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HEARING DATE(s):	24 July 2000
JUDGMENT OF:	Mr Greg Cavanagh SM

CATCHWORDS:

REPRESENTATION:

Counsel:

Counsel assisting the Coroner:	Mr Michael Grant
Counsel for Territory Health Service:	Ms Sally Seivers

Solicitors:

Territory Health Service:	Cridlands

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IN THE CORONERS COURT
AT DARWIN IN THE NORTHERN
TERRITORY OF AUSTRALIA

No. D0024/1999

AN INQUEST INTO THE DEATH OF

FRANCIS KAMARANGA

FINDINGS

(Delivered 6 September 2000)

Mr Cavanagh SM:

THE NATURE AND SCOPE OF THE INQUEST

1. Francis Kamaranga ("the deceased") was pronounced dead at the Royal Darwin Hospital at 2.31pm on 18 February 1999. He was 28 years old at the time of his death. He was a male of Aboriginal descent.

2. Section 12(1) of the *Coroners Act* ("the Act") defines a "reportable death" to mean a death that:

> "appears to have been unexpected, unnatural or violent, or to have resulted directly or indirectly from an accident or injury".

3. For reasons that appear in the body of these Findings, the death fell within the ambit of that definition and this Inquest is held as a matter of discretion pursuant to s15(2) of the Act. Section 34(1) of the Act details the matters that an investigating Coroner is required to find during the course of an Inquest into a death. That section provides:

> "(1) A coroner investigating -

(a) a death shall, if possible, find -

(i) the identity of the deceased person;

(ii) the time and place of death;

(iii) the cause of death;

(iv) the particulars needed to register the death under the Births, Deaths and Marriages Registration Act;

(v) any relevant circumstances concerning the death."

4. Section 34(2) of the Act operates to extend my function as follows:

> "A coroner may comment on a matter, including public health or safety or the administration of justice, connected with the death or disaster being investigated".

5. The duties and discretions set out in subs 34(1) and (2) are enlarged by s35 of the Act, which provides as follows:

(1) A coroner may report to the Attorney-General on a death or disaster investigated by the coroner.

(2) A coroner may make recommendations to the Attorney-General on a matter, including public health or safety or the administration of justice connected with a death or disaster investigated by the coroner.

(3) A coroner shall report to the Commissioner of Police and the Director of Public Prosecutions appointed under the *Director of Public Prosecutions Act* if the coroner believes that a crime may have been committed in connection with a death or disaster investigated by the coroner.

6. The public Inquest into the death was held at the Magistrates Court complex in Darwin on 24 and 25 July 2000. Counsel assisting me was Mr Michael Grant. Ms Sally Seivers appeared on behalf of Territory Health Services, the agency responsible for the administration of the Royal Darwin Hospital.

Mr Eric Hutton appeared on behalf of the Australian Red Cross Blood Service. Mr Michael Grove appeared on behalf of the St John Ambulance. I granted leave to those parties to appear pursuant to s40(3) of the Act.

7. The family of the deceased was not represented at the Inquest. At the commencement of the Inquest I was informed by counsel assisting that he had had discussions with Mr John Duguid of the North Australian Aboriginal Legal Aid Service in relation to the matter. Mr Duguid apparently indicated that NAALAS had made arrangements with the Deputy Coroner to procure a copy of these Findings, and on the basis of that arrangement the family of the deceased did not seek to be represented at the Inquest.

8. Counsel assisting me also advised that he had had discussions with Hoechst Marion Roussel Australia ("HMR"), the manufacturer of the agent Haemaccel, which in the months following the death was identified by the Victorian Institute of Forensic Pathology as connected with the death. HMR were aware of the conduct of the Inquest and similarly did not seek to be represented.

FORMAL FINDINGS

(i) The identity of the deceased person was Francis Kamaranga. It is also apparent from various material tendered during the course of the Inquest that his Aboriginal name was at times spelt “Kamarunga”. Police records disclose that the deceased was also known as Albert Jimmy or Jimmy Albert. The deceased was a male Aboriginal Australian born at Barunga in the Northern Territory of Australia. The deceased's hospital records indicate that his date of birth was 23 April 1970.

(ii) The time and place of death was at the Royal Darwin Hospital in the Northern Territory at 0231 hours on 18 February 1999.

(iii) The cause of the death was asphyxiation following severe swelling to the larynx due to anaphylactoid reaction.

(iv) The particulars required to register the death are:

(1) The deceased was a male.
(2) The deceased was of Australian Aboriginal origin.
(3) The death was reported to the Coroner.
(4) The cause of death was confirmed by post-mortem examination.
(5) The death was caused in the manner described in paragraph (iii) above.
(6) The pathologist viewed the body after death.
(7) The pathologist was Dr Terence John Sinton of Royal Darwin Hospital.
(8) The deceased had no fixed place of abode.
(9) The deceased was unemployed.

RELEVANT CIRCUMSTANCES CONCERNING THE DEATH INCLUDING COMMENTS, REPORTS AND RECOMMENDATIONS

9. The evidence disclosed that the deceased had attended at flat 113 at the Kurringal Complex on the night of 17 February 1999. A number of people were present in the flat on that night in varying states of intoxication. The deceased became involved in a dispute with another guest by the name of Tanya Kelly. There was a slight physical altercation during which Ms Kelly broke a ceramic cup from which she had been drinking and threw it at the deceased. The cup hit the deceased on the right wrist and caused a deep laceration. Subsequent medical examination indicated that the laceration severed the radial artery.

10. The injury to the deceased was sustained at some time around midnight. It is impossible to fix a precise time as the recollections the various witnesses interviewed by police were not sufficiently specific in that respect. In any event, it would appear that the injury to the deceased was not immediately brought to the attention of the people in attendance at the flat or, if it was, there was no appreciation of its severity.

11. At about 12.26am a security guard attended the flat 113 in response to noise emanating from the area. The security guard observed that the deceased was unconscious and had apparently lost a large amount of blood. The security guard called the St John Ambulance. An ambulance unit arrived at 12.43am. The ambulance officers applied a pressure bandage to the wound and administered oxygen, saline and Haemaccel, the last two agents intravenously. Evidence tendered during the course of the Inquest shows that Haemaccel is a plasma volume expander. It is used to prevent or treat shock associated with a reduction in blood volume, in this case due to the haemorrhage at the deceased's wrist. Once the patient had been stabilised, the ambulance departed for the Royal Darwin Hospital at 1.08am.

12. The ambulance arrived at the Royal Darwin Hospital at about 1.20am. On arrival the deceased's blood pressure was 98/68 and his pulse was 100 beats per minute. The deceased was fitted with two intravenous tubes. The Fluid Balance Chart in the Royal Darwin Hospital records shows that a number of fluids were then administered. At 1.22am the patient was given Haemaccel and saline. At 1.30am 500mls of Haemaccel were administered through each tube. At 1.37am a further 500mls of saline and 500mls of Haemaccel were administered. At 1.45am the deceased was given 450mls of O negative blood. A blood sample had been taken from the deceased at the time of his arrival at the Royal Darwin Hospital. There had been insufficient time to determine the deceased's blood type by 1.45am and for that reason the deceased was given “universal donor” O negative blood.

13. At 1.47am the deceased became distressed and restless and complained of thirst and throat pains. At that time his tongue was noted to be swollen. This presentation was recorded contemporaneously by Sister Marienne Shanahan, the clinical nurse consultant in charge of the nursing team attending to the patient on that night. At 1.55am the deceased's abdomen was noted to be distended. An in-dwelling urinary catheter was fitted by nursing staff. That treatment was only partially successful in alleviating the distention.

14. By 2.00am the deceased was noted to be making gurgling sounds on respiration and Dr Simon Maffey, the Intensive Care Registrar, was called to the Accident and Emergency Unit to assist. At that same time a further litre of O negative blood was administered.

15. Dr Maffey arrived in the Accident and Emergency Unit at approximately 2.05am. On arrival he saw the patient struggling on the bed and observed that he was incontinent. Dr Leong, the Surgical Registrar, was already in attendance on the patient. Dr Leong was attempting to insert a nasogastric tube. Dr Maffey went to the deceased's head and attempted to assist the deceased in his respiratory effort by use of a self-inflating bag. Dr Maffey noted that the deceased had a grotesquely swollen face and neck and a pale, protruding tongue which was swollen to something in the order of 4 or 5 times its normal size.

16. Dr Maffey had only been in attendance as the Accident and Emergency Unit for a matter of seconds before the deceased went into respiratory arrest. He immediately suspected anaphylaxis (in lay terms a gross allergic reaction) and informed the rest of the team. Intravenous administration of adrenaline was commenced immediately (the administration of adrenaline being the standard medical response). Dr Maffey then attempted to intubate

the patient. He was unsuccessful in those efforts as the deceased's pharyngeal tissue and tongue were swollen to such an extent that the tube could not be introduced to the deceased's mouth.

17. It was apparent to both Dr Maffey and Dr Leong that a surgical airway had to be established. There followed a very brief discussion between Drs Maffey and Leong as to who was the appropriate person to perform the procedure. It was resolved that Dr Leong would do so. Dr Leong made a vertical incision in the deceased's neck and located the trachea. A needle cricothyroidotomy was then attempted. It was hoped by this procedure to introduce oxygen to the deceased's lungs through a large canula. This procedure failed as there was no jet ventilating equipment in the Accident and Emergency Unit, and attempts to ventilate the patient by adapting a tube to the oxygen outlet on the wall were not successful. Dr Leong then extended the incision by way of a transverse cut. An airway was established at 2.15am.

18. By that time the deceased still had a rhythm on the ECG machine and a weak and occasional pulse. Further adrenaline was administered together with lignocaine, atropine and hydrocortisone. The patient was also treated with DC shocks. Despite these efforts the patient lapsed into an asystole rhythm and was pronounced dead at 2.31am.

19. An autopsy was performed at 9.30am on 18 February 1999. The forensic pathologist concluded that the deceased died as a result of acute respiratory obstruction following severe swelling to the larynx after an acute anaphylactic reaction. The forensic pathologist was unable to identify the agent that caused the anaphylaxis. In May 1999 the matter was referred to Dr Michael Burke, a forensic pathologist at the Victorian Institute of Forensic Medicine. He found the cause of death to be asphyxia secondary to an anaphylactoid reaction to Haemaccel.

20. A number of issues arise from this tragic course of events. I will deal with each in turn below.

The cause of the anaphylactoid reaction

21. The first matter calling for some consideration is the likely cause of the reaction. This is a matter which did not at first instance appear to be attended by any doubt. As stated above, Dr Burke of the Victorian Institute of Forensic Medicine formed the view that Haemaccel was the causative agent. That finding was subsequently queried by Dr Sinton, the pathologist who performed the autopsy, in discussion with the Coroner's Constable. Dr Sinton expressed the view that an anaphalaxis would be expected to manifest shortly after the administration of the causative agent. In that the Haemaccel was first administered at 1.02am and the reaction did not manifest until 1.47am, Dr Sinton expressed some doubt as to whether the timeframe was consistent with Haemaccel causing the reaction.

22. The matter was raised with Dr Burke prior to the Inquest. Dr Burke conceded that he had not considered any other agent as a possible or likely cause. Specifically, he did not consider the administration of the O negative blood some minutes before the symptoms of anaphylaxis first manifested. Dr Burke also indicated that any determination as to whether Haemaccel or blood was the more likely cause of the reaction was beyond the bounds of his expertise. These qualifications were reduced by Dr Burke to the form of a letter dated 18 July 2000, a copy of which was tendered during the course of the Inquest.

23. During the course of the Inquest I took evidence from Associate Professor Anthony Brown, a physician specialising in emergency medicine, and Dr Robert Heddle, a specialist immunologist. Dr Brown has a special interest in anaphylaxis and has lectured on its recognition and management

nationally and overseas. Dr Heddle specialises in allergic reactions including anaphylaxis, although not specifically in relation to reactions in the transfusion context. Both gentlemen were, in my assessment, amply qualified to give an opinion in relation to the likely cause of the reaction in this case.

24. That Dr Burke had initially seized upon Haemaccel as the likely causative agent is not surprising in the circumstances. It is apparent from Associate Professor Brown's evidence that in late 1998 and early 1999 there was a sudden increase in adverse reactions to Haemaccel reported worldwide, particularly in Czechoslovakia, Columbia and Australasia. There were 72 adverse drug reactions to Haemaccel documented in Australia between October 1998 and April 1999. Associate Professor Brown's report of 19 July 2000 describes the cause of the problem as:

> "… a change early in 1998 to the sterilisation time and an increased temperature of the filter medium, leading to the release of small negatively charged particles that then triggered the anaphylactoid reactions."

25. Against that background, and given that the death in this case occurred in February 1999, Haemaccel presented as the obvious cause. In that same report, however, Associate Professor Brown went on to consider the possibility that the reaction was triggered by the blood product administered at 1.45am. He stated relevantly:-

> "Blood has also long been recognised as a potential cause of anaphylactic and anaphylactoid reactions. These are more likely with whole blood rather then packed red blood cells, suggesting that plasma protein constituents are involved as triggers. Reactions may occur in any individual, irrespective of whether blood has been fully cross-matched or given as 'universal donor' O negative blood (as in Mr Kamaranga's case) ….. Severe or fatal anaphylactoid reactions to O negative blood are exceedingly rare. None are known to have occurred in Queensland in the last 12 years and none in the UK in 1997 …. Only

three or four have been known to Professor J Isbister of Royal North Shore Hospital, Sydney in his extensive 20+ year clinical haematology career."

26. In his written report, Associate Professor Brown opined that the cause of the reaction was more likely to be the O negative blood rather than the Haemaccel, even though at the time Haemaccel was causing a higher incidence of anaphylactoid reactions. The basis for that view was that most reactions would be expected to occur within five to ten minutes of delivery. That opinion was provisional in nature and was revised by Associate Professor Brown prior to giving his oral evidence. This revision followed a further consideration of the literature and certain material that came to light during the course of the Inquest, which material was not available to Associate Professor Brown at the time he framed his initial opinion. During the course of his oral evidence Associate Professor Brown made three crucial observations.

27. First, he noted that whilst most reactions would occur within five to ten minutes, it was recorded in the literature that certain reactions or symptomatology might be delayed by up to 60 minutes following the administration of the causative agent.

28. Secondly, Associate Professor Brown made the observation that the time of first administration of the agent might not be the critical factor. The reaction might be triggered not simply by the first introduction of a particular agent, but by the volume or rate of flow of that introduction. Thus, although the Haemaccel was first introduced at 1.02am, either of the large doses administered at 1.30am or 1.37am may have been the triggering dose.

29. Thirdly, Associate Professor Brown agreed with an observation made by Dr Heddle in the course of his evidence to the effect that swelling of the tongue

due to vascular leakage would have taken longer than two minutes to manifest following administration of the causative agent. In that the O negative blood product was first administered at 1.45am, and swelling of the tongue was first observed at 1.47am, the blood product was the less likely cause of that oedema. In light of those matters, Associate Professor Brown expressed the opinion that the Haemaccel was, on balance, the more likely cause.

30. That opinion was in accord with that expressed by Dr Heddle. Dr Heddle also provided a written report which was tendered during the course of the Inquest. Dr Heddle's approach in the written document was extremely cautious in that he considered himself unable to find whether the Haemaccel or the blood was the more probable cause of the reaction. Again, that opinion was expressed to be provisional and subject to a continuing search of relevant data. During the course of his oral evidence, Dr Heddle was taken to the observation in his written report to the effect that he had some doubt as to whether swelling of the tongue would develop within two minutes of the administration of the causative agent. He agreed that if the observation of a swollen tongue at 1.47am was correctly recorded, then Haemaccel was the more likely cause of the reaction. As I have already stated, Sister Shanahan gave evidence during the course of the inquest that leaves me in no doubt as to the fact that some swelling of the tongue was observed at 1.47am and recorded in the notes contemporaneously with that observation. Dr Fergus, the treating doctor, made notes after the incident which indicated that tongue swelling was first noticed at 1:55am. I do not accept that particular note and prefer the evidence of Nurse Shanahan that the tongue swelling was first noticed at 1:47am.

31. In all the circumstances I have no difficulty in finding on the balance of probabilities that Haemaccel was the cause of the anaphylactoid reaction in this case. The evidence of both Associate Professor Brown and Dr Heddle

is to that effect. In saying this I note and commend the caution and diligence with which each of these medical practitioners has approached the issues. I am fortified in my conclusion by the fact that at the very time these events transpired there had been a change in the manufacturing process for Haemaccel, and it is now well-documented this change gave rise to a manifold increase in the number of reactions to the substance. I do not make my finding solely on the basis of that coincidence, but in conjunction with the evidence of Associate Professor Brown and Dr Heddle, the conclusion is compelling.

32. I should also note by way of addendum that Associate Professor Brown and Dr Heddle both expressed the view that the deceased's high blood alcohol content at the time he was admitted to the Royal Darwin Hospital may well have potentiated the release of histamines and contributed to the reaction. Neither suggests that the alcohol could have been the sole cause of the reaction and the matter does not call for any other comment.

33. Having found that the reaction in his particular case was attributable to Haemaccel, the question then arises whether any consequent recommendation is necessary. I am of the view that it is not. Associate Professor Brown's evidence was to the effect that following the increase in adverse reactions, HMR instigated an "extensive high-quality and exemplary investigation" into the cause of the problem. The manufacturing process was immediately revised such that strict new temperature and sterilisation times were set and mandatory biological Plasma Activating Substances testing of every batch was instituted. It would appear that nothing remains to be done in that respect.

34. The only other question is whether the inherent tendency of some patients to react to Haemaccel militates against its use. Quite clearly it does not. The substance is an important agent for use in emergency medicine. It

would appear from the evidence that it is impossible to guard against the slight risk of reaction. The overwhelming clinical judgment is that it is a risk necessarily taken in order to save lives. There is no suggestion that its administration by either St John or the Royal Darwin Hospital was inappropriate in these circumstances. The potentiality for an adverse patient reaction is simply one of those properly assumed risks that attend emergency medical treatment.

Treatment by the St John Ambulance

35. The course of the attendance and treatment administered by the St John Ambulance is set out briefly above. At the time of the first attendance by the St John Ambulance the deceased had a barely discernible pulse and no verbal or motor responses. The St John paramedics spent approximately 25 minutes at the scene stabilising the patient. The deceased was delivered to the hospital in a normal state of consciousness and with satisfactory pulse, blood pressure and respiratory rates. The evidence shows that the deceased was displaying no signs of anaphylaxis at the time of his delivery to the Royal Darwin Hospital and there is no suggestion of any causal nexus between the treatment administered by the St John Ambulance and the subsequent death of the deceased.

36. These matters being so, their involvement may have passed without comment or consideration but for two matters raised by Associate Professor Brown during the course of his evidence. First, Associate Professor Brown expressed the view that 25 minutes on-scene time was excessive given that the Royal Darwin Hospital was only an 11 minute drive away. Secondly, Associate Professor Brown questioned the role for pre-hospital intravenous fluids in trauma patients. He suggested that the better treatment would have been to administer only airway care, oxygen and a pressure bandage prior to departure for the hospital. These views were endorsed by Dr Didier Palmer,

the Director of Accident and Emergency at the Royal Darwin Hospital, who also gave evidence during the course of the Inquest.

37. During the course of their oral evidence Associate Professor Brown and Dr Palmer both qualified those views by observing that the appropriate treatment is largely a matter of judgment that falls to be made by the paramedic on the scene. The treatment that was given in this particular case in pursuance of that judgment fell well within the standard guidelines for managing a patient with this type of presentation.

38. Mr David Hoschke, one of the attending paramedics, was called to give evidence during the course of the Inquest. Mr Hoschke was an impressive witness and his evidence was instructive. Mr Hoschke agreed that the scene time of 25 minutes was longer than optimum and that he preferred to keep scene times to somewhere around 10 minutes. He painted a vivid picture, however, of the chaotic scene that presented on arrival. Security guards and police officers were already in attendance. The patient was covered in blood and this, together with various other aspects of the scene, gave rise to a significant biological hazard. It was not easy at first instance to discern the full extent of the trauma suffered by the deceased. Moreover, the paramedics were subject to various forms of harassment and interference by a number of intoxicated people present at the scene.

39. In all the circumstances, I am satisfied that the attending paramedics understood the importance of conveying the patient to the hospital with a minimum of delay. They were thwarted in that effort by the factors detailed above.

40. A number of matters are clear from the evidence. First, the treatment administered by the St John Ambulance paramedics was appropriate and within standard guidelines. The paramedics made a judgment call at the

time as to the extent of on-scene care to be administered to the deceased. That judgment was not shown to be inappropriate. Secondly, there was no causal nexus between the treatment administered by the St John paramedics and the subsequent death of the deceased. If anything, the treatment administered by the paramedics brought the patient to a stable condition prior to his delivery to hospital.

41. That leaves the suggestion by Associate Professor Brown and Dr Palmer that pre-hospital care may not be all that useful in cases of penetrating injuries. This is a debate that apparently ensues in emergency medicine circles. I do not consider that it is necessary or appropriate for me to enter into that debate, nor is it something that falls within the parameters of this Inquest.

The treatment administered at the Royal Darwin Hospital

42. The final matter that falls for consideration is the appropriateness or otherwise of the treatment administered at the Royal Darwin Hospital. On all the evidence the treatment progressed in satisfactory fashion up to 1.47am. The administration of both the blood product and the Haemaccel was appropriate in the circumstances. As I have already observed, the slight risk of some reaction to those products quite clearly did not militate against their use.

43. It is from 1.47am, however, that the matter becomes problematic. I have already found that the deceased was exhibiting symptoms of anaphylaxsis from that time. It was Dr Maffey's evidence that the deceased was obviously in the throes of anaphylaxsis when he arrived at the Accident and Emergency Unit at 2.05am, yet the anaphylaxis was not diagnosed until that time.

44. There was some suggestion during the course of the evidence and during the submissions of counsel for the Territory Health Services that the failure to recognise the anaphylaxis was understandable in that alcohol masked the full extent of the reaction, and that the symptoms manifested by the deceased were equally explicable by reference to other diagnoses. Whilst that may have been the case for part of the time after 1.47am, the reaction should have been identified prior to 2.05am. Certainly the severe swelling of the facial region and the gurgling breathing that was present at 2.00am should have alerted hospital staff to the true nature of the situation.

45. It was also suggested by counsel for Territory Health Services that even if it were the case that anaphylaxis was not identified until 2.05am, the treatment given up to that time was nevertheless an appropriate treatment regime for anaphylaxis. I do not wholly accept that to be the case. The appropriate treatment included the immediate administration of adrenaline. As it transpired, adrenaline was not administered until 2.05am.

46. Having said this, I do not seek to suggest that the omission stemmed from some deficiency in the procedures in the Accident and Emergency Unit. It was a matter of simple human error in the context of an emergency medical situation. I also do not seek to suggest that had adrenaline been administered earlier in the piece that the need for the attempt at establishing a surgical airway would have been averted, or that the adverse patient outcome would have been avoided.

47. Two further matters call for some attention.

48. First, it took some ten minutes to establish the surgical airway. It is clear from the evidence given by Dr Maffey that the magnitude of the swelling rendered the task difficult in the extreme. It was Associate Professor Brown's opinion that the performance of such a procedure in these

circumstances would be highly problematic, and he expressed sympathy for any emergency medicine practitioner called on to do so. That the procedure took ten minutes was entirely understandable and cannot properly be subject to criticism.

49. Secondly, the attempt at needle cricothyroidotomy was thwarted by the absence of jet ventilating equipment in the Accident and Emergency Unit. There was no suggestion in evidence that this absence was untoward. Dr Maffey's evidence was that he attempted to ventilate the patient from a wall outlet but was unable to do so. Associate Professor Brown gave evidence to the effect that the preparation of emergency packs containing tube and jointing would avert such difficulty. Dr Palmer subsequently indicated in evidence that such packs are now available in the Accident and Emergency Unit.

50. I also note from Dr Palmer's evidence that this case now forms part of the training regime for accident and emergency staff at the Royal Darwin Hospital. I trust that inclusion will go some way to assisting in the identification and treatment of any similar case that might present in the future.

Dated this 5th day of September 2000.

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GREGORY R CAVANAGH

Territory Coroner