

# WORKERS COMPENSATION COMMISSION

## CERTIFICATE OF DETERMINATION

Issued in accordance with section 294 of the *Workplace Injury Management and Workers Compensation Act 1998*

**Matter Number:** 5673/18  
**Applicant:** Gaylene Ann Reakes  
**First Respondent:** State of New South Wales (NSW Police Force)  
**Second Respondent:** Jaiden Colin Reakes  
**Date of Determination:** 10 April 2019  
**Citation:** [2019] NSWCC 134

The Commission determines:

1. Ian Mark Reakes (the deceased) died on 13 September 2014 as a result of psychiatric injury, namely post-traumatic stress disorder, arising out of and in the course of his employment.
2. At the date of the deceased's death his widow, Gaylene Ann Reakes, and his son, Jaiden Colin Reakes were dependent upon him for support.
3. At the time of his death the amount payable in respect of the death of a worker pursuant to section 25(1) (a) of the *Workers Compensation Act 1987* was the sum of \$510,800.
4. Liberty to apply in relation to the apportionment of this sum and generally.
5. Order the respondent to pay the second respondent compensation in accordance with section 25 (1) (b) of the *Workers Compensation Act 1987*.
6. Respondent to pay the applicant's costs as agreed or assessed.

A brief statement is attached setting out the Commission's reasons for the determination.

Paul Sweeney  
**Arbitrator**

I CERTIFY THAT THIS PAGE AND THE FOLLOWING PAGES IS A TRUE AND ACCURATE RECORD OF THE CERTIFICATE OF DETERMINATION AND REASONS FOR DECISION OF PAUL SWEENEY, ARBITRATOR, WORKERS COMPENSATION COMMISSION.

S Naiker

**Sarojini Naiker**  
**Senior Dispute Services Officer**  
As delegate of the Registrar



# STATEMENT OF REASONS

## INTRODUCTION

1. Ian Mark Reakes (the deceased) died on 13 September 2014 of ischaemic heart disease. He was 51 years of age.
2. During his lifetime, the deceased was a police officer. He last performed active duty on 2 August 2011. He was medically discharged by the NSW Police Force (the respondent) on 15 March 2012. He suffered from post-traumatic stress disorder (PTSD), a condition which was caused by the execution of the duties of his office.
3. By these proceedings, Gaylene Reakes (the applicant), the deceased's widow, alleges that his death from ischaemic heart disease resulted from his post traumatic stress disorder. Thus, the dependents of the deceased are entitled to the compensation provided by section 25 of the *Workers Compensation Act 1987* (the 1987 Act) in respect of the death of a worker.
4. It is not disputed that the applicant or the child of her marriage to the deceased, Jaiden Colin Reakes, were dependent upon him for support at the date of his death. The respondent, however, disputes that the death of the deceased results from injury.

## PROCEEDINGS BEFORE THE COMMISSION

5. When the matter came on for conciliation and arbitration on 6 February 2019, Mr Callaway, of counsel, appeared for the applicant and Mr Flett, of counsel, appeared for the respondent. I was informed by counsel that the parties were unable to resolve the issue of whether the death of the deceased resulted from employment injury. I have used my best endeavours to bring the parties to a mutually acceptable agreement. I am satisfied that the parties had sufficient opportunity to explore settlement and have been unable to resolve the matter.

## EVIDENCE

6. The following documents are in evidence before the Commission;
  - (a) The Application to Resolve a Dispute (Application) and the attached documents;
  - (b) The Reply and attached documents;
  - (c) Applications to admit late documents of 17 December 2018 and 30 January 2019 and the documents attached to each.
7. Mr Flett indicated, at the commencement of the arbitration, that he wished to cross examine the applicant. Mr Callaway did not object. Given the complexity of the history in the matter, I had no doubt that it was in the interests of justice to permit cross examination. Accordingly, I granted leave to both counsel to adduce oral evidence.
8. Mr Flett also foreshadowed that the respondent may object to aspects of the applicant's written evidence. The parties agreed that this matter should be revisited after the oral evidence. Following his cross examination of the applicant, Mr Flett stated that he no longer wished to object to the written evidence.

9. After the arbitration hearing, I issued a Direction by which I provided both counsel with the opportunity to address the issue of whether section 9A of the 1987 Act applied in the circumstances of the case. Only the applicant provided written submissions.

## **ISSUES FOR DETERMINATION**

10. The respondent could not dispute that the deceased suffered from PTSD during his lifetime. By a Medical Assessment Certificate (MAC), dated 4 March 2014, an Approved Medical Specialist (AMS), Dr Steele, certified that the deceased suffered from PTSD. No medical practitioner, who saw the deceased in his lifetime, expressed a contrary view. On 3 April 2014, the Commission entered an award in favour of the deceased against the respondent, in the sum of \$25,300 in respect of 17% permanent impairment resulting from injury deemed to have occurred on 23 July 2011. Accordingly, the beguilingly simple task confronting the Commission is to determine whether death results from that injury.
11. The applicant asserts that the condition of PTSD materially contributed to the development of the deceased's ischaemic heart disease. The injury also caused the deceased to increase his intake of cigarettes and inhibited him from ceasing to smoke. Similarly, it led to an increase in his alcohol consumption, so that he became dependent on or abused alcohol. One, or more, of these factors resulted in his death.
12. The respondent's denial of liability is based both on the opinion of Dr Keogh, a cardiologist and a Professor of Medicine at the University of New South Wales, and on a challenge to the historical basis upon which the applicant's doctors have accepted a connection between the deceased's PTSD and his use of alcohol and cigarettes, and his death.
13. The submissions of counsel are recorded and I do not propose to reiterate each of those submissions in these short reasons. I will, however, set out the main thrust of counsels' arguments below. I propose to commence, however, by reviewing the evidence of the applicant, the other lay evidence in the case, and the contemporaneous medical evidence concerning the deceased's use of tobacco and alcohol.

## **GAYLENE ANN REAKES**

14. By her statement, the applicant says that she met the deceased in June 1999. They were married on 7 September 2002. Jaiden Colin Reakes was born on 11 February 2003.
15. Ms Reakes recounts that the deceased re-joined the police service in 1999 and "around 2000 was working as a highway patrolman in the Penrith/Windsor area." Ms Reakes addresses the deceased's alcohol and cigarette consumption at that time as follows:

"If Ian was not working an afternoon shift he often enjoyed having one or two beers after dinner. Ian would also have a cigarette. From my understanding, Ian started smoking when he was about 19 years old."

The applicant describes the deceased as "a fun-loving guy" and a loving husband.

16. The applicant recounts that she was told of two work incidents by the deceased. The first, in 2004, involved the death of a four-year-old girl in a high-speed motor vehicle accident. She says:  
  
"I remember Ian attended the inquest and found the questioning difficult. Even though he was exonerated by both the coroner and his superiors, Ian often said he felt guilty and responsible for her death. Ian took approximately three months stress leave after the death of Tabitha."

17. Ms Reakes recalls that from this time the deceased “started to have difficulty sleeping” experienced nightmares and images of Tabitha during the day. His personality changed. He made:

“excuses to avoid catching up with friends. He no longer talked and laughed as much. He became irritable towards me and started to pick fault at small things.”

18. Ms Reakes recounts that the deceased was involved in another motor vehicle accident, in which a mother and daughter were killed, in September of 2008. He was off work for some three months following that incident and sought treatment from Dr Shelly Bannon, a psychologist. He was “particularly hard to live with” after this incident. He became “increasingly moody and irritable”. The applicant and the deceased argued and separated for a brief period in 2010. The deceased also became over protective. Ms Reakes recounts:

“Ian seemed preoccupied and rarely laughed or joked and was spending more time alone. Ian was drinking and smoking more.”

19. Following a third incident, in July 2011, when the deceased drove through a red light he experienced further nightmares about his work and the deaths he had encountered during it. Ms Reakes states:

“Ian had more nightmares about his work and the deaths he had attended. He would often talk of having flashbacks to ‘Tabitha’. He was having difficulty concentrating, and had poor energy levels and motivation. He seemed to be depressed.”

20. Ms Reakes states that the deceased was drinking on average between 8 to 12 standard drinks a day and his cigarette intake had also doubled to about 25 cigarettes per day. The deceased stopped work in August 2011. He came under the care of Dr Looi, who prescribed anti-depressant medication and referred him to Dr Bannon, the psychologist and Dr Pusic, a psychiatrist.

21. The applicant says that the deceased “seemed to improve a little” towards the end of 2012. At that time, he was experiencing fewer nightmares and was starting to watch the news and crime shows once more. She states:

“It was around this time I remember pleading with Ian to reduce his alcohol and smoking intake. Ian reduced his drinking to about three or four standard drinks five days a week for a brief time.

Within a couple of months Ian had resumed drinking, on average, ten to twelve beers a day – sometimes more. He would smoke around a packet and a half of cigarettes a day. Ian would not eat saying he had ‘butterflies’ in his stomach. Ian lost about eight to ten kilograms in weight.”

22. Ms Reakes recounts that she and the deceased purchased a caravan park at Laurieton in 2013. She says that for “a short while” after moving to Laurieton Ian seemed to get a little better however he continued to have flashbacks and experienced distressing memories. She states that she would often see him “looking sad”. He neglected his appearance. His sleep pattern deteriorated and he would experience nightmares which caused him to “get out of bed sweating”.

23. Ms Reakes says that the deceased's memory and concentration diminished. She states:

"I would not let Ian have anything to do with the administrative running of the caravan park as on the few occasions he did this he would get angry with customers, double book sites or forget to confirm a booking. I also had to take over managing the family finances."

24. Ms Reakes states that from January 2014, the deceased ceased taking his medication and would not leave the park. He continued to drink 12 to 14 standard drinks a day and 25 cigarettes a day.

25. In his cross examination, Mr Flett challenged the reliability of applicant's observations of the deceased's use of alcohol and tobacco. The applicant agreed that she worked full time and that the deceased worked shift work. The following exchange took place:

MR FLETT: How many cigarettes do you think he was smoking per day after 2004?

MRS REAKES: Well, I know when he'd come home from work he'd sit there and he'd have his beer and then he'd have his cigarettes but I never really counted them.

MR FLETT: O.K. So, in that period of time, say from 5 o'clock if he wasn't working a night shift, he would have how many beers?

MRS REAKES: Are we talking about 2004?

MR FLETT: After 2004 up to 2011.

MRS REAKES: It could be ten during the day.

MR FLETT: Well, if you're at work you wouldn't be able to see - - -

MRS REAKES: Well that's - - -

MR FLETT: - - - during the day.

MRS REAKES: - - - correct.

MR FLETT: So, you're just making an approximation?

MRS REAKES: Well that's correct because I wasn't there during the day. I would see him after work sitting there with his beer and cigarettes.

26. Mr Flett then cross examined the applicant on her evidence that there had been a reduction in the deceased's use of cigarettes and alcohol around 2012. He put to her that from mid-2012 until April 2013 there was a reduction in his drinking. She acceded to that proposition. He also suggested that after they moved to Laurieton, the deceased performed a variety of jobs at the caravan park. The applicant agreed. It was put to her that he was more active at Laurieton than he'd been before the move. Again, the applicant acceded to that proposition.

27. Mr Flett cross examined the applicant to suggest that she and the deceased were required to work "24/7 for the two of you" in the caravan park. The applicant assented to this proposition. The applicant was cross examined on the history that the deceased gave to Professor Robertson, a psychiatrist in 2013, that he went fishing with acquaintances he had met since moving to Laurieton. The applicant agreed that was correct. It was put that the deceased went fishing on a weekly basis. She also acceded to that proposition. The following exchange then took place:

MR FLETT: And is it true that he'd go fishing on a number of days per week from time to time?

MRS REAKES: For the first six months and then after that everything ceased.

MR FLETT: Right. August would have been about the time are you saying, that he started drinking again?

MRS REAKES: He was always drinking but it was quantity that he was drinking.

28. It was put to the applicant that while she was in the office she did not have the opportunity to see the deceased during the day. She accepted that position, with the qualification that it was a small caravan park and that she would see him from time to time.
29. Mr Flett cross examined the applicant to suggest that her evidence that the deceased never left the caravan park was incorrect. The applicant agreed that it was not, as the deceased had seen Dr Mina, a general practitioner, only a few days before his death. The following exchange occurred:

MR FLETT: My question though is if he had been seeing a doctor in Laurieton why didn't you think it was important enough to tell us or tell your solicitors in April 2016?

MRS REAKES: Well, I know he went to the doctor two days prior to his death.

MR FLETT: And of course, the estimates as you have told us about the whole consumption and smoking of cigarettes they are all guesses, if I could put it that way, on your behalf?

MRS REAKES: Well you know how many times you go out and get a carton of beer and you know how many times you buy a carton of cigarettes.

#### **KRISTI BLANNING**

30. In a short statement, Miss Blanning says that she knew the deceased for eight years. She says that he attempted to give up cigarettes on several occasions and did well "until the stress would overcome him". She continues:

"I vividly remember Ian he was a chain smoker and would often ask what's bothering you but I would never get a straight answer.

I would see him with patches, gum and Champix until he would be so stressed that he couldn't cope so he would take up smoking again."

The witness does not state when she first met the deceased.

#### **LEIGH ELLIOT**

31. Mr Elliot is the deceased's son in law. He states that he had a close relationship with the deceased, even after he started his own family. He continues:

"He was always a drinker and a smoker from the time I meet him, but as time went on I noticed he started changing and appeared to be depressed and was continuously talking about flashbacks in his past. As time pasted [sic] I noticed that he was smoking more and more of the 16mg cigarettes (Winfield Red)."

32. Mr Elliot states that the deceased attempted to give up cigarettes by chewing Nicabate gum or taking Champix but “when he became distressed, he would always go back to cigarettes for relief”.

## **MEDICAL EVIDENCE OF TOBACCO AND ALCOHOL CONSUMPTION DURING THE DECEASED’S LIFETIME**

### **Dr Bannon, Clinical Psychologist**

33. Dr Bannon treated the deceased following the incident in 2011. By her report of 16 November 2011, she expressed the opinion that the deceased was not psychologically fit to attempt any work. She recorded that he suffered anxiety on a regular basis. He reported his alcohol consumption had increased to “approximately a carton of beer every two days (24)”. Dr Bannon recommended that the deceased be medically discharged from the police force.

### **Dr Bertucen, Psychiatrist**

34. Dr Bertucen saw the deceased on 28 November 2011, at the request of the respondent. He expressed the opinion that the deceased was totally and permanently incapable of returning to work as an operational police officer owing to the high likelihood of regression of symptoms “with potentially disastrous consequences”. He recorded that the deceased had chronic sleep disturbance and that his attention, concentration, energy levels and motivation had been significantly impaired by his disease. He recorded the following:

“Alcohol has also increased over the last eighteen months to hazardous levels (now on average twelve standard drinks per day). His cigarette intake has also doubled to twenty-five cigarettes per day.

### **Dr Pusic, Psychiatrist**

35. On 19 December 2011, Dr Pusic, the deceased’s treating psychiatrist, wrote to the respondent’s insurer. He also diagnosed PTSD as a result of multiple incidents during the deceased’s employment. He expressed the opinion that the deceased was unfit to return to any form of remunerative employment. Curiously, in view of the suggestion that the doctor recommended detoxification, the report contains no reference to alcohol or tobacco use.

### **Dr Dinnen, Psychiatrist**

36. Dr Dinnen saw the deceased on 24 July 2012. He diagnosed chronic PTSD with associated depressant disorder and excessive use of alcohol and cigarettes. He stated that:

“All of these associated conditions can be subsumed under the diagnosis of chronic post-traumatic stress disorder.”

In respect of alcohol and tobacco, he recorded the following history:

“He told me he drinks ‘a fair bit’. Currently that can amount to eight beers a day. His consumption has increased since he stopped work. When he was working if he was on an afternoon shift he would have no beer for a week. Usually he would have a couple of beers after dinner.

With regard to cigarette use he told me he is currently smoking a packet of cigarettes daily. He started smoking at 19. He is smoking a little more now since he stopped work, because he smokes when he drinks and he is drinking more.”

### **Dr George, psychiatrist**

37. On 21 September 2012, the deceased saw Dr George a psychiatrist, at the request of the respondent. Dr George diagnosed chronic PTSD. He expressed the opinion that the deceased needed “an inpatient admission in order to become abstinent from alcohol.” He noted that Dr Pusic had already suggested a detoxification/rehabilitation program but this had not been undertaken to date. Dr George recorded the following history:

“He said he has ‘eight to ten stubbies a day’ and said that this has been his pattern of drinking over the last twelve or perhaps, eighteen months. He said that his psychologist had attempted to have him reduce his alcohol intake but has not been successful to date. He said Dr Pusic had discussed with him an inpatient admission to St John of God Hospital where he could be treated for PTSD as well as his alcohol dependence and hopefully, move him towards total abstinence.”

### **Dr Pusic - 7 November 2012**

38. Dr Pusic wrote to the respondent’s insurer on 7 November 2012, doubting that the deceased suffered from alcohol dependence. He recorded this:

“On specific questioning Mr Reakes tells me that he would consume some twenty-four cans a beer [sic] over a one and a half to two-week period. This is substantially less than the amount quoted by Dr George. I do not agree that Mr Reakes requires inpatient or outpatient treatment for alcohol dependency as he does not show any evidence of alcohol dependency.”

39. The doctor opined that the deceased did not display evidence of alcohol dependency or excessive alcohol consumption. It does not appear that he was aware of the histories recorded by Dr Bannon or Dr Dinnen.

### **Dr Dinnen**

40. On 28 November 2012, Dr Dinnen wrote to the deceased’s solicitors stating that he did not agree that the deceased suffered from alcohol dependence. He said this:

“I provided a diagnosis of chronic post-traumatic stress disorder with associated depressive disorder and excessive use of alcohol and cigarettes. I advised that the associated conditions could be subsumed under the diagnosis of chronic post-traumatic stress disorder.”

### **Dr Bannon**

41. On 6 December 2012, Dr Bannon wrote to Dr Looi. She observed that the deceased had “reported an improvement in his PTSD symptoms”. He reported a reduction in the frequency of his nightmares to every one or two weeks. The report also that:

“He reported a reduction in his alcohol intake to 3-4 standard drinks five days a week.”

Dr Bannon referred the deceased to a Ms Turner, psychologist for psychological pain management in respect of his shoulder.

### **Dr Powell, Orthopaedic Surgeon**

42. Dr Powell saw the deceased for the respondent with respect to his shoulder injury on 13 June 2013. He recorded the following relevant history:

“He smokes 12 cigarettes a day and has two beers after dinner.”

### **Associate Professor Robertson, Psychiatrist**

43. On 15 August 2013, Associate Professor Robertson, a consultant psychiatrist saw the deceased at the request of the respondent regarding his permanent impairment claim. The deceased gave a positive account of his existence at Laurieton. The doctor recorded that:

“He is usually out of bed by 6.30am and in addition to attending to the family business, he spends time interacting with his son, including attending and taking an active interest in his son’s fledgling interest in football. He and his wife are slowly building social networks. Mr Reakes has befriended a number of residents in the caravan park and goes fishing with them on a weekly basis. He reports his marital relationship has been stable, with minimal conflict and the couple have adjusted well to their lifestyle changes.”

The doctor continued:

“He has modified his alcohol use significantly and now consumes alcohol to just above target level. He is contemplating ceasing smoking. The deceased was to see Dr Pusic for a ‘final session’ in November. Professor Robertson expressed the opinion that the deceased had recovered significantly. He anticipated that this would continue although there was always the risk of ‘further instances of depression’, particularly if he is retraumatised”.

### **Dr Steele, AMS**

44. Dr Steele saw the deceased on 4 March 2014. His history is strikingly different to that obtained by Professor Robertson only eight months previously. In respect of alcohol, Dr Steele recorded that consumption was reduced but “he will still drink somewhere in the vicinity of 12 standard drinks per day”. Dr Steele recorded that the deceased’s symptom complex “remains in the main present with flashbacks, distressing memories, and marked avoidance of social situations and propensity to anger being the major features of his ongoing psychiatric illness.”
45. Dr Steele considered the deceased to be an “honest and frank reporter of his condition.” He did not consider that the very different history recorded by Professor Robertson in 2013, cast any doubt on the reliability of the deceased’s evidence.

### **MEDICAL OPINION EVIDENCE**

46. I summarise below the salient aspects of the medical opinion evidence addressing the issue of causal nexus between PTSD and the deceased’s death. What follows is not intended as a comprehensive account of the entirety of the expert evidence. I record the principal hypotheses of the doctors, so that the parties can understand the way in which the Commission has resolved the dispute.

## Professor Duflou

47. Professor Duflou, a consulting forensic pathologist, provided a report, dated 9 May 2018, in which he reviewed much of the medical evidence which I have summarised above. He concluded that the cause of the deceased's death was "best attributed to ischaemic heart disease". He accepted that "the mechanism of death was very likely an arrhythmia."
48. The applicant's solicitors asked Professor Duflou to express an opinion as to whether there was a causal link between the deceased's ischaemic heart disease and chronic PTSD. After citing several epidemiological studies, he referred to the meta-analysis by Edmondson et al, and stated:

"When the influence of confounding variables such as depression and heavy smoking or excessive alcohol consumption are removed from a study population, the risk of fatal and non-fatal ischaemic heart disease is increased by 55% in persons with PTSD relative to control populations."

49. Turning his attention to Mr Reakes, he conceded that it was difficult to extrapolate studies involving populations to individuals. He continued:

"He certainly has accelerated ischaemic heart disease and there is a strong link between development of coronary atherosclerosis and death due to ischaemic heart disease both in persons with PTSD and persons who are heavy tobacco smokers. The link with excessive alcohol consumption is not as strong, although there certainly is good evidence that prolonged excessive alcohol consumption causes both accelerated ischaemic heart disease and hypertension. In my opinion, it is more likely than not that the various neuroendocrine and autonomic abnormalities typically expected in cases of chronic PTSD, especially when taken together with the deceased's excessive alcohol and cigarette use typical of persons with PTSD, contributed significantly to the onset of ischaemic heart disease in the case of Mr Reakes, and as such it is more like than not the case that the deceased's heart disease is causally linked to his chronic PTSD."

50. Professor Duflou was then asked to consider whether PTSD with associated depressive disorder and excessive use of alcohol and cigarettes contributed to or precipitated the deceased's death. He expressed the following opinion:

"There is in my opinion strong evidence on epidemiological grounds to link the deceased's PTSD and associated depressive disorder and excessive alcohol and tobacco use to his death due to ischaemic heart disease. However, it is more difficult to make this statement in an individual case, where other confounders and risk factors have been major or minor contributors to the development of the deceased's ischaemic heart disease."

51. After considering aspects of Professor Keogh's opinion, he concludes thus:

"It is my opinion that it is very likely that the deceased's post-traumatic stress disorder with associated depressive disorder and excessive use of alcohol and cigarettes was a significant underlying cause of his ischaemic heart disease, and that death was the result of that ischaemic heart disease."

52. Professor Duflou was also asked to consider whether the deceased's heavy smoking and use of alcohol accelerated or exacerbated ischaemic heart disease. He replied thus:

"The development of ischaemic heart disease is multi factorial, and there is usually no single factor which is the cause of ischaemic heart disease in a patient. Also, in many patients with ischaemic heart disease, no specific cause or predisposing factor is found. In general, though, heavy smoking has a marked effect of development of ischaemic heart disease, with heavy smokers developing the disease earlier, having more advanced disease and developing more complications of the disease (including sudden death) than non-smokers. There is a similar but not as well studied association between excessive alcohol consumption and the development of ischaemic heart disease and death due to ischaemic heart disease."

### **Associate Professor Richards**

53. Associate Professor David Richards provided a report of 3 September 2018 in which he considered the medical evidence, which I have summarised above and the opinion of Dr Keogh, the respondent's cardiologist. He expressed his opinion thus:

"Although I agree that Mr Reakes was at risk for coronary artery disease because of his family history of vascular disease and his personal history of cigarette smoking and hyperlipidaemia, it is my view that increased cigarette smoking and alcohol consumption associated with PTSD could only have aggravated ischaemic heart disease (other equivalent terms include coronary artery disease, atherosclerotic cardiovascular disease). It is my view highly likely that the increased cigarette and alcohol consumption made it more likely that Mr Reakes would experience myocardial infarction when he did, than would have been the case if cigarette smoking and alcohol consumption had not increased because of post-traumatic stress disorder.

At the very least, additional cigarette smoking and alcohol consumption due to PTSD could not have reduced Mr Reakes' predisposition to myocardial infarction. Rather, autopsy evidence of heart weight above normal made it more likely that Mr Reakes had hypertension during life, likely aggravated by excessive alcohol consumption, in turn due to PTSD, and use of non-steroidal anti-inflammatory agents, probably used because of shoulder pain secondary to an injury at work. Any incremental increases in cigarette consumption, and blood pressure due to increased alcohol and Celebrex consumption as a result of work related post-traumatic stress and physical injury, would have increased the risk of earlier complication of ischaemic heart disease than would have been the case in the absence of increased cigarette, alcohol and Celebrex consumption as a result of work-related post-traumatic stress disorder and physical injury."

54. Associate Professor Richards opined that the deceased's alcohol consumption could aggravate his hypertension, which in turn aggravated ischaemic heart disease. He expressed the opinion that the cigarette smoking aggravated ischaemic heart disease in a dose-dependent manner. He concluded by stating:

"It is my view that in the absence of increased alcohol and cigarette consumption Mr Reakes would not have died when he did."

## **Dr Bertucen**

55. Dr Bertucen provided a report to the applicant's solicitors on 28 March 2017, following his consultation with the applicant on 14 March 2017. He took a history from the applicant that the deceased smoked "up to a packet a day of cigarettes" i.e., 25-30 cigarettes until his death. Dr Bertucen remained of the opinion that the deceased suffered chronic PTSD and comorbid major depression at the time of his death. He also suffered from secondary alcohol dependence/abuse and excessive smoking.
56. When asked to express an opinion as to whether there was a causal link between the deceased's psychological injury and his ultimate death as a result of ischaemic heart disease, Dr Bertucen said this:

"This is naturally a difficult and somewhat speculative question. Mr Reakes was (as has been established) a smoker since the age of 19, i.e., five years prior to his initial employment with the NSW Police Force. The question naturally, therefore, is whether or not Mr Reakes tobacco addiction would have resulted in his premature death irrespective of being employed with the NSW Police Force, or any other occupation from 1987 to 2011."

After considering several epidemiological studies, the doctor said this:

"While no such causal link can be incontrovertibly demonstrated, I would argue that given the current knowledge of bidirectional influence of smoking and mental illness that Mr Reakes psychological condition (manifested from early 2000 onwards) would have significantly influenced his tobacco intake and would have been likely to have inhibited smoking cessation."

57. Dr Bertucen then went on to consider the possibility that chronic PTSD results in over production of catecholamine chemicals within the bloodstream because of constant sympathoadrenal arousal. He expressed the opinion that incessant catecholamine release over time promotes arterial injury via haemodynamic (turbulence and shear stress) and metabolic (e.g., platelet aggravation and lipolysis) changes and eventually causes coronary atherosclerosis due to plaque rupture and thrombus formation.
58. Dr Bertucen concluded his report thus:

"This question, in my opinion, is subsumed within the answers to the questions above. As previously, I am unable to make any 'courageous' statement regarding an actual causal link between smoking, chronic post-traumatic stress disorder and Mr Reakes death; however, there is, in my opinion a significant layer of attribution that lends persuasion to this theory."

## **Dr Anthony Dinnen**

59. Dr Dinnen provided a supplementary report to the applicant's solicitors dated 30 April 2018 in which he reviewed some of the relevant literature. He expressed the following opinion:

"It has been my opinion that there is an association which is unarguable between PTSD and substance abuse (including alcohol and cigarettes). I also believe that there is clinical evidence and research evidence to support the argument that psychiatric conditions such as PTSD are causally related to heart disease."

60. Dr Dinnen also addressed a question from the applicant's solicitors who asked him to assume that the deceased was loath to attend regular check-ups from his medical practitioner. He said this:

"Many people are avoidant with regard to medical check-ups and attendances. I wouldn't class this as part of PTSD avoidance. However, avoidance generally of matters which remind the individual of traumatic experience is a central feature of PTSD and to that extent Mr Reakes may have wanted to avoid seeing the doctor for fear of being reminded of service matters and traumatic experiences".

## **PROFESSOR KEOGH**

61. The respondent's declinature of liability to pay compensation in respect of the death of the deceased was primarily based upon the opinion of Professor Keogh. By a report of 21 April 2015, Professor Keogh considered the coroner's report, some of the psychiatric evidence brought into existence during the deceased's lifetime and the notes of Dr Looi, the deceased's general practitioner. She expressed the opinion that the deceased died of arrhythmia secondary to acute thrombus on top of previously stable plaque in the LAD coronary artery. She continued:

"The arrhythmia was almost certainly ventricular tachycardia or fibrillation arising as the result of ischemia. According to the Coroner, the heart at autopsy showed early myocardial infarction and the lungs were congested due to back pressure from acute heart failure in the second of the rhythm disturbance. The course of the chronic coronary artery disease was his large number of risk factors. The chronic coronary artery disease would have started to develop from as early as 10/20 years of age."

62. Professor Keogh cited several risk factors for coronary artery disease. They are male gender, smoking from age 19 until death aged 51, early history (father having had a CVA) hyperlipidaemia and hypertension. She stated:

"Over the years covered in GP notes, there was clear evidence of lack of control of risk factors for coronary artery disease (smoking and high cholesterol) with probably lack of compliance with Crestor."

63. Professor Keogh stated that she did not believe there was any causal relationship between a work injury and the death of the deceased. He died of coronary artery disease, a result of the process of atherosclerosis which began early in his life. She referred to the autopsy finding that of the three main epicardial coronary arteries "2 were found at autopsy to carry a heavy burden of diffuse and also localised stenosis". The doctor reiterated her view that the deceased would have died of ischemic heart disease around the same age, regardless of any work injury.
64. By a supplementary report dated 20 July 2018, Professor Keogh reiterated that the risk factors referred to above were the substantial and material cause of the deceased's coronary artery disease. Even an acceptance that the deceased's cigarette consumption "increased or fluctuated", in accordance with the evidence of the applicant, did not undermine the hypothesis that these risk factors were the cause of his death. In respect of alcohol intake, she opined that drinking alcohol was not a "risk factor for the acute thrombus which formed in the LAD just prior" to the death of the deceased.

65. Professor Keogh conceded that the medical literature established a possible relationship between psychological factors and coronary artery disease. However, the evidence in support of that connection “is mixed with only some studies showing a possible link.” Accordingly, Professor Keogh preferred the view that the cause of the deceased’s death was the large number of “traditional” constitutional risk factors. She observed that psychological factors seem to have been considered (by medical experts in the applicant’s case) to the virtual exclusion of “usual medical risk factor intervention and control”.
66. By a supplementary report, dated 10 December 2018, Professor Keogh addressed the opinion of Associate Professor David Richards. She expressed the opinion that the deceased’s increased drinking and smoking following his development of PTSD did not “**materially** contribute” to his death. (The emphasis on the word materially is that of Professor Keogh). She agreed that additional smoking could “not have reduced Mr Reakes predisposition to myocardial infarction”. It did not follow, however, that it was a material cause of death. Further, Professor Keogh doubted that heavy alcohol usage “had a provable meaningful role”.
67. Professor Keogh observed that the smoking history was somewhat inconsistent. She referred to the obvious discrepancy between the history of smoking in a submission made on behalf of the deceased, in August 2012, and the history obtained by Dr Powell, an orthopaedic surgeon less than a year later, on 13 June 2013. The former document recorded cigarette usage of 25 per day, whereas Dr Powell took a history of 12 cigarettes per day.
68. Accepting, however, that the deceased’s smoking increased by an additional 15 cigarettes per day between 2011 and 2014 for the purposes of argument, Professor Keogh calculated that this represented a 10 to 15% “increase in smoking above his baseline rate”. The baseline rate is the measure of the deceased’s smoking in his lifetime, on the assumption that he had not suffered PTSD. Professor Keogh expressed the opinion that the increase in smoking would not represent a “significant or material increase in risk above his usual habit”.
69. Professor Keogh then further considered the risk factors of untreated hyperlipidaemia and hypertension. She agreed with Professor Richards about the first of these risk factors. She noted that there was no evidence that the deceased was prescribed statins after 25 February 2010. She was unsure whether this was due to non-compliance or medical oversight. She canvassed the possibility that the deceased’s hyperlipidaemia was inherited from his father, so that it was a significant risk factor for ischaemic heart disease.
70. In respect of hypertension, Professor Keogh, having reviewed the documentation, “did not see any definite evidence”. She postulated that there may be information from Dr Mina, who the deceased saw two days before his death, or earlier blood pressure readings. However, the material before her did not prove the existence of hypertension. Accordingly, she did not accept the opinion of Professor Richards that the deceased probably suffered from hypertension.
71. In respect of alcohol abuse, Professor Keogh observed that alcohol does not directly increase the risk of myocardial infarction. It can contribute to hypertension. But as the evidence did not establish the probability of the existence of hypertension, the role of alcohol in the deceased’s ischaemic heart disease and in his myocardial infarction was not significant.

## SUBMISSIONS

72. Mr Flett submitted that the applicant's evidence as to the deceased's smoking and drinking was unreliable. He argued that her evidence that the deceased smoked "a cigarette" after dinner was inconsistent with the other evidence. It was unlikely that the deceased would have approached a medical practitioner for advice and assistance in giving up smoking in 2001, if he was an extremely light smoker. The applicant's evidence in respect of the deceased's consumption of alcohol was equally unreliable.
73. An analysis of the medical histories of the consumption of alcohol necessitated a finding that the deceased was "not drinking the quantities which the applicant would have you believe".
74. The respondent submitted that the applicant's written evidence that the deceased would only leave the caravan park to visit a doctor was also inconsistent with other evidence. The deceased clearly visited Dr Mina in the days before his death.
75. The unreliability of the applicant's evidence made it difficult to establish the "starting point" in terms of the deceased's drinking and smoking. It could be inferred from the evidence of Mr Elliott, for example, that he was always a heavy smoker. If the starting point was unreliable, it was difficult to draw inferences as to increases in smoking and drinking from 2004 onwards.
76. Mr Flett submitted that the best evidence of the deceased's cigarette and alcohol consumption were the multiple contemporaneous accounts of medical practitioners. These also established alcohol and tobacco abuse. They suggest, however, some lessening of the deceased's alcohol and cigarette consumption after severing his ties with the police in 2012.
77. Mr Flett submitted that some of the variations in histories might be explained by the "purpose" for which the medical consultation was held. Mr Flett argued:

"The MAC is a significant because it's a court process to and the end result being that you're wittingly or unwittingly tend to maximise your complaints".

I understand that submission to mean that the Commission should discount the history recorded by the AMS, as it was coloured by the deceased's desire to maximise his permanent impairment compensation. That would explain the considerably greater use of alcohol and tobacco recorded by the AMS in 2014 than that earlier recorded by Professor Robertson.

78. Mr Flett then submitted that the Commission would accept the evidence of Professor Keogh in relation to hypertension. At autopsy, the heart weight of the deceased was not abnormal. Hence it was not possible to say whether the deceased suffered from hypertension. Given the objective basis of Professor Keogh's opinion, the Commission would not accept the contrary view of Dr Duflou and Dr Richards.
79. Mr Flett proceeded to attack the basis of Dr Richards's opinion. The "mays and coulds" employed by the doctor in his report necessitated a finding that it was not persuasive. His assertion that altered abdominal sensation and shoulder pain may be evidence of PTSD aggravating ischaemic heart disease was speculative. Dr Richards was "over cooking the condition". There were other medical reasons for these symptoms. The deceased, for example, had a significant shoulder injury.
80. Finally, Mr Flett addressed the significance of the absence of evidence or information from Dr Mina, who had seen the deceased two days before his death. The absence of Dr Mina's records undermined the hypothesis that the deceased suffered hypertension in his lifetime for the reasons propounded by Professor Keogh.

81. Mr Callaway submitted that the Commission would accept the applicant's evidence. There was no reason to conclude that she was not telling the truth. Her evidence was corroborated by the MAC and by the other lay evidence.
82. The clinical record, particularly that of Dr Looi, demonstrates that the deceased certainly suffered from PTSD from 2008. There were "multiple attendances" on the doctor in respect of that condition. Mr Callaway submitted that the notes of Dr Looi demonstrated the deceased's unsuccessful attempts to cease smoking, after he developed PTSD. He was prescribed Champix in 2009. His inability to cease smoking "was made more difficult" by his injury.
83. Mr Callaway submitted that the evidence of Dr Looi, who accepted a connection between the deceased's increased drinking and smoking and his death was important, as he had been the deceased's general practitioner for more than 10 years. Mr Callaway also referred to the evidence of Professors Richards and Duflou. He submitted that when the issue case was approached on the basis of the approach to causation approved in *Kooragang Cement Pty Ltd v Bates* (1994) 35 NSWLR 452 (*Kooragang Cement*), the Commission would find a causal connection between the proven injury and the death of the deceased. He made only cursory submissions on the medical evidence.

## DISCUSSION AND FINDINGS

### The relevant law

84. As Mr Callaway submitted, the leading authority on causation in workers compensation cases is *Kooragang Cement*. In that case, Kirby P notably said this:

"The result of the cases is that each case where causation is in issue in a workers compensation claim, must be determined on its own facts. Whether death or incapacity results from a relevant work injury is a question of fact. The importation of notions of proximate cause by the use of the phrase 'results from' is not now accepted. By the same token, the mere proof that certain events occurred which predisposed a worker to subsequent injury or death, will not, of itself be sufficient to establish that such incapacity or death 'results from' a work injury. What is required is a common-sense evaluation of the causal chain."

85. Subsequently, in *Sutherland Shire Council v Baltica General Insurance Co Limited and Others* (1996) 39 NSWLR 87 (*Sutherland Shire Council*), Clarke JA stated that the phrase "results from" might be equated with the common law test of causation and "that the relevant inquiry directs attention to whether the injury caused or materially contributed to the incapacity". In expressing that opinion, his Honour relied on the reasoning of Brennan J, in the High Court, in *Accident Compensation Commission v CE Heath Underwriting and Insurance Australia Pty Limited* (1994) 68 ALJR 525 (*Accident Compensation*). In a judgment with which all members of the High Court concurred, the judge explicitly stated that liability to pay weekly payments or lump sum compensation under the Victorian Workers Compensation Legislation fell on any employer/insurer where the worker had sustained injury which materially contributed to his incapacity.
86. The reasoning of Clarke JA in the *Sutherland Shire Council* case has been unreservedly accepted by the Presidential Unit of the Commission. While the decision in *Comcare v Martin* [2016] HDA 43 has cast doubt on whether the principle of "common sense" causation has universal utility, there is no compelling reason to doubt that the reasoning in the *Sutherland Shire Council* case and in the *Accident Compensation* case remain good law. Nonetheless, in conducting the enquiry as to causal nexus in this case, it is best to utilize the words of the Act. The question is whether death results from injury.

87. It is unnecessary to consider whether employment is a substantial contributing factor to the injury in this case. As indicated above, the relevant injury is not in dispute.

### **PTSD and Ischaemic Heart Disease**

88. While there is some debate about terminology in the medical evidence, it is common ground that the deceased died of myocardial infarction, consequent upon ischaemic heart disease. The opinion expressed in the autopsy report, dated 7 October 2014, identified ischaemic heart disease as the direct cause of death and coronary artery atherosclerosis as the antecedent cause.
89. Both the cardiologists in the case accept that the deceased's ischaemic heart disease was multi-factorial. Professor Keogh identifies seven "traditional" risk factors which contributed to the deceased's ischaemic heart disease. These were his male gender, his family history, smoking, hyperlipidaemia, hypertension, alcohol use and medical oversight or the deceased's non-compliance with medical advice and, possibly, with prescribed medication.
90. The applicant's medical case suggests that PTSD is another risk factor. Dr Bertucen and Dr Dinnen propose that there is a direct causal connection between PTSD and heart disease. Dr Dinnen puts the argument this way:
- "I also believe that there is clinical evidence and research evidence to support the argument that psychiatric conditions such as PTSD are causally related to heart disease. I have seen the arguments put on either side of this proposition, that my opinion is as given." [sic].
91. Dr Bertucen, after referring to several studies, states that the mechanism of heart disease in chronic PTSD relates to the overproduction of catecholamine chemicals causing arterial injury.
92. Neither doctor expresses an unequivocal opinion that the deceased's PTSD directly accelerated his ischaemic heart disease, or caused his death. As I read his report, Dr Dinnen, concedes that there are "arguments put on both sides" in respect of this hypothesis.
93. Dr Bertucen also describes a mechanism whereby stress may cause ischaemic heart disease. But his opinion on causation in this case, which is heavily qualified, introduces smoking as an additional factor in the hypothesis. In other words, there is no clear statement of a direct causal nexus between PTSD and the deceased's death from ischaemic heart disease.
94. I was not taken specifically to any of the studies referred to by Dr Dinnen or Dr Bertucen. My impression is that the studies referred to do no more than raise the possibility of a connection between PTSD and "early-age heart disease mortality". One difficulty with these studies, is that it is not clear whether the chronic PTSD sufferers surveyed may have also had one, or more, of the traditional risks for ischaemic heart disease identified by Professor Keogh. They may, for example, have gross pre-existing ischemia, used tobacco and alcohol to excess because of a pre-existing addiction, and suffered from hypertension. Some may also suffer from hyperlipidaemia and have a family history of ischaemic heart disease. If that were the case, the studies may not assist in establishing causal connection.
95. Professor Duflou also supports an association between PTSD and ischaemic heart disease. He cites a study from Edmondson, which made allowance for the "confounding factors", which I have referred to in the above paragraph, and reaches the conclusion that the risk of fatal and non-fatal ischaemic heart disease is increased by 55% in persons with PTSD relative to a control population.

96. Obviously, such evidence cannot be as easily discounted. It is probably evidence of what has been referred to as “general causation”: see the discussion in the reasons of Spiegelman CJ in *Seltsam Pty Limited v McGuinness; James Hardie & Coy Pty Limited v McGuinness* [2000] NSWCA 29. It is evidence that PTSD is capable of causing ischaemic heart disease. But even Professor Duflou concedes that it is difficult to argue from general to specific causation. When asked to express an opinion on causal nexus in this case, he refrains from making an unambiguous statement that PTSD directly caused the deceased’s death. Rather, he opines that PTSD “when taken together with the deceased’s excessive alcohol and cigarette use” contributed to his ischaemic heart disease and death.
97. Professor Duflou also states that hypertension “is more commonly seen in persons with chronic PTSD”. The evidence, however, suggests another significant cause of hypertension is alcohol consumption. The evidence strongly suggests a connection between PTSD and alcohol abuse. That may explain why hypertension is more commonly seen in persons with PTSD.
98. Ultimately, neither Professor Keogh or Professor Richards, the cardiologists retained by the parties in the case, postulate that PTSD, without the interposition of alcohol and tobacco is a likely direct cause of the deterioration of the deceased’s ischaemic heart disease and death. Dr Keogh concedes that psychosocial factors “may possibly contribute” to the development of atherosclerosis, but opines that the evidence is weak with “only some studies showing a possible link”.
99. Professor Richards does not rebut that proposition. It is possible that he assents to Professor Duflou’s opinion of a connection. But his own formulation of causal nexus between PTSD and ischaemic heart disease is through the medium of the increased use of tobacco and cigarettes.
100. While the evidence of psychiatrists and pathologists may provide valuable insight, the opinion of cardiologists on the cause of heart disease will ordinarily prevail. Professor Richards does express the opinion that “depression may be aggravated by myocardial ischaemic”. That is the reverse of the hypothesis which the applicant presses: that PTSD directly causes heart disease.
101. Accordingly, I am not persuaded, on the evidence in this case, that the argument put by the applicant that PTSD was a direct cause of the deterioration of the deceased’s ischaemic heart disease has been proven.

### **Consumption of alcohol and cigarettes**

102. On the other hand, it is accepted by all the medical practitioners in the applicant’s case and, probably, by Professor Keogh that PTSD may lead to increased tobacco and alcohol consumption. That is really a question for the psychiatrists. There is no evidence which contradicts Dr Dinnen or Dr Bertucen opinion on this issue.
103. Acceptance that PTSD can increase the use of tobacco and alcohol gives rise to two questions. First, whether the evidence establishes that the deceased used tobacco and alcohol more following his contraction of PTSD. Secondly, whether increased use of cigarettes and alcohol, in fact, accelerated the deceased’s ischaemic heart disease to the extent that it caused his death.
104. It was the first question that Mr Flett addressed, when he submitted that the evidence of the applicant as to the deceased’s tobacco and alcohol consumption was unreliable. He argued that the evidence did not provide any valid basis for concluding that there was an increase in the deceased’s alcohol and tobacco use. Although Mr Flett denied that he was challenging the applicant’s veracity, it is, nonetheless, necessary to consider her credibility.

105. It is often the case that a party will put his/her case in the most positive possible way in both written and oral evidence. That tendency is probably exacerbated when witness statements are prepared with the assistance of solicitors. That may be true of the applicant's evidence in this case. There are important differences between her written and oral evidence. I do not accept, however, that the substantial criticisms that Mr Flett made of her evidence destroys or significantly diminishes its force. Rather, I formed the view that the applicant was probably doing the best that she could to assist the Commission during Mr Flett's testing cross-examination.
106. In my opinion, the applicant's written evidence that the deceased had "a smoke" after dinner prior to 2004 should not be interpreted to mean that he only had one smoke each evening after work. It certainly should not be taken to mean that the deceased only had one smoke a day. Having "a smoke" is idiomatic English. I believe that the applicant was trying to convey her impression that the deceased smoked considerably less early in their marriage than he did after 2004.
107. The applicant's failure to record in her statement that the deceased left the caravan park to visit a medical practitioner on one, or more, occasions is troubling. However, it does not undermine the entirety of the applicant's evidence. Once again, in my opinion, the applicant was attempting to convey an impression that, by and large, the deceased did not wish to leave the caravan park in the last year of his life.
108. Aspects of the applicant's evidence in respect of the deceased's alcohol consumption were quite compelling. When it was put to her that her evidence estimating the deceased's drinking and smoking was "all guesses", she responded that you "know how many times you buy a .... carton of beer" and "buy a carton of cigarettes". Certainly, her evidence is not an exact or unerring account of the quantity of alcohol and cigarettes which the deceased consumed each week of his life. Obviously, her evidence as to the period in 2012 and 2013, when the deceased's psychological condition improved, is not entirely consistent. When cross-examined, the applicant gave quite different accounts of the time and duration of this period to that contained in her written evidence.
109. There is some corroboration of the applicant's evidence in relation to the deceased's smoking in the short statements of Ms Blanning and Mr Elliott. The former stated that the deceased was "a chain smoker". Mr Elliott stated that as time passed, "he was smoking more and more". There is also corroboration of the applicant's evidence as to the extent of the deceased's alcohol consumption in the last years of his life in the MAC of Dr Steele. It is, of course, possible, as Mr Flett argued, that all this evidence is unreliable. On reflection, I do not believe it is appropriate to reach that conclusion. On the other hand, the contemporaneous evidence cannot be ignored.
110. The respondent sought to rely upon the contemporaneous reporting, before the MAC to counter the applicant's observations that there was a considerable increase in the deceased's use of alcohol and tobacco, which persisted up to his death. Dr Bannon last saw the deceased in early 2013, probably just before he and the applicant moved to the caravan park. She reported considerable improvement in his mood. She recorded that he was consuming three to five standard drinks, five days a week. In June, shortly after he moved to the caravan park, Dr Powell reported that he was smoking 12 cigarettes a day and has two beers after dinner. Then, on 15 August 2013, Professor Robertson recorded that the deceased had modified his use of alcohol considerably and was contemplating ceasing smoking.
111. The history recorded by Dr Powell suggests the deceased smoked slightly less in mid-2013 than he did in 2007, when Dr Looi recorded that he smoked 16 cigarettes a day.

112. As I understand the applicant's oral evidence, there was some improvement in the deceased's psychological symptoms from shortly after the time he was discharged by the respondent in March 2012. When Mr Flett pressed the applicant to be more precise in respect of the period, she responded that it was:

"From the period of him getting out of the police force to shortly after moving up the coast".

The applicant also stated that it was probably about six months after moving to the caravan park that "he went back into drinking and smoking heavily again".

113. That evidence suggests a lengthy period. The deceased was discharged from the Police Force on 15 March 2012. A period "six months after" the deceased and the applicant moved into the caravan park probably ends around September 2013. Certainly, on this evidence, the deceased smoked and drank less for a period of, at least, a year and probably longer. That is consistent with the histories recorded by Dr Bannon, Dr Powell and Professor Robertson. It is inconsistent with the evidence in the applicant's statement.
114. Professor Keogh is the only medical practitioner who attempts to establish the increase in the deceased's cigarette consumption in the last years of his life. She assumed, based on her survey of the evidence, that his smoking increased by 15 cigarettes a day between 2011 and his death in 2014. In one respect, her calculations are favourable to the applicant's case, as they make no allowance for the undoubted diminution in the deceased's use of alcohol and tobacco in 2012 and 2013. Neither the applicant's oral evidence, nor the medical histories, support a conclusion that, during this period, the deceased's smoking was significantly greater than it would have been had he not suffered PTSD.
115. On the other hand, Professor Keogh has confined the deceased's increase in smoking to the period from 2011 to his death. That is not consistent with the evidence. The evidence supports a conclusion that the deceased's alcohol and cigarette consumption increased from, at least, 2008 and possibly before. The notes of Dr Looi records a clear diagnosis of PTSD as early as the 8 September 2008. The applicant's evidence is consistent with the deceased smoking and drinking more after the incident during his employment in 2008.
116. The evidence is probably consistent with some gradual increase in smoking and alcohol use between 2008 and 2011. The deceased's accounts of drinking voluminous quantities of alcohol to Dr Bannon in 2011 and to Dr George in 2012, in my opinion, are likely to be reliable. There is no compelling reason why the deceased would have lied about a marked increase in his alcohol consumption. As Mr Callaway submitted, however, there is a possible explanation as to why he might subsequently downplay his alcohol consumption, when consulting medical practitioners.
117. The evidence is important for two reasons. It, of course, suggests a gradual but substantial increase in the deceased's alcohol consumption commencing in 2008. The deceased also said that he smoked more when he drank.
118. In my opinion, the evidence, taken as a whole, provides a proper basis to find that there was a significant increase in both alcohol and cigarette consumption from 2008 until 2012, and from 2013 until the deceased's death in 2014. While it is not possible to calculate increased consumption of either substance with mathematical certainty, it is likely that the deceased's increased cigarette consumption was similar to that assumed by Professor Keogh. That leads to a consideration of the question of whether the deceased's death resulted from PTSD through the agency of increased smoking and drinking.

## The cause of the deceased's death

119. Some of the evidence, which addresses this issue, does so on unproven assumptions. Dr Bertucen, for example, offers a tentative opinion of a causal relationship between injury and death on an assumption that the deceased suffered from PTSD throughout both periods during which he served as an officer. There is no acceptable evidence that the deceased suffered from PTSD before 2004. Even after that date, he was capable of uninterrupted work, for many years. He may have received medical treatment following the incident in 2004. But there is no record of continuous medical treatment until September 2008.
120. One of the assumptions put to Professors Richards and Duflou by the applicant's solicitor is also inconsistent with the evidence proven in the case. Nonetheless, it is apparent from his reports that Professor Richards carefully considered the evidence and his ultimate opinion is based upon correct factual assumptions. That is possibly true of Professor Duflou's opinion but it is less clear.
121. The opinions of Professor Richards and Duflou, on the one hand, and Professor Keogh, on the other are reconcilable on many of the issues in dispute. They are diametrically opposed on the ultimate issue. Professor Richards accepts that increased drinking and smoking caused the deceased to die earlier than if he had not suffered a psychological injury. Professor Duflou agrees. Professor Keogh expresses the opinion that had he not been injured, he would have died at about the same time.
122. Mr Callaway referred me to *EMI (Australia) Ltd v Bes* [1970] 2 NSWLR 238 in his written submissions. I assume he was suggesting that I should draw an inference or inferences in favour of the applicant from the lay evidence in the case. I doubt, however, whether there is much scope for the Commission to draw inferences from lay evidence in respect of the issue whether death results from injury. There are competing opinions from medical practitioners who possess undoubted expertise in the areas of medical science relevant to the issue; see *Wiki v Atlantis Relocations (NSW) Pty Ltd* (2004) 60 NSWLR 127 and *Tudor Capital Australia Pty Limited v Christensen* [2017] NSWCA 260 (17 October 2017). It is necessary to engage with the medical evidence addressing the contested issues.
123. Within the larger issue of whether death results from injury, there is disagreement between the medical witnesses on two subsidiary issues. First, whether the deceased suffered from hypertension. Secondly, whether he had symptoms of ischaemic heart disease in his lifetime. The latter must be speculation on Professor Richard's part, as it is impossible to determine now whether the discomfort the deceased suffered in his torso, before his death, was a manifestation of ischaemic heart disease: or his significant shoulder problem; or any number of other ailments. While Professor Richard's must be rejected on this issue, it is not of critical importance for the outcome of the case.
124. Conversely, the issue of hypertension, is of fundamental importance. Professor Keogh initially raised the possibility that the deceased may have suffered hypertension, exacerbated by his abuse of alcohol. She referred to the size of his heart at autopsy, the presence of small cerebral infarcts and the record of the coroner that the deceased had a history of hypertension.
125. In his initial report, Professor Duflou stated that it was not possible to establish whether the deceased suffered from hypertension, as his heart size at autopsy was "above the normal range but only slightly so". He referred to the likelihood that Dr Mina would have tested the deceased's blood pressure, at the consultation in the days before his death.
126. By her supplementary report of 20 July 2018, Professor Keogh stated that it was not possible to be definitive as to whether the deceased suffered "systemic hypertension" as there was only one blood pressure reading, during his lifetime. Nonetheless, she reiterated that:

“the likelihood of uncontrolled hypertension is indicated by the increased left ventricular wall thickness and the presence on autopsy of brain of old infarcts and macrophages and reactive astrocytes in the motor cortex and basal ganglia of the brain.”

Thus, while Professor Keogh could not offer a definitive opinion, it is evident that she thought it likely that the deceased had hypertension.

127. Professor Richards had access to the reports of Professors Keogh and Duflou when preparing his primary report. He was in no doubt that the material available suggested hypertension. He said this, on the issue of hypertension:

“At post-mortem examination, his heart was of above normal weight, consistent with long-standing hypertension, likely aggravated by excessive alcohol use related to post-traumatic stress disorder”.

128. By her supplementary report of 10 December 2018, Professor Keogh stated that she did not see any definite evidence of hypertension. She observed that the deceased’s heart at autopsy weighed 474 gm which was inside the “population normal”. The average heart size for a man of the deceased’s size was 400 grams. But, 10% of males of the deceased’s size had hearts of “around 500 gm”. Absent an enlarged heart, the other signs of hypertension were equivocal.
129. By an email of 20 December 2018, Professor Duflou commented on the reports of Professors Keogh and Richards. He “didn’t think there is much difference of opinion” between the doctors. For his part, he would prefer an older paper on heart size than that cited by Professor Keogh. According to his calculations, the deceased’s heart was “only slightly outside” the normal range, but 142 gm above the mean for the deceased’s height made “it 43% heavier than average. In short, it’s a bigger heart than normal which I would typically see at autopsy.” Professor Duflou does not specifically resile from his opinion that it was not possible to establish whether the deceased had hypertension given his heart size. Nonetheless, it is clear from his reports, that he thought increased alcohol consumption was implicated in the deceased’s death.
130. Professor Richards considered these opinions in a report of 11 January 2019. He said that he agreed with Professor Duflou in respect of the deceased having larger than normal heart size and disagreed with Professor Keogh on the incremental damage caused to the coronary arteries by increased drinking and smoking. This report does not greatly assist in resolving the question of whether the deceased had hypertension. The reports give the impression that, by this stage of the medical debate, the expert witnesses were entrenched in their respective positions.
131. Mr Flett emphasised the absence of evidence from Dr Mina, which may have resolved the hypertension issue as part of his submission that the applicant’s case was “undercooked”. The relevance of Dr Mina’s evidence is apparent from Professor Duflou’s initial report. Its importance to the resolution of the controversy between Professors Keogh and Richards may not have been evident until Professor Keogh’s final report.
132. In my opinion, it is not open to the Commission to draw an inference in accordance with the principle in *Jones v Dunkel* [1959] HCA 8; (1959) 101 CLR 298. It was open to both parties to obtain the clinical notes of Dr Mina, by way of a Direction for Production, if they thought it was relevant. Obviously, I would have made orders for such a direction in the circumstances of the case. On the other hand, it is appropriate that a party adduces all the essential evidence on an issue on which it carries the burden of proof. More so when the evidence on that issue is meagre: see *Australian Securities and Investments Commission v Hellicar & Ors* [2012] HCA 17 (3 May 2012) at par 167 et seq.

133. The pathologist who undertook the autopsy report for the coroner observed that the deceased had a “mildly enlarged heart”. That probably offers some comfort to each side of the debate. The autopsy report is compiled from “a variety of sources, which may include police and medical records.” It contains a warning that “some personal and other details may be inaccurate.” Nonetheless, the pathologist, who prepared the report, recorded that the deceased had a history of hypertension. The relevant part of the report states:

“Significant past medical history included hypertension, hypercholesterolaemia treated with Rosuvastatin 10mg each evening, gastro- oesophageal reflux disease treated with Esomeprazole 20mg daily. He was also a heavy smoker of cigarettes.”

134. The above is a fairly insightful account of the deceased’s past medical treatment. It is not clear whether it came from a medical practitioner, but one would not anticipate that a colleague would have such detailed knowledge of the medications, which the deceased had been prescribed in the past. I appreciate that it is hearsay evidence, with the undoubted limitations of such evidence. Nonetheless, it appears to be a reliable account of relevant parts of the deceased’s medical history. Certainly, the pathologist accepted that hypertension was a “background” factor in the deceased’s death. The pathologist concluded, of course, that the deceased died of:

“ischaemic heart disease due to coronary artery atherosclerosis on a background of alleged hypertension and treated hypercholesterolaemia.”

135. In my opinion, this evidence, the history of hypertension and the opinion of the pathologist, lends some support to the opinions of Professors Duflou and Richards. Apart from the history recorded by the pathologist, there were other indications in the post-mortem that are consistent with the existence of hypertension. Professor Keogh set these out in some detail in her initial reports. It was only in her subsequent reports that she came to doubt the presence of hypertension. In her final report, she concluded that there “was no definite evidence of it.” I have concluded that the evidence favours the contrary view of Professor Richards. My conclusion may, in part, reflect the difference between the medical expert’s quest for “definite evidence” and the Commission’s obligation to determine issues on the balance of probabilities.

136. In those circumstances, the weight of the evidence favours the opinion expressed by Professor Richards and supported by Professor Duflou that the deceased’s increased use of tobacco and alcohol accelerated the deceased’s ischaemic heart disease and caused him to die earlier than he would have had he not suffered from PTSD. If both the deceased’s alcohol and cigarette consumption were operative causes of his ischaemic heart disease, the case that they accelerated his death is more cogent. Accordingly, I find that the deceased’s death results from injury. This finding does not negate the likelihood that the deceased had pre-existing ischaemic heart disease at the time of his psychological injury or that he was likely to die prematurely because of that disease.

137. The effect of a finding in favour of the worker on causation is still best illustrated by the reasoning of Latham CJ in *Ward v Corrimal-Balgownie Collieries Ltd* [1938] HCA 70; (1938) 61 CLR 120 (23 December 1938). He said this:

“Compensation is paid under the Act in respect of death or total or partial incapacity resulting from an injury (secs. 8 and 9) not in respect of the injury itself (See *Harwood v. Wyken Colliery Co.* [1]; *King v. Port of London Authority*[2]). Incapacity under sec. 9 is measured by loss of earning power (*Wicks v. Union Steamship Co. of New Zealand Ltd.*[3]; *Ball v. William Hunt & Sons Ltd.*[4]).

In determining whether incapacity results from an injury the law necessarily adopts an idea of causation which, in a sense, isolates the injury as a causative element from other elements which are taken for granted or ignored. If the addition of the injury to other concurrently existing facts brings about the incapacity, then the incapacity is regarded as resulting from the injury, although in fact it results from the injury taken together with the other circumstances.

The legal doctrine may be illustrated by considering the case of a worker who has a condition of heart disease which is not an injury within the meaning of the Act and which has not produced any incapacity. Although the worker has heart disease, he is able to earn full wages, and, as his earning capacity is not diminished, he is suffering from no incapacity within the meaning of the Act (*Wicks v. Union Steamship Co. of New Zealand Ltd.* [5]). If such a worker then receives an injury within the meaning of the Act and suffers incapacity as the consequence of the injury added to his heart disease, then the incapacity (total or partial) in those circumstances results from the injury. The injury is an element which only completes the tale of circumstances which constitutes the cause of the incapacity in the non-legal sense: but in the legal sense it is itself the cause of the incapacity which therefore is said to 'result' from it (See *Clover, Clayton & Co.Ltd. v. Hughes* [6]; *Partridge Jones and John Paton Ltd. v. James* [7]—cases on 'arising out of the employment'). If an employer employs a man who suffers from some defect, though the defect produces no incapacity, the employer runs the risk that incapacity may more readily result from an injury to such a man than from an injury to a man who does not suffer from any such defect."

138. There are aspects of Mr Callaway's argument, which I have not addressed in the above reasoning. In my opinion, the evidence does not support a finding, on the balance of probabilities, that the deceased's psychological injury precluded him from giving up tobacco. Certainly, both the lay and medical evidence establish that the deceased unsuccessfully attempted to cease smoking after the onset of his disease. But, he had unsuccessfully attempted to cease smoking in 2001, prior to the onset of PTSD. In those circumstances, it is difficult to draw an inference that the injury played a material role in his attempts to cease smoking.
139. There is also a good deal of evidence that the deceased was non-compliant with medical advice after the onset of his illness and ceased to take medication, at least, in the last year of his life. There is evidence from Professor Keogh that non-compliance was a risk factor which contributed to the development of his ischaemic heart disease. The evidence that non-compliance resulted from the injury is not strong. Once again, it is difficult to make a positive finding that non-compliance caused by his injury materially contributed to the deceased's premature death.
140. As both the applicant and the second respondent were dependent upon the deceased at the time of his death, I propose to make orders that the respondent pay the applicant the compensation applicable to the death of a worker pursuant to section 25 of the 1987 Act. I will grant liberty to apply in respect of the issues of apportionment and the calculation of the award of weekly compensation. I also order that the respondent pay the applicant's costs. Any issue in respect of those costs, can also be addressed at a telephone conference.