

*McMillan v Territory Insurance Office Board* [2011] NTSC 72

PARTIES: ELIZABETH McMILLAN

v

TERRITORY INSURANCE OFFICE  
BOARD

TITLE OF TRIBUNAL: MOTOR ACCIDENTS  
(COMPENSATION) APPEAL  
TRIBUNAL

JURISDICTION: REFERRAL UNDER S 28E OF THE  
MOTOR ACCIDENTS  
(COMPENSATION) ACT

FILE NO: 7 of 2010 (21036395)

DELIVERED: 23 September 2011

HEARING DATES: 5 & 6 SEPTEMBER 2011

JUDGMENT OF: MILDREN J

**CATCHWORDS:**

MOTOR ACCIDENTS TRIBUNAL – appeal de novo – compensation – whether blood alcohol level exceeded 0.08% - deceased driving at high speed – two passengers deceased – high risk involved resulting in death – whether compensation payable to dependants reduced – amount to be reduced – relevant factors.

*Motor Accidents (Compensation) Act* ss 4A(1), 7(1), 20, 20A, 20A(4), 21 A (1)(a)(b)(i)(c), 20A(2), 20A(3), 28E(1)(a), 29(2)

*Briginshaw v Briginshaw* (1938) 60 CLR 336; *Haskett v Territory Insurance Office*; *Robertson v Territory Insurance Office*; *Shepperbottom v Territory Insurance Office*; applied

*Klein and Domus Pty Ltd* (1963) 109 CLR 467; referred to

**REPRESENTATION:**

*Counsel:*

Appellant:	G Barns
Respondent:	S Brownhill

*Solicitors:*

Appellant:	Central Australian Aboriginal Legal Aid Service
Respondent:	Minter Ellison Lawyers

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IN THE MOTOR ACCIDENTS  
(COMPENSATION) APPEAL  
TRIBUNAL

*McMillan v Territory Insurance Office Board* [2011] NTSC 72  
No. 7 of 2010 (21036395)

BETWEEN:

**ELIZABETH McMILLAN**  
Appellant

AND:

**TERRITORY INSURANCE OFFICE  
BOARD**  
Respondent

CORAM: MILDREN J

REASONS FOR JUDGMENT

(Delivered 23 September 2011)

- [1] This is a referral by a claimant, Elizabeth McMillan, on her own behalf and on behalf of the infants Glen Smith, Eva Smith and Clinton Smith, pursuant to s 28E(1)(a) of the Motor Accidents (Compensation) Act (the Act).

**Facts**

- [2] The basic background facts are set out in the Agreed Statement of Facts and other documents tendered by consent.
- [3] The claimant, Elizabeth McMillan, is the spouse of the late Derek James Smith, with whom she bore three children, Glen Smith (born 4/3/1994), Eva Maria Smith (born 21/4/1996) and Clinton Anthony Smith (born 12/10/1998).

- [4] At around 1730 hours on 23 August 2008, the deceased was the driver of a Holden Commodore sedan in which there were two other passengers, which departed from Amoonguna Community, intending to travel to Alice Springs a short distance away. At around 1740 hours, the deceased was driving the vehicle north along the Ross Highway towards the intersection of Ragonesi Road, Alice Springs. In this vicinity, Ross Highway was an urban road comprising a bitumen sealed dual carriageway, with a single lane for each direction. The speed limit was 80 kph. Approximately 400 metres south of the intersection, the speed limit changed to 60 kph, and there was a 60 kph sign post clearly visible to approaching traffic. Approximately 310 metres south of the intersection was a prominent red sign stating “reduce speed” and a yellow roundabout sign. Approximately 210 metres south of the intersection was another sign indicating the existence of a roundabout at the intersection.
- [5] The deceased drove the vehicle towards the roundabout at approximately 123 kph. About 30 metres from the roundabout, the near side of the vehicle scraped along the kerb. It then mounted the roundabout and travelled directly across it, rolling several times, until it came to rest on its wheels approximately 85 metres from the southern edge of the roundabout, and into Palm Circuit on the northern side of the roundabout.
- [6] The deceased’s passengers were both ejected from the vehicle and died. The deceased remained in the vehicle still harnessed into the driver’s seat

by his seat belt, but he had also died from injuries he received in the accident.

[7] Subsequently, an autopsy was performed on the deceased's body, and a blood sample was taken from the heart by the pathologist Dr Sinton, which was sent for analysis. The result of the analysis revealed a blood alcohol reading of 0.318%. There were no drugs found in the sample.

[8] The vehicle was later examined. The vehicle was not affected by any defects or failures which contributed to the accident.

[9] There is no dispute that:

(1) the deceased was a "qualifying person" within the meaning of s 20 of the Act; and

(2) the applicants are the qualifying spouse of the deceased and that each of the children were his dependent children.

There is no evidence to the contrary. Accordingly, the applicants are entitled to benefits payable under the Act.<sup>1</sup> The benefits payable are dealt with in Part V of the Act. However, the benefits are liable to be reduced if the provisions of s 20A of the Act apply. This section provides:

#### **20A. Reduction of benefits in certain cases**

(1) The Board may reduce benefits payable under this Part if:

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<sup>1</sup> See s 7(1).

- (a) the accident resulting in the death of the qualifying person occurred while that person was driving a motor vehicle; and
  - (b) the qualifying person was under the influence of alcohol or a drug to such an extent that the person:
    - (i) was, by driving a motor vehicle, committing an offence against relevant laws regulating road traffic; or
    - (ii) would have been committing such an offence if the person had been driving the motor vehicle in a public street or public place; and
  - (c) the influence of the alcohol or drug contributed, in the Board's opinion, to the accident.
- (2) If the concentration of alcohol in the qualifying person's blood is shown to have been 80 milligrams or more in 100 millilitres of blood, the Board is to presume that the influence of the alcohol contributed to the accident unless the contrary is established.
- (3) The Board may reduce benefits payable under this Part if:
- (a) the accident occurred while the qualifying person was engaged in conduct that created a substantial risk of injury to the qualifying person; and
  - (b) the qualifying person recklessly ignored the risk.

*Note*

*This subsection applies whether the qualifying person was the driver, a passenger, a cyclist or pedestrian or involved in the accident in some other way.*

- (4) A reduction of benefits under this section is to be a proportion of the benefits otherwise payable considered

appropriate by the Board having regard to the extent to which the influence of alcohol or the drug or the qualifying person's risky conduct (as the case requires) contributed to the accident.

[10] There is no dispute that ss 20A(1)(a)(b)(i)(c) and (2) applies to the facts of this case, there is no evidence to the contrary and I formally so find.

Counsel for the respondent submitted that in the circumstances s 20A(3) may also apply. The main issue is the extent to which the benefits should be reduced in accordance with the terms of s 20A(4). In order to resolve these questions it will be necessary to consider the evidence led at the hearing of the reference in some detail. The factual issues include:

- (1) What was the concentration of alcohol in the blood of the deceased at the time of the accident?
- (2) What were the causes which contributed to the accident?
- (3) Was the deceased engaged in conduct that created a substantial risk of injury to himself?
- (4) If yes to (3), did the deceased recklessly ignore that risk?
- (5) What proportion of the benefits should be reduced in terms of s 20A(4)?

### **The standard and burden of proof**

[11] On the hearing before the Tribunal, the Board bears the legal onus of establishing that s 20A applies, on the balance of probabilities applied

according to the standard of satisfaction required by the test in *Briginshaw v Briginshaw*.<sup>2</sup> It is not in dispute that the hearing before this Tribunal is a hearing *de novo*.<sup>3</sup>

### **The deceased's blood alcohol concentration**

[12] As noted previously the deceased was killed instantly in the accident. The time of his death was therefore at around 1740 hours. Senior Sergeant Potts arrived at the scene at 1847 hours and remained there until the bodies of the deceased persons were removed. He departed the scene at 2050 hours or about 3 hours 10 minutes after death. No evidence was led from the ambulance attendants who removed the bodies and transported them to the Alice Springs Hospital. The emergency department notes from the hospital were not tendered. There is therefore no evidence as to the precise time when the body was placed in the morgue. The distance between the accident scene and the hospital is not such as to require a period of time to arrive at the hospital of more than half an hour. There is no evidence as to when the body was stored under refrigeration. For all I know the body may have been left on a gurney in the hospital for examination by a medical practitioner before it was stored. This is not improbable given that only a medical practitioner can certify that death has occurred. How long this may have taken is a matter of speculation, but given that hospitals are busy places, it would not be surprising if it took an hour or so before the body

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<sup>2</sup> (1938) 60 CLR 336; applied to s 20A in *Robertson v Territory Insurance Office* [2005] NTSC 74 at [12]; *Shepperbottom v Territory Insurance Office* (2005) MUR 403 at [9]; *Haskett v Territory Insurance Office* [2009] NTSC 37 at [4].

<sup>3</sup> See s 29(2).

was examined, and then shifted by an orderly to the refrigeration equipment where it was stored. According to the pathologist, Dr Sinton, who conducted the autopsy on 29 August 2008, the body had been stored at 4°C to minimise bacterial changes. He did not find any post-mortem hypostasis (change of colour); nor was there any bloating of the body, or other signs of putrefaction. On the evidence, I find that it is most likely that the body was placed in refrigeration probably not later than five to six hours after death, and that there were no signs of decomposition or putrefaction of any kind at subsequent post mortem.

- [13] The evidence of Dr Sinton was that a blood sample was taken from the deceased's heart, and a urine sample was also taken. So far as the blood sample is concerned, Dr Sinton was not asked precisely which part of the heart the sample was taken from. Professor Starmer, a retired former Associate Professor of Pharmacology at Sydney University, who was called by the respondent, gave evidence that the sample, if taken from one of the chambers of the heart, would not pose any concern as to the accuracy of the sample, but it could well be otherwise if the sample was taken from the pericardial sac, which is a closed membranous sac enveloping the heart. The reason for this is that if the deceased had been imbibing beer shortly before death, there could be neat beer in the stomach which could diffuse into the sample, and therefore increase the blood alcohol readings taken by the toxicologist. Professor Starmer said that samples taken from the heart are usually taken from one of the chambers to the heart. Dr Sinton's report

states that the pericardium was intact and unremarkable. There is no reference to any injuries to the heart, stomach or bowels, all of which are noted as “unremarkable”. Dr Sinton is an experienced pathologist. I think it is extremely unlikely that he would have taken the blood sample from the pericardial sac. If he had done so, I expect that he would have said so, consistently with the precise manner in which he gave his evidence.

[14] There is no evidence strictly proving the chain of evidence from the sample taken to the sample tested by the toxicologist. The toxicologist’s report notes that the blood sample tested was labelled “SMITH, Derek C216/2008” and contained in a sealed cardboard box. No submission was made by counsel for the appellant that the sample tested was not the same sample, and the manner in which he conducted his case assumed as much. I therefore find that the chain of evidence is not in issue. It is not in dispute that the sample, when analysed, revealed a blood alcohol reading of 0.318%.

[15] Mr Barns called evidence from Professor Jason White, Professor of Pharmacology from the University of South Australia. Professor White’s evidence is that estimating the blood alcohol concentration from a deceased person is complicated by the potential for post-mortem changes. The most important factors are diffusion of alcohol from organs to the blood, and the possibility of endogenous production of alcohol due to bacterial activity after death. There is no evidence of diffusion affecting the sample. Professor White referred to a study published in Forensic Science

International 106 (1999) at pp 157-162 by de Lima and Midio. The results of the study were based on 27 corpses, all in various stages of decomposition; nine were slightly decomposed, nine were in the moderate range and nine were severely decomposed. The purpose of the study was to investigate the feasibility of using vitreous humour “as the complementary fluid for the determination of aetiology of post mortem blood ethanol concentration in decomposed bodies for forensic purposes”. Essentially the study revealed that samples taken from the vitreous humour were likely to be more reliable. Professor White’s conclusion in this case was, according to his report, that although there was no evidence of putrefaction occurring, and accepting that the body was removed (and stored appropriately) in a little over three hours, it was unlikely that a significant degree of putrefaction occurred, and that it is highly unlikely that the blood alcohol concentration due to post mortem changes would have exceeded 0.08%. In the result, the deceased’s blood alcohol “can be estimated at 0.238%”. The logic of this conclusion escapes me. The best that might be said by this process of reasoning is that the reading was *at least* 0.238%. During his evidence in chief, Professor White stated that this was a “reasonable” albeit conservative assessment. In cross examination he conceded that any endogenous alcohol was highly unlikely to exceed 0.06%, and that the results “could be anywhere between 0% to 0.08%. Professor White’s evidence is that endogenous alcohol produced after death is the result of chemical changes caused by bacteria entering the

body. The more wounds there are, the greater the chance for bacteria to enter the body and cause these changes. The extent of the changes depends upon the time taken until it is stored in refrigeration. On the basis that the body was not stored for at least three hours, he considered that there was likely to be “some contribution”, although he considered that the trauma to the body (which was to the head) was not a major factor in this case.

[16] Professor Starmer agreed that bacterial fermentation can occur after death, depending on the temperature at which the body was kept, for how long, and whether there was any fermentable bacteria in the individual’s blood. He did not consider that a period of three hours before the body was placed in refrigeration was long enough to cause any “serious” generation of alcohol to have occurred, and that the risk of generation of alcohol in this case was small. However he agreed that a sample taken from the vitreous humour would have “enhanced” the toxicological assessment conducted by the gas chromatography method employed by the toxicologist. Neither side explored the possibility that the body may not have been placed in a refrigerated environment for longer than three hours.

[17] Evidence was given by two witnesses who had been drinking with the deceased at Amoonguna. According to Nanette McMillan, the deceased and others had been consuming Passion Pop and VB beer before the deceased and his passengers left Amoonguna. The deceased intended driving to the football at Alice Springs to see his wife. In her statement to the police, she said that the deceased was very drunk, and in her opinion,

too drunk to drive. However, she had intended travelling in the vehicle, and only missed out because she went to the toilet and whilst there, the deceased left without her. Her evidence was that the deceased was not “all that wobbly” and that she would have been happy to get in the car. This witness admitted that she was drunk at the time.

[17A] The other witness, Patrick Smith, was not called, but his police statement was tendered. According to his statement, he met the deceased and others at some time earlier in the day. The deceased had already been drinking, and he was told by the group that they had been drinking most of the night before. He confirms that he, the deceased and others consumed a carton of Passion Pop as well as a carton of VB beer before the deceased drove off in the vehicle. This witness stated that they were “all a bit smashed” even before they had started to consume the beer. There are some differences between this witness’s account of the total number of bottles of Passion Pop, and cans of beer were in the cartons, but for my purposes this does not matter as it is not possible to make an estimate of the likely blood alcohol levels of the deceased from the information provided, particularly if the deceased had already been drinking beforehand. The evidence does support the conclusion that when the deceased left Amoonguna he was already heavily affected by alcohol. The evidence is not inconsistent with a high range blood alcohol reading.

[18] Doing the best I can on the evidence, I think it is likely that some bacterial changes occurred which inflated the toxicological result somewhat.

Bearing in mind the onus of proof is on the respondent, I find that the difference is not likely to be more than 0.03% which would decrease the blood alcohol content to about .29% (in round figures), and I so find.

### **What were the causes which contributed to the accident?**

[19] In broad terms the evidence shows that the vehicle entered the intersection at a high speed. The approach to the intersection required manoeuvring around a bend to the right immediately before entering the roundabout. To continue on towards Alice Springs via Palm Court, the vehicle would have needed to turn left into the roundabout, follow the roundabout towards the right, then exit the roundabout to the left. The evidence of Senior Sergeant Potts, whose qualifications as an expert accident reconstruction investigator were not challenged, was that the vehicle was travelling at a speed which exceeded the critical speed for the curve in the road leading up to the roundabout, and therefore was incapable of negotiating that bend. I accept this opinion which is entirely consistent with the path which the vehicle actually travelled through the roundabout, as detailed by Senior Sergeant Potts, and as verified by the eye witness, Jared Ewin who was not cross examined. I find therefore that speed was a critical factor which contributed to the accident.

[20] I accept the evidence that the vehicle was in a roadworthy condition before the accident. This was not challenged. The state of repair of the vehicle was not a factor.

[21] There is no evidence that the vehicle was fitted with airbags. The photograph taken of the deceased still in the vehicle after the crash does not reveal any airbags. Whether or not airbags would have, or might have, prevented his death I cannot determine. That matter was not explored in evidence. It may be that a consideration of this issue is not relevant, as it is, under s 20A(4), necessary to consider the factors which “contributed to the accident” rather than the factors which contributed to the deceased’s death.<sup>4</sup> This matter was not raised in argument and I make no findings relevant to this potential issue.

[22] The evidence of Senior Sergeant Pott was that the driver’s vision of the road signs and of the intersection was not impeded by the sun, and the signs were clearly visible and not obscured by vegetation. The photographs accompanying his report confirm this. Therefore the deceased either failed to see the signs for some reason, or deliberately chose to ignore them. I think that the latter is unlikely. There is no evidence that the deceased was involved in a race with any other vehicle. At the time, there were no other vehicles on the road in the near vicinity. There is no evidence that the deceased was suicidal. There is no evidence that the deceased had fallen asleep. On the other hand, there is evidence that he deceased had tried to correct the path of the vehicle by a steering correction to the right after the vehicle hit the soft gravel section in the

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<sup>4</sup> But see the definition of “motor accident” in s 4A(1) of the Act. It may be arguable that an accident is “an occurrence ... resulting in the death ...” of the deceased, and therefore any factor arising out of the use of a motor vehicle which contributes to the death is part of the concept of what is an “accident”.

centre of the roundabout. There is also evidence that the deceased had attempted to negotiate the approach entry for the roundabout with a steering input of approximately 4°. The calculated steering impact required to negotiate the actual road curvature was 15°. Senior Sergeant Potts' analysis suggests that the deceased had not fallen asleep. Further, the journey to the point of the crash from Amoonguna was not far, and would normally not take longer than 10 minutes, there were a number of bends in the road to that point which had to be negotiated, the last being 1.53 km from the crash. It is more probable than not that the deceased was awake at all times before the crash, notwithstanding the amount of liquor he had consumed.

[23] The other possible explanations for ignoring or not seeing the signs are (1) distraction or (2) misjudging the distance available to him to slow the vehicle down before entering the intersection. As there were no survivors from the crash, it is not possible to find whether or not there was something, either in the car or on the road, which distracted the deceased. If the deceased had been distracted, a prudent driver would have applied the brakes and slowed down. This did not occur. The possibility of misjudging the distance is a real one, not uncommon in inexperienced drivers nor in drivers under the influence of alcohol.

[24] Whatever may be the explanation, the prime cause of the accident was a failure to keep a proper lookout, and slow the vehicle down in time to negotiate the bend into the roundabout.

[25] The evidence of Professor Starmer is compelling that excessive alcohol consumption is a significant factor as a cause of this accident. Before considering that evidence in more detail, I find that the deceased had a high tolerance to alcohol. This is based upon the observations of Nanette McMillan that the deceased was “not wobbly”, my finding that it is likely that he was awake, the evidence concerning the bends he had negotiated, and the evidence of the witnesses, Ronald Mummery and Amanda Lucas, who were on the Ross Highway shortly before the crash. Neither of these witnesses claimed that the car which they saw, (which I find was the deceased’s vehicle) was veering all over the road. The witness Lucas noted that the driver, a male Aboriginal, appeared to be sitting well forward “in a way that you would expect a driver to sit if they had something coming up in front of them and they couldn’t make out what it was ...”. If the deceased did not have a high tolerance to alcohol, it is likely that his level of intoxication would have affected his ability to maintain a straight path, and probably would have led to death, or at least unconsciousness, according to Professor Starmer.

[26] The observations of Ms Lucas are telling. They strongly suggest that this may be the likely explanation for not seeing the signs. Professor Starmer in his report states:

... alcohol has been shown to impair the three most important driver functions, perception, decision-making and response. There is usually an alcohol-induced inability to efficiently divide attention among two or more sub-tasks which is a classical expression of alcohol-induced driving impairment ... Intoxicated drivers almost

invariably feel that they need to concentrate on events in the centre of the visual field and to progressively ignore those in the periphery. When events in the periphery are sampled, the centre field is ignored for an inappropriately long time ... There is also an alcohol-induced slowing of information-processing which affects every action which the driver is called upon to perform. It should also be noted that those deficits frequently occur in conjunction with an increase in driving aggression and risk-taking behaviour ... [which] is usually manifested in speeding, especially in young males,<sup>5</sup> and there appears to be an attenuation of any appreciation of the dangers involved in driving a motor vehicle.

[27] In cross examination, Professor Starmer conceded that if the deceased had a high level of tolerance to alcohol (and he accepted that this was likely to be so) the deceased was less likely to be risk-taking or reckless. In evidence in chief, he said that it did not surprise him that he could have negotiated other bends in the road before the crash. He described driving a vehicle as an “over learned task”, i.e. that an intoxicated driver can drive when the levels of attention required are so not great as to require his whole attention. Reference was made to the common experience of drivers continuing to drive along a familiar route and being surprised to find themselves further along the way than expected, even when sober. This is because the brain does not require the driver’s whole attention to operate the controls. But when an untoward event occurs, an intoxicated driver is unable to make the series of manoeuvres needed to minimise crash risk.

[28] I accept the broad thrust of Professor Starmer’s evidence. The level of intoxication provides a rational explanation for the deceased’s speed, failure to observe the signs, a failure to react to them and slow down. I

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<sup>5</sup> The deceased was born on 29/5/1976 and therefore aged 32 at the time of his death.

find that the deceased's level of intoxication was also a contributing factor to the accident.

**Was the deceased engaged in conduct which created a substantial risk of injury to himself?**

[29] I find that he was, because of the speed at which he approached the intersection, his failure to slow down, and his failure to observe and comply with the warning signs.

**Did the deceased recklessly ignore that risk?**

[30] The expert evidence did not suggest that the deceased's driving was reckless. Proof of recklessness requires actual foresight of the risk and a deliberate or conscious decision to take the risk in circumstances where an ordinary sober individual having the deceased's capabilities and skill as a driver would not have taken that risk. It is not proved that the deceased had actual foresight of the risk and consciously made a decision to take the risk. This was not pressed by Ms Brownhill.

**What proportion of the benefits should be reduced in terms of s 20A(4)?**

[31] This requires an assessment of what is appropriate "having regard to the extent to which the influence of alcohol ... contributed to the accident". The key words in s 20A(4) are "appropriate", "reduced" and "having regard to".

[32] ‘Appropriate’ implies a discretion after considering and balancing all of the relevant considerations. The only consideration which is specifically mentioned and to which the Tribunal must have regard, is the “extent to which the influence of alcohol contributed to the accident”. That enquiry requires a consideration of all of the factors which contributed to the accident, and the extent to which alcohol was a contributing factor. Counsel for the appellant argued that the other key word, “reduced”, suggests that there is to be a reduction but not a complete denial of benefits. It is to be noted that elsewhere in the Act, the effect of alcohol as a contributing factor to an accident disentitles a person under the influence to any benefits to which s 9 applies.<sup>6</sup>

[33] The principal argument of counsel for the appellant was that s 20A(4) should be interpreted beneficially, and it was not to be used as a “punitive sanction”. In exercising its discretion, the Tribunal must be careful to ensure that the dominating or actuating reason for its decision was not outside the scope or purpose of the Act, citing *Klem and Domus Pty Ltd.*<sup>7</sup> The purpose of the Act is to provide a “no fault” scheme for persons injured in motor vehicle accidents.

[34] On the facts of this case, Mr Barns submitted that it had not been proved that alcohol was the only factor responsible for the accident. He submitted that the cause of the crash which led to the deceased’s death was the

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<sup>6</sup> See s 9(i). The effect s 9 (8) is that benefits for loss of earning capacity and lump benefits for permanent disability are excluded entirely, but other benefits are not affected.

<sup>7</sup> (1963) 109 CLR 467 at 473 per Dixon CJ

deceased's error of judgment in approaching the roundabout at 123kph, and that the reason why he did so is speculative because there are no witnesses available.

[35] Mr Barns' ultimate submission was that the only consideration in this type of case is "the proper regard to the extent to which alcohol contributed to the accident". Counsel for the respondent, Ms Brownhill also submitted that the extent of contribution of alcohol was the only relevant matter. I accept these submissions, but of course in arriving at that conclusion I must have regard to all of the factors which contributed to the accident.

[36] Ms Brownhill submitted that the factors which contributed to the accident were the appellant's excessive speed, failure to perceive and respond to the road signs, and excessive alcohol consumption, and that the first and second of these factors were the direct and natural consequences of the appellant's high blood alcohol reading. In all the circumstances it was submitted that the appropriate reduction was at least 75%.

[37] I accept the general thrust of Ms Brownhill's submission. I also accept Mr Barns' submission that I cannot find that these were no other relevant factors which also contributed to the accident, such as the possibility that the appellant was distracted by some unknown factor inside or outside the vehicle, or the possibility that the appellant was, to use Professor Starmer's expression, allowing the car to find its own way home, ie, failing to give his full attention to driving because he is thinking about other things which

were on his mind at the relevant time. Statistical evidence, whilst helpful can only go so far towards proof in any individual case, because human behaviour is infinitely variable. On the evidence I find that excessive alcohol consumption was the major contributing factor and that a reduction of 75% is appropriate.

[38] I was invited by Ms Brownhill to consider endorsing a scale of reductions as the appropriate reduction depending on blood alcohol levels. The proposed scale is:

<u>BAC</u>	<u>REDUCTION</u>
0.08% - 0.133%	30%
0.134% - 0.186%	45%
0.187% - 0.240%	60%
Over 0.240%	75%

[39] It is no part of my function to endorse a scale of this kind. If Parliament had wanted to provide a scale it could have done so. The fact that no scale is provided recognises that there are cases where a driver, although intoxicated, did not cause the accident at all, and therefore no reduction at all would be appropriate. In other cases, particularly where more than one vehicle is involved, the intoxication of the deceased driver may have played only a relatively minor role in causing the accident. Ms Brownhill recognised this, but submitted that the scale would only be a guide. I do not consider any such guide would be helpful. The Parliament has wisely decided to allow each case to be considered on its merits.

[40] I will hear counsel as to the form of the orders sought and as to costs.

