascular diseases cannot be compared to the inflammation suffered by the disease.

[34] A critical part of Dr du Toit evidence in dealing, with what he referred to Dr Tyrell hypothesis, was that, if this hypothesis was true, Dr du Toit would have expected the deceased to have suffered a myocardial infarction when he was at his worst condition, that is in February 2003 when his CRP was 226, when he was suffering from infection, when there was a reduced haemoglobin and a low sodium level and where he was at his least mobile. According to Dr du Toit, three months thereafter, when there were clear improvements in all of these markers, when he was

mobile, albeit using the Zimmer frame, and where there was evidence of a slow recovery, it was highly unlikely that the inflammation would have precipitated a rupture of the plaque.

[35] Dr du Toit's evidence was supported by Dr van der Spuy, a specialist surgeon and orthopedic surgeon who have been head of National Trauma Research Programme from 1987 until August 2000. In essence his evidence was captured in the following passage of evidence. Having been asked to comment on the proposition that surgery and/or trauma leads to a myocardial infarction he answered:

> *"My Lord, I will do this as briefly as I can. In a long career in trauma surgery, I have never seen trauma and surgery, and, for that matter, inflammation, per se leading to myocardial infarction in a patient who had not been a particular ischemic heart disease risk. I cannot say that it cannot possibly occur. What I can say, is, in quite a number of thousand of operations of people not having manifest risk factors, I have not seen a myocardial infarction. The second part of my reply would be, on 3, 4, 5, I can't remember exactly but I have had postoperative deaths from myocardial infarction, but those usually occur intra-operatively. When I say post-operatively, I mean peri-operatively. They either occur during surgery, or within the first few days. I am not aware of – certainly not in my experience, I am not aware though in close colleagues, or*

in my unit, when I was at Groote Schuur, of a much delayed myocardial infarction arising from trauma and surgery."

[36] He was then asked about a comment offered in his expert report in which he had stated that the injuries from the accident could have been no more than 'a minor contributory factor to his death. His reply was as follows:

"To what extent it had been contributory, I don't think any of us, in the absence of a post-mortem, we would know. But I cannot see that seven months down the line, and three months after the last surgery, and when the sepsis was declining, was being controlled, with no ischemic heart disease episode having occurred earlier when the first operations were done, later on with the arthroscopy of the knee, later on even in – correct me – January or February ... when it was manifest staphylococcal sepsis and septicaemia haven't occurred, I can't see, in my own analysis, how this now could have occurred belatedly three months down the line, when, as far as one can see, other than for a complaint about his left knee, why this would now, for the first time, in a patient who had been at serious risk, ischemic heart disease risk, why it would now suddenly, when things were better than at several other stages, why it would now cause a myocardial infarction. What I say, is, if it played a part, it must have been a minor contributory part."

[37] The third key witness, who testified on behalf of the defendant, was Dr Mabin. Dr Mabin testified that, with the deceased's profile, he had a 50% chance of surviving for five years; that is with his medical profile, only 50% of patients presenting with such symptoms would be alive after a five year period.

[38] Differing from Dr Tyrell, he distinguished various types of inflammation and differentiated between systemic inflammation and arterial inflammation. Thus he informed the court:

> *"The inflammatory process in rheumatoid arthritis is a completely different inflammatory process to what was dealt with in this particular patient."*

In short, Dr Mabin contended that there was no medical basis by which to use the condition of rheumatoid arthritis to justify a conclusion that the deceased's myocardial infarction had been increased and the fact that he succumbed seven months later, was not a major obstacle to accepting Dr Tyrell's explanation.

Mr Gamble then asked Dr Mabin:

> *"But on the one hand you've got that, you've got surgery or trauma, and on the other – and the other end of the spectrum, of the continuum, you have rheumatoid arthritis, chronic condition. Why is there no room for something in between, such as we have in the present case?"*

Dr Mabin replied:

> *"I don't see any room. We are talking about two completely different issues they are completely different diseases. To try and connect the two, I think is – it is very difficult to do in a logical term."*

Dr Mabin furhter testified that, in an acute phase of surgery, trauma and acute infection, there is a high incidence of myocardial infarction in that the body reacts by releasing pro thrombotic particles in the blood such as platelets. The body then tries to seal the wound. If there is plaque that is already active, this inflamed plaque will be at high risk of causing a thrombosis. Accordingly, there is a high incidence of acute myocardial infarction within the first two or three days after the surgical procedure. For this reason patients are admitted after such major surgery to an intensive care unit. Thus, he testified:

> "*I find it very difficult to believe that temporal relationship existed, such a far distance temporarily, from that last surgical event. I think the man was at high risk of having a myocardial infarction at any time*".

[39] Dr Mabin's evidence concentrated, in part, on an analysis of a series of articles which had been handed up into evidence in an attempt to support or debunk the hypothesis of Dr Tyrell. Much was made of this literature by both counsel. Dr Mabin's attitude to the available academic literature was as follows:

"I don't believe there is anything in these articles that support that contention. I have done my best to try and search literature, looking for evidence for that, and is just not forthcoming. Most of these articles were publishes several years ago, and there is nothing in the current literature, or current conferences that I have been to, and I have specifically looked at this issue, because this intrigued me. And there is just no valid evidence connecting a systemic non-specific inflammatory process to progression of vascular disease. Vascular diseases inflammation progresses as a separate entity. What the actual provocative factors are, we still don't know, but it certainly cannot, at this stage, be claimed that there is any direct evidence between a generalised systemic inflammation and inflammation of the arteries themselves."

In summary, Dr Mabin said of Dr Tyrell's evidence:

"*I think it's an attractive hypothesis but there is no data to support it."*

Amplifying upon this statement, he said:

"If it accepted the myocardial infarction was the result of plaque rupture, my contention is that there is no hard, scientific evidence that the plaque rupture would be precipitated this late after an incident, in a man who is, if anything, getting better."

In his view:

"From the literature survey I did there was no cause or relationship that I could find between a chronic inflammatory or infected state and an increase risk of myocardial infarction."

Evaluation

[40] In order to evaluate the evidence, it is necessary to examine the legal basis of plaintiff's claim. To recapitulate: the occurrence required to trigger an obligation on the part of the defendant to pay was 'bodily injury caused solely by violent accidental external and visible means which injury shall independently of any other cause be the sole cause of any of the result'. The contract provided for a series of exceptions which included the following:

"The general exception shall not apply to this section and this insurance shall apply to any occurrence consequent upon .....any pre-existing physical defect or infirmity."

As Mr Gamble, who appeared on behalf of the plaintiff, correctly submitted defendant did not plead the exception cause and therefore was not entitled to rely upon it. Had it done so it would have borne the onus of proof. See Agiakatsikas NO v Rotterdam Insurance Limited 1959 (4) SA 726 (C) at 77-78.

[41] The occurrence clause included two critical components:

1. Bodily injury caused solely by violent accidental external and visible means;
2. That bodily injury was 'the sole cause of death' and occurred 'independently of any other cause'

[42] It did not appear to be disputed that the deceased had sustained 'bodily injury' which was 'caused by violent accidental external and visible means having sustained extensive and serious injuries in the motor vehicle accident which occurred on 10 October 2002. Neither was there a dispute that the bodily injuries were caused solely by the accident.

[43] The key question for determination was whether the death of the deceased on 18 May 2003 was not only causally linked to these injuries but, independently of any other cause was the sole cause of his death. The complication in this case it that the deceased suffered from PVD at the time of the accident as the factual matrix as set out earlier in this judgment has made abundantly clear.

[44] Mr Gamble conceded wisely that the deceased was more pre-disposed to a myocardial infarction than an ordinary healthy person by virtue of his PVD as well as his general life style, as emerged from the evidence of Mr Mower.

[45] Mr Gamble relied heavily on the decision in Concord Insurance Company Limited v Oelofsen NO 1992 (4) SA 669 (A). For this reason before proceeding with an analysis of the evidence, it is necessary to examine this case in some detail. The facts were as follows. The insured was involved in a motor vehicle accident he assumed to have suffered no physical injury pursuant to the collision. A few hours thereafter, he died of a heart attack. For approximately two years before the accident he had suffered from a coronary disease known as 'triple artery atherosclerosis', that is a narrowing of the arteries. Medical evidence revealed that the actual mechanism of the heart attack was 'myocardial ischaemia due to a constriction in the area where the arteries have already been narrowed by sclerosis'. The medical experts disagreed as to the probable cause of this constriction. Whereas the expert on behalf of the insurer denied that occurred naturally in the progression of the disease, the plaintiff's expert maintained that 'the constriction was probably caused by a biological process whereby, due to the shock of the accident the sympathetic nervous system released chemicals substances into the blood.

[46] The claim was based on a following policy clause:

> *"If during any period of insurance an insured person sustains bodily injury which, independently of any other cause, results in the death, or permanent disablement of the insured person, the company will*

pay to the insured person or his estate the compensation state below."

The trial court found in favour of the insured and the matter proceeded on appeal. The insurer contended that there was no bodily injury as envisaged in the policy. This argument was rejected. The question then arose as to the causation in relation to the provision in the insurance policy, which I have already cited.

[47] Hefer JA (as he then was) said the following, in a passage which given its significance to the present dispute, requires citing in full:

"*In the context of the cover clause it may similarly be said that the bodily injury constituted the proximate cause of death, but in view of the words 'independently of any other cause' this is plainly not enough. If the insured's pre-existing condition was a contributory "cause" within the intended meaning of this word, Concord must be absolved.*

The emphasis of the intended meaning of the word "cause" serves to indicate what I conceive to be the correct approach to the problem at hand. Legal causation is not a logical concept and the law does not ascribe causative effect to every logical sine qua non *(cf International Shipping Co (Pty) Ltd v Bentley 1990 (1) SA 680 A at 700 E – I). Basically this is so because complex legal questions*

– often involving considerations of policy – cannot be solved satisfactorily by a general positive application of the simple logical proposition that a particular fact or state of affairs cannot be regarded as the cause of another unless the former is a sine quo non *for the latter. Such questions usually arise where several factors concurrently or successively contribute to a single result and it is necessary to decide whether any particular one of them is to be regarded legally as a cause. In criminal law and the law of* delict *legal policy may provide an answer but in a contractual context, where policy considerations usually do not enter the enquiry, effect must be given to the parties' own perception of causality lest a result be imposed upon them which they did not intend. What must accordingly be decided in the present case is whether the parties, by referring in the cover clause to 'any other cause' of an insured's death or disablement, intended to include his infirmity.*

That they could not possibly have attached meaning to the word "cause" which would embrace every conceivable sine qua non *is clear. Mr Trengove conceded that such a construction would make a mockery of the agreement. The enquiry must accordingly proceed on the basis that the word was used in a restricted sense. But there is no express indication of the extent of the contemplated limitation nor can its ambit be gauged by way of implication from*

the other terms. Why then should we favour an interpretation which would specifically include the insured's infirmity? To this question Mr Trengove supplied no answer. Not a word is said in the policy about the insured's sate of health either at the time of his application for insurance not at any time thereafter and one is left with the firm impression that it is something which simply did not concern the parties. Because it obviously affects the risk, an insured's state of health is commonly known to be of decisive importance to any life insurer. Indeed provision in a life policy whereby the application for insurance, containing the applicant's answers to certain questions regarding his medical history and the state of his health, is incorporated in the policy. Moreover, it is not unusual for accident policies to contain specific provisions excluding liability for the insured's death or disablement arising from or traceable to any physical defect or infirmity existing prior to the accident. (Such a provision appeared for example in the policy before the court in Jason v Batten (1930) Limited [1969] 1 LLR 281 (QB) – a case on which Mr Trengove relied but which is clearly distinguishable – and in a number of other cases). Bearing this in mind, the significance of the absence from the present policy of any reference whatsoever to the insured's state of health is patent. It is difficult to accept, to say the least, that the parties meant to express in the simple words "independently of any other cause" an intention

similar in effect to the once evinced by the elaborate provisions in the policies in cases like Jason v Batten.

In any event we must apply the rule in the Latin phrase verba fortius accipiuntur contra proferentem. *In French Hairdressing Saloons Limited v National Employers Mutual General Insurance Association Limited 1931 AD 60 at 65 it was said that* "it is an accepted principle in interpreting insurance contracts that it is the duty of the insurer to make it clear what particular risks he wishes to exclude".

Accordingly, as Kotze J A said in Norwich Union Fire Insurance Society Limited v South African Toilet Requisite Co Limited 1924 AD 212 at 222,

"It is laid down that, as insurance is a contract of indemnity, it is to be construed reasonably and fairly to that end. Hence conditions and provisos will be strictly construed against the insurers because they have for their object the limitation of the scope and purpose of the contract." (*See also Pereira v Marine and Trade Insurance Co Limited 1975 (4) SA 745 (A) at 752 F – 753 A and cases cited there; Price and Another v Incorporated General Insurances Limited 1983 (1) SA 311 A at 315 G).*

Although the independent cause provision does not appear in the present policy under the conditions or exceptions, its object is

plainly to limit the liability and the same principle applies. On a strict and reasonable interpretation of the policy I have no doubt that an insured's ill health at the time of the accident was not intended to constitute another "cause" of his death or disablement. I say this for the reason stated earlier and bearing in mind the frailty of the human body and the great variety of physical conditions that may develop at any time and may aggravate the effect of an injury or in one way or another contribute to death or disablement. Reasonably speaking, I find it inconceivable that the parties intended to exclude liability in every case in which such a condition occurs. The Trial Court's conclusion that the insured's death was caused by bodily injury independently of any other cause is accordingly correct." at 673 H – 675 D

[48] In his judgment, Hefer JA distinguished the facts of Concord from an English decision in Jason v Batten (1930) Limited [1969] 1 LLR 281 (QB) which the court had found against an insured, again on facts similar to the present dispute primarily because the policy in that case included the following provision:

> "*(3) that the bodily injury result in the disablement and is, independently of all other causes, the exclusive, direct and immediate cause of the disablement."*

Fisher J then said:

"This provision is expressed to be 'subject as hereinafter provided' and the policy goes on to provide that no benefit shall be payable in respect of disablement directly or indirectly caused by, arising or resulting from or traceable to a number of tings, including (2) (iii)(b): Any physical defect or infirmity which existed prior to an accident". at 289

Fisher J concluded:

"*However, it seems to me quite clear that the claim must fail for other reasons. The bodily injury was not "independently of all other causes the exclusive ... cause of the disablement". There were two concurrent causes, the pre-existing arterial disease and the formation of the clot. These two causes were independent of each other, and the thrombosis would not have occurred on June 21, 1965, unless both had operated. If there were any doubt about this, it would be removed by the words of the later provision. It seems to me plain that the disablement was ... directly or indirectly caused by or arising or resulting from or traceable to-*

(b) Any physical defect or infirmity which existed prior to an accident, namely, arterial disease." at 290.

Mr Gamble emphasized the far more restrictive nature of the relevant clause in Jason's case and the reliance in Jason on the exemption clause in order to contend that Concord was the more applicable approach to the present dispute.

[49] Further assistance for this indicated approach with regard to causation can be found in the case of Incorporated General Insurances Ltd v Shooter t/a Shooter's Fisheries 1987 (1) SA 842 (A) at 862 CD where Galgut AJA said:

> "*No difficulty arises when one cause only has to be considered. The difficulty arises when there are two or more possible causes. In such a case the proximate or actual cause or effective cause (it maters not which term is used) must be ascertained, and that is a factual issue. I cannot put it better than was done by Ivamy at 255, where it is said that an earlier event may be the dominant cause in producing the damage or loss; it may be the causa sine qua non but the issue is, is it the causa causans? Ivamy at the above page, Arnould at 773 and Gordon and Getz at 383 all stress that the rule to be applied is causa proxima non remota spectatur*".

[50] Given a distinction which is evident in the policy between a provision for cover and the exemption clause, it would appear that the intention of the parties was to cater for a pre-existing physical defect or infirmity by way of an exception; or as Hefer JA put in Concord:

> "*an insured's ill health at the time of the accident was not intended to constitute another 'cause'.*"

Furthermore, had defendant called for a medical examination of the deceased under the policy, it would then have been able to take further steps, such as the insertion of an additional clause to restrict the cover afforded under the policy.

[51] There is no suggestion in this case that the dispute was predicated on non disclosure. Therefore, the battle ground between the parties was based on the following narrow issue: Notwithstanding any medical history or precondition, absent the accident, would the insured then have taken place in the manner in which it did.

[52] Within this context, it is possible to return to the expert evidence which was critical to the resolution of the dispute. There was considerable debate about the three levels of evidence employed by medical science, levels A, B, C (sometimes referred to as 1, 2 and 3) Level A evidence is essentially scientific proof that a well held hypothesis is correct. According to Dr Tyrell, level C evidence includes case reports, expert opinions, committee reports and non randomised cohort studies used as a guide for treatment and prevention without being able to be used to prognosticate patient outcomes.

[53] There was a considerable debate between the experts as to whether there was a viable prospect of being able to present level A evidence in this

case, owing to the non availability of a sufficiently large sample group. Dr Tyrell testified that such a study would require the assessment of persons with PVD were then subject to the type of multiple insults that the deceased endured. Dr Tyrell further testified that, in his view, level C evidence would be more congruent with proof on a balance of probabilities, as opposed to level A evidence which arguably equated to proof beyond a reasonable doubt.

[54] By contrast, Mr Dane submitted that Dr Tyrell's hypothesis, unsupported by the academic literature or other expert evidence was no more than a possibility rather than a probability. Mr Dane therefore submitted that a far more probable explanation was that the deceased's death was precipitated by a rupture of the plaque as a result of his severe vascular disease. Dr Mabin's uncontested evidence was that within five years of the finding of PVD at the second visit which the deceased made to Dr Tyrell, he had a mortality rate of 50%; that is within five years of this finding, there was a 50% chance of him being dead.

[55] Mr Dane submitted that, on the totality of the evidence, that probability had materialised. As the deceased had a silent myocardial infarction prior to 1999 and further, having due regard to Dr Tyrell's evidence that plaque can rupture and embolise continually this was the probable explanation of what happened on 18 May 2003. The deceased was in a far more

precarious situation when he consulted Dr du Toit on 6 February 2003 then when Dr Lategan last saw him on 16 April 2003. It was at this time that his indicators were at their worst. On 18 February 2003 he been taken to theater where the infected hardware in his right hip had been removed. At this point, he was far more ill that at the time immediately leading up to his death. Had the accident been the cause of death, it was to have been expected that he would have died at that stage. But he did not die the nor did he have a heart attack during this perilous period which, on the basis of Dr Mabin and Dr du Toit's evidence, would have been far more likely. Therefore, no explanation had been preferred within the context of plaintiff's version as to why the myocardial infarction suffered by the deceased could be ascribed to the trauma resulting from the surgery and subsequent inflammation when this had occurred some three months prior to his death and his indicators had suggested an improvement during this period. In summary defendant contended thus:
If Dr Tyrell' hypothesis had merit, it is far more probable that it would have happened earlier or within the acute phase on 18 February 2003 than on 18 May 2003. According to Mr Dane, everything pointed to the myocardial infarction on 18 May 2003 not being related at all to the surgery, trauma and inflammation.

[56] The court is this confronted with two versions: The probabilities are that the deceased would not have died in May 2003, had it not been for the

accident and the complications which resulted therefrom *versus* the complications which resulted from the accident were at their worst in February 2003, yet the deceased survived. When he finally succumbed in May 2003, the probabilities are that his death was as a result of his precarious medical condition which existed independently of the events surrounding the accident.

Expert Testimony: its application

[57] Much was made by Mr Dane, and experts Dr du Toit and Dr Mabin, that, were the court to uphold the Tyrell hypothesis, it would run counter to prevailing medical evidence. In this regard, it is helpful to refer to the approach of the US Supreme Court in dealing with expert scientific evidence. In the seminal case of Daubert v Merrell Dow Pharmaceuticals Inc 509 US 579 at 485 Blackmun J said:

> "*It is true that open debate is an essential part of both legal and scientific analyses. Yet there are important differences between the quest for truth in the courtroom and the quest for truth I the laboratory. Scientific conclusions are subject to perpetual revision. Law, on the other hand, must resolve disputes finally and quickly. The scientific project is advanced by broad and wide-ranging consideration of a multitude of hypotheses, for those that are incorrect will eventually be shown to be so, and that in itself is an advance. Conjectures that are probably wrong are of little use,*

however, in the project of reaching a quick, final, and binding legal judgment – often of great consequence – about a particular set of events in the past. We recognise that in practice, a gatekeeping role for the judge, no matter how flexible, inevitably on occasion will prevent the jury from learning of authentic insights and innovations. That, nevertheless, is the balance that is struck by Rules of Evidence designed not for the exhaustive search for cosmic understanding but for the particularised resolution of legal disputes."

[58] However at footnote 13 to Justice Blackmun's judgment, the following appears:

"*This is not to say that judicial interpretation, as opposed to adjudicative factfinding, does not share basic characteristics of the scientific endeavor: 'The work of a judge is in one case enduring and in another ephemeral ... In the endless process of testing and retesting, there is a constant rejection of the dross and a constant retention of whatever is pure and sound and fine.' B Cardozo, the Nature of the Judicial Process 178 – 179 (1921).*"

[59] This dictum from Daubert needs, of course, to be seen within the context of the US Supreme Court's development of four factors for admissibility of expert evidence,

1. The process of arriving at some propositional hypothesis about a set or group of events which can be tested.
2. The known or potential rate of error associated with using the particular scientific techniques adopted in (1).
3. Peer review and publication; that is whether the theory or technique has been subjected to peer review and publication; and
4. General acceptance, that is a measurement of the extent to which the experts' methods produce information that qualifies the scientific knowledge's recognized within the relevant scientific community.

[60] A strict application of Daubert may, in my view, favour defendant in that Dr Tyrell's hypothesis has not been rigorously tested and the scientific literature presented by plaintiff only provides a general basis for the acceptance of this hypothesis.

[61] But consider firstly the facts of Daubert:

> The plaintiffs, two children and their parents, sued Merell Dow in California state court alleging that the children's limb defects were caused by their mothers' use of Bendectin. Bendectin was a drug which was manufactured by Merell Dow and prescribed routinely to alleviate "morning sickness" during pregnancy. Science could not

identify a precise causal mechanism linking Bendectin to birth defects. Statistical evidence derived from epidemiological studies were offered in lieu of more "concrete" evidence, but it was vulnerable to manipulation and produced contradictory conclusions. Merrell Dow claimed that because there was no epidemiological evidence linking Benedectin to birth defects, then the plaintiffs would not be able to offer any kind of "good" (i.e. "generally accepted") scientific evidence. Daubert countered this by offering a reanalysis of the same epidemiological data as well as several other types of evidence. In doing so, Daubert implicitly argued that the more liberal admissibility standard applied.

In siding with plaintiff in Daubert, the court accepted much of the argument put up by a group of eminent scientists (the "Boembergen group') who placed an *amicus* brief on before the court, as is evident from the following passage in the judgment:

> "*That scientific truth is neither absolute nor constant. They invoked Sir Karl Popper's The Logic of Scientific Discovery to make their point that "[a]n hypothesis can be falsified or disproved but cannot, ultimately, be proven true because knowledge is always incomplete... Thus, scientific statements or theories are never final and are always subject to revision or rejection. In other words, while tests may corroborate a hypothesis, they do not confer upon it the stamp of absolute truth.*

Further, the Bloembergen group distinguished between the meaning of truth in science and the meaning of truth in law. They emphasised that truth in science is extremely "mutable", but truth in law must "become final and immutable in a relatively short time. Most importantly, Bloembergen argued that truth has different functional definitions in science and law. Consequently, there are important differences "between the purposes of science in the laboratory and scientific testimony in court. The nature and breadth of scientific evidence used for establishing legal truth in court is vastly different from that which is necessary to pursue scientific truth in the laboratory. Moreover, the Bloembergen group emphasised the differences between science and law which circumstances their relationship. They differ in purpose, with science constructing "descriptive general theories based on particular data" and law consisting of "a system of normative general rules that are individualised to apply to particular cases. The two disciplines also differ in a temporal sense. Science deals with predictive notions, while legal processes deal with unique, unrepeatable past events."

It should also be remembered that Daubert was concerned with the question of whether certain expert witness's testimony should be accepted. In this case, Dr Tyrell most certainly qualified to give expert

opinion evidence on the matter. He was the consulting cardiologist. He had a better understanding of the deceased condition than any of the other experts, particularly by Dr Mabin who had never seen the deceased and who was reduced to presenting an analysis of the available literature and Dr van der Spuy, who appeared at times to cross the line between an expert and an enthusiastic advocate on behalf of defendant. Mr Dane criticised Dr Tyrell as follows:

> "*The inference to be drawn is that he was trying to further the Plaintiff's case and lost his sense of objectivity in so doing.*"

That description far better suited Drs' Du Toit and van der Spuy. Furthermore, it was not as if Dr Tyrell's evidence was presented without any justificatory support from the academic literature, albeit not in terms of a controlled study.

[62] In any event, this court is required to adopt a legal approach to the question of causation in its evaluation of the evidence. A decision in favour of plaintiff can hardly be construed as establishing a scientific precedent. Viewed legally, it is significant that Dr Tyrell was insistent that there was no reason to adopt the view, absent the events of October 2002, that the deceased would have died in May 2003. For this conclusion he was by far the best placed expert. Although it is correct that the deceased's indicators improved during the period after February 2003, there was no suggestion that the deceased was a healthy man or had

recovered to any extent to that level of health which he enjoyed prior to October 2002.

[63] In my view therefore, the evidence read as a whole justifies the conclusion that it was more probable that the thrombosis occurred because of the repeated insults to the deceased body, flowing from the accident, the body's natural responses thereto and the trauma associated with all the medical treatment which flowed directly from the accident as opposed to a natural progression of his PVD. The only defendant expert who was qualified to provide clear testimony on behalf of the defendant and the particular medical condition of the deceased (and then only after the accident) was Dr du Toit. He had the benefit of consulting the deceased. Dr du Toit was certainly a less impressive witness than Dr Tyrell. He was aggressive, he was far less thoughtful in attempting to assist the court than was Dr Tyrell. As his expert 'summary' was no more than a summation of correspondence to defendant's legal team, his expert evidence in court often shifted to the exigencies of the moment rather than being an amplification of the report. Furthermore, in his letter to defendant's attorney on 16 November 2004 there appears to be some significant ambivalence about the key issue of causation. The relevant passage reads thus:

> *"Ek moet by vorige opinie bly. In die lig van Mnr Keith Compton-James se vorige kardinale geskeidenis hou ek by my besluit oor die*

finale oorsaak van dood soos gestel in sy doodsertifikaat. Hy is waarskynlik dood as gevolg van 'n kardinale incident. Ek kan nie sê dat die motor voertuig ongeluk, die enigste oorsaak van sy dood is nie. Alhoewel die motor ongleuk groot inpak op sy algemene gesondheid gehad het, met verskeie operasies en herhaalde infeksies.

Sy sieklike sterfte egter, byna 7 maande na die motor ongeluk, is steeds van kardinale oorsaak."

Under cross examination he did not appear to be very convincing as to the explanation of the contents of this letter. He argued that the referred sentence in the letter could be explained by his use of a negative in the relevant passage yet he was forced to concede the following:

"At the end of the day you are quoted as saying here that the motor accident had a considerable impact upon his overall health. --- That is true."

Once the evidence of Dr Tyrell is accepted, read with the benefit of Dr du Toit's equivocal letter, this dispute falls clearly within the framework of both the facts and approach adopted in Concord Insurance, supra.

Costs

[64] On 30 October 2007, the matter was postponed for trial after defendant abandoned its special plea, and its objection to plaintiff's intention to amend the particulars of claim aimed at annexing the correct pages of the applicable contract of insurance. In terms of the agreed order of that date the wasted costs of the postponement including all costs postponed from the filing and withdrawal of defendant special pleas stood over for later determination.

[65] It is clear that these costs should be awarded to plaintiff. In this connection, I agree with Mr Gamble that the approach adopted by defendant was unnecessarily vexatious and accordingly, the award of these wasted costs should be on the attorney and client scale.

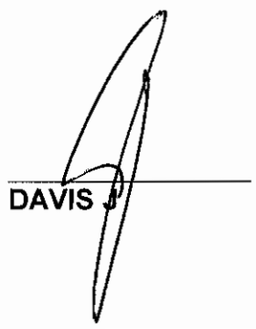
[66] I am also satisfied that defendant should be ordered to pay the plaintiff's costs of trial including the qualifying expenses of Dr Tyrell. I have not dealt with Dr Jeffery's evidence which, in my view, given the concession made by defendant was unnecessary and certainly did not appear to add much up to plaintiff's case.

[67] For these reasons therefore, the following order is made:

1. Defendant is ordered to pay the agreed sum of R 611 693 .24 together with VAT.
2. Interest on this sum shall commence on 1 June 2003 at the

prescribed rate determined in terms of Act 55 of 1975;

3. Defendant is ordered to pay plaintiff's costs, including the costs of the postponement of 30 October 2007 at an attorney and client scale and the cost of trial including the qualifying expenses of Dr Tyrell.



DAVIS J