SUPREME COURT OF QUEENSLAND

CITATION:	*Masson v State of Queensland* [2018] QSC 162
PARTIES:	**THE ESTATE OF THE LATE JENNIFER LEANNE MASSON** (*Plaintiff*) **v** **STATE OF QUEENSLAND** *(Defendant)*
FILE NO/S:	306 of 2005
DIVISION:	Trial
PROCEEDING:	Claim
ORIGINATING COURT:	Supreme Court at Cairns
DELIVERED ON:	23 July 2018
DELIVERED AT:	Cairns
HEARING DATES:	12, 13, 14, 15, 16, 19, 20, 21 February 2018, 8 March 2018
JUDGE:	Henry J
ORDERS:	**1. Claim dismissed.** **2. I will hear the parties as to costs at 9.15 am 8 August 2018 (out of town parties having leave to appear by telephone) if costs are not agreed in the meantime.**
CATCHWORDS:	TORTS – NEGLIGENCE– ESSENTIALS OF ACTION FOR NEGLIGENCE – STANDARD OF CARE – GENERALLY – whether the ambulance officers exercised the standard of care expected of an ambulance officer operating in the field in an emergency – whether reasonable care and skill was exercised – whether adrenaline should have been administered in preference to salbutamol – whether the response to the risk was reasonable – whether the timely administration of adrenaline would have avoided the injury. TORTS – NEGLIGENCE – ESSENTIALS OF ACTION FOR NEGLIGENCE – DUTY OF CARE – GOVERNMENT AND PUBLIC AUTHORITIES – whether the State owed the plaintiff a duty of care to ensure that Queensland Ambulance Service officers acted in accordance with their obligations under the Queensland Ambulance Service guidelines and duty of patient care – where the guideline invited

consideration of the administration of adrenaline – whether the guideline mandated the administration of adrenaline.

Ambulance Service of NSW v Worley [2006] NSWCA 102, cited
CGU Insurance Ltd v Porterhouse (2008) 235 CLR 103, cited
Roads & Traffic Authority (NSW) v Dederer (2007) 234 CLR 330, cited
Robinson Helicopter Co Inc v McDermott (2016) 90 ALJR 679, cited
Rogers v Whitaker (1992) 175 CLR 479, cited
Vairy v Wyong Shire Council (2005) 223 CLR 422

Crown Proceedings Act 1980 (Qld), s 8.
Succession Act 1981 (Qld), s 66.

COUNSEL: D Campbell SC with S Longhurst for plaintiff
D H Tait QC with C Fitzpatrick for defendant

SOLICITORS: RMB Lawyers for plaintiff
Crown Law for defendant

[1] Jennifer Masson suffered hypoxic brain damage as a result of a severe asthma attack. It is alleged she would have avoided the injury if ambulance officers who attended upon her had administered adrenaline promptly to her. Their decision not to do so is said to have been contrary to Queensland Ambulance Service ("QAS") guidelines and a breach of the QAS's duty of patient care.

[2] The injury occurred in 2002. Ms Masson lingered, catastrophically brain damaged in around the clock care, for many years before her death in 2016. Her personal injuries claim against the State of Queensland, per s 8 *Crown Proceedings Act 1980* (Qld), had not been resolved by then.

[3] The action survived in the hands of her estate, pursuant to s 66 *Succession Act 1981* (Qld). Quantum was agreed in the amount of $3,000,000 prior to the nine-day trial of her action on the issue of the State's liability in negligence.

Facts

[4] Ms Masson was a 25-year-old chronic asthmatic. Asthma is an episodic respiratory disease characterised by acute exacerbations of airflow obstruction due to airway inflammation and airway hypo responsiveness to a wide range of stimuli.[1] It is not known what stimuli prompted the asthma attack with which this case is concerned.

[1] Ex 4 p 8.

[5] Ms Masson had previously suffered severe asthma attacks which, unbeknown to the ambulance officers in this case, had been successfully treated by the administration of adrenaline. She carried a Ventolin puffer and had been prescribed an EpiPen (a patient administered adrenaline auto injector[2]) but there is no evidence she possessed or attempted to use an EpiPen during the events in question.[3]

[6] Turning to those events, on the night of 21 July 2002 Ms Masson drove to the home of her friend Jonathon Turner in Brinsmead, Cairns. On walking into the house, she was wheezing badly and announced she was returning to her car, apparently to look for her Ventolin puffer. When she returned to the house she asked Mr Turner to take her to the hospital. As they walked outside she collapsed on the front lawn. Another friend who was present, David Denman, telephoned for ambulance assistance and Mr Turner tended to Ms Masson, performing mouth to mouth resuscitation.

[7] According to the QAS trip history record[4] and the QAS ambulance report form ("ARF")[5], Mr Denman's call was received at 22.52 and ambulance officers were at the scene at 22.58, six minutes later.

[8] Two ambulance crews attended: intensive care paramedic Clinton Peters with third year paramedic Tania Stirling, and advance care paramedic Glen Timmins with first year paramedic Neville Weazel. Of them, Mr Peters and Ms Stirling gave evidence. Mr Peters' witness statements of 2 February 2006 and 5 July 2009 were also exhibited.[6] Mr Peters was the senior officer and the determinative decision maker amongst the officers.

[9] On arrival, Mr Peters noticed Ms Masson lying supine on the grass while a male performed external compressions on her.[7] The ambulance officers were given a history of Ms Masson having had a history of severe asthma,[8] suffering an asthma attack, having used her puffer to no effect, asking to be taken to hospital and collapsing into respiratory arrest, after which a friend performed "EAR/CPR" (expired air resuscitation/cardiopulmonary resuscitation).[9] Mr Turner and Mr Denman did not give evidence, but their unsigned statements became exhibits by consent. Those statements only spoke of Mr Turner performing "mouth to mouth". Whether it was Mr Turner or another bystander who was the male Mr Peters saw performing CPR is unclear.

[10] The presenting history section of the ARF notes that on arrival Ms Masson was in respiratory arrest. Mr Peters observed Ms Masson's eyes were open and her pupils

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2 Adrenaline is also known as "epinephrine".

3 Notes made by a treating doctor on Ms Masson's eventual arrival at Cairns Base Hospital recorded Ms Masson's presenting history as: "asthma with two life-threatening episodes in past carries adrenaline" (Ex 22 p 517). The source of that information is unclear. If it was conveyed by the ambulance officers, it was likely conveyed to them by one of Ms Masson's friends at the scene but telling against that is the absence of such a record in the Ambulance Report Form. In any event, the plaintiff did not allege the ambulance officers knew Ms Masson carried an EpiPen or knew that her asthma was specifically known to respond to adrenaline.

4 Ex 23 p 1324.

5 Ex 5.

6 Ex 6.

7 T2-56 L12.

8 T2-64 L27.

9 Ex 5.

responsive to light. However, she had trismus (lockjaw)[10] and was centrally cyanosed (her face was blue), flaccid and unresponsive. In the observations section of the ARF, her respiratory rate was recorded at 22.58 as being only 2 retracted (laboured) breaths per minute. In evidence Mr Peters said her respiratory rate was almost non-existent.[11] Her Glasgow Coma Scale ("GCS") was recorded as only six, consisting of the highest possible mark of four for eye opening and lowest possible marks of one each for verbal and motor response. Her blood pressure was high, at 155/100 (systolic/diastolic readings).

[11] Mr Peters checked Ms Masson's carotid pulse, detecting a very high heartbeat of 150 beats per minute. She was connected to a heart monitor which also revealed her sinus tachycardia was the same rate (the reference to sinus means the measurement was taken from the sinus node). Tachycardia is a rapid heartbeat, greater than 100 beats per minute. It is the opposite of bradycardia, in which the heart rate is less than 60 beats per minute.

[12] Mr Peters concluded Ms Masson was "hypoxic and deprived of oxygen and required oxygen immediately".[12] His response to the risk inherent in oxygen deprivation was to ventilate and oxygenate her by the application of a bag valve mask.[13] The operator of the mask indicated she was difficult to oxygenate.[14] Mr Peters applied an intravenous cannula into her cubital fossa (elbow pit) to administer intravenous drugs.[15] One minute after arrival at 22.59 he commenced administering intravenous salbutamol in aliquots (portions) of 250 micrograms.[16]

[13] It is this decision upon which the case largely turns. The two frontline drugs in the treatment of asthma attacks are adrenaline and salbutamol. The plaintiff contends adrenaline should have been administered at the scene after the arrival of ambulance officers. The defendant contends the administration of salbutamol was a reasonable response to the known circumstances at that time, particularly bearing in mind Ms Masson was tachycardic and was hypertensive (a state of high blood pressure).

[14] In all, eight aliquots, a total of two milligrams, of salbutamol were progressively administered from 22.59 to 23.20.[17] This was actually twice the maximum dose of one milligram recommended in the Salbutamol drug data sheet in the Clinical Pharmacological section of the QAS Clinical Practice Manual.[18] However the plaintiff did not rely upon that as a particular of the alleged negligence.

[15] The administration of salbutamol appeared at first to have been effective. Of that effect, Mr Peters testified:

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[10] T2-57 L21.
[11] T2-57 L16.
[12] T2-60 L6.
[13] T2-59 L45 – T2-60 L3.
[14] T2-64 L25.
[15] T2-60 L10.
[16] T2-60 L17.
[17] T2-65 L37.
[18] Ex 2 p 1052.

> "So it's – she – it improved her condition. So, initially, upon auscultation (listening for sounds within the body), we were unable to detect any breath sounds or there – initially, we had – we could hear an expiratory wheeze with a single breath but then, basically, her chest was very silent with no breath sounds and she was very difficult to ventilate. With multiple doses we could actually hear – I can – I can recall she went to an inspiratory/expiratory squeak, then from an inspiratory squeak to an expiratory wheeze, then an inspiratory wheeze/expiratory wheeze which – and Timmins advised me that she was becoming easier to ventilate. So … to summarise, her respiration was improving."[19]

[16] Mr Peters opined this was because the IV salbutamol was effective in bronchodilating her airways and allowing air movement in and out of her lungs.[20]

[17] The apparent improvement in some of Ms Masson's symptoms continued through to and beyond the point when she was loaded into the ambulance and transportation commenced. Shortly before departure, at 23.14, the ARF records she had a regular pulse rate of 94, improved but still high blood pressure of 140/100, a respiratory rate of 14 which was still retractive and her colour was normal rather than cyanosed. Her GCS score remained at six.

[18] Mr Peters testified transportation from the scene was initiated at 23.15, some 17 minutes after arrival. He explained the time taken at the scene reflected the fact Ms Masson was "actual time critical" as distinct from "transport critical".[21] The former involves a patient who has altered vital signs through some condition; the latter involves a patient whose abnormal vital signs indicate they will die unless transported to surgery.

[19] Once the transportation from the scene to the hospital was underway, Mr Peters noted an unexpected increase in Ms Masson's heart rate to 136 beats per minute as at 23.17. The ARF records that at that time she was again cyanosed and her GCS score had descended to three, reflecting the fact that her eyes were no longer opening. By 23.19 her heart rate had dropped markedly to 40 beats per minute, her respiratory rate had reduced to 12 retractive breaths per minute and blood pressure was absent. Cardiac arrest was imminent.[22]

[20] A minute later at 23.20 Mr Peters administered 300 micrograms of adrenaline. Of his reasoning for doing so he testified:

> "I then changed my pharmacology. I changed from IV salbutamol to low dose IV adrenaline … in accordance with the clinical practice manual for adrenaline at that time. … Her vital signs had deteriorated to the point where adrenaline was the most appropriate drug for her clinical presentation … [S]he was now bradycardiac. She had a slow heart rate; less than 60.

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19 T2-65 L40 – T2-66 L1.
20 T2-66 L4.
21 T2-67 LL5-22.
22 T4-54 L13, T4-56 L2.

> And – although it's not recorded there, she either was or about to be hypotensive."[23]

[21] Mr Peters explained the adrenaline was administered in three 100 microgram aliquots, 60 seconds apart. The initial dose had no effect.[24] Intubation commenced in the meantime. A second dose totalling two milligrams at 23.24 produced some return of cardiac output but only for 30 seconds or so.

[22] Mr Peters diagnosed Ms Masson was suffering bilateral tension pneumothoraces (air trapped in the pleural space causing collapsed lung).[25] The ambulance was stopped, not far from the hospital, and Mr Peters conducted an emergency left side thoracostomy (an incision of the chest wall allowing trapped air to escape). This achieved the decompression of the left lung accompanied by immediate improvement in heart rate and blood pressure.[26] In light of that improvement and the proximity of the hospital, a right sided thoracostomy was not attempted and instead, to avoid further delay, the ambulance proceeded to the hospital.[27]

[23] The QAS trip history initially recorded the ambulance's arrival at the hospital as being 23.33, though this was subsequently changed to 23.29. The triage time documented in the hospital records is 23.37, a difference between the ambulance and hospital records of eight minutes. The expert evidence in the case suggested that discrepancies such as this are not uncommon.[28] Quite apart from the variable of different time pieces being set inaccurately, the ambulance and hospital would not necessarily have chosen the same point in time for the purpose of the recording the time of "arrival". It would have taken time after the ambulance first arrived at the hospital for Ms Masson to be unloaded and conveyed into the emergency department, the part of the hospital where her arrival would in due course have been noted by hospital staff. In any event the time taken to transport Ms Masson to hospital was not pressed as a particular of negligence.

[24] At the hospital Ms Masson was noted to be centrally and peripherally mottled and cyanosed. She had no respiratory effort. There was no carotid pulse. She was bagged with resistance with inspiration. Adrenaline was administered at 23.41, 23.43 and 23.45, provoking an immediate response with a carotid pulse becoming discernible and increasing. Other measures were taken, including the relief of a right sided pneumothorax, and she was transferred from emergency to intensive care at 00.30.

[25] Subsequent to her arrival at the hospital, it became apparent Ms Masson had suffered severe hypoxic brain damage as a result of oxygen deprivation.[29] It is not in dispute that this was caused as a result of events prior to her arrival at hospital. The plaintiff contends the deprivation of oxygen giving rise to the hypoxic brain injury occurred in the course of her treatment by the ambulance officers because adrenaline was not administered during the initial phase of her treatment at the scene. On the other hand

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23 T2-70 L40 – T2-71 L26.
24 T2-74 L45.
25 T2-75 L4.
26 T2-77 L40.
27 T2-78 L25.
28 Eg. Ex 4 p 7.
29 Compare second amended statement of claim [6(h)] and amended defence [3(a)].

the defendant contends Ms Masson's severe hypoxic brain damage had already irreversibly occurred prior to intervention by the ambulance officers.

Issues for determination

[26] The case falls for determination by reference to the common law, the relevant events having occurred prior to the commencement of the *Civil Liability Act 2003* (Qld).

[27] Some issues in the pleadings fell away in the course of the trial and some are not in dispute. In particular, the decision not to attempt to decompress the probable right-sided tension pneumothorax, having decompressed the left side, was not pressed as a particular of the defendant's alleged negligence. The real focus of the case was the ambulance officers' assessments and decision making at the scene, specifically, the decision that the pharmacological intervention at the scene should involve salbutamol and not adrenaline.

Duty of Care

[28] The defendant admits it is vicariously liable for the acts, omissions and breaches of duty of its servants or agents, including the ambulance officers who attended upon Ms Masson, which attendance was also admitted. More particularly the defendant admits the plaintiff's pleading that:

> "3.(a) At all material times the Defendant:
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> (i) Trained and instructed ambulance officers of the QAS in the manner in which they were to carry out their duties as ambulance officers, including the provision of emergency medical treatment and transportation of medically ill persons.
>
> (ii) Provided guidance as to the manner in which ambulance officers carted (sic – carried) out their duties by provision of a manual, namely the "*QAS Clinical Practice Manual*" Version 2.1 (revised January 2002).
>
> (ii) In the premises aforesaid the Defendant owed a duty of care to members of the public to whom its ambulance officers were rendering assistance to:
>
> (i) Take reasonable care to prevent reasonably foreseeable risk of injury to members of the public.
>
> (ii) Take reasonable care that the Defendant's training and instruction of ambulance officers was adequate and sufficient to allow ambulance officers to perform their duties safely and adequately.
>
> (iii) Provide adequate guidelines and sufficiently specific guidelines and manuals for ambulance officers. …

5. In the premises pleaded aforesaid, the Defendant, and its servants or agents, the said ambulance officers, owed the Plaintiff a duty of care to:

   (a) Take reasonable care to prevent reasonably foreseeable risk of injury to the Plaintiff.

   (b) Take reasonable care in the assessment of the Plaintiff's medical condition and diagnosis of the Plaintiff's medical condition.

   (c) Administer proper, adequate and timely medical treatment and medication.

   (d) Transport the Plaintiff in a timely and safe fashion to a suitable medical facility or hospital."[30]

Standard of Care

[29] Those admitted propositions reflect the standard of care to be observed by a person with some special skill or competence, as ambulance officers have, namely, "that of the ordinary skilled person exercising and professing to have that special skill".[31] It is well established that standard is not determined solely or primarily by reference to the practice followed or supported by a responsible body of opinion in the relevant profession.[32] Nonetheless, as was observed in *CGU Insurance Ltd v Porterhouse*[33]*:*

> "Evidence of a particular practice or standard of conduct, whether laid down by a professional body or sanctioned by common usage, may be relevant to establishing a standard of care in a case of professional negligence …"

This heralds the significance of QAS guidelines and the common approach of skilled ambulance officers in respect of the administration of adrenaline for asthma.

[30] Importantly the standard of care under consideration here is not as high as that expected of a medical practitioner or an emergency physician in the hospital setting. It is that expected of an ambulance officer, operating in the field, in an emergency.[34]

Risk and Response

[31] An obvious consideration in the ambulance officers' exercise of reasonable care and skill in their treatment of Ms Masson on the occasion in question was the risk of serious harm to Ms Masson from the continued progress of her acute asthma attack. The self-evident risk of the continued impact of that attack was oxygen deprivation and consequent brain damage and death.

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30 Second amended statement of claim [3], [5].
31 *Rogers v Whitaker* (1992) 175 CLR 479, 487.
32 Ibid.
33 (2008) 235 CLR 103, 122.
34 *Ambulance Service of NSW v Worley* [2006] NSWCA 102.

[32] In assessing whether there was a breach of duty it is necessary to assess what a reasonable response to that risk would have been.[35] Of that assessment it was observed by Hayne J in *Vairy v Wyong Shire Council* (2005) 223 CLR 422, 461:

> "When a plaintiff sues for damages alleging personal injury has been caused by the defendant's negligence, the enquiry about breach of duty must attempt to identify the reasonable person's response to foresight of the risk of occurrence of the injury which the plaintiff suffered. That enquiry must attempt, after the event, to judge what the reasonable person *would* have done to avoid what is now known to have occurred. Although that judgment must be made after the event it must seek to identify what the response would have been by a person looking forward at the prospect of the risk of injury."

[33] At the time of the initial assessment and treatment of Ms Masson by the ambulance officers, the risk of oxygen deprivation and consequent brain damage, and potentially death, was readily apparent. The real issue is whether it was a reasonable response to that risk in the initial stages of treatment to administer salbutamol or whether the only reasonable response was to administer adrenaline from the outset.

[34] In assessing the reasonableness of the actual response, it is important to bear in mind that what is under consideration is not only emergency medical decision-making, but emergency medical decision-making by ambulance officers in the front yard of a suburban residence at night, not doctors in a hospital.

Breach

[35] If the risk was such that the exercise of reasonable care and skill in emergency treatment by ambulance officers called for the administration of adrenaline at the time of the treatment administered at the scene by the ambulance officers ("the time of initial treatment") then the defendant was in breach of its duty of care in not then administering adrenaline. This aspect of the case requires consideration in a general sense of what was warranted by the known circumstances, as well as consideration of the QAS Clinical Practice Manual, particularly the QAS asthma guideline.

[36] As to the known circumstances, setting aside the QAS Clinical Practice Manual, the plaintiff's case is that adrenaline is the drug of first resort when the sufferer of a severe asthma attack is "in extremis", that is, near the point of dying. The defendant contends salbutamol is equally an effective option in such circumstances. In the alternative the defendant argues Ms Masson's high heart rate and high blood pressure at the time of initial treatment told against the administration of adrenaline, and in favour of the administration of salbutamol, as a reasonable clinical choice in this particular case.

[37] As to the QAS Clinical Practice Manual the plaintiff maintains that in the presenting circumstances the non-administration of adrenaline at the time of initial treatment was contrary to the QAS asthma guideline.

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[35] *Roads & Traffic Authority (NSW) v Dederer* (2007) 234 CLR 330, 338, 351.

[38] It follows the real issues for determination relevant to breach may be shortly stated as follows:

(i) In 2002 was adrenaline ordinarily the preferred drug to administer to asthmatics in extremis?

(ii) Did Ms Masson's condition render salbutamol a preferred or equally acceptable option to adrenaline?

(iii) Was the non-administration of adrenaline at the time of the initial treatment contrary to the QAS asthma guideline?

[39] To the extent discussion of the above issues does not dispense with some pleaded breach matters they will be addressed as miscellaneous issues at the end of these reasons.

Causation

[40] If a breach of duty is established it is necessary to establish the damage – in this case the hypoxic brain damage – resulted from the breach.

[41] The High Court recently affirmed in *Robinson Helicopter Co Inc v McDermott*:[36]

> "Although proof of causation may sometimes entail the robust, pragmatic drawing of inferences, especially where there are a number of possible causes and there is difficulty in ascertaining which of them was the cause of damage suffered, proof of causation still requires proof on the balance of probabilities that the alleged breach of duty was the cause of the damage suffered."

[42] Proof of causation requires proof that the timely administration of adrenaline would likely have worked, so that Ms Masson would have recovered, as the evidence shows she had in her previous episodes of severe asthma attack. Further, it requires proof that it was not too late by the time of the ambulance officers' intervention for the administration of adrenaline to prevent Ms Masson from suffering hypoxic brain damage. If by that time Ms Masson was too far gone, such that hypoxic brain damage had occurred or was inevitable, then proof of the chain of causation would be unsustainable.

[43] It follows the real issues for determination relevant to causation may be shortly stated as follows:

(i) Would timely administration of adrenaline have avoided the injury?

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[36] (2016) 90 ALJR 679, 694.

(ii) Was the time of initial treatment too late for the injury to be avoided by administering adrenaline?

The experts

[44] Consideration of a number of the issues for determination is informed by some of the expert evidence adduced in the case. Generally speaking, this was not a case in which I felt concerns about the credibility or reliability of the expert evidence. While different opinions were espoused they were broadly within the admittedly diverse range of responsible medical opinion regarding adrenaline.

[45] The plaintiff called the following experts:

(i) Professor Gordian Fulde

Professor Fulde is a dual professor in emergency medicine and specialist in surgery at two universities and author of the book *Emergency Medicine, The Principles of Practice*. He is a founding fellow of the Australasian College for Emergency Medicine, a member of the senior court of examiners for the Australasian College for Emergency Medicine. He is director of the emergency department at St Vincent's Hospital in Sydney, a surgeon at St Vincent's Private Hospital Sydney, and senior medical officer-in-charge at the Sydney Hospital emergency department.

Professor Fulde was at times distractingly emphatic in the witness box – probably a product in part of his manner and in part of defensiveness because of some unusually strident criticisms he would have seen in the reports of the defence expert Professor Brown. In the end his emphatic demeanour did not undermine his credibility.

(ii) Associate Professor John Raftos.

Associate Professor Raftos, a conjoint associate professor of medicine at the University of New South Wales, has held a variety of positions as a specialist and senior specialist in emergency medicine, including at St Vincent's Hospital and Sydney Hospital and was director of emergency services at Sutherland Hospital for many years.

(iii) Dr John Vinen

Dr Vinen, an emergency physician, was head of the Department of Emergency Medicine at Royal North Shore Hospital from 1987 to 1999. He assumed a new role as Director of Emergency Support Services and eventually left Royal North Shore Hospital in 2008 to commence as a Senior Staff Specialist in emergency medicine at Blue Mountain District ANZAC Memorial Hospital.

Dr Vinen's reports were premised on an understanding of records to the effect that Ms Masson had suffered a cardiac arrest four minutes after the administration of salbutamol, giving rise to an understanding that the initial dose of adrenalin was not administered until 17 minutes after that cardiac

arrest.[37] That understanding was wrong. Cardiac arrest did not occur on the ambulance officers' watch until Ms Masson was being transported. That error did not compromise the foundation of all of Dr Vinen's evidence, though such of his opinions as were founded on it shall be disregarded.

(iv) Mr Jeff Kenneally

Mr Kenneally testified as an expert paramedic. He qualified as an ambulance officer in 1988 and a mobile intensive care paramedic in 1993 and worked as team manager at intensive care and advance life support paramedic teams from 2000. He is now senior lecturer in para medicine at Victoria University and in medical and traumatic emergencies for the Royal Australian College of General Practitioners and is director of a company delivering training and education to those who work in the medical first respondent setting.

[46] The defendant called the following expert witnesses:

(i) Professor Anthony Brown

Professor Brown is senior staff specialist for the department of emergency medicine in the Royal Brisbane and Women's Hospital and a professor in the field of anaesthesiology in critical care at the University of Queensland's school of medicine.

Professor Brown's reports contained unusually and unnecessarily strident criticisms of the content of the reports of some of the plaintiff's expert reports, for example, labelling some content "factually, morally and ethically wrong and misleading"[38] and having "absolutely no merit in terms of veracity or reliability".[39] He even went so far as to assert violations of the expert witness code of conduct.[40] The stridency of his criticism raised a concern he had unwittingly become an advocate for the defendant's cause but that concern was allayed by his apparent professionalism in the witness box. His strident criticisms more likely resulted from misunderstanding the distinction, discussed below, between the law's interest in accepted clinical practice regarding the administration of adrenalin and the evidentiary support justifying that practice.

(ii) Associate Professor Rob Boots

Associate Professor Boots has been an intensive care specialist and thoracic physician since 1994. He is an associate professor in the University of Queensland's department of critical care and deputy director of the department of intensive care in Royal Brisbane Hospital.

(iii) Dr Geoffrey Ramin

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[37] Compare, eg. Ex 4 p 7 and T5-4 L13.
[38] Ex 10 p 168.
[39] Ex 10 p 187.
[40] Ex 10 p 185.

Dr Ramin has been a specialist emergency physician since 1994, after which time he was staff specialist in emergency medicine at the Gold Coast Hospital and network director for emergency medicine in the Tweed Byron network. He is general manager of health services and medical director Aeromedical and critical care services in the Royal Flying Doctor Service Queensland, a medical director in emergency medicine at John Flynn Hospital, a visiting medical officer in extensive care at the Tweed Hospital, and chief medical officer to Allianz Global Assistance.

(iv) Mr Tony Hucker

Tony Hucker is a long experienced critical care paramedic and is now the director of clinical quality and patient safety for the QAS. He had involvement in preparing the QAS manual, including its 2002 version.

Breach issue 1: In 2002 was adrenaline ordinarily the preferred drug to administer to asthmatics in extremis?

[47] Professor Fulde testified that in emergency medicine adrenaline is, and was in 2002, widely regarded as "the go-to" drug, the "drug of first resort", for asthma attack sufferers in extremis.[41] Associate Professor Raftos explained it had always been his experience that adrenaline was more effective in treating patients in extremis with acute asthma than salbutamol and that adrenaline is the drug of first choice to use upon such patients.[42] Dr Vinen expressed the opinion that the use of adrenaline in life-threatening asthma is common in clinical practice and recommended in a variety of respected reference texts.[43]

[48] Professor Brown was critical of the plaintiff's experts expressing such opinions in the absence of published medical evidence showing that the administration of intravenous adrenaline "has any absolute, or relative beneficial outcome effects over and above nebulised or intravenous salbutamol in severe or critical asthma".[44] However it is quite clear those opinions were founded upon common clinical practice in emergency medicine, rather than upon scientific papers or testing.

[49] As Associate Professor Ramin explained, it is not unusual there is no good quality conclusive evidence either way about the respective merits of adrenaline versus salbutamol. He described it as but one of many examples in medicine "where one is required to treat life-threatening and very complex conditions with medications for which there is little or poorly developed evidence".[45] On this issue Associate Professor Raftos also observed:

> "[T]here is no conclusive evidence by way of controlled clinical trials for the efficacy of most medical treatments. This does not mean the treatments

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41 Eg. T5-21 LL15-45.
42 Ex 9 p 111.
43 Ex 4 p 32.
44 Ex 10 p 164, T6-83 L40.
45 Ex 17 p 220.

> are not effective. When a treatment is obviously effective, there is usually no need or incentive to statistically prove its efficacy."[46]

[50] Associate Professor Boots opined there was no clear benefit of adrenaline over salbutamol.[47] He opined that despite a historical reliance upon adrenaline as the go to drug for patients in extremis, there had been little comparison of adrenaline and salbutamol[48] and when there had been, they had been shown to be equivalent.[49] However he did opine there exists a preference for the use of adrenaline initially when there is a cardiac arrest or if there is anaphylaxis.[50]

[51] In a similar vein, Professor Brown was also prepared to concede adrenaline was the "go to" drug in severe asthma resulting from anaphylaxis, despite the absence of evidence in peer reviewed journals or trials.[51] However he and Associate Professor Boots did not agree it is the drug of first choice generally in dealing with severe asthma attacks.[52] In addressing Professor Fulde's position to the contrary, Professor Brown testified:

> "Certainly, Professor Fulde has been practising emergency medicine a few years longer than I have. Doctors are very reticent to change their practice and a lot of clinicians of an older age group – and I'm placing myself near to that now – have always used adrenaline for asthma because they're used to it. The younger generation who are more evidence based and look at the research literature to inform their practice, they prefer salbutamol. But absolutely, it is used. It's almost interesting. It's almost as though New South Wales has a cartel on use of adrenaline. There's a lot of hospitals – Royal North Shore where I have worked with Dr – adrenaline was used for asthma there. Now, it's predominantly salbutamol. St Vincent's, as you pointed out, RPA. So, yes, people use it. No question."[53]

[52] As with the above quoted testimony of Associate Professor Boots, that testimony implicitly acknowledged that adrenaline had a history of general acceptance in the medical profession as the ordinarily preferred drug to administer to patients in extremis. There has evidently been a shift over time in the preference of some practitioners.

[53] Dr Ramin testified that some clinicians use adrenaline preferentially whereas others favour salbutamol. Like Professor Brown and Associate Professor Boots he acknowledged there was "clearly absolutely no role for salbutamol once a cardiac arrest has occurred" and that most clinicians would use adrenaline preferentially in a situation where breathlessness is thought to be directly related to an anaphylactic reaction.[54] He went on to explain:

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46 Ex 9 p 111.
47 Ex 12 p 149.
48 T7-25 LL29-35.
49 T7-25 L38.
50 Ex 12 p 148.
51 T8-29 L39.
52 T8-29 L35, T7-26 L22.
53 T8-30 LL1-12.
54 Ex 17 pp 220-221.

> "At the time of initial presentation drug therapy was being used to try and achieve a degree of bronchodilation so as to improve oxygenation and decrease work at breathing. From a pharmacological point of view both salbutamol and adrenaline have effects that theoretically can help to achieve this. In the context of bradycardia, decreased perfusion and decreased cardiac output the mechanism of action of salbutamol offers no theoretical or actual benefit whereas adrenaline does have alternative mechanisms of action that can theoretically act to improve cardiac output and overall perfusion."[55]

[54] It was put to Associate Professor Ramin that it is common practice in emergency departments of hospitals in Australia where there are respiratory emergencies concerning severe pre-arrest asthma for practitioners to administer adrenaline intravenously, to which he responded:

> "I would be aware that there were emergency physicians that would consider using and would use adrenaline in such circumstances."[56]

This was not a concession that it is the predominant or majority practice. While he did not accept the use of adrenaline in that context was a "generically held belief across the whole body of emergency medicine", he did accept there was certainly a "reputable body of medicine" that believes it.[57]

[55] It is obvious that the practising medical profession traditionally regarded adrenaline as the ordinarily preferred drug to administer to asthmatics in extremis but that there has been a shift in the extent of that preference. It clearly remains so regarded by a credible body of medical practitioners but there is also a credible body of medical practitioners who regard salbutamol as an at least equally preferable drug to administer to asthmatics in extremis.

[56] The events of present concern occurred 16 years ago. The focus in some of the expert evidence on the lack of scientifically proved justification for the traditional preference seemingly diverted attention from any detailed exploration of the timing and degree of the shift in preferences in clinical practice. Nonetheless, while there were doubtless credible views in 2002 favouring the equivalent utility of salbutamol for asthmatics in extremis, it may reasonably be inferred from the whole of the expert evidence that the practising medical profession's traditional view in favour of ordinarily administering adrenaline to asthmatics in extremis was then likely a predominant view in the profession.

[57] That said, such a view was hardly binding and could at best have been be no more than a default starting point, for the mere characterisation of a patient being extremis is to say nothing of the broader detail of the patient's condition. The traditional view would not have precluded the administration of salbutamol in preference to adrenaline if that was medically appropriate having regard to the discrete aspects of the patient's condition.

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55 Ex 17 pp 221-222.
56 T7-43 L27.
57 T7-44 LL16-22.

[58] Illustrating the relevance of discrete conditions, it is clear from the expert evidence that adrenaline is and likely was regarded as preferable to salbutamol for administration to asthmatics in extremis when suffering cardiac arrest or anaphylactic reaction. Further, salbutamol would have been regarded then, as now, as unlikely to assist with bradycardia, decreased perfusion and decreased cardiac output, whereas adrenaline may. This demonstrates the significance of the discrete detail of the patient's condition. Here of course cardiac arrest or anaphylactic reaction were not known to be present at the time of initial treatment. It was unknown whether Ms Masson's asthma was the product of anaphylaxis and at the time of initial treatment she was not in cardiac arrest. She was apparently perfused, with a high blood pressure and cardiac output, indeed she was tachycardic, not bradycardic. As will be seen, whether her high heart rate and blood pressure were conditions mitigating against the administration of adrenaline is a relevant issue here.

[59] The above conclusions derive from the expert opinions of medical practitioners rather than ambulance officers, the opinions of the two expert ambulance officers adding nothing material in this context. However, it is reasonable to expect the approach of ambulance services in the training and guidance of their officers would be informed by widely accepted medical opinion.

Breach issue 2: Did Ms Masson's condition render salbutamol a preferred or equally acceptable option to adrenaline?

Discussion

[60] Dr Vinen opined that the early administration of adrenaline was called for in this case having regard to the fact that when initially assessed Ms Masson was in extremis with cardiac output having experienced a respiratory arrest secondary to severe asthma. [58] He repeatedly expressed the view that because, as at the time of initial assessment, Ms Masson was found to have already had a respiratory arrest, had minimal air movement and was in extremis, the early administration of adrenaline was essential in order to increase the likelihood of a good outcome.[59]

[61] Dr Vinen accepted in cross-examination that the term "in extremis" was a wider term than respiratory arrest, acknowledging there could be other causes as to why a patient would be in extremis.[60] He explained however that the term "in extremis" meant that death was imminent.[61] He opined the fact that Ms Masson had tachycardia and good blood pressure did not detract from the fact that she was in extremis when initially assessed by the ambulance officers.[62]

[62] Professor Fulde opined a respiratory rate of only two was not compatible with life.[63] He considered such a respiratory rate in combination with cyanosis constituted a respiratory

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58 Ex 4 p 21.
59 Ex 4 pp 15, 16, 19 and 21.
60 T3-56 L20.
61 T3-56 L28.
62 Ex 4 p 29.
63 Ex 8 p 69.

arrest.[64] Further he considered the GSC score of six could only have been due to hypoxia due to inadequate respiration in Ms Masson.[65] He opined adrenaline was clearly indicated by the circumstances, whereas salbutamol was not indicated.[66]

[63] Mr Kenneally concluded from the information received, and Ms Masson's state when the ambulance arrived, that she had clearly fallen into respiratory failure and required assisted ventilation.[67] He expressed some doubt whether Ms Masson had definitely been pulseless when her friend performed CPR. He also questioned whether Ms Masson's open eyes, which may have been uncontrolled eye opening, were misinterpreted as warranting a GSC sub-score of four rather than one. In any event, as to the latter he expressed a view it did not matter because, whether her GSC was six or three, she was in extremis in either case.[68] Mr Kenneally opined the circumstances prompted adrenaline as the front-line drug to administer to Ms Masson.[69]

[64] There was in substance no dispute from the defendant's experts that Ms Masson was in extremis, that is, near the point of dying, at the time of initial treatment.

[65] Associate Professor Ramin considered it was likely Ms Masson had effectively had a respiratory arrest at the time the ambulance were called, though unlikely that she had experienced true cardiac standstill, it being difficult for laypeople to detect pulses in emergency circumstances.[70] He opined that respiratory arrest will eventually lead to cardiac arrest potentially at any time.[71] He agreed Ms Masson was at a high risk of progression to cardiac arrest potentially within seconds.[72]

[66] Professor Brown accepted that Ms Masson was critically ill and suffering a life-threatening attack.[73] He did not bicker with the observations of the ambulance officers that at the time of arrival Ms Masson was in respiratory arrest.[74] He conceded that in patients who have had a respiratory arrest and are cyanosed, have depressed levels of consciousness and have restricted breathing there is very real risk of imminent cardiac arrest.[75] He explained that cyanosis is a sign of low oxygen in the blood[76] and accepted where acute immediate symptoms are being experienced in consequence of insufficient oxygen, death can result.[77] He ultimately acknowledged the words "imminent arrest", a term used in the QAS Clinical Practice Manual discussed below, could apply equally to a respiratory arrest or a cardiac arrest and accepted that a respiratory arrest will cause cardiac arrest.[78]

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64 Ex 8 p 69.
65 Ex 6 p 70.
66 Ex 8 p 56.
67 Ex 7 p 125.
68 Ex 7 p 125.
69 Ex 7 p 127.
70 Ex 17 p 218.
71 T7-40 L10.
72 T7-40 LL22-26.
73 T6-44 L46, T6-45 L17.
74 T6-45 L44.
75 T6-48 L1.
76 T6-57 L10.
77 T6-56 L15.
78 T6-74 LL35-43.

[67] The defendant's position in cross-examination of the plaintiff's experts was not that adrenaline was per se an inappropriate drug to have administered at the time of initial treatment but that the risks associated with its administration, because of Ms Masson's high heart rate and high blood pressure, meant that salbutamol was a more or at least equally appropriate drug to administer

[68] Dr Vinen rejected the suggestion that salbutamol was an equally appropriate drug to adrenaline to give in a case of a respiratory arrest with no signs of cardiac arrest. He acknowledged salbutamol is a highly selective beta agonist, its so-called beta-2 effect helpfully acting as a bronchodilator, dilating the respiratory system in the lungs.[79] He explained a negative effect of salbutamol, its so-called beta-1 effect, is that it causes vasodilatation, taking blood away from the essential circulation areas of the heart and brain, moving it to peripheral locations such as the muscles and abdomen with reduction in blood pressure.[80] He testified for this reason, that the use of salbutamol is thought to be associated with sudden cardiac death in patients who are acidotic (a reference to respiratory acidosis resulting from respiratory retention of carbon dioxide).[81] He opined Ms Masson was very likely acidotic.[82]

[69] I place no material weight on Dr Vinen's evidence that salbutamol is thought to be associated with sudden cardiac death in patients who are acidotic. The real significance of Ms Masson's likely acidosis in this case lay not in mitigating against the administration of salbutamol but in potentially supporting the administration of adrenaline. As will be seen, it is the negative risk associated with adrenaline, rather than salbutamol which is of relevance here.

[70] In any event that evidence, which emerged from Dr Vinen in cross-examination, involved no identification of the published papers it was purportedly premised on, which is reason enough to place little weight on it. For the same reason I place little weight on Associate Professor Boots bare evidence, premised on uncited literature or emergency room studies, that while IV salbutamol and IV adrenaline are of equivalent utility, there is literature to support greater adverse reactions to adrenaline.[83] That said, the risk of one of the reactions he mentioned – cardiac arrhythmia – was supported by other evidence.

[71] Mr Kenneally acknowledged salbutamol was a well-regarded bronchodilator drug with similar bronchiole actions during asthma attacks as adrenaline but noted adrenaline has other advantageous properties "including improving venous return, maintaining cardiac output and positive reduction of the immune response involved in establishing asthmatic attacks".[84] Mr Kenneally reasonably acknowledged salbutamol's usefulness in treating asthma but explained he considered adrenaline was better in a case like the present because:

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79 T3-58 L28.
80 T3-58 L35.
81 T3-59 L24.
82 T3-59 L44.
83 Ex 12 p 159, T7-23 L14, T7-25 L39.
84 Ex 7 p 127.

> "The – salbutamol is very specific in that it works, as you said, in beta-2 in the – particularly in the airways, but that's all it does. Salbutamol is effectively a little tiny part of adrenaline… that's taken off. The other effects of adrenaline, particularly some of its anti-immune response, its mast cell stabilising responses and features are very good in asthma that's been precipitated quickly, by you know, some sort of immune response to a trigger…"[85]

[72] Mr Kenneally explained his reference to an "immune response to a trigger" was not specifically to anaphylaxis but a reaction like anaphylaxis.[86]

[73] Professor Brown did not bicker with Dr Vinen's evidence about salbutamol's beta-2 effect of bronchodilating or opening up the airways to start oxygen being transmitted through the lung[87] and he asserted that effect made it a drug of choice in opening up airways in asthma respiratory arrest.[88] As to Dr Vinen's appraisal of salbutamol's beta-1 negative peripheral effects Professor Brown testified:

> "The beta-1 effects of salbutamol are minimal. They are responsible for a direct action on the heart to speed the heart up. There is some speeding up or increased rate, known as tachycardia, from beta-1 effect of salbutamol, but this is minimal."[89]

[74] He rejected the proposition that because salbutamol is a vasodilator it has a negative effect on a patient with severe asthma, asserting that the vasodilation effect of salbutamol is trivial, insignificant, almost non-existent.[90]

[75] When asked if salbutamol was ever an appropriate drug to use in the treatment of severe asthma Professor Fulde testified:

> "I'd have to redefine the word severe. … In other words, if I make it very simple: if a patient is still breathing, right?... In other words, the ability – the ability to inhale, nebulise or whatever agent, salbutamol is very much – that's where it's got its place. It's an excellent drug and, through the puffer, everybody etcetera – Australia with its massive asthma problem and New Zealand, compared to the rest of the world – that's where it is. But if a patient is not breathing, salbutamol and imminent arrest critically you would think that salbutamol is not an entity. It's adrenaline."[91]

[76] When asked if there was a time when IV salbutamol should be used Professor Fulde explained:

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85 T4-70 LL17-24.
86 T4-70 L27.
87 T6-30 LL17-32.
88 T6-30 L17, L47.
89 T6-31 L36.
90 T6-36 L40.
91 T5-8 LL29-39 (The quoted passage as transcribed included an "indistinct" reference which on replaying the recorded evidence can be heard to be "you would think". For completeness, there was some querying at trial as to whether the words "an entity" should read "indicated" but the words "an entity" are clearly audible.).

> "Yes, it's a good drug and the thing is it's – once again, as a generalisation, it's used in a more stable patient, such as an intravenous infusion. Once you've got a patient who may even be intubated, but you've got an airway, you've got a patient breathing to some extent, that's the critical factor. … Right? If a patient's not breathing – and I consider, in this case, just to cut to the chase, the respiratory rate of 2, cyanosis, the patient had had a respiratory arrest clearly document the patient was not breathing. The patient had a respiratory arrest. IV salbutamol, which is appropriately used in critically ill patients in intensive care and so forth, in the emergency department – but it was not the place for it here. … Because she has of – had arrested and not breathing."[92]

Professor Fulde went on to say:

> "And I think I've discussed – salbutamol was not the drug that this lady – young lady needed, when she was cyanosed. Right? In other words, the oxygen was depleting through her body. She was not moving in air into her lungs. Right? And salbutamol is not the agent to treat that."[93]

When asked if anyone would have used IV salbutamol he responded:

> "No one is absolute, but I would be very amazed in that situation that anybody would've even seriously considered it."[94]

[77] When Professor Fulde was asked about what would have happened if Ms Masson with her pulse rate of 150 was given adrenaline he acknowledged :

> "Of course, adrenalin stimulates the cardiovascular system. It would've gone up. All right. … Her blood pressure would've gone up… but that is what adrenalin does. That is part of the thing and that is all part and known when the people who put together this clinical case management guidelines – of course, that's what adrenalin does but let's – you are taking that with what it also does for the air–occluded airways."[95]

[78] Associate Professor Raftos opined the ambulance officers should have administered adrenaline, given Ms Masson was cyanosed, only taking two breaths per minute and in respiratory arrest:

> "They should have given adrenaline as soon as they had established venous access. Their failure to do so represents a departure from what would have been widely accepted by peer professional opinion in Australia in 2002 to be competent professional practice."[96]

[79] That opinion, contained in his report, was not maintained so forcefully in the witness box. Associate Professor Raftos accepted in cross-examination that it was reasonable to have used salbutamol.[97] He qualified that concession somewhat, by explaining the

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92 T5-8 L 45 - T5-9 L8.
93 T5-10 L40.
94 T5-12 L40.
95 T5-15 LL23-31.
96 Ex 9 p 103.
97 T5 -28 L18.

administration of salbutamol should have been followed by the administration of adrenaline much sooner,[98] because, notwithstanding the changes in some aspects of Ms Masson's condition, she was effectively unconscious, likely as a result of the carbon dioxide levels, meaning that more aggressive therapy in the form of the administration of adrenaline was called for promptly.[99] This was a rather unconvincing qualification to the above concession given that for a while after the administration of salbutamol there was actually an improvement in respiratory rate and auscultation indicated improved air movement,[100] making it reasonable to think the salbutamol was having a positive effect.

[80] Professor Brown explained that unlike salbutamol, adrenaline does not have a significant safety margin, observing:

> "Adrenaline is probably the most potent drugs doctors use day to day and at a therapeutic dose, in some people, it can have the feared side effects. Particularly if they are hypoxic or tachyarrhythmias, myocardial infarction, a stroke."[101]

[81] Professor Brown's third report noted a 2006 case series and a recent review article showed:

> "[A]drenaline has increased side effects compared with the other pure beta agonist drugs such [as] salbutamol. These particular side effects of adrenaline use can include tremor, palpitations, hypertension that itself may cause a stroke from either an intracranial haemorrhage or from an intracranial infarction, and or cardiac arrhythmias or a myocardial infarction."[102]

[82] Professor Brown also highlighted a 2009 article by Holley and Boots, "Management of acute severe and near fatal asthma", which concluded:

> "At the current time no recommendations regarding adrenaline infusions can be made, except its use would be reasonable as a rescue therapy in severe asthma complicated by hypotension that is not secondary to dynamic hyperinflation."[103]

Professor Brown emphasised that Ms Masson was not hypotensive (a state pertaining to low blood pressure) when first being treated and, to the contrary, had high blood pressure.

[83] Professor Brown opined that Ms Masson's tachycardia and high blood pressure made it unsafe to administer adrenaline, saying:

> "You don't give adrenaline to somebody who has got a rapid pulse and a high blood pressure, because it is a dangerous drug that will cause, in the face of hypoxia, a dangerous arrhythmia such as a ventricular tachycardia or

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98 T5-33 L24.
99 T5-34 L40.
100 T2-66 L30.
101 T8-32 L15.
102 Ex 10 p 199 (footnotes omitted).
103 Ex 10 p 200.

> ventricular fibrillation. And that's why the safety of adrenaline is difficult, and you wouldn't use when somebody still has a perfusing rhythm…that's why salbutamol is given."[104]

[84] Associate Professor Ramin confirmed that adrenaline can increase the heart rate which, depending on the context, may be a benefit or a negative.[105]

[85] Associate Professor Boots testified that Ms Masson would have been severely acidotic which would have tended to compromise the function of any drug she was given.[106] Associate Professor Boots acknowledged it was correct practice to give adrenaline if there is cardiac arrest[107] although he noted the absence of data in support of the practice.[108] As to the giving of adrenaline when cardiac arrest is imminent, he acknowledged it could have a positive effect on the circulatory system, but also testified that it could have a negative effect as well.[109] He did not agree that the negative effects of adrenaline were far outweighed by its positive effects, testifying:

> "The issue is that this girl was severely hypoxic. Until you correct the hypoxaemia, any drug you give, including adrenaline, may just make the whole thing worse. The drug – the poor heart can't respond to adrenaline or anything else without the correction of hypoxia. …. [I]f you do give it, you run the risk of this poor heart just going…with the adrenaline, because it's got no substrate, no nutrition, the oxygen, to make it start beating again. … [I]t can make it worse by degenerating it into a ventricular tachycardia; been there, done that. And then the heart just continues to stop."[110]

[86] The effect of Associate Professor Boots' evidence was that once a patient has gone into cardiac arrest there is not much to be lost by the use of adrenaline, whereas prior to that point, where the patient is hypoxic as was likely the case here, he explained the administration of adrenaline "can make it quite worse by stunning the heart into all manner of funny rhythms".[111]

[87] Mr Kenneally was dismissive of the theory that adrenaline was contraindicated in this case because it would have put Ms Masson's blood pressure and pulse rate up, although his evidence hinted that the position was not so clear in 2002. He testified:

> "Again, in the context of why her pulse rate and her blood pressure are high. Looking at it in 2002, there was probably less information available. Looking at it from now, there probably isn't anyone around who considers that to be a risk. You – her blood pressure and her heart rate will probably not go up with adrenaline. They'll probably come down, because you will increase the amount of air getting into her lungs. … So you will fix the reason – the reason her vital signs are so markedly disturbed is her body is

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[104] T6-75 LL34-43.
[105] T7-44 L35.
[106] T7-18 L25.
[107] T7-18 L35.
[108] T7-19 L11.
[109] T7-20 L36.
[110] T7-20 L40 – T7-21 L6.
[111] T7-21 LL8-37.

> already surging with her own adrenaline… .Okay. She's trying to fix her own problem, which she has no hope of doing, so we supplement that with a significantly increased amount of drug. … And that, in fact, corrects the reason that all those numbers are up. So it's not unreasonable to think that her vital signs will actually go the complete opposite direction to what the textbook says. That's what happens if I give it to someone just sitting here, with nothing else go on. Yes, things will go up. If I give it to you when you're in extremis and fix your problem, they will come back to normal. And we now know that. We've been doing that for years. It's--- No one ever has a problem with adrenaline in asthma."[112]

[88] Mr Kenneally conceded that many people in Mr Peters' position in 2002 would have chosen salbutamol, commenting:

> "Salbutamol has always been – we discussed at the start, the two have been fighting each other for that pole position."[113]

[89] Mr Kenneally did acknowledge that adrenaline had a tendency to cause possible arrhythmia, saying:

> "[W]hen you look…at what adrenaline does on paper, that's an obvious concern that you would always have in your mind."[114]

However, he explained:

> "If you stayed within the required drug doses, time intervals, drug route, so if it's supposed to be in a vein or whatever, you are generally in an area where you shouldn't cause those problems…. The ambulance service don't give you guidelines that are going to cause more trouble than they're worth"[115]

Conclusion

[90] At the time of initial treatment Ms Masson was not known to be in cardiac arrest or suffering an anaphylactic reaction, so it was not inevitable that adrenaline should have been administered. Nor was she suffering from conditions known to mitigate against the utility of salbutamol, such bradycardia, decreased perfusion and decreased cardiac output.

[91] Ms Masson was however in extremis, which meant that adrenaline would ordinarily have been the preferred drug to administer, subject to her discrete conditions. Of those conditions her cyanosis and likely acidosis were conditions reinforcing the extremely dire state she was in and tending to confirm the appropriateness of administering adrenaline. On the other hand, her high heart rate and blood pressure were conditions founding a legitimate concern that the administration of adrenaline might worsen her

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[112] T4-71 LL22-42.
[113] T4-72 L10.
[114] T4-79 L23.
[115] T4-79 L30.

state by plunging her into a dangerous arrhythmia or causing her heart to stop – that is, that it would heighten the risk of death.

[92] That concern provided a logical basis to prefer the administration of salbutamol and, if Ms Masson's condition did not improve, or if it worsened, revert to considering the administration of adrenaline. That course carried risks, particularly that salbutamol would not improve her condition and hypoxia may continue with irreversible results. Reasonable minds may differ as to whether those risks were outweighed by the above risks associated with administering adrenaline to an asthmatic in extremis with high heart rate and blood pressure.

[93] I conclude that there would have existed a responsible body of opinion in the medical profession in support of the view that Ms Masson's high heart rate and high blood pressure, in the context of her overall condition, provided a medically sound basis to prefer the administration of salbutamol to the administration of adrenaline at the time of initial treatment.

[94] This conclusion heralds obvious difficulty for the plaintiff's case.

Breach issue 3: Was the non-administration of adrenaline at the time of the initial treatment contrary to the QAS asthma guideline?

The Manual

[95] The QAS Clinical Practice Manual[116] in operation at the time of these events ("the manual"), contains in its foreword an introduction relevantly including the following:

> "**Introduction**
> The purpose of this manual is to provide Queensland Ambulance Officers with a comprehensive guide to prehospital clinical practice. The broad scope of professional ambulance practice has been considered throughout each section of the manual.
>
> Patient Care Principles
> This section covers the broad principles to be applied in all patient care situations.
>
> Case Management Guidelines
> This section covers a range of clinical conditions common to the prehospital setting. It provides diagnostic patterns of each listed condition to assist in arriving at an ambulance provisional diagnosis, and guiding principles to assist in patient management. Flow charts have been included to guide all officers in considering appropriate patient care options.
>
> Within each flow chart:
>
> – The diamond icon represents key clinical decision points. 
>
> – Shaded text boxes list the range of prehospital treatments.

[116] Ex 2.

– The reader is directed to other relevant guidelines/procedures. 

Clinical Pharmacology
This section lists all the pharmacological agents approved for use in the Queensland Ambulance Service. Drug Data Sheets for each agent provide parameters for prehospital administration. …"[117] (emphasis added)

[96] It is noteworthy that on the manual's own terms its case management guidelines are not proscriptive and rather are provided to guide and assist patient diagnosis, management and care.

[97] The introductory part of the manual goes on to provide information about "Patient Care Principles", including basic care principles, the emergency medical system, patient assessment, time/transport criticality, clinical judgment/problem solving, variables that influence patient care, clinical quality improvement and medico legal issues.[118]

[98] Significantly, in the context of the present case, the section of patient care principles in respect of clinical judgment/problem solving, includes this note:

> "Officers must consider the best possible care for the patient. The QAS Clinical Practice Manual is designed to assist clinical judgment, using the problem solving approach, to achieve best practice. It is acknowledged that every situation is different. Deviations from the guidelines will occur but must be documented and audited, and officers must be able to justify that their treatment was in the patient's best interest."[119] (emphasis added)

QAS asthma guideline

[99] Section A of the manual contains the case management guidelines. Its table of contents contains major headings followed by relevant sub-headings. Under the major heading "Dyspnoea" (laboured breathing), one of the sub-headings is "Asthma". The two pages of the manual's case management guidelines dealing with asthma ("the QAS asthma guideline") contain at the first page, information including:

> "**Diagnostic pattern:**
> - Past history of asthma
> - Wheeze; Initially apparent on expiration but as the disease progresses there may be both inspiratory and expiratory wheeze evolving into only inspiratory wheeze and then no wheeze.
> - Leaning forward braced posture
> - Prolonged expiratory phase of ventilation
> - Pursing of lips
> - Hyper inflated thorax
> - Cyanosis (*late sign*)
>
> **Guiding principles:**

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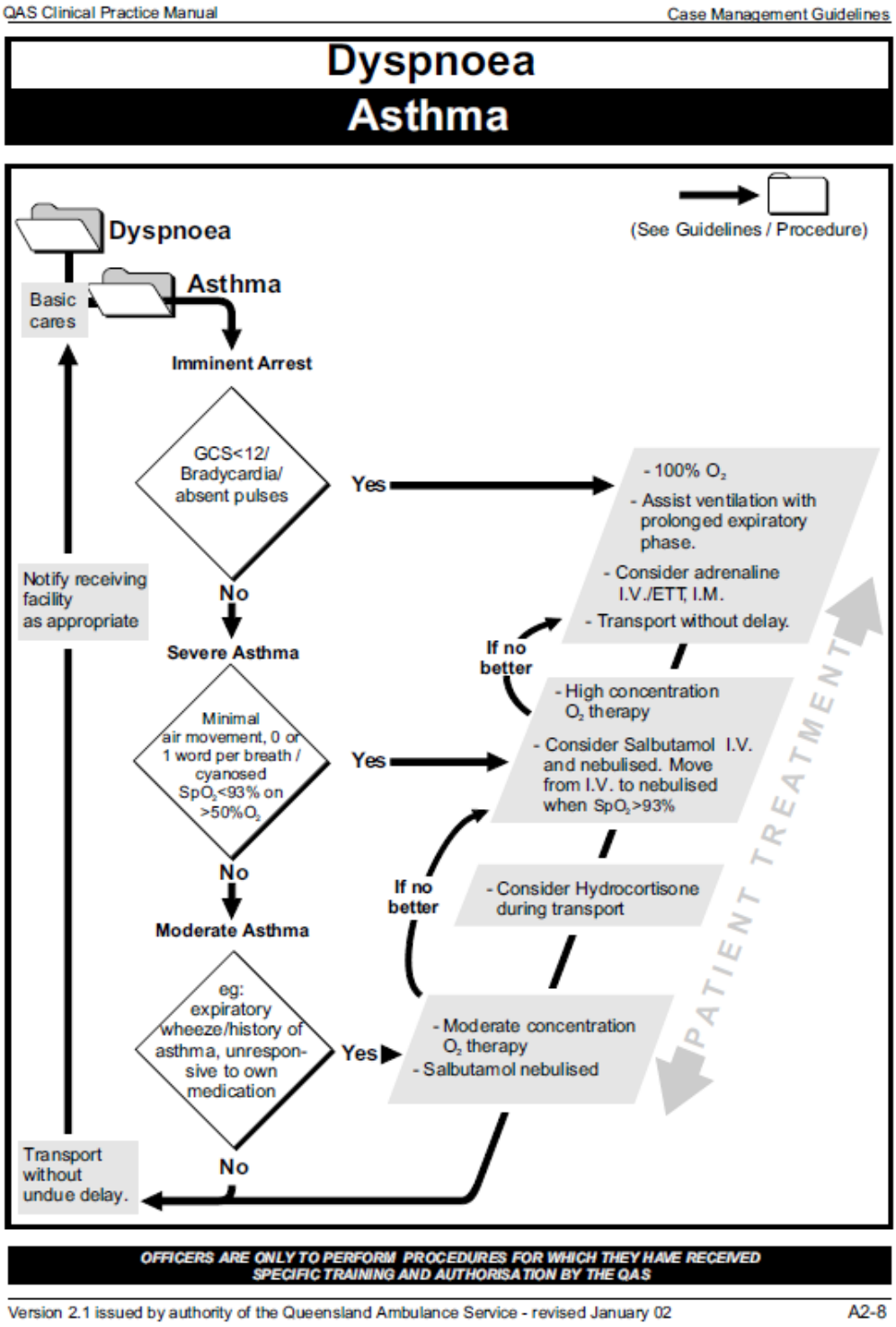
[117] Ex 2 p 878.
[118] Ex 2 pp 879-896.
[119] Ex 2 p 887.

- "All that wheezes is not asthma." Consider other causes of wheeze, eg. bronchiolitis, acute pulmonary oedema, c.o.a.d., chest infection (particularly if there is no history of asthma).

A patient with apnoea or near arrest requires ventilation assisted with slow gentle, shallow breaths of 100% oxygen at a rate of 4-8 per minute – very slow to allow passive exhalation. IPPV carries the risk of creating a pneumothorax (pulmonary barotrauma from high inflation pressure from air trapping) and then converting this into a tension pneumothorax."[120] (emphasis added)

[100] The second page of the QAS asthma guideline is the following flow chart ("the flowchart"):

[120] Ex 2 p 911.



Asthma data sheet

[101] The manual's chapter on clinical pharmacology also contains information about adrenaline, in the form of a two-page drug data sheet.[121] Its first page includes a section headed "Indications", against which there is a large tick (as distinct from cross) symbol, indicating the situations listed are apt for the administration of adrenaline, as follows:

[121] Ex 2 pp 1007, 1008.

"**INDICATIONS**
- Cardiac arrest.
- Bradycardia and/or poor perfusion unresponsive to other measures.
- Anaphylactic reactions.
- Bronchospasm unresponsive to Salbutamol.
- Croup with life-threatening airway compromise (nebulised)"

[102] The first page contains a variety of other information on the topics of presentation, pharmacology, action, metabolism, precautions, routes for administration, side effects, drug effect and contra-indications. The second page of the drug data sheet contains usual dosages for adults and children. It begins with the following caution:

"**Caution:**
The use of adrenaline may lead to hypertension, stroke, MI or a life-threatening arrhythmias."

[103] Such a caution is of course consistent with the expert evidence discussed above.

[104] It then provides details in respect of dosages pertaining respectively to "cardiac arrest", "bradycardia (unresponsive to atropine/pacing) or poor perfusion/cardiogenic (shock)", "anaphylaxis" and "asthma or severe bronchospasm with imminent arrest". As to the latter, which is the descriptor of relevance in this case, it provides:

"d) **Asthma or severe bronchospasm with imminent arrest:**
0.25-0.5 mg adrenaline I.M.I.
Paramedic 3, Paramedic ECP (on medical consult), Paramedic 4 only.
50-100 mcg (ug) adrenaline IVI every 60 seconds until perfusion and respiratory status normalises. Max. dose 1 mg.
Paramedic 4 only"

[105] As Professor Fulde and Mr Kenneally correctly noted, "Asthma or Severe Bronchospasm With Imminent Arrest", explicitly describes the situation Ms Masson was in at the time of initial treatment. In contrast the manual's drug data sheet for salbutamol contains no reference to "imminent arrest" or "severe bronchospasm" and merely lists one of the indications for use as "bronchospasm associated with asthma".[122]

Adrenaline when "Imminent Arrest"

[106] The medical profession's above discussed traditional preference for the use of adrenaline when a patient is in extremis is obviously reflected in the flowchart in the QAS asthma guideline. In respect of the action at the end of the "Yes" arrow from the diamond headed "Imminent Arrest", the only drug mentioned for consideration is adrenaline. It appears, consistently with the prevailing traditional medical orthodoxy, the QAS did not regard salbutamol as ordinarily as effective as adrenaline when a patient is in extremis. If it was not of that view it no doubt would have included, to the right of the first diamond, the pharmacological option of "or salbutamol".

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[122] Ex 2 p 1051-1052.

[107] Ms Masson's death was imminent when ambulance officers arrived on the scene. To adopt the language of some experts, she was in extremis, or, to adopt the terminology of the QAS asthma guideline at the heading of the first diamond, her arrest was imminent.

[108] There was some debate in submissions (and between some experts) as to whether the reference to "Imminent Arrest" in the guideline is to cardiac arrest or respiratory arrest. The debate verges on academic in that the heading does not stand alone and falls to be read in the context of the rest of the flowchart and the broader manual. However, on any view of it, Ms Masson was extremely close to respiratory arrest, indeed she may earlier have suffered one respiratory arrest, and was, at best for the defendant's position, barely breathing. Accepting that she was at best barely breathing, it is obvious that respiratory arrest was imminent. If a person stops breathing, cardiac arrest will soon follow. As Professor Fulde explained, if there is not enough oxygen for the brain the heart will imminently arrest due to the lack of oxygen.[123] It follows that where respiratory arrest is imminent, cardiac arrest is likewise also imminent, that is, death is imminent.

[109] The defendant argued against the imminence of arrest on the basis that Ms Masson was tachycardic, which is less suggestive of imminent cardiac arrest than bradycardia. That argument falls away when it is appreciated respiratory arrest was imminent, with the consequence cardiac arrest was logically also imminent.

When to take the arrow right?

[110] The heading "Imminent Arrest" is not the sole entry prompting the following of the flowchart's first "Yes" arrow to the right. Beneath the heading appears a diamond referring to three indicia, namely a GCS score under 12, bradycardia and absent pulses, each separated by a forward slash. There was considerable debate in submissions as to whether one or all of the indicia within the diamond ought be present in order to prompt the following of the "Yes" arrow to the right. Before turning to that debate two preliminary points ought be emphasised.

[111] Firstly, there was a tendency during argument for the flowchart to be analysed in the same manner that lawyers tend to analyse statutes and legal documents such as contracts. The flowchart does not call for analysis of that kind. It is a document intended for use by ambulance officers and should be considered in the context in which it fell for consideration by ambulance officers in 2002 (if its ordinary meaning is not apparent from that context then that would raise a separate problematic issue for the defendant, namely a failure to provide clear guidance). That context necessarily includes the broader manual. It must be remembered the manual's case management guidelines, of which the flowchart is a part, are intended to guide and assist rather than proscribe decision-making. Further the manual contains drug data sheets which also contain information relevant to decision-making about the circumstances in which drugs, including adrenaline, should be administered.

[112] Secondly, as to other interpretative assistance, it may have assisted for the court to have been informed of the content of any specifically relevant education or training provided

[123] Ex 8 p 82.

to the ambulance officers in respect of the manual, including the interpretation of the flowchart. There was no such evidence, save for some brief evidence from Mr Peters that was so general as to be of no material assistance.[124] A number of the medical experts purported to opine whether the indicia in the first diamond ought be read collectively or individually but with the arguable exception of the opinions expressed by the ambulance experts, discussed below, those opinions carried no probative force. The medical experts did not profess to be experts in respect of the interpretation of such QAS documents. Their evidence could only assist the interpretation process indirectly in the sense of aiding the court's understanding of the effect and potential significance of medical conditions and pharmacology of relevance to the decision making process with which the flowchart is concerned.

[113] Turning to the content of the first diamond in the flowchart, the debate largely went to whether the forward slashes between the three indicia meant "or" or "and". At one point the defendant effectively sought to cherry-pick the three indicia in the top diamond, seemingly taking the position that it would only be if a patient was bradycardic or had an absent pulse that the right arrow would be followed. This would make an irrelevancy of the presence of a GCS score under 12 in the indicia for, if that position were correct, there would be no need for that descriptor to appear in the diamond at all.

[114] The broader argument against the plaintiff, that all three indicia need to be present to prompt the following of the right-hand arrow, is unsustainable on the face of the manual. The forward slash is used both elsewhere in the same flowchart, and with consistency elsewhere in pages of the case management guidelines,[125] as indicative of alternatives, that is, as meaning "or". The obvious meaning of the diamond's content is that the presence of any of the three listed conditions will prompt the following of the right arrow.

[115] The argument is also unsustainable because it characterises the debate as turning solely upon the content of the diamond as a prompt and ignores the significance of the heading, "Imminent Arrest". The conditions listed in the diamond are not the only conditions relevant to determining whether the suffer of an asthma attack is in a state in which arrest is imminent. For instance, none of them make any direct reference to respiration or lack thereof, yet a patient in respiratory arrest or imminent respiratory arrest is, as earlier explained, in a state of imminent arrest. It is conceivable, as this case demonstrates, that patients in such a state may not (yet) be bradycardic or have absent pulses, but their GCS score will invariably be less than 12, which is one of the indicia listed in the first diamond. I conclude the presence of that indicia should have prompted the following of the right arrow, thus inviting consideration of the potential administration of adrenaline.

The expert ambulance officers' opinions

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[124] To remove doubt, while the defendant's ambulance expert, Mr Hucker evidently had involvement in the drafting of the QAS Clinical Practice Manual, his evidence did not detail any specifically relevant education or training in respect of it.

[125] Ex 2, eg. pp 922, 923, 925, 930, 946, 948, 982, 984, 990, 996.

[116] That interpretative conclusion is consistent with the interpretation apparently given to the manual by both ambulance experts.

[117] The plaintiff's ambulance expert, Mr Kenneally, noted the ambulance crews found Ms Masson to be unconscious, unresponsive and in respiratory arrest. He noted, consistently with the manual's case management guidelines Ms Masson was recognised as suffering an altered level of consciousness due to lack of oxygen from an asthma attack, which would have prompted the following of the asthma guideline. He opined the officers correctly documented the presence of one of the indicia in the flowchart's first diamond, namely a GCS score of six. Of that score he opined a score of three was more likely in a non-breathing patient, noting it is common to misreport uncontrolled eye opening in unconscious patients as being controlled, but considered that was of no moment because either way Ms Masson was in extremis. He observed that the first part of the response prompted by the following of the flowchart's first right arrow was in fact initiated but that there an error in not then opting for the administration of adrenaline, the drug mentioned in the first arrow:

> "The first management response documented appears to be consistent with the guideline by correctly providing intermittent positive pressure ventilation (IPPV) therapy. This should have then prompted following the remainder of the 2-7 Dyspnoea Asthma guideline. They then either underestimated her illness severity or made an error of guideline interpretation since they subsequently initiated the incorrect pharmacotherapy."[126]

[118] The response in the report of the defendant's ambulance expert, Mr Hucker, was not to disagree with the applicability of the first arrow but to disagree with Mr Kenneally's conclusion there was an underestimation of the condition or error of guideline interpretation. He explained:

> "By the crew's immediate actions, they clearly appreciated the severity of Jennifer's condition. The responding Paramedics immediately provided high concentration oxygen with intermittent positive pressure ventilation (IPPV). Simultaneously vascular access was gained and she received multiple doses of intravenous salbutamol. This was given in preference to adrenaline as she had palpable pulses, a tachycardia and high blood pressure. This decision making is allowed for with the asthma flowchart A2-8."[127]

[119] Mr Hucker's report later elaborated on his disagreement with Mr Kenneally's view that the crews either underestimated Ms Masson's condition or did not follow the asthma guideline:

> "Neither is true. Mr Kenneally continues to be of the view that the QAS guideline was not followed as he believes adrenaline was the only drug of choice. Adrenaline was an option. However, being an option does not mean it must be used. The QAS guidelines are designed to be flexible and

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[126] Ex 7 p 126.
[127] Ex 18 [7e].

> used by well-educated paramedics practising sound clinical judgment for the many various ways a patient can present with a disease or injury."[128]

[120] I agree with that reasoning. The following of the right arrow invites consideration of the option of administering adrenaline, it does not mandate that it must be administered. The fact it was not administered does not per se indicate the asthma guideline was not followed or that there was an underestimation of Ms Masson's condition.

[121] Before leaving the opinions of the ambulance experts I record for completeness that in cross-examination, Mr Hucker emphasised the importance of matters of degree in applying the flowchart, noting that the assessment of how imminent an arrest is may bear upon deciding the appropriate treatment.[129] He also emphasised the flexible contextual importance of all of the flowchart's content, noting the following of the arrow to the right of the first diamond did not preclude assistance being gleaned from the second diamond, which for instance refers to cyanosis.[130] While reasoning the second diamond also had potential to be followed in this case, he did not abandon the above mentioned interpretation of the flowchart, namely that imminent arrest in combination with a GCS score under 12 triggers the following of the first arrow to the right.[131]

"Consider adrenaline"

[122] The word "consider" to the right of the first diamond in the guideline indicates it remains for the ambulance officer to exercise a matter of professional judgment. This is hardly a surprising outcome for what is intended to be a guideline. Nonetheless, the plaintiff urges an interpretation that the administration of adrenaline is mandated and that the word "consider" relates only to considering which of the three means of administering adrenaline, there set out, ought be selected. Put another way, the plaintiff submits that what falls for consideration is not whether adrenaline ought be administered – the plaintiff contends it should be – and rather, what falls for consideration is how adrenaline should be administered.

[123] This submission finds some support in the presence after the words "Consider adrenaline" of three acronyms, namely, IV, ETT and IM. In the clinical pharmacology section of the manual in the Adrenaline Drug Data Sheet, the nominated routes of administration suggest those acronyms respectively stand for intravenous, endotracheal tube and intramuscular. Further, no drug other than adrenaline is mentioned for consideration to the right of the first diamond. It would be a surprising outcome for a patient in extremis or in imminent arrest to receive no administered drug.

[124] As against these features, it is readily apparent that the words "Consider adrenaline I.V./ETT, I.M." represent the third of four other actions listed to the right of the first diamond, the others being "100% 0^2", "assist ventilation with prolonged respiratory phase" and "transport without delay". On the face of it, all four actions are prompted.

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[128] Ex 18 [7h].
[129] T8-8 L45 – T8-9 L9
[130] T8-10 LL13-22.
[131] T8-7 L10, T8-9 L44, T8-10 L15, T8-11 L8.

Moreover, the word "consider" also appears against various drugs' names further down the right-hand side of the flowchart in contexts also inconsistent with it mandating the administration of the named drug.

[125] The natural meaning of the third action, described as "Consider adrenaline I.V./ETT, I.M.", in the context in which those words and acronyms are used in the flowchart, is "Consider administering adrenaline intravenously, via endotracheal tube or intramuscularly", not, "Administer adrenaline and consider whether to do so intravenously or via endotracheal tube or intramuscularly". If the plaintiff's urged interpretation were correct, the words in the guideline would not be as they are and instead would simply be "Administer adrenaline I.V./ETT, I.M." or merely "Adrenaline I.V./ETT, I.M".

[126] The notion that, even with arrest imminent, the administration of adrenaline is something to be considered, rather than to be done automatically, is entirely consistent with the broader content of the manual in which the guideline is found. As already indicated in earlier quotes, the Case Management Guidelines are expressly designed to "assist" in patient management, diagnosis and clinical judgment and there will be situations in which they are deviated from. Significantly, the introduction to the Clinical Pharmacology section of the document also includes the following:

> "This section of the clinical practice manual lists **all** the pharmacological agents approved for use in the Queensland Ambulance Service. …
>
> Officers of various levels of training may administer various subsets of these agents …
>
> Ambulance Officers must always consider the implications of any drug administration. Any drug can sometimes have an unpredictable adverse effect on a patient and consequently the Ambulance Officer will have to manage these effects as well as the original condition. Before use of any drug the Officer must:
>
> - Weigh up the potential benefits of the drug and the potential adverse effect;
> - Check previous drug therapies and effects and any potential drug interaction; and
> - Check any previous adverse drug effects.
>
> Sound clinical judgment is as much about **when not** to administer drugs as when to give them." (emphasis added)

[127] That passage makes plain the importance of clinical judgment and caution in not only determining what drug to administer, but also in determining "when not" to administer a particular drug, bearing in mind its "potential adverse effect". This point is further reinforced by the content of the clinical pharmacology drug data sheet about adrenaline[132] which, as already mentioned, cautions that the use of adrenaline may lead to hypotension, stroke, MI (myocardial infarction, i.e. heart attack) or life-threatening

[132] Ex 2 pp 1007, 1008.

arrhythmias. This is significant given that Ms Masson was tachycardic at the time Mr Peters was considering pharmacological intervention in the initial stage of treatment.

[128] These contextual considerations in respect of the broader content of the manual in which the flowchart is found, tend to confirm, rather than undermine, the above conclusion that the word "consider", when used against drug names in the flowchart, does indeed mean "consider" the administration of the named drug, not "administer" the drug.

[129] Even if this conclusion is incorrect, there would remain the difficulty for the plaintiff that the patient care principles in the introductory section of the manual clearly indicate it is designed to "assist clinical judgment", not to dictate it, and acknowledges that deviations from the guidelines may occur. It follows that even if the interpretation of the QAS asthma guideline urged by the plaintiff is correct, its status as a guideline as distinct from a proscriptive requirement inevitably leads to the conclusion that the QAS adrenaline guideline did not of itself require the administration of adrenaline at the time of the initial treatment.

[130] In any event, for the reasons already given, the effect of the guideline was not to require the administration of adrenaline but to require the treating ambulance officers to "consider" administering adrenaline, in addition to administering 100% 0^2, assisting ventilation with prolonged respiratory phase and transporting without delay.

Was adrenaline considered?

[131] This raises the question of whether the attending officers considered the administration of adrenaline at the time of initial treatment. That requires focus upon their decision-making as explained by Mr Peters. While that decision-making inevitably involved some contribution by other officers, Mr Peters was the senior officer, he played the lead role and the only witness accounts of the decision-making process came from him. The only other ambulance officer called, Ms Stirling, proffered no substantive evidence on the decision-making at the scene.

[132] Mr Peters' reasons for the decision-making at the scene fall to be considered in light of his own experience and expertise as an ambulance officer. He is now one of about 14 critical care paramedics with the QAS High Acuity Response Unit. He holds a Diploma as well as an Advanced Diploma in Applied Science in Paramedicine. He worked as a volunteer ambulance officer from 1990, becoming a full-time ambulance officer in 1996. After completing further studies in 2000 he became an intensive care paramedic in 2001. A lengthy record of his certifications and completed courses with QAS bespeak an obviously successful history of ongoing training and assessment in his time with QAS.[133] It also shows he became authorised to administer salbutamol in 1996 and adrenaline in 2000. Mr Peters testified his training included the use of the manual.

[133] Mr Peters explained he decided to administer IV salbutamol in the initial phase of the treatment of Ms Masson because of her history of severe asthma and his assessment of

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[133] Ex 1.

her clinical presentation.[134] This included her depressed respiratory rate, cyanosis, a hyper-inflated chest, she was difficult to oxygenate and tight in her airways, was hypertensive (pertaining to high blood pressure)[135] and tachycardic and had an altered level of consciousness.[136] He testified this presentation led him "fairly convincingly to the fact that she required immediate pharmacological intervention through salbutamol".[137] With multiple doses her respiration improved, and she continued to improve until after she had been loaded and the transportation from the scene had commenced.

[134] Mr Peters conceded in cross-examination that the dose of salbutamol administered – eight aliquots of 250 mcg over about 20 minutes – exceeded the dose recommended in the manual's clinical pharmacology drug data sheet for salbutamol.[138] Indeed, it was twice the recommended dose.[139]

[135] Associate Professor Ramin opined that while the salbutamol dose exceeded the time and dose limitations within the QAS drug data sheet "it is difficult to conceive that it would have contributed to Jennifer's subsequent outcome".[140] He testified of that high dosage:

> "Clearly the higher the dose used the greater the potential for side effects. The dose administered was in excess of QAS protocol at the time. Potential side effects would include hypoglycaemia, changes in serum potassium, tachycardia, hypotension and increased metabolic demands. Increasingly we are recognising the adverse metabolic effects that can be caused by salbutamol, particularly in relation to the fact that it can increase oxygen consumption at a time when oxygen availability is severely compromised. However, it should be noted that there was not a great emphasis in the literature on this particular point in 2002…"[141]

[136] Mr Peters explained that when adrenaline was administered at a later stage of treatment it was introduced because her "vital signs had deteriorated to the point where adrenaline was the most appropriate drug for her clinical presentation".[142] When asked to identify those signs he replied:

> "So she was now bradycardic. She had a slow heart rate; less than 60. And – although its not recorded there, she either was or about to be hypotensive. …[H]er vital signs were tending towards both bradycardia and hypotension."[143]

[137] It is noteworthy that just as the presence of tachycardia and hypertension was influential in initially causing Mr Peters to favour the administration of salbutamol, the subsequent

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134 T2-64 LL16-27.
135 Contrast "hypotensive" which pertains to abnormally low blood pressure.
136 T2-64 L25.
137 T2-64 L20.
138 T3-35 L14.
139 Ex 2 p 1052.
140 Ex 17 p 225.
141 Ex 17 p 220.
142 T2-71 L21.
143 T2-71 LL20-31.

presence of the opposite of those conditions was influential in causing him to favour the administration of adrenaline.

[138] It is unsurprising in light of the expert evidence discussed above that the presence of a high heart rate and high blood pressure was influential in the determination of the initial pharmacology.

[139] The plaintiff submits that the administration of adrenaline was not considered at all during the initial treatment phase, when the decision was made to administer salbutamol. That submission is unsustainable. On any view adrenaline was considered, albeit in the context of considering it should not be administered because of the presence of tachycardia and hypertension.

[140] There were aspects of Mr Peters' evidence which support a view that he believed the asthma guideline prevented him from administering adrenaline at the time of initial treatment. There were also aspects of his evidence which support a view that he considered adrenaline but concluded it should not be administered because Ms Masson was tachycardic and hypertensive. Such views are not mutually exclusive.

[141] In a witness statement by Mr Peters, of 5 July 2009, he described the choice of initial treatment:

> "We then commenced oxygen therapy via a bag valve mask resuscitator at a flow of 14 litres per minute which delivered approximately 100% oxygen. As Ms Masson's respiratory rate was only two breaths per minute, we immediately commenced intermittent positive pressure breathing ('IPPB') or assisted ventilation."[144]

It is noteworthy that this response, also apparent from the content of the ARF, involved the first two of the actions prompted if the arrow to the right of the first diamond in the flowchart is followed. They are not actions prompted by the arrow to the right of the second diamond, which relevantly only refers to high concentration oxygen therapy, not to 100% oxygen and assisted ventilation. This is powerful evidence in support of the inference that the flowchart's first arrow to the right was followed.

[142] Mr Peters' statement, given seven years after the event, continued:

> "As we were …commencing IPPB with 100% oxygen, Officer Stirling secured cardiac monitoring electrodes to Ms Masson's chest and attached the monitor/defibrillator … The monitor revealed that Ms Masson's cardiac rhythm was sinus tachycardia and the rate was 150 per minute.
>
> The QAS guidelines for the management of a patient with asthma, in July 2002, are set out in section A2-7 to A2-8 of the CPM, a copy of which is annexed .... The management is determined having regard for the patient's clinical presentation and vital sign recordings and may include: high concentration oxygen therapy, intravenous Adrenaline if bradycardic (pulse rate less than 60 beats per minute) or absent pulses; nebulised and intravenous Salbutamol; and consideration for intravenous Hydrocortisone.

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[144] Ex 6 p 249 [25].

> In view of the fact that Ms Masson was tachycardic, that is she had a heart rate that was greater than 100 beats per minute and peripheral pulse were palpable, intravenous Adrenaline was not permitted under the Asthma protocol. I therefore elected to administer intravenous Salbutamol."[145] (emphasis added)

[143] The above passage curiously introduces the term "protocol", which is not a term used in the manual. I doubt it is term favoured by Mr Peters, whose evidence showed he understood a protocol requires the taking of a fixed course whereas a guideline permits a greater flexibility of response allowing for the application of clinical judgment.[146] In the context of the above statement the reference to protocol was apparently a reference to the asthma guideline and the flowchart within that guideline. The reasons proffered in Mr Peters' statement for asserting the administration of adrenaline "was not permitted" are confusing. They were seemingly the presence of the high heart rate and the presence of peripheral pulse. It will be recalled the indicia in the first diamond of the flowchart include two indicia which are the opposite of those, namely bradycardia and absent pulses. This suggests that by the time of the statement Mr Peters was favouring an interpretation of the first diamond of the flowchart to the effect that the presence of indicia opposite to any of those listed in the diamond precluded the following of the arrow to the right of the diamond.

[144] There is reason to doubt whether that was the interpretation given at the time of treatment, in light of the fact some of the initial treatment was the very treatment contemplated by the arrow to the right of the first diamond. In any event, such an interpretation is wrong at two levels. Firstly, it is the presence in a patient of at least one of the indicia listed in a flowchart diamond which is the operative consideration, not the presence of any indicia opposite to that listed. Secondly, the asthma guideline does not proscribe, in the sense of permitting or precluding. Rather, on the manual's own terms, it is provided to guide and assist diagnosis and patient management and it may be deviated from.

[145] By the time of Mr Peters' evidence at trial, 16 years after the event, he explained that in the interim "this case has been brought up consistently in a potential legal forum and review".[147] The question of whether Mr Peters considered the administration of adrenaline and whether he believed the application of the asthma guideline meant he was not permitted to administer adrenaline was taken up during his testimony:

> "[A]s July of 2002 it was your belief, was it not, that you were prohibited from administering adrenaline because of the content of the asthma guidelines?--- The guideline stated a maximum dose of one milligram, that's correct.
>
> No, that you were prohibited from administering adrenaline was your understanding based on your reading of the guidelines as at July of 2002. That's the position, isn't it?--- No, I was prohibited from administering adrenaline with a patient's vital signs as Jennifer's were presenting.

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[145] Ex 6 p 250.
[146] T2-49 LL40-45.
[147] T2-512 L5.

Well, you were prohibited from administering it is what you believe the position was?--- Yes, that's correct.

HIS HONOUR: At the time is that what your belief was?--- At the time the guideline indicated that I should give IV salbutamol and that adrenaline was the inappropriate drug for Jennifer's presentation. …

Do you recall one way or the other, whether when you were initially making your decision that led you introducing salbutamol---?--- Yes.

---whether or not in making that decision you gave consideration to the option of using adrenaline? Do you remember one way or the other whether you considered that option then?--- Not really. It was – it was very clear which pathway I was required to go down.

So does "Not really" mean you didn't – you don't remember one way or the other or you recall that you did not consider that option?--- I would have considered both adrenaline and IV salbutamol, and IV salbutamol was clearly the defined pathway I was required to go down.

So you're saying you would have considered the option of adrenaline. Do you recall actually considering it?--- Certainly. So if Jennifer was initially presenting bradycardic/hypotensive, would have been straight into adrenaline. So it certainly would have been considered. So both options would have been in any mind in preparation for my actions dependent on how she presented.

Thank you. Yes, Mr Campbell.

MR CAMPBELL: You, as I understand your evidence – because she was tachycardic said you were prohibited, you were not permitted, you were unable to administer adrenaline?--- That's one of the parameters. The other one was her blood pressure. So she was tachycardic and hypertensive.

And if the position were that the guidelines permitted the administration of adrenaline in circumstances where the patient had a Glasgow Coma Score of less than 12, you, because of the fact of there being tachycardia, believed you could not implement those guidelines. Is that right?--- Tachycardia and hypertension, considering all the components of the – of the assessment tool.

And is that as a result of something that you were trained in or was this a result of your interpretation of the words in the guideline?--- Both. So there was quite a – a specific module in the appropriate pharmacological treatment of asthma in the intensive care paramedic program. It was both some reading material, there was verbal tutorials and there was case scenarios."[148] (emphasis added)

[146] The tone and manner in which the above answers were given made it obvious that Mr Peters had difficulty, doubtless because of the very long lapse of time (if not perhaps the contaminating contributions of others in legal forums), in distinguishing between hindsight assumption and actual recollection. It is clear however that Mr Peters well

[148] T3-35 L41 – T3-37 L2.

appreciated that salbutamol and adrenaline were potential pharmacological options in treating an asthma attack. It similarly obvious on the whole of his testimony that he considered Ms Masson's tachycardia and hypertension mitigated against the administration of adrenaline. As he said in the above quoted passage had she instead been bradycardic and hypotensive, he would have administered adrenaline.

[147] I have already concluded there would have existed a responsible body of opinion in the medical profession in support of the view that Ms Masson's tachycardia and hypertension, in the context of her overall condition, provided a medically sound basis to prefer the administration of salbutamol as an acceptable option to the administration of adrenaline. The clinical assessment that the use of adrenaline in a tachycardic, hypertensive patient might matters worse, for instance causing the heart to stop, provided a reasonable clinical basis to consider that adrenaline was too risky and to first favour the more conservative course of administering salbutamol.

[148] On a careful consideration of the whole of Mr Peters' testimony I am satisfied he did make such a clinical assessment, considering the possibility of administering adrenaline, deciding not to administer it because of the risk of serious adverse reaction to it raised by the presence of tachycardia and hypertension and instead deciding to administer salbutamol. The fact that he considered the administration in the context of rejecting it, because of the presence of those conditions, does not mean it was not considered at all. Such a clinical assessment and decision was a reasonable response to the risk associated with Ms Masson's oxygen deprivation as well as the risk of pharmacological intervention worsening her already dire condition.

[149] In arriving at that conclusion, I am conscious of the curious and erroneous references in Mr Peters' evidence to the asthma guideline prohibiting or not permitting the administration of adrenaline. However, they are not inconsistent with him also having considered, as I find he did, that the administration of adrenaline was too risky by reason of Ms Masson's tachycardia and hypertension.

Conclusion re breach

[150] The plaintiff's complaint of an alleged failure to administer proper, adequate and timely treatment was in substance a complaint that during the initial treatment there was a failure to administer adrenaline, contrary to the asthma guideline and or contrary to the standards of ordinary skilled ambulance officers.

[151] The asthma guideline did not require adrenaline to be administered. It prompted consideration of the administration of adrenaline. The administration of adrenaline was considered but rejected by reason of the concerning presence of tachycardia and hypertension. The presence of those conditions would have been regarded by a responsible body of opinion in the medical profession as supporting the view that Ms Masson's high heart rate and high blood pressure, in the context of her overall condition, provided a medically sound basis to prefer the administration of salbutamol to the administration of adrenaline at the time of initial treatment. Opting to administer salbutamol in preference to adrenaline in those circumstances was a reasonable response to the known risks.

[152] True it is, many doctors (perhaps less now than then) presented with Ms Masson's condition in a hospital setting may have elected to administer adrenaline. However, it is important to bear in mind ambulance officers are not medical practitioners who are specialists in emergency medicine.

[153] The urgent reality presenting itself on a suburban front lawn at night was that Ms Masson was certainly going to die if ambulance officers did not administer immediate treatment. In determining the pharmacological component of the broader treatment being administered they were presented with the dilemma that the administration of the drug ordinarily favoured for administration to a patient in extremis would, because of Ms Masson's tachycardia and hypertension, carry a real risk of worsening her condition and hastening her death. As against this there was the option of administering a drug known to be effective for severe asthma which did not carry the same risk.

[154] In a different but similar hypothetical case if adrenaline was administered and the patient soon suffered cardiac arrest and death, it can readily be imagined that the decision to administer adrenaline would in hindsight be characterised as having been too risky in light of the patient's particular condition. The reality is that this was a decision which could properly have gone either way in light of the competing risks.

[155] The treatment which was administered did not fall below the standard of care to be observed by ambulance officers and was not contrary to the QAS asthma guideline. No breach of the duty of care has been established and the claim must fail.

[156] Lest I be wrong in that conclusion I should for completeness address causation, on the premise, contrary to my view, that the non-administration of adrenaline did result from a breach.

Causation issue 1: Would timely administration of adrenaline have avoided the injury?

[157] Ms Masson was admitted to Shoalhaven District Hospital on 4 May 1995[149] against a background where she had woken up with an exacerbation of asthma and her father had administered adrenaline. The hospital records noted inter alia:

> "Woke up with exacerbation of asthma. Father administered 1 ampule 1:1000 adrenaline due to patient being cyanosed. …"[150]

[158] This suggests as early as 1995 her family had been equipped by medical practitioners to administer adrenaline to her in the event of an asthma attack.

[159] On 10 July 1997 Ms Masson had an acute asthma attack and was attended upon by New South Wales ambulance. They found her in severe respiratory distress, cyanosed and apnoeic, and administered two lots of adrenaline,[151] the result of which was recorded as being an improved response. The ambulance record form also records her father had

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[149] Ex 21 p 415.
[150] Ex 21 p 417.
[151] Ex 21 p 333.

given her an adrenaline injection.[152] On admission at Shoalhaven District Memorial Hospital, her condition on arrival was noted as:

> "History of asthma past respiratory arrests requiring home adrenaline. Tonight sudden onset asthma – found apnoeic by ambo's, given adrenaline by father and ambo's. On arrival inspiratory and expiratory wheeze, cyanosed, restless."[153]

[160] Ms Masson was admitted again to Shoalhaven District Memorial Hospital on 12 February 2000. The notes of that admission record that she had presented for the second time in the last few hours. She had inspiratory and expiratory wheeze and had used her nebuliser 10 times at home without any improvement. The record notes that following her presentation she became unable to breathe and unresponsive.[154] Oxygen ventilation was commenced and 0.5 ml of 1 mg in 1000 adrenaline was given and recorded as having a "good response".[155]

[161] The evidence of the successful use of adrenaline to treat Ms Masson's apparently severe asthma attacks in 1997 and 2000 is relied upon as supporting the inference the timely administration of adrenaline in the present instance would again have been successful. No evidence other than records was led in respect of these past episodes. Each obviously involved life threatening attacks of asthma, although it is not apparent there had been respiratory arrest or that either episode involved the prolonged period in extremis awaiting intervention which this case involves.

[162] That said, the evidence of Ms Masson's past positive responses, of which the ambulance officers were unaware, trends against the conclusion that the risk of her condition worsening in consequence of the administration of adrenaline was likely to manifest and in favour of the conclusion adrenaline would have had a positive effect.

[163] The competing expert opinions on this issue were unavoidably general. For instance, Dr Vinen opined in the majority of cases patients presenting as Ms Masson did at the time of initial assessment will recover without sequelae, provided the required dosage of adrenaline is administered without delay. Professor Fulde espoused a similar view:

> "It can be stated, in my opinion, even in the absence of specific knowledge of the patient's past history that most probably had adrenaline been administered early, subsequent events – deterioration during transport, arrest (respiratory and cardiac), and hypoxic brain damage, would not have eventuated."[156]

[164] Other experts were of a different view, as is enlarged on below when the significance of the lapse of time prior to treatment is discussed.

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[152] Ex 21 p 333.
[153] Ex 21 p 317.
[154] Ex 21 p 233.
[155] Ex 21 p 234.
[156] Ex 8 p 56.

[165] Associate Professor Raftos did have regard to the past positive responses prompted by the use of adrenaline by Ms Masson, observing:

> "It had been shown previously that Ms Masson's Asthma was responsive to adrenaline and she carried an EpiPen for that reason. If she had been treated with intravenous or intramuscular adrenaline in appropriate doses at about 23:00 hours on 22 July 2002, she would, on the balance of probabilities, have recovered from the severe Asthma attack without requiring incubation and without developing hypoxic ischaemic encephalopathy."[157]

Associate Professor Raftos noted, as did Dr Vinen, that when eventually given adrenaline there was a prompt return of spontaneous circulation in Ms Masson, suggesting that had she been given adrenaline earlier she would probably have recovered from her severe life threatening asthma without significant neurological deficit.[158]

[166] In contrast, Associate Professor Boots noted from the hospital medical records that the asthma took many days to settle in intensive care. Against that background he opined it was unlikely the administration of adrenaline at the time of initial treatment would have reversed the process. He did however concede the administration of adrenaline may have improved the ability to ventilate.[159]

[167] In the robust drawing of inferences called for in the present process I infer, by reason of Ms Masson's past positive responses to the administration of adrenaline in the 1997 and 2000 episodes, it was more likely than not that the timely administration of adrenaline would have avoided Ms Masson's injury. In the present case though there was no opportunity for a "timely" administration of adrenaline. There was a material intervening period before the arrival of ambulance officers.

Causation issue 2: Was the time of initial treatment too late for the injury to be avoided by administering adrenaline?

[168] If by the time of the ambulance officers' initial treatment Ms Masson had already been so oxygen deprived that hypoxic brain damage had occurred or was inevitable because her hypoxia was so entrenched and unlikely to quickly reverse, then the alleged failure to administer adrenaline at that time would not have been causative of the hypoxic brain damage.

[169] It will be recalled Mr Denman called for an ambulance. According to the tendered records, that must have been at 10.52 pm. The statements of Mr Denman and Mr Turner,[160] who were not called as witnesses, do not indicate how long had elapsed between Ms Masson's collapse on the front lawn and Mr Denman's telephone call, however it is reasonable to infer from their content and the circumstances which prevailed that Mr Denman placed the call very soon after Ms Masson's collapse. Mr

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[157] Ex 9 p 91.
[158] Ex 9 p 95, Ex 4 p 12.
[159] Ex 12 p 161.
[160] Ex 15, 16.

Denman's description of Ms Masson's state prior to the arrival and intervention of the ambulance officers was as follows:

> "John was taking her to the car and yelled out "Dave come help". I saw Jen sit down and then lie down. She had collapsed on the lawn so I called an ambulance on my mobile.
>
> Jen's eyes rolled into the back of her head and she stopped breathing.
>
> I was on the phone being talked through things by emergency. John was giving her mouth to mouth. The guy on the phone told me that "Right about now she should be opening her eyes", and she did open her eyes at around that time. She couldn't talk, but she was looking at us as though she wanted us to save her.
>
> I think it was about 10 minutes before the ambulance arrived. The ambulance stopped on the corner of the street for about a minute. I had to run up to the ambulance, waving, to get its attention. …
>
> By the time the first ambulance arrived Jen was conscious again. They gave her oxygen and I believe they gave her liquid Ventolin. They treated her for about 20 minutes to half an hour before they put her in the ambulance."[161]

[170] Mr Turner's recollection of the same period was as follows:

> "As we were walking to the car Jen collapsed. I caught her and laid her on the ground, and that's where she stayed.
>
> Dave called an ambulance. …
>
> From the time when Jen lay on the ground she didn't say another word. She was still breathing and looking at me but she was gasping for breath and was unable to communicate.
>
> I don't know how long she was there for. I was in a state of shock. Dave was walking around with his phone talking to the ambulance.
>
> Then Jen stopped breathing. Her eyes were starting to roll back in her head. She looked at me as though she wanted me to help her. It was very distressing.
>
> I checked her pulse and saw that it had stopped.
>
> Dave was on the phone still and told me that I had to give her mouth to mouth.
>
> I rolled her onto her side and then tilted her head back. I then spoke to the ambulance officer on the phone and he told me to give her mouth to mouth.
>
> It was difficult to give her mouth to mouth because she wasn't taking the air, but after a few breaths she came back and started breathing again, but very shortly and erratically.

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[161] Ex 15.

> I remember that there were two ambulances there. I didn't look to see when they arrived. In fact, I thought they both came at the same time. …
>
> A guy and a girl came in first. I asked them what they were giving her. I saw the first shot they gave her, and a paramedic told me it was liquid Ventolin, like what was in a puffer but that it acts immediately."[162]

[171] Further to Mr Turner's witness statement indicating he gave Ms Masson mouth to mouth, he or another layperson was witnessed performing CPR upon Ms Masson at the time of the arrival of ambulance officers. Ms Stirling was informed that person had been a surf lifesaver. [163] This was obviously hearsay. Though not objected to, I accord it no material weight as enabling any conclusion as to how necessary or how effective that treatment was.

[172] While Mr Denman's recollection is a period of 10 minutes elapsed before the ambulance arrived, the QAS trip history suggests it was a period of 6 minutes.[164] The call was taken at 22.52 and they were recorded as "at scene" at 22.58. As against this, a note by Dr Cameron in the hospital records about Ms Masson's presenting history recorded "People @ scene CPR = 15/60" followed by an arrow in the direction of QAS,[165] which suggests the ambulance may have been informed by bystanders at the scene that CPR had been performed for 15 minutes prior to the ambulance's arrival. Dr Cameron was not called as a witness and the source of this hearsay information is unclear. The better evidence is the trip history times and the evidence of Mr Denman, suggesting a range in the period between Ms Masson's collapse and the attendance of ambulance upon her of between six to 10 minutes. Allowing for the apparently short delay between when Ms Masson first collapsed and when the subsequent call went through to the QAS that range was likely seven to 10 minutes.

[173] Dr Vinen opined that while Ms Masson was in severe respiratory distress when the ambulance officers arrived the degree of hypoxia was not severe enough to have resulted in a cardiac arrest and enough oxygen was being circulated to allow breaths to be taken for good cardiac function of 150 beats per minute.[166] He considered the fact that Ms Masson's friends could not find a pulse or evidence of breathing prior to the arrival of the ambulance was unsurprising, in effect, because laypersons may be unable to feel pulses or detect evidence of breathing despite their presence.[167] In any event Dr Vinen emphasised at the time of the ambulance's arrival Ms Masson was still taking breaths, even if barely, and had a good cardiac output. Dr Vinen opined this meant "that while she was by definition comatose, cerebral function was still present".

[174] Professor Brown asserted severe hypoxic brain damage had probably already occurred prior to the arrival of the ambulance officers. In unconvincing support of that assertion he opined the apparent passage of at least six minutes of inadequate or absent oxygen supply would be "more than enough" time to have caused irreversible and devastating

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[162] Ex 16.
[163] T4-40 L45.
[164] Ex 23 p 1324.
[165] Ex 22 p 517.
[166] Ex 4 p 10.
[167] Ex 4 p 38.

hypoxic brain damage.[168] In substance this was merely an assertion of a possibility. It said nothing as the probability of the rival possibility that hypoxic brain damage had not yet occurred or become inevitable. Professor Brown also alluded in support of his assertion to the difficulty the ambulance officers had in ventilating Ms Masson who was cyanosed from lack of oxygen.[169] That underscores the perilous state Ms Masson was in when the ambulance arrived, but it does not mean she had reached the point of no return by that time.

[175] Professor Fulde disagreed with Professor Brown's above assertion, observing that some oxygen, even if a little, to the brain was very different to anoxia, that is no oxygen, when trying to determine brain damage.

[176] Associate Professor Raftos explained that oxygen stores in a previously well individual will be sufficient to prevent brain injury for up to 10 minutes after sudden respiratory arrest alone and for up to three minutes after combined cardiac and respiratory arrest. He also noted the effect of CPR provides some protection from hypoxic ischaemic brain injury and may extend the period of time following cardiac and or respiratory arrest before brain injury occurs.

[177] Associate Professor Raftos considered, given ambulance officers arrived about six minutes after Ms Masson had collapsed, it was likely her oxygen stores provided her adequate oxygen to prevent hypoxic brain injury before the ambulance's arrival.[170]

[178] Associate Professor Boots was reluctant to commit to definitive opinions in his reports because of the paucity of clinical information available.[171] As to whether Ms Masson's brain damage occurred prior to the intervention of the ambulance officers, he opined:

> "This is an impossible question to answer. … The cyanosis and GCS of six with eyes opening spontaneously in a setting of the recorded cardiovascular parameters would suggest the unconscious state was from hypoxia…We can state there was significant hypoxia at the time of the ambulance officers' arrival. The effectiveness of the bystander's CPR would most likely be poor in the circumstances and unable to effectively overcome the airway's resistance of asthma and bag valve masking was described as difficult in the ambulance reports in the ambulance officers' statements. Certainly the subsequent events of a cardiac arrest and the need to decompress tension pneumothoraces all contributed to the hypoxic brain injury."[172]

[179] It ought not be overlooked that the prospect of tension pneumothoraces was heightened by the nature of the other required intervention during the initial treatment, namely the intermittent positive pressure breathing.[173] This highlights the point that Ms Masson faced other serious risks involved in the treating of her condition. Admittedly the fact that one such risk did manifest itself, with further urgent medical consequences, falls for

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[168] Ex10 p 172. In a similar vein see Ex 10 p 187.
[169] Ibid.
[170] Ex 9 p 106.
[171] Ex 12 p 151.
[172] Ex 12 p 158.
[173] See eg. Ex 2 p 911.

consideration in the context that adrenaline had not been administered at the time of initial treatment. Whether its administration would have mitigated the risk of a tension pneumothorax being triggered by IPPB cannot be determined on any informed basis.

[180] It is also necessary to bear in mind that, on any view, Ms Masson's state of hypoxia was well underway. It is unlikely that the effects of that state would have ended instantaneously upon the administration of adrenaline. As Associate Professor Boots put it, the effects of such a state do not "reverse on the end of a needle".[174]

[181] The report of Associate Professor Ramin helpfully sums up the difficulty in reaching a scientific conclusion on this issue:

> "In regards to the question I was asked as to whether the hypoxia that resulted in Jennifer's brain injury occurred prior to the arrival of the Paramedics I must say that nobody could provide a definitive answer to that question. Jennifer's collapse occurred at some point close to 22:53. The minimum amount of time prior to the arrival of QAS would have been 6 minutes. Bystander reports indicate that her friends commenced CPR almost immediately as they could not feel a pulse or see effective breathing. At the time of QAS arrival they noted a spontaneous respiratory rate of 2 and that it was extremely hard to ventilate Jennifer. There is no doubt, therefore, that in the 6 minutes prior to the arrival of QAS Jennifer would have been receiving insufficient amounts of oxygen to her brain. Whether that was enough to cause irreversible brain injury prior to QAS intervention is impossible to say with certainty. All I can say is that it is possible. It is equally possible and in my view perhaps more likely that hypoxic brain injury developed as a consequence of the continuum of events over an unknown period of time both before and after QAS arrival. All one can say with certainty is that Jennifer's brain was receiving suboptimal quantities of Oxygen from around 22:53 and that this would not have been improved by bystander CPR and not improved to a significant degree by the QAS interventions although not as a result of inappropriate management but rather the severity of her condition. At what exact point irreversible catastrophic brain injury occurred cannot be determined.[175] (emphasis added)

[182] This opinion is consistent with my own overall view of the evidence. While the issue is finely balanced, I conclude that if, contrary to my findings, there was a breach by reason of a failure to administer adrenaline during the initial treatment, that breach was likely a material contributing cause of Ms Masson's injury. Put differently and aided by information the ambulance officers did not have, I conclude on the balance of probabilities that it was not too late at the time of initial treatment for Ms Masson's injury to be avoided by administering adrenaline.

Miscellaneous issues

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[174] T7-17 p 33.
[175] Ex 17 p 227.

[183] The above analysis dispenses directly or indirectly with the live issues in the case but to remove doubt it is best to say a little more about some miscellaneous issues.

[184] The plaintiff contended that if the QAS asthma guideline was confusing or ambiguous, that would prove another alleged particular of negligence, namely a failure to provide adequate or sufficiently specific and detailed guidelines. These reasons show the guideline was not confusing or ambiguous.

[185] The plaintiff pleaded the defendant failed to provide its ambulance officers with adequate or sufficient training and instruction, resulting in allegedly negligent acts by those officers. There is no evidence of such a failure. The approach to proving the supposed lack of adequate training and instruction appears to have been that it ought be inferred from the fact of the allegedly negligent acts of the ambulance officers. The core complaint as to those acts, expressed in multiple ways in the pleadings, was the alleged failure to administer adrenaline during the initial treatment phase. That complaint has failed.

[186] Of the alleged acts of negligence by the ambulance officers which are not covered by the above core complaint there remains allegations of a lack of urgency at the scene and a failure to take pulse oximetry readings. As to the lack of urgency it appears only to be pressed in the sense of a generalised complaint that the QAS asthma guideline's reference to "transport without delay", was not followed at the scene.[176] There is no substance to that complaint. The decision to administer treatment for a period at the scene before loading and transporting Ms Masson was obviously taken with the intention of immediately easing her life-threatening symptoms. The evidence does not suggest there was malingering or undue delay at the scene and nor, on balance, does the expert evidence.[177]

[187] In respect of the failure to take oximetry readings, it appears there was no pulse oximeter available on this occasion, notwithstanding that such a device was ordinarily part of an ambulance's array of equipment. Mr Peters testified the availability of such a device would not have altered his treatment.[178] It was apparent from the expert evidence that while the data such a device would have provided may have been helpful it is unlikely to have materially altered the course taken.[179] Its absence did not constitute a breach of the duty of care and in any event had no causative contribution.

[188] The defence included the pleading of contributory negligence as follows:

> "6. Further, the defendant will, at trial, contend that the incident, and the plaintiff's injury, loss and damage were caused or contributed to by the plaintiff's own negligence.
>
> Particulars

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[176] Plaintiff's written submissions [54.1.5].

[177] Dr Vinen expressed the view that the ambulance officers took too long at the scene although he acknowledged "it is a difficult call" in determining whether the patient should be extracted before being fully stabilised (T3-57 L38). As against this Mr Kenneally was untroubled (Ex 7 p 132).

[178] T2-81 L37.

[179] Ex 7 p 130.

(i) Failing to have on her person when attending the Banning Avenue address an EpiPen which had been prescribed for her use in circumstances of acute asthma attack;

(ii) Failing to make use of her EpiPen during the onset of her acute asthma attack on the evening in question."

[189] In the end result, no such contention was advanced at trial. I infer that was because the defendant was unable to identify evidence sufficient to prove the allegation of contributory negligence on the balance of probabilities. This is not an occasion which there is such obvious and persuasive evidence in support of a pleaded allegation that it can be said to have been established at trial notwithstanding the absence of any submissions in support of it. In circumstances where the conclusion is not of an obvious character and where there have been no submissions made in support of the pleaded allegation, the allegation is rejected. I conclude the allegation has not been established.

Conclusion

[190] My finding there was no breach of the duty of care has the consequence the plaintiff's claim should be dismissed.

[191] While costs would ordinarily follow the event, I will out of caution give the parties an opportunity to be heard.

Orders

[192] My orders are:

1. Claim dismissed.

2. I will hear the parties as to costs at 9.15 am 8 August 2018 (out of town parties having leave to appear by telephone) if costs are not agreed in the meantime.