

SOUTH AFRICA / JUDGMENT

**Mutual & Federal Insurance Company Ltd
v SMD Telecommunications CC (486/09)
[2010] ZASCA 133; 2011 (1) SA 94 (SCA) ;
[2011] 2 All SA 34 (SCA) (1 October 2010)
[2010] ZASCA 133**

COURT

Supreme Court of Appeal

BEFORE

Navsa, Cloete, Mhlantla, Shongwe, Tshiqi

CASE / FILE NUMBER

486/09

Source: saflii.org · Research copy typeset by Esheria. Not court-issued or certified.

THE SUPREME COURT OF APPEAL OF SOUTH AFRICA

JUDGMENT

Case no: 486/09

In the matter between:

MUTUAL & FEDERAL INSURANCE COMPANY LTDAppellant

and

SMD TELECOMMUNICATIONS CCRespondent

Neutral citation: Mutual & Federal Insurance Company Ltd v SMD Telecommunications CC
(486/09) [2010] ZASCA 133 (1 October 2010)

Coram: NAVSA, CLOETE, MHLANTLA, SHONGWE and TSHIQI JJA

Heard: 19 August 2010

Delivered: 1 October 2010

Summary: Insurance contract – Occurrence Clause covering bodily injury caused ‘solely’ and
‘independently’ of any other cause – Exception Clause excluding any pre-existing death or
infirmity.

ORDER

On appeal from: Western Cape High Court (Cape Town) (Davis J sitting as court of first instance):

The appeal is dismissed with costs.

TSHIQI JA (Navsa, Cloete, Mhlantla, Shongwe JJA concurring):

[1] On 1 July 2002, the appellant and the respondent concluded a contract of insurance. The clauses relevant to the present dispute

are the Occurrence and Exception Clauses. In terms of the Occurrence Clause, the appellant undertook to compensate the respondent in the event of disability or death of one of its managerial staff, occurring as a result of ‘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’ (which included death). The Exception Clause specifically excluded cover for ‘any occurrence consequent upon any pre-existing physical defect or infirmity’.

[2] On 10 October 2002 Mr Keith Compton-James (‘the deceased’), who held the position of Chief Executive Officer of the respondent, sustained orthopaedic injuries in a motor vehicle collision. On 18 May 2003, seven months later, he died. His death was precipitated by a plaque rupture which caused a myocardial infarction (a heart attack). It was not in dispute that the deceased fell within the ambit of managerial staff as contemplated in the contract.

[3] Before the collision, the deceased had a history of coronary problems. In 1999 and 2001 he was diagnosed by a cardiologist, Dr Tyrell, to be suffering from high blood pressure and presented with a cystolic murmur. He had also presented clinical features of aortic stenosis (narrowing of the aortic valve of the heart) together with a slightly leaking mitral valve and evidence of left ventricular hypertrophy (the heart muscle was thicker than normal). He was advised to change his lifestyle and to stop smoking and drinking. An electrocardiogram and echogram indicated he had had a previous myocardial infarction. On both occasions Dr Tyrell also noted that the deceased had developed mild claudication (a narrowing of the arterial supply to the muscles of the legs).

[4] On the day of the collision he was admitted to the Milnerton Medi Clinic for trauma care and treatment. On 11 October 2002, he was taken to theatre where a doctor carried out an open reduction and internal fixation of the right femoral fracture and an open reduction and internal fixation of the distal left femur and a patellectomy of the left knee were performed. He remained there until 25 October 2002 when he was transferred to the Panorama Rehabilitation Unit for after care. On 29 November 2002, it appeared that his left knee had flared up and was painful. His doctor carried out an arthroscopy and a wash out of turbid fluid of the left knee. He was taken back to the Panorama Rehabilitation Unit.

[5] On 13 December 2002, he was discharged to his home. He walked with the aid of a walking frame and at times required the use of a wheelchair. While at home, it appeared that there was a wound on the left heel and toe. He utilised the services of a nurse to change the dressings. It became necessary for him to be referred to a vascular surgeon, who, on 15 January 2003, carried out a femero-popliteal bypass of the leg. He was discharged on 23

January 2003. On 27 January 2003 he commenced at his home with a process of physiotherapy.

- [6] On 30 January 2003 (more than three months later) the deceased was readmitted to Panorama Rehabilitation Unit because it appeared

that his health condition had deteriorated. He could not get out of bed, his speech was slurred and he looked pale and drawn. Dr Rossouw, a general practitioner, was consulted. On 5 February 2003, Dr Rossouw referred him to Dr Du Toit, a physician. After setting out the deceased's condition and his prescribed medication, he commented: 'Ek mis iets hier ek hoop jy kan help'. In the referral, Dr Rossouw also described the deceased's condition as anaemic, that he stammered, and that his C-Reactive Protein (CRP) which had been 226 had reduced rapidly to 195, after five days of anti-biotic treatment. His sodium level was 120. (CRP is found in blood in small quantities and the normal level is less than five mg per litre. It is an important measurement of inflammation in the body.) On 6 February 2003 he was readmitted at Panorama Hospital for treatment of sepsis that developed in relation to a plate in the right hip by Dr Lategan.

- [7] After a series of tests, Dr Du Toit determined that the deceased had developed a methicillin resistant staphylococcal infection. He placed the deceased on vancomissien and rifampesien, which are two powerful antibiotics. On 18 February 2003 an orthopaedic surgeon, Dr Lategan, removed the hardware from the right hip because it was infected. The deceased consulted Dr Du Toit again on 7 March 2003. Dr Du Toit noted that the deceased's condition had improved. He could walk with a frame, his CRP had dropped to 40, his sodium level had improved and his blood pressure was under control because he was taking a prescription drug called norvasc. On 14 April 2003, on a further visit to Dr Du Toit, he noted that the left knee was sore, that the right hip had improved, that the CRP level had improved to 30, and that his haemoglobin had improved from 9.1 to 11.4, but the sodium level was still low at 114 ml per litre. On 15 April 2003, his physiotherapist discontinued her sessions because she believed that no further improvement appeared to be possible. At that stage he walked a maximum of approximately 20 metres with a zimmer frame. He attended work on a daily basis, but only for a few hours. He continued to attend work on a relatively regular basis until 18 May 2003 when he was found dead, at home.

- [8] During the deceased's lifetime, the Insurance Company paid out an amount to the respondent in respect of temporary disablement.

Subsequent to his death, the respondent lodged a claim arising out of the contract of insurance. The appellant repudiated the claim and denied that the death fell within the scope of the contract of insurance. The respondent instituted a claim in the Western Cape High Court. That court (Davis J) held in favour of the respondent. This appeal is directed, with leave of the court below, against that finding.

- [9] Put simply, having regard to the Occurrence Clause referred to in paragraph 1, the dispute between the parties in the court below and before us can be telescoped as follows: The appellant contends that the deceased was at high-risk before the collision, that he had

already suffered a heart attack in the past and had maintained a lifestyle that was not conducive to good health and that the plaque rupture had occurred naturally because of these factors and cannot be said to be due to the collision as required by the Occurrence Clause. This, they submitted was especially so given the lapse of approximately seven months from the time of the collision to the time of his death. The respondent, on the other hand, contends that the serious injuries that were directly due to the collision were such that they developed complications, including infection, and that this led to a marked deterioration in his health and ultimately caused his death within the terms of the Occurrence Clause.

[10] The divergence of views set out in the preceding paragraph, were predicated on the views of the parties' respective experts. The court below favoured the views of Dr Tyrell, the deceased's cardiologist, against the views of Dr Mabin, a cardiologist who testified in support of the appellants' case.

[11] At the outset it is necessary to consider the use of the words 'bodily injury' in the Occurrence Clause. There is a long line of cases in which it has been recognised that even if the loss is not felt as the immediate result of the peril insured against, but occurs after a succession of other causes, the peril remains the proximate cause of the loss, as long as there

is no break in the chain of causation. One such case is *International Shipping Co (Pty) Ltd v Bentley*.¹ In that case it was stated:

"The enquiry as to factual causation is generally conducted by applying the so called "but-for" test, which is designed to determine whether a postulated cause can be identified as a *causa sine qua non* of the loss in question. In order to apply this test one must make a hypothetical enquiry as to what probably would have happened but for the wrongful conduct of the defendant. This enquiry may involve the mental elimination of the wrongful conduct and the substitution of a hypothetical course of lawful conduct and the posing of the question as to whether upon such an hypothesis plaintiff's loss would have ensued or not. If it would in any event have ensued, then the wrongful conduct was not a cause of the plaintiff's loss; aliter, if it would not so have ensued. If the wrongful act is shown in this way not to be a *causa sine qua non* of the loss suffered, then no legal liability can arise. On the other hand, demonstration that the wrongful act was a *causa sine qua non* of the loss does not necessarily result in legal liability. The second enquiry then arises, viz whether the wrongful act is linked

sufficiently closely or directly to the loss for legal liability to ensue or whether, as it is said, the loss is too remote. This is basically a juridical problem in the solution of which considerations of policy may play a part. This is sometimes called "legal causation".

[12] Returning to the question of which of the two views referred to in paragraph 10 above should win the day, it is necessary to consider the evidence of Drs Tyrell and Mabin.

[13] According to Dr Tyrell, a person suffering from chronic vascular disease has by definition widespread areas of plaque in arteries

throughout the body (atherosclerosis or, in common parlance, hardening of the arteries). The most common arteries to be affected

by plaque are those supplying the lower limbs, heart and brain. Coronary heart disease is a condition characterized by long periods

of chronicity and stability, and also at times by periods of instability and acute events, such as myocardial infarction. In cases

of myocardial infarction the event is caused by rupture of plaque in a coronary artery, which then leads to the formation of thrombus

(‘coronary thrombosis’), which in turn causes occlusion of the artery and leads successively to myocardial ischaemia (lack of blood supply to the heart muscle which also implies shortage of oxygen) and infarction, and – in some cases – to ventricular fibrillation and death. Dr Tyrell’s opinion was that a major event such as the accident in which the deceased was seriously injured has a number of significant patho-physiological consequences over and above the injuries which a patient may sustain. These include the development of pro-inflammatory and pro-thrombotic states in the body.

[14] Dr Tyrell referred to the fact that the deceased had evidence of persistent infection and inflammation in the months before his death, and to the fact that the deceased’s CRP level was significantly above normal over a protracted period. In February 2003 the CRP level was measured as 97mg/l, in March it was 38mg/l and in April (about four weeks before his death) it was 30mg/l. As I have said, the normal CRP level is less than five mg/l. He regarded it as significant that the deceased not only suffered major trauma resulting from the accident but also underwent multiple surgical procedures and developed infection. According to Dr Tyrell, each of these consequences of the accident (‘triggers’), by causing activation of pro-inflammatory and pro-thrombotic pathways of themselves considerably increased the risk of plaque rupture. He stated that in a vulnerable person such as the deceased (one more pre-disposed to a heart attack than the average person), the concurrence of these events greatly increased the risk of a heart attack.

[15] Dr Tyrell concluded that trauma, surgery, infection and inflammation are well-known and potent triggers for plaque rupture,

and thus heart attack – and that it is beyond doubt that these triggers were directly attributable to the accident because they rendered the chronic stable condition of the deceased to become acute, unstable and lethal. He stated that although the death of the deceased occurred some seven months after the motor vehicle accident, he regarded the above multiple occurrences as various triggers of his cause of death. He found it significant that as recently as four weeks before the deceased’s death there were still signs of a systemic inflammatory state (as shown by persistently high CRP levels) and concluded that it was probable that this condition persisted up

to the time of death. Dr Tyrell accordingly held the opinion that the deceased's death was a late or delayed consequence of the accident and that the deceased would not have died in May 2003 if it had not been for the motor vehicle accident.

[16] Dr Mabin's opinion, on the other hand, was that the pre-existing health complications of the deceased put him at a high

risk of an ischaemic heart attack at any time. In support of this he referred to the fact that the deceased was already suffering from established atherosclerotic vascular disease at the time of the accident. Further factors, so he stated, were the fact that in October 2002 his disease had already manifested as a previous heart attack and the fact that the deceased also had disease of the arteries in his legs. Dr Mabin stated that the fact that the deceased died seven months after the accident at a time when he was improving as indicated by his clinical state and reduction in his CRP level, also indicates that the CRP and inflammation reaction were unlikely to be directly responsible for his death. He regarded it as an important factor that the deceased survived in February when his condition of inflammation was acute, but died four months later when it was described as chronic rather than acute – thus showing signs of improvement. Dr Mabin stated that the release of pro-inflammatory chemicals that make the blood more likely to clot, occurred in the acute phase after surgery and/or trauma, either during the event or a day or two thereafter – hence his opinion that death was more likely to have occurred during February, during the acute phase and around the period of the last surgical intervention. He concluded that the timing of death showed that the deceased was most likely to have died from pre-existing ischaemic heart disease, and not from the consequences of the accident. According to him the deceased had sufficient evidence of serious pre-existing cardiac disease to have resulted in the deceased dying at any time, irrespective of the accident – and that it cannot therefore be said that the deceased would not have died but for the accident.

[17] The approach that is helpful in resolving the divergence between Dr Tyrell and Dr Mabin is the approach adopted in *Michael & another v Linksfield Park Clinic (Pty) Ltd & another 2* where the court held:

'In the course of the evidence counsel often asked the experts whether they thought this or that conduct was reasonable or unreasonable, or even negligent. The learned Judge was not misled by this into abdicating his decision-making duty. Nor, we are sure, did counsel intend that that should happen...' (para 34).

'That being so, what is required in the evaluation of such evidence is to determine whether and to what extent their opinions advanced are founded on logical reasoning. That is the thrust of the decision of the House of Lords in the medical negligence case of *Bolitho v City and Hackney Health Authority* [1997] UKHL 46; [1998] AC 232 (HL (E)). With the relevant dicta in the speech of Lord Browne-Wilkinson we respectfully agree. Summarised, they are to the following effect...'

'The court is not bound to absolve a defendant from liability for allegedly negligent medical treatment or diagnosis just because

evidence of expert opinion, albeit genuinely held, is that the treatment or diagnosis in issue accorded with sound medical practice.

The Court must be satisfied that such opinion has a logical basis, in other words that the expert has considered comparative risks and benefits and has reached “a defensible conclusion” (at 241G – 242B).

If a body of professional opinion overlooks an obvious risk which could have been guarded against it will not be reasonable, even if almost universally held. (at 242H).’ (para 36-38).

‘...This essential difference between the scientific and the judicial measure of proof was aptly highlighted by the House of Lords in the Scottish case of *Dingley v The Chief Constable, Strathclyde Police* 200 SC (HL) 77 and the warning given at 89D-E that “(o)ne cannot entirely discount the risk that by immersing himself in every detail and by looking deeply into the minds of the experts, a Judge may be seduced into a position where he applies to the expert evidence the standards which the expert himself will apply to the question whether a particular thesis has been proved or disproved – instead of assessing, as a Judge must do, where the balance of probabilities lies on a review of the whole of the evidence”.’ (para 40 F-H).

[18] Before I review the evidence and consider the probabilities in the present enquiry, it is helpful to consider the decision of this court in *Concord Insurance Co Ltd v Oelofsen* NO.3. In that case the executrix in a deceased estate instituted action for payment under a personal accident policy issued by Concord. The policy had an Occurrence Clause which provided that Concord would provide cover if during the period of insurance the insured sustained ‘bodily injury which independently of any other cause results in the death’ of the insured. The Occurrence Clause is similar to the one in issue except that the parties in Concord did not incorporate ‘sole cause’ in their policy. Nothing turns on this omission because there is no difference between

‘independently of any other cause’ and ‘be the sole cause’. The use of both terms in the clause is tautologous. The deceased was subsequently involved in a motor vehicle accident from which he emerged apparently unscathed. Within a few hours after the accident, he suffered a heart attack. He was rushed to hospital where he was treated, but ventricular fibrillation developed and he died later the same evening. The deceased had been suffering from a coronary disease known as triple artery atherosclerosis for a period of about two years before the accident and his condition was such that he might have experienced a heart attack at any time.

[19] The medical experts in Concord were agreed that the immediate cause of the fibrillation was myocardial ischaemia due to a constriction in the area where the arteries had already been narrowed by sclerosis; they differed on the probable cause of the constriction. Concord’s witness, Dr Baskind, was of the view that it had occurred naturally in the progression of the disease and was unrelated to the accident. The executrix, on behalf of the estate, called a Professor Simson who testified that the constriction was probably caused by a biological process whereby, due to the shock of the accident, the sympathetic nervous system released chemical substances into the blood. The High Court preferred Professor Simson’s view. On

his evidence it was held 'that the ischaemia of the heart was caused by the stress of the collision coupled with the pre-existing disease which together resulted in a vasoconstriction with the resultant chain of events described above'.

[20] In Concord, this court in considering the question of causation in connection with the cover clause, found it significant that the insurance contract did not contain an Exception Clause specifically excluding pre-existing infirmities. The court reasoned as follows:

'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...' (at 673 G-H).

'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 a meaning to the word "cause" which would embrace every conceivable sine qua non is clear. Mr Trengrove 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 extend 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 Trengrove supplied no answer. Not a word is said in the policy about the insured's state of health either at the time of his application for insurance nor 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 one can almost describe it as standard practice for insurers to insert a provision in a life policy whereby the application for insurance, containing the applicant's answers to searching 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) Ltd [1969] 1 L1 LR 281 (QB) – a case on which Mr Trengrove 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 one evinced by the elaborate provisions in the policies in cases like *Jason v Batten*.' 4 (at 674 B-H).

[21] The essence of the distinction the court was referring to in the terms of the contracts in Concord and Jason v Batten is that, in the latter case, the policy contained an Exception Clause that specifically excluded the pre-existing health condition of the insured. If the intention of the parties is to exclude pre-existing infirmities, this should be unequivocally stated in the insurance contract. The Exception Clause in Jason v Batten did so. It provided that

‘No benefit shall be payable under this [p]olicy in respect of [d]eath, [i]njury or [d]isablement directly or indirectly caused by or arising or resulting from or traceable to –

.....

(iii)

(a)

(b) Any physical defect or infirmity which existed prior to an accident’.

[22] In Jason v Batten the court was impressed by the fact that Mr Jason had not suffered from the thrombosis despite his matrimonial problems and the stressful life he was leading. Because of these factors, the court found in favour of Mr Jason, despite the Exception Clause. The court stated at page 288:

‘... and if there is not a casual connection with the accident it is a very remarkable coincidence that the thrombosis should have occurred just when it did. But I also find it established that he would have had a thrombosis quite soon even if the accident had not occurred. Dr Gibson thought “he could have gone for five years without a thrombosis”, but considering all the evidence, both about Mr Jason’s temperament and the kind of life he led, I think that this is an outside figure and I find as a fact that at the date of the accident his expectation (using that word in the sense in which it is used in the phrase “expectation of life”) was of a coronary in three years’ time.’

[23] Jason v Batten is distinguishable from Concord because the agreement in Concord did not contain an Exception Clause. Because of the similarity of the present case to Jason v Batten one would reasonably expect we would follow the reasoning in that case. The problem for the appellant, however, is that it did not plead the Exception Clause and no reliance can therefore be placed on its terms. Having failed to place reliance on the Exception Clause before or during the trial, the appellant is not able to do so now nor can it seek an amendment on appeal. This is so because if the clause had been pleaded, the onus would have been on the insurer⁵, during the trial, to prove that the respondent’s occurrence fell within the terms of the exception and the respondent would not be prejudiced because it would have had an opportunity of rebutting this evidence. It will be recalled that during the deceased’s

lifetime, the appellant paid out a sum in respect of disability without any reliance on the non-disclosure of the previous myocardial

infarction.

[24] The enquiry in this matter is two-fold. First, the insured would have to prove on a balance of probabilities that the injury sustained in the accident was the proximate cause of the deceased's death and that his pre-existing condition was not a contributory 'cause' within the intended meaning of this word. The insured is greatly assisted in this task by the decision of this court in Concord which construed a policy for practical purposes identical to the present as not including a pre-existing condition. Once the causal

nexus between the accident and the death has been established, the onus would then shift to the insurer to show on a balance of

probabilities that the proximate cause of the accident was excluded by the Exception Clause. In this matter it will not be necessary to engage in the second enquiry, because of the failure to place reliance on the Exception Clause.

[25] Both Dr Tyrell and Dr Mabin agreed that the mechanism of death and the cause lay within the domain of cardiology. Both of them did not dispute that the deceased's health before the accident was stable. Their dispute was in respect of the temporal relationship between the injury and the plaque rupture, which was effectively linked to what appeared to be the deceased's improving medical status. According to Dr Mabin the risk of a triggering of a heart attack was in the early phases of acute trauma, surgery and infection but it diminished with time; whilst Dr Tyrell's view was that the risk persisted and was present even at the chronic phase. Dr Tyrell made reference to medical journals to support his hypothesis. Dr Mabin noted these and could not criticise nor disagree with them. He agreed that he participated in the content of the journals as an editor. His reluctance to embrace Dr Tyrell's opinion and the proposals contained in the journals was based on the fact that there was no clinical proof to support the hypothesis. His evidence on the issue proceeded as follows:

'Ja, look, I don't think any of us dispute the pathogenesis of arterial inflammation, plaque rupture etcetera. The question is what the triggers are, and its association with generalised systemic infection. I think we accept that, in the early phases of acute trauma, surgery, infection, there is a risk, and there is a risk of triggering a heart attack. But not beyond that. I think it diminishes with time, but not beyond what would be a few days. So in a chronic inflammatory state, [it] would imply a long, protracted inflammatory state, and I don't think it is well recognised.'

On being asked to comment directly on Dr Tyrell's hypothesis, he stated:

'I think had it happened in the acute immediate period, then I would accept that it's an attractive hypothesis, but not months after the event, and certainly not at the time that he was improving. I just find it difficult to accept the relationship between the two.'

Under cross examination, Dr Mabin again conceded that he found the hypothesis of Dr Tyrell attractive and stated:

‘It lends itself to logical thought in terms of pathogenesis, and I don’t think any of us disagree with the pathogenesis. What we disagree with, is how protracted those acute factors are in the chronic stage. And I’ve just been unable to find a causal factor relating to chronic inflammation and acute myocardial infarction.’

On being questioned further, Dr Mabin appeared to embrace Dr Tyrell’s opinion and was even constrained to concede that it may be more than just hypothesis. This he did in the following responses to counsel:

“As far as acute events are concerned, is it generally accepted now that acute events may lead to plaque rupture?”

---You mean acute trauma?

Yes...(intervention)...And surgery?

Trauma or surgery---It can increase the likelihood of plaque rupture.

Is that more than just hypothesis?---I think that’s again epidemiological, and it’s well known and well established in medicine that in the acute phase or major surgery, the likelihood of an acute myocardial infarction in a patient at risk is increased.

So that’s more than just hypothesis---That is.’

[26] Dr Mabin was also constrained to concede that the deceased had a further protracted difficult time after his initial surgery. Although he seemed reluctant to agree that the other incidences of surgery or trauma could be termed insults to the deceased’s body he was constrained to concede this when they were tabulated. The importance of this concession is that according to Dr Tyrell, it was the cumulative effect of these insults arising out of the progressive series of surgical interventions which caused the trauma that triggered the rupture. Dr Mabin accepted that Dr Tyrell’s hypothesis is logical, but lacks medical data to prove it. He stated:

‘...I’ve analysed what’s happened and I’ve looked for medical data to support what might have happened and the sequence of events and I must say I was in some way almost disappointed not to be able to find the corroborative evidence that we are looking for---It’s a logical hypothesis.’

[27] Clearly Dr Mabin was stating that the only reason why he was reluctant to agree with Dr Tyrell was because of the absence of corroborative empirical evidence to support what he himself regarded as a logical hypothesis. On the probabilities, it can be safely concluded that the opinion of Dr Tyrell is logical, was attractive to a fellow cardiologist and is supported by journal articles and studies on closely related areas of cardiology. His opinion in line with *Michael v Linksfield* was in my view correctly accepted by the court a quo.

[28] The respondent has therefore proved that the accident was the proximate cause of the deceased’s death. It not being open to the appellant to raise the Exception Clause, the appeal must fail.

[29] The remaining issue is the costs order made by the court a quo pertaining to a postponement on 30 October 2007, after the appellant had abandoned its special plea, and its objection to the respondent's intention to amend the particulars of claim so as to annex the correct pages of the contract of insurance. The court exercised a narrow discretion in awarding attorney and client costs against the appellant and there is no submission that this discretion was not exercised judicially. The appellant must accordingly fail on this aspect as well.⁶

[30] The appeal is dismissed with costs.

Z L L Tshiqi

Judge of Appeal

APPEARANCES

APPELLANT: W R E DUMINY SC

Instructed by Mellows & De Swardt

Cape Town;

Symington & De Kok, Bloemfontein.

RESPONDENT: D F IRISH SC

Instructed by Otto Krause Inc,

Cape Town; Honey & Partners, Bloemfontein.

1 1990

(1) SA 680 (A) at 700F-I

2 2001

(3) SA 1188 (SCA) para 34; para 36-38; para 40.

3 1992

(4) SA 669 (A).

4At p 673 G-H; 674 B-H.

5Agiakatsikas NO v Rotterdam Insurance Co Ltd 1959 (4) SA 726 (C); See also Rabinowitz & another NNO v Ned-Equity Insurance Co Ltd & another 1980 (1) SA 403 (W); Aegis Assuransie BPK v Van der Merwe 2001 (1) SA 1274 (T).

6Naylor & another v Jansen 2007 (1) SA 16 (SCA) at 23F-24D and authorities referred to in footnotes 15 to 23.

Source & reproduction note

Judgment wording, source notices and original paragraph numbers are retained. Esheria supplies the layout and this PDF's page numbers; these are not the source pagination. This is a research copy, not an official or certified court document. Check quotations, citations and publication restrictions against the original source before relying on it.

ORIGINAL SOURCE <https://www.saflii.org/za/cases/ZASCA/2010/133.html>

CASE PAGE <https://chat.esheria.ai/case-law/south-africa/mutual-federal-insurance-company-ltd-v-smd-unknown-topic-asca-2010>

Prepared on 14 October 2026 · Esheria