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| **JURISDICTION** | **:** | SUPREME COURT OF WESTERN AUSTRALIA |
| **TITLE OF COURT** | **:** | THE COURT OF APPEAL (WA) |
| **CITATION** | **:** | CHILD AND ADOLESCENT HEALTH SERVICE -v- SUNDAY JOHN MABIOR by next friend MARY KELEI [2019] WASCA 151 |
| **CORAM** | **:** | QUINLAN CJ<br>MURPHY JA<br>PRITCHARD JA |
| **HEARD** | **:** | 19-20 MARCH 2019 |
| **DELIVERED** | **:** | 27 SEPTEMBER 2019 |
| **FILE NO/S** | **:** | CACV 21 of 2018 |
| **BETWEEN** | **:** | CHILD AND ADOLESCENT HEALTH SERVICE<br>Appellant<br><br>AND<br><br>SUNDAY JOHN MABIOR by next friend MARY KELEI<br>Respondent |

**ON APPEAL FROM:**

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| **Jurisdiction** | **:** | DISTRICT COURT OF WESTERN AUSTRALIA |
| **Coram** | **:** | DERRICK DCJ |
| **Citation** | **:** | MABIOR by her Next Friend MARY KELEI -v- CHILD AND ADOLESCENT HEALTH SERVICE [2018] WADC 12 |
| **File Number** | **:** | CIV 3062 of 2008 |

*Catchwords:*

Tort - Negligence - Medical negligence - Allegation of failure of doctors to recognise that patient had sepsis - Whether patient was suffering from sepsis - Challenge to findings of fact - Advantages of trial judge - Whether adequate reasons for findings of fact

Negligence - Breach of duty of care - Standard of care for health professionals - Whether act or omission in accordance with a practice widely accepted by peers as competent professional practice - Requirement for evidence of a practice

*Legislation:*

*Civil Liability Act 2002* (WA), s 5B, s 5PB

*Result:*

Appeal dismissed

*Category:* A

**Representation:**

*Counsel:*

| | | |
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| Appellant | : | Mr G R Donaldson SC |
| Respondent | : | Mr T Lampropoulos SC |

*Solicitors:*

| | | |
|---|---|---|
| Appellant | : | Panetta Mcgrath Lawyers |
| Respondent | : | Maurice Blackburn |

**Case(s) referred to in decision(s):**

Abalos v Australian Postal Commission [1990] HCA 47; (1990) 171 CLR 167
Allied Pastoral Holdings Pty Ltd v Commissioner of Taxation [1983] 1 NSWLR 1
Brett v Rees [2009] WASCA 159

Browne v Dunne (1894) 6 R 67
Chief Executive Officer, Department for Child Protection and Family Support v IGR [2019] WASCA 20
Christos v Curtin University of Technology [2017] WASCA 110
Davids Holdings Pty Ltd v Attorney-General (Cth) (1994) 49 FCR 211
Dobler v Halverson [2007] NSWCA 335; (2007) 70 NSWLR 151
DRA Industries Pty Ltd v Kuredale Pty Ltd [2018] WASCA 17
Kakavas v Crown Melbourne Ltd [2013] HCA 25; (2013) 250 CLR 392
Lee v Lee [2019] HCA 28
Lightfoot v Rockingham Wild Encounters Pty Ltd [2018] WASCA 205
Makita (Aust) Pty Ltd v Sprowles (2001) 52 NSWLR 705
McKenna v New England & Hunter Local Health District [2013] NSWCA 476
New England & Hunter Local Health District v McKenna [2014] HCA 44; (2014) 253 CLR 270
Smart v Power [2019] WASCA 106
South Western Sydney Local Health District v Gould [2018] NSWCA 69; (2018) 97 NSWLR 513
Sparks v Hobson [2018] NSWCA 29; (2018) 361 ALR 115
Sydney South West Area Health Service v MD [2009] NSWCA 343; (2009) 260 ALR 702
Taylor v The Owners - Strata Plan No 11564 [2014] HCA 9; (2014) 253 CLR 531
Western Australia v Watson [1990] WAR 248
Wyong Shire Council v Shirt [1980] HCA 12
X and Y (by her tutor X) v Pal (1991) 23 NSWLR 26

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**JUDGMENT OF THE COURT**:

## Introduction

1 On 2 February 2018, Derrick DCJ (as his Honour then was) ordered that judgment be entered for Sunday John Mabior (the **respondent**) against the Child and Adolescent Health Services (the **appellant**) with damages to be assessed.[1] Judgment was entered in respect of serious injuries suffered by the respondent while she was a patient at Princess Margaret Hospital (the **Hospital**) in December 2005.

2 The respondent, who was 16 months of age at the time, was being treated at the Hospital as a result of having suffered superficial and partial thickness burns to approximately 18% of her total body surface area (**TBSA**).[2] The injuries in relation to which the respondent sued the Hospital were a consequence of her having developed Acute Respiratory Distress Syndrome (**ARDS**). ARDS is an acute, diffuse and inflammatory lung injury that can lead to hypoxia. The ARDS in turn caused the respondent to suffer cardiac arrest, multi-organ failure, brain damage and cerebral palsy.[3]

3 The appellant is the legal entity responsible for medical and nursing care provided by the Hospital, including the team of doctors responsible for the care of the respondent while she was in the burns ward at the Hospital from 9 to 11 December 2005.

4 The learned trial judge found that the respondent's injuries were caused by the negligence of the doctors in the burns ward responsible for the care of the respondent. His Honour was satisfied on the balance of probabilities that, but for the negligence of those doctors, the respondent would not have developed ARDS, to the extent that she did, and consequently would not have suffered the injuries.[4]

5 The appellant appeals the learned trial judge's decision.

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[1] ***Mabior v Child and Adolescent Health Service*** [2018] WADC 12 (Primary reasons). The Primary reasons, at [74] to [95] contain a glossary of relevant medical terms. Unless otherwise stated, those definitions have been used in these reasons.
[2] Primary reasons [9].
[3] Primary reasons [61].
[4] Primary reasons [844].

## The appeal in overview

6 The appellant's amended grounds of appeal include 16 individual grounds of appeal. One of those grounds (ground 8) was not pursued at the hearing of the appeal.

7 The grounds of appeal relate to two broad issues.

8 First, a number of the grounds (grounds 1 to 11) are directed at challenging a critical finding of fact made by the learned trial judge; namely, that by the evening of 10 December 2005 the respondent had sepsis and that her sepsis continued to evolve up until the time she was transferred to, and treated in, the Intensive Care Unit (**ICU**) at the Hospital.[5]

9 The significance of that finding of fact arises in this way: the respondent's case at trial was that the cause of the respondent's ARDS was Systemic Inflammatory Response Syndrome (**SIRS**) in the presence of sepsis. Sepsis is a serious complication of, and a systemic response to, the presence of infection. SIRS is an inflammatory response of the body that can, relevantly, be caused by infection or sterile burns (or both). ARDS is a condition that can be caused by sepsis, but can also be caused by SIRS (as a result of burns), with or without the presence of sepsis.

10 There was no contest at trial that the respondent developed both SIRS and ARDS. The contest was whether she developed those conditions solely by reason of her burns or whether they were also the result of her having sepsis (ie. a serious complication of, and systemic response to, the presence of infection).

11 If, as the respondent contended, she had sepsis by the evening of 10 December 2005, she could, on the respondent's case, have been treated with antibiotics, so as to potentially avoid her developing ARDS.

12 If, however, the respondent did *not* have sepsis, and the cause of her SIRS and ARDS was (as the appellant contended at trial) solely due to the respondent's burns, the administration of antibiotics could have made no difference to her condition.

[5] Primary reasons [667].

13 Accordingly, whether the respondent in fact had sepsis was, as the case was fought, a necessary finding for the respondent to succeed at trial. In detailed reasons, the learned trial judge identified thirteen matters that, in combination, persuaded him, to the required standard, that the respondent in fact had sepsis by the evening of 10 December 2005 (the **sepsis finding**). Grounds 1 to 11 challenge that ultimate finding and many of the individual matters that led to that finding.

14 The second broad issue, raised by grounds 12 to 16, concerns the learned trial judge's finding that the appellant was in breach of its duty of care to the respondent. The relevant breach was, in summary, the failure of the doctors treating the respondent to recognise, by around 2.00 am on 11 December 2005, the possibility that the respondent had sepsis and by failing to administer antibiotics as quickly as was reasonably possible.[6]

15 The principal focus of the appellant's case in relation to these grounds concerns the proper construction, and application, of s 5PB(1) of the *Civil Liability Act 2002* (WA) (*Civil Liability Act*). Section 5PB(1) provides that:

> An act or omission of a health professional is not a negligent act or omission if it is in accordance with a practice that, at the time of the act or omission, is widely accepted by the health professional's peers as competent professional practice.

16 Aside from the challenge to the sepsis finding itself, there is no appeal from the learned trial judge's finding that the negligence found by him, relevantly, 'caused' the respondent's ARDS and injuries, within the meaning of s 5C of the *Civil Liability Act*. In that regard, the learned trial judge found that but for the failure by the doctors to administer antibiotics to the respondent, she would not have developed ARDS to the extent that she did and consequently would not have suffered the injuries.[7] Underpinning that finding, of course, is the finding that the respondent had sepsis and that it was negligent not to have administered antibiotics, both of which are challenged by the grounds of appeal.

17 For the reasons set out below, none of the grounds of appeal have merit and the appeal must be dismissed.

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[6] Primary reasons [804].
[7] Primary reasons [844].

18 Before turning to the grounds of appeal, it is necessary to set out, in some detail, the relevant background, including the learned trial judge's reasons for the critical findings challenged by the appellant.

## Background

### The course of the respondent's condition

19 The learned trial judge set out the non-contentious factual background at [3] to [56] of the Primary reasons.

20 The relevant timeline can be summarised as follows:

| Date | Time | Event |
|---|---|---|
| 9/12/2005 | 1700 hrs | The respondent suffers hot water scald burns to chest, forearms, and proximal regions of both hands and knees.[8] |
| | 1820 hrs | The respondent arrives at the Hospital.[9] |
| | 1900 hrs | The respondent is seen by a surgery burns registrar in the emergency department.<br>Registrar assesses superficial and partial thickness burns to approximately 18% of the respondent's total body surface area.<br>Temperature: 36.9º C.<br>Heart rate: 190 beats per minute (bpm).<br>Respiratory rate: 50 breaths per minute.<br>The respondent is put on oxygen therapy. Her lungs were clear.[10] |
| | 2040 hrs | The respondent is admitted to the Hospital's burns ward.<br>Temperature: 36.6º C.<br>Heart rate: 144 bpm.<br>Respiratory rate: 28.<br>The respondent is taken off oxygen therapy.[11] |
| | 2050 hrs | Swabs taken from the respondent's chest, left and right knee burns and a screen for Multi Resistant Staphylococcus Aureus (MRSA) conducted.<br>All swabs and MRSA screen came back negative, save for right knee swab.<br>Right knee swab produced positive result for Enterobacter bacteria with '+++ growth'.[12] |

[8] Primary reasons [6].
[9] Primary reasons [8].
[10] Primary reasons [9].
[11] Primary reasons [10] - [12].

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| | 2105 hrs | Blood samples are taken from the respondent to ascertain full blood picture (FBP).[13]<br><br>White cell count: 10.7.<br>Neutrophil count: 6.0.<br>Neutrophils showed 'left shift with toxic changes'.<br>The respondent's burns are dressed with silver sulfadiazine cream.[14] |
| 10/12/2005 | 0800 hrs | Temperature: 38.6º C.<br>Heart rate: 182 bpm.<br>Respiratory rate: 40.<br>Oxygen saturation: 95%.<br>The respondent is put on oxygen therapy.[15] |
| | 0820 hrs | Temperature: 39.4º C.[16] |
| | 0900 hrs | Temperature: 39.3º C.<br>Heart rate: 178 bpm.<br>Respiratory rate: 36.<br>Oxygen saturation: 100%.[17] |
| | 0900 - 2100 hrs | Heart rate: 157 - 197 bpm.<br>Respiratory rate: 22 - 38.<br>Oxygen saturation: 89 - 99%.[18] |
| | 1000 hrs | Temperature: 39.4º C.[19] |
| | 1000 - 2100 hrs | Temperature: 36.7 - 38.8º C.[20] |
| | 1825 hrs | Blood samples are taken from the respondent - C-reactive protein level (CRP) was 108 mg/L.[21] |
| | 2100 hrs | Chest X-ray of the respondent is performed. X-ray reveals mild patchy change in right lung base medially. Lungs otherwise 'well expanded and essentially clear'.[22] |
| | 2200 hrs | Blood samples are taken from the respondent<br>White cell count: 4.9<br>Neutrophil count: 0.7.[23]<br><br>Haematology report notes 'mild neutropenia'.[24]<br><br>The respondent remains on oxygen therapy.[25] |

[12] Primary reasons [13] - [15].
[13] All FBP results would have been available to medical staff within hours of the sample taken: Primary reasons [16].
[14] Primary reasons [16] - [17].
[15] Primary reasons [19].
[16] Primary reasons [20].
[17] Primary reasons [21].
[18] Primary reasons [24].
[19] Primary reasons [22].
[20] Primary reasons [23].
[21] Primary reasons [26].
[22] Primary reasons [27].
[23] Primary reasons [28].
[24] Primary reasons [28].

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|---|---|---|
| 11/12/2005 | 0800 hrs | Temperature: 38.4º C.<br>Heart rate: 178 bpm.<br>Respiratory rate: 34.<br>Oxygen saturation: 98%.<br><br>The respondent remains on oxygen therapy.[26] |
| | 0900 hrs | The respondent receives a burns bath and her wounds are dressed.[27] |
| | 1100 hrs | Heart rate: 182 - 192 bpm.<br>Respiratory rate: 40.<br>Oxygen saturation: 84 - 95%.[28] |
| | 1200 hrs | Oxygen saturation: 72 - 92%.[29] |
| | 1300 - 2100 hrs | Heart rate: 167 - 192 bpm.<br>Respiratory rate: 32 - 74.<br>Oxygen saturation: 89 - 100%.[30] |
| | 1600 hrs | Temperature: 37.8º C.[31] |
| | 1755 hrs | The respondent receives Frusemide (a diuretic).[32] |
| | 1800 hrs | The respondent is noted to have crepitation (crackling sounds) at base of both lungs.[33] |
| | 2000 hrs | Temperature: 37.1º C.[34] |
| | 2030 hrs | Chest X-ray of the respondent is performed. X-ray reveals widespread patchy areas of alveolar consolidation in both lungs, particularly left lung.[35] |
| | 2055 hrs | The respondent receives Frusemide.[36] |
| | 2145 hrs | The respondent is admitted to the Hospital's paediatric intensive care unit (ICU).[37] |
| | 2200 hrs | Temperature: 37.8º C.<br>Heart rate: 187 bpm.<br>Respiratory rate: 42.<br>Oxygen saturation: 95%.<br><br>The respondent remains on oxygen therapy.[38] |
| | 2300 hrs | Heart rate: 187 bpm.<br>Respiratory rate: 48.<br>Oxygen saturation: 85 %.<br><br>The respondent remains on oxygen therapy.[39] |

[25] Primary reasons [29].
[26] Primary reasons [30].
[27] Primary reasons [31].
[28] Primary reasons [33].
[29] Primary reasons [33].
[30] Primary reasons [34].
[31] Primary reasons [34].
[32] Primary reasons [35].
[33] Primary reasons [36].
[34] Primary reasons [34].
[35] Primary reasons [37].
[36] Primary reasons [35]
[37] Primary reasons [38].
[38] Primary reasons [40].

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|---|---|---|
| | 2315 hrs | Blood samples are taken from the respondent for FBP and culturing.<br>White cell count: 1.3.<br>Neutrophil count: 0.1.<br>CRP level: 242 mg/L.<br>Blood culture negative for bacteria.[40] |
| | 2400 hrs | The respondent is intubated. |
| 12/12/2005 | 0100 hrs | The respondent is administered Ceftriaxone (a broad-spectrum antibiotic).[41] |
| | 0200 hrs | The respondent is placed on a ventilator.[42] |
| | 0300 hrs | The respondent is administered Gentamicin (a broad-spectrum antibiotic).[43] |
| | 0500 hrs | The respondent receives blood infusion of packed red cells.[44] |
| | 0600 hrs | The respondent is administered Vancomycin (a broad-spectrum antibiotic).[45] |
| | 0800 hrs | The respondent is administered Meropenem (a broad-spectrum antibiotic).[46] |
| | 0900 hrs | Oxygen saturation: 33%.[47] |
| | 1100 hrs | Oxygen saturation: 68%.<br>The respondent goes into cardiac arrest and is resuscitated.[48] |
| | 1330 hrs | The respondent goes into cardiac arrest and is resuscitated.[49]<br>The respondent develops multi-organ failure and brain damage secondary to hypoxia (lack of oxygen to bodily tissues).[50] |
| | 1430 hrs | Blood samples are taken from the respondent for FBP.<br>White cell count: 6.3.<br>Neutrophil count: 0.5.[51] |
| | 1830 hrs | Blood samples are taken from the respondent for FBP.<br>White cell count: 12.6.<br>Neutrophil count: 1.6.<br>Neutrophils showed a left shift with marked toxic changes.[52] |

[39] Primary reasons [41].
[40] Primary reasons [42].
[41] Primary reasons [45].
[42] Primary reasons [44].
[43] Primary reasons [45].
[44] Primary reasons [46].
[45] Primary reasons [45].
[46] Primary reasons [45].
[47] Primary reasons [44].
[48] Primary reasons [47].
[49] Primary reasons [48].
[50] Primary reasons [49].
[51] Primary reasons [50].
[52] Primary reasons [51].

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| 13/12/2005 | | Samples are taken from the respondent's peritoneal fluid, post-nasal aspirate, tracheal aspirate, pleural fluid and endotracheal aspirate and tested for bacteria.[53] |
| | Prior to 0900 hrs | Blood samples taken for FBP analysis.<br>White cell count: 8.5.<br>Neutrophil count: 2.0.[54] |
| | 1045 hrs | Swabs are taken from the respondent's burn wounds to right and left arm, right and left leg, chest and left knee.<br><br>Culturing of all swabs produced negative results for bacteria.<br><br>Blood cultures produced negative results for bacteria.[55] |
| 29/12/2005 | | The respondent is extubated and transferred to the Hospital's rehabilitation ward.[56] |

## The expert evidence

21 Leaving aside (for the moment) the legal issues arising under s 5PB of the *Civil Liability Act*, the findings of the learned trial judge challenged by the appellant largely turned upon his Honour's assessment of the expert evidence. The expert evidence was critical to the determination both as to whether the respondent had sepsis by the evening of 10 December 2005 and in relation to whether the appellant was in breach of its duty of care.

22 The respondent called the following expert witnesses:

(a) Dr Andrew Numa, an intensive care and paediatric respiratory physician;

(b) Professor Alison Kesson, a paediatric infectious diseases physician; and

(c) Professor Michael Ditchfield, a paediatric radiologist.

[53] Primary reasons [52].
[54] Primary reasons [53].
[55] Primary reasons [54] - [55].
[56] Primary reasons [56].

23 The appellant called the following expert witnesses:

(a) Dr Jeffrey Prebble, a consultant paediatrician;

(b) Dr Hugh Allen, a respiratory and general paediatrician;

(c) Associate Professor Mike Starr, a consultant paediatrician, infectious diseases physician and consultant in emergency medicine;

(d) Professor Roy Kimble, a paediatric surgeon;

(e) Associate Professor John Harvey, a general, thoracic and burns paediatric surgeon;

(f) Dr Fiona Bettenay, a consultant radiologist; and

(g) Dr Conor Murray, a paediatric cardiothoracic and general radiologist.

24 It will be necessary to refer in detail to parts of the experts' evidence in the context of the grounds of appeal. For present purposes, it is sufficient to summarise the effect of their evidence by reference to their written reports. [57]

### *Dr Andrew Numa*

25 Dr Numa provided three expert reports prior to trial, dated 5 November 2016,[58] 26 December 2016[59] and 27 July 2017[60] respectively.

26 Dr Numa's opinion was that the deterioration in the respondent's condition from 7.00 pm on 9 December 2005 to 9.30 pm on 11 December 2005 was almost certainly related to either sepsis or toxic shock syndrome. He was of the opinion that the former diagnosis (sepsis) was more likely, by a significant margin, as an organism was identified on one of the wound swabs taken from the respondent soon after her admission to the Hospital (the organism being 'Enterobacter species +++ growth').[61]

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[57] The radiological evidence did not feature in the issues raised in the appeal, nor were the reports of the radiologists (Professor Ditchfield, Dr Bettenay and Dr Murray) in the appeal books. The evidence of those witnesses is, accordingly, not included in the summary that follows.
[58] Exhibit 19.4; GAB 585 - 626.
[59] Exhibit 19.6; GAB 627 - 661.
[60] Exhibit 19.8; GAB 662 - 716.
[61] Exhibit 19.4; GAB 585; Primary reasons [110].

27 Dr Numa's report of 5 November 2016 identified and discussed a variety of evidence in support of that opinion, including several significant spikes of temperature over the first 48 hours, persistent tachycardia, elevated inflammatory markers (elevated C-reactive protein), evolving neutropenia, evolving respiratory distress, a requirement for oxygen, and subsequent progression to multi-organ failure including circulatory collapse and ARDS in the ICU.[62]

28 Two particular matters in Dr Numa's opinion require further explanation.

29 First, his reference to evolving neutropenia. This is a reference to low neutrophil counts (neutrophils being a sub-set of white blood cells). Dr Numa stated that 'Low neutrophil counts (neutropenia) are a hallmark of sepsis' and that 'neutropenia is highly correlated with sepsis (but not burns alone) in animal models as well as adult and paediatric case series', in support of which he cited a number of publications.[63]

30 Secondly, in a passage particularly relevant to a number of the grounds of appeal, Dr Numa expressed a view as to the improbability of a patient developing ARDS as a result of burns alone (i.e. in the absence of sepsis), with burns to the extent suffered by the respondent (18% TBSA). In this regard, Dr Numa stated: [64]

> [T]he risk for ARDS is closely related to the area and severity of burns and in the case of [the respondent], described as having 12% to 18% superficial and partial thickness (*i.e.* no full thickness), the probability of ARDS developing purely as a response to the tissue injury alone is vanishingly small. In a recent series, burns patients who developed ARDS had substantially more full thickness burn area than patients who did not develop ARDS (median 20.5% full thickness burn area in ARDS patients vs median 7% full thickness burn area in non-ARDS patients). Similarly, the total burn area was significantly larger in patients developing ARDS … In this case where the plaintiff had no full thickness burns and burns to no more than 18% of her TBSA, ARDS is unlikely to have developed as a result of the burn itself. (citations omitted)

31 Dr Numa returned to this opinion in his subsequent report of 26 December 2016, which responded to opinions expressed by Dr Prebble, Professor Kimble and Professor Harvey.

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[62] Exhibit 19.4; GAB 586 - 588; Primary reasons [111].
[63] Exhibit 19.4; GAB 587; Primary reasons [118].
[64] Exhibit 19.4; GAB 588; Primary reasons [120].

32 In relation to the suggestion that the respondent's burns alone may have caused her developing ARDS, Dr Numa repeated that this was not supported by the literature. He stated that he not been able to find any case reports of ARDS secondary to burns of the nature and area suffered by the respondent, unless accompanied by sepsis. He concluded that the balance of probabilities overwhelmingly favoured sepsis as the cause of the respondent's ARDS rather than the burns per se.[65]

33 He again referred to the 'quite striking neutropenia' as proving strong evidence for sepsis and noted that none of the other experts had addressed the heavy growth of Enterobacter.[66] Dr Numa reiterated the various evidence he identified for infection, adding an additional factor: the respondent's severe coagulopathy on her admission to the ICU, which he described as a common finding in severe sepsis. [67]

34 In that report Dr Numa expressed the view that it was 'beyond any doubt' that the respondent's deterioration was related to sepsis. [68]

35 Dr Numa's final report, dated 27 July 2017, responded to reports from a number of the other experts. He adhered to his view as to the cause of the respondent's ARDS, which he stated was never a primary problem but was always secondary to some triggering disease or insult, for which, in the paediatric population, the most common cause was infection. [69]

36 In relation to the treatment provided by the Hospital (i.e. beyond the question of whether the respondent's ARDS was caused by sepsis), Dr Numa expressed the opinion that the constellation of signs and symptoms present over 10 to 11 December 2005 should have been sufficient to trigger a clinical concern of sepsis and that on the evening of 10 December 2005 (or at the very latest the morning of 11 December 2005), a prudent physician would have obtained blood cultures and commenced antibiotic therapy.[70]

[65] Exhibit 19.6; GAB 629-630; Primary reasons [123].
[66] Exhibit 19.6; GAB 630; Primary reasons [124] - [125].
[67] Exhibit 19.6; GAB 630-631; Primary reasons [126].
[68] Exhibit 19.6; GAB 631; Primary reasons [127].
[69] Exhibit 19.8; GAB 662; Primary reasons [131].
[70] Exhibit 19.4; GAB 588-589; Primary reasons [720] - [721].

37 Dr Numa agreed with Professor Starr that prophylactic antibiotics should not be given to every burns patient but should be reserved for cases where there is a suspected or proven infection. Dr Numa believed that the respondent's was such a case. [71]

38 Dr Numa stated that the key issue was: at what stage did that the diagnosis of sepsis become sufficiently likely to warrant commencement of antibiotics; at the time of the precipitous collapse that culminated in the ICU admission, or earlier? He believed that there were enough symptoms and signs to justify commencement of antibiotics at an earlier stage. [72]

### *Professor Alison Kesson*

39 Professor Kesson provided two expert reports prior to trial, dated 25 January 2017[73] and 21 July 2017[74].

40 Professor Kesson's opinion was that the respondent's vital signs on 10 December 2008 (particularly her temperature and pulse rate) suggested clinical sepsis. She considered that the respondent's full blood count (showing 'Marked neutropenia. Moderate leucopaenia') was a very strong indication of overwhelming sepsis.[75]

41 Professor Kesson confirmed these views in her subsequent report. She stated that, individually, the observations of the respondent's temperature, heart rate and respiration could have a multitude of causes, but taken together with the burn injury had a very strong positive predictive value for sepsis.[76]

42 In relation to the treatment provided by the Hospital, Professor Kesson expressed the opinion that a two-hour delay between prescribing antibiotics and administering antibiotics to a patient with a high risk of having sepsis, a life-threatening condition, was in 2005, and still is, unacceptable and poor medical care. She said that, as sepsis is a life-threatening condition, any delay in the administration of antibiotics increases the risks of morbidity (injury to tissues and organs) and mortality. When a patient is considered septic, antibiotics should be administered immediately and without delay.[77]

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[71] Exhibit 19.8; GAB 663; Primary reasons [728].
[72] Exhibit 19.8; GAB 663; Primary reasons [727].
[73] Exhibit 9.2; GAB 353 - 371.
[74] Exhibit 10.3; GAB; Primary reasons [217] - [220].
[75] Exhibit 9.2; GAB 354; Primary reasons [212] - [215].
[76] Exhibit 10.3; GAB 383; Primary reasons [220].
[77] Exhibit 10.3; GAB 382; Primary reasons [738].

43 In her second report Professor Kesson also drew the distinction, referred to by the learned trial judge,[78] between sepsis and septicaemia (a distinction that was not consistently observed in some of the evidence). 'Sepsis', Professor Kesson said, was a clinical syndrome caused by infection where the patient is hyperthermic (body temperature above normal) or hypothermic (body temperature below normal) and has tachycardia (fast heart rate), tachypnoea (fast breathing rate), together with a high neutrophil count (neutrophilia) or a low neutrophil count (neutropenia). 'Septicaemia', on the other hand, was the presence of replicating bacteria or their products in the bloodstream itself with a systemic response and evidence of altered organ function.

44 There was no suggestion in the present case that the respondent had septicaemia, as so defined.[79]

### *Dr Jeffrey Prebble*

45 Dr Prebble provided four reports prior to trial, dated 5 May 2010,[80] 18 May 2010,[81] 20 February 2016[82] and an undated report prepared in August 2017.[83]

46 Dr Prebble stated that, under most circumstances, the respondent's burns would not have been life threatening. He said that she was most unfortunate to develop ARDS which, although a recognised complication of burns of her severity, was more commonly associated with more extensive burns. [84]

47 Dr Prebble stated that the cause of the respondent's hypoxia (lack of oxygen reaching the tissues) was ARDS. ARDS usually develops within 72 hours of the initial illness. ARDS can be associated with sepsis, but in the case of the respondent, he said, septic screens were always negative. Dr Prebble said that ARDS was more usual in more extensive burns, but certainly had been associated with burns of similar severity to the respondent's. [85]

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[78] Primary reasons [86] - [91].
[79] Primary reasons [238], [300]. As the learned trial judge noted, however, at least Professor Harvey used the terms 'sepsis' and 'septicaemia' interchangeably (Primary reasons [91], [552]).
[80] Exhibit 3; GAB 24 - 28.
[81] Exhibit 4; GAB 2 9- 31. This report was substantially concerned with whether the respondent had fluid overload, an allegation that was abandoned prior to trial (Primary reasons [66]).
[82] Exhibit 5; GAB 32 - 40. This report was also substantially concerned with the abandoned allegation that the respondent had fluid overload.
[83] Exhibit 6; GAB 41 - 71.
[84] Exhibit 3; GAB 25; Primary reasons [298].
[85] Exhibit 3; GAB 25; Primary reasons [299].

48 Dr Prebble returned to the question of whether the respondent had sepsis in his August 2017 report. He stated that sepsis is 'SIRS + documented infection site' and that in the respondent's case there was no documented infection site. Dr Prebble observed that the blood culture performed on the blood sample obtained on 11 December 2005 showed no growth, that the pleural fluid sample obtained on 12 December 2005 showed no growth, that the peritoneal fluid sample obtained on 13 December 2005 showed no growth and that endotracheal tube aspirate obtained on 13 December 2005 showed no growth. [86]

49 Dr Prebble stated that Enterobacter (from the skin swab taken from the respondent's right knee) was a skin contaminant not an infection. He stated that, while it is acknowledged that negative results can uncommonly occur in overwhelming sepsis, 'there still remains no proof' of sepsis as the cause of the respondent's collapse. [87]

50 Finally, Dr Prebble remarked on Dr Numa's opinion that the probability of ARDS developing purely as a response to the tissue injury alone was 'vanishingly small' (see [30] above). Dr Prebble stated that while it was true that ARDS is seen more frequently in larger surface area burns, there were several reports of ARDS in burns to less than 20% of the TBSA. [88]

### *Dr Hugh Allen*

51 Dr Allen provided four reports prior to trial, dated 15 July 2010,[89] 9 May 2016,[90] 18 October 2016[91] and 26 April 2017.[92]

52 Dr Allen found, in his report dated 15 July 2010, that the measurement of the respondent's burns (18%) was accurate and, therefore, large.[93] As such, the extent of the burns to the respondent meant that there were irreversible local thermal injuries and a systemic response.[94] In relation to the respondent's diagnosis of ARDS, he found that the 'large burn and the metabolic response to it caused the ARDS'.[95]

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[86] Exhibit 6; GAB 42; Primary reasons [307].
[87] Exhibit 6; GAB 42; Primary reasons [307].
[88] Exhibit 6; GAB 41; Primary reasons [306]
[89] Exhibit 13.2; GAB 407 - 415.
[90] Exhibit 13.3; GAB 416 - 447.
[91] Exhibit 13.4; GAB 448 - 472.
[92] Exhibit 13.5; GAB 473 - 476.
[93] Exhibit 13.2; GAB 410.
[94] Primary reasons [357] - [359].
[95] Exhibit 13.2; GAB 412; Primary reasons [361].

53 In his report dated 9 May 2016, Dr Allen stated that the greater the percentage of the burn, the greater the risk of ARDS and infection, including pneumonia, and that these outcomes do occur in burns of 15% or more.[96] He found that, while there may have been pulmonary oedema, there was also ARDS, and possible infection, complicating the respondent's pulmonary conditions.[97]

54 In his report of 18 October 2016, Dr Allen expressed the opinion that the respondent's system failure occurred in the time frame consistent with ARDS.[98] There was biochemical evidence of increasing CRP, which suggested 'a massive inflammatory response suggestive of ARDS'.[99] Dr Allen observed that the radiology reports confirmed that the most likely diagnosis was ARDS, not pulmonary oedema.[100] He remarked that, on the basis of the clinical progress, the respondent suffered from ARDS, which was supported by the multi-organ failure that occurred.[101]

55 Dr Allen's final report of 26 April 2017, considered expert witness medical reports prepared by Dr Numa and Professor Kesson.[102]

56 In relation to Professor Kesson's report, Dr Allen found that in retrospect, while administering antibiotics earlier may have made a difference (as suggested by Professor Kesson), equally it may not have altered the outcome.[103]

57 Dr Allen's preferred explanation for whether the failure to treat the infection caused septic shock (and in turn the acquired brain injury), was that the hypoxic brain injury was caused by the septic shock and may have been alleviated by earlier administering of antibiotics.[104] He agreed with Professor Kesson's recognition that the blood cultures taken produced negative results, and that there are commonly false negative results in blood cultures.[105]

[96] Exhibit 13.3; GAB 419; Primary reasons [362].
[97] Exhibit 13.3; GAB 419; Primary reasons [362].
[98] Exhibit 13.4; GAB 449; Primary reasons [365].
[99] Exhibit 13.4; GAB 450; Primary reasons [365].
[100] Exhibit 13.4; GAB 450.
[101] Exhibit 13.4; GAB 450; Primary reasons [368].
[102] Exhibit 13.5; GAB 473; Primary reasons [369].
[103] Exhibit 13.5; GAB 474.
[104] Exhibit 13.5; GAB 475.
[105] Exhibit 13.5; GAB 475; Primary reasons [370].

58 Dr Allen identified the other experts' reports as identifying an 'inflammatory cytokine cascade-like response' being the cause of the respondent's deterioration, in relation to which he agreed that there is no doubt that infection can cause such a deterioration.[106] Dr Allen surmised that the medical and nursing staff believed that the respondent's tachycardia and tachypnoea were caused by the activation of the inflammatory cascade, not by impending septicaemia. In the context of the inflammatory cascade, Dr Allen recognised that the blood cultures did not grow a pathogenic bacteria, and while there are false negatives, in severe sepsis one would expect to find a pathogen.[107]

### *Associate Professor Mike Starr*

59 Professor Starr wrote a report dated 25 June 2017[108] which referenced four articles, including some referred to by Dr Prebble.

60 Professor Starr considered that the respondent's primary problem may have been neither sepsis nor fluid overload, but rather ARDS.[109] While fever, tachycardia and raised inflammatory markers are 'almost universal in burns patients', and can be signs consistent with infection, Professor Starr found those signs to also be consistent with an inflammatory response to significant burns with associated ARDS.[110]

61 Professor Starr did not agree with Professor Kesson that the respondent most likely had overwhelming sepsis.[111] Given the respondent's presentation, Professor Starr found sepsis to be a possible complication,[112] however, not the primary problem.

### *Professor Roy Kimble*

62 Professor Kimble prepared two reports dated 28 October 2016[113] and 2 June 2017.[114]

63 Professor Kimble found the respondent's percent body surface burn to be 19%, but that the initial assessment of 18% was very accurate.[115] It was his opinion that the respondent had a systemic

[106] Exhibit 13.5; GAB 475.
[107] Exhibit 13.5; GAB 476; Primary reasons [370].
[108] Exhibit 12; GAB 393 - 398.
[109] Exhibit 12; GAB 394.
[110] Exhibit 12; GAB 395; Primary reasons [403].
[111] Exhibit 12; GAB 397; Primary reasons [404].
[112] Exhibit 12; GAB 397.
[113] Exhibit 16.2; GAB 574 - 556.
[114] Exhibit 16.3; GAB 577.
[115] Exhibit 16.3; GAB 574; Primary reasons [453].

inflammatory response to her burns, possibly with a concurrent infection (early high fever within the first 24 hours of her burn injury).[116]

64 Upon reading the reports of Dr Numa and Professor Kesson, Professor Kimble prepared a short second report dated 2 June 2017, where he confirmed that neither experts' report altered his original statement,[117] and that he was in complete agreement with Professor Harvey.

65 Professor Kimble was of the opinion that the burn was a mixture of superficial partial thickness and deep dermal burns, and that the depth and size of the burn in a child of the respondent's age, frequently leads to a systemic inflammatory response which can result in ARDS.[118] He stated that he had witnessed many similar scenarios in small children with moderate size burns where they had developed similar severe systemic inflammatory responses resulting in ARDS.[119]

***Associate Professor John Harvey***

66 Professor Harvey prepared four reports dated 13 April 2015,[120] 9 September 2015,[121] 23 March 2016,[122] and 27 April 2017.[123]

67 In his 13 April 2015 report, he supported the finding that the respondent suffered an 18% burn to her TBSA.[124] As Professor Harvey found no evidence that the respondent was given excess fluid,[125] he was of the opinion that acute respiratory deterioration occurred after the bath of the respondent on 12 December 2015, which was most likely due to rapid progression of an unusually severe systemic inflammatory response which led to ARDS.[126]

68 In his report dated 23 March 2016, Professor Harvey confirmed his original opinion that there was no evidence that too much fluid was given to the respondent to cause fluid overload,[127] and that the rapid

[116] Exhibit 16.3; GAB 575; Primary reasons [455].
[117] Exhibit 16.3; GAB 577.
[118] Exhibit 16.3; GAB 577; Primary reasons [458].
[119] Exhibit 16.3; GAB 577; Primary reasons [458].
[120] Exhibit 21.2; GAB 723 - 728.
[121] Exhibit 21.3; GAB 729.
[122] Exhibit 21.4; GAB 730 - 731.
[123] Exhibit 21.5; GAB 732 - 737.
[124] Exhibit 21.2; GAB 723; Primary reasons [504].
[125] Exhibit 21.2; GAB 725, 726.
[126] Exhibit 21.2; GAB 726 - 727; Primary reasons [506].
[127] Exhibit 21.4; GAB 731; Primary reasons [512].

progression of the respiratory distress and pulmonary oedema, despite proper management, predominately reflected the unusual severity of the SIRS in the respondent.[128]

69 Professor Harvey's fourth report was a response to the reports of Dr Numa.[129] He found that the respondent did not deteriorate over the first 48 hours.[130] Such deterioration was related to signs of respiratory distress with basal lung crepitations, suggestive of pulmonary oedema. The differential diagnosis was sepsis/ARDS.[131]

70 At the time of admission to PICU, Professor Harvey was of the opinion that there was no absolute evidence of sepsis. While the elevation of CRP and neutropenia may represent sepsis, in the setting of burns Professor Harvey stated that there was no evidence from the literature that either of those parameters were specific to sepsis, rather than the SIRS associated with burns.[132]

71 After reviewing the evidence relating to sepsis, Professor Harvey found that there was not 'good evidence' to invoke sepsis as a cause of the respondent's deterioration, in the face of 'repeated clinical evidence of a respiratory basis for her deterioration'.[133] He then turned to the evidence of the respondent's respiratory distress and outlined that, as diuretics had failed to resolve it, her deterioration was likely related to an evolving ARDS.[134] Further, he stated that while ARDS is more common in major burns and associated with inhalation injury, it may occur in more minor burns (20%), although the exact incidence is unknown.[135]

72 Professor Harvey stated that in retrospect it is likely that ARDS was the cause of the respondent's deterioration. Finally, he stated that while ARDS may be exacerbated by the presence of sepsis and antibiotics may have benefited the respondent, there was no evidence at the time of infection nor of signs that would lead clinicians to start antibiotic treatment before 11 December 2005.[136]

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[128] Exhibit 21.4; GAB 731; Primary reasons [512].
[129] Exhibit 21.5; GAB 732.
[130] Exhibit 21.5; GAB 732; Primary reasons [514].
[131] Exhibit 21.5; GAB 732; Primary reasons [514].
[132] Exhibit 21.5; GAB 732; Primary reasons [515].
[133] Exhibit 21.5; GAB 732; Primary reasons [516].
[134] Exhibit 21.5; GAB 732; Primary reasons [517].
[135] Exhibit 21.5; GAB 732; Primary reasons [518].
[136] Exhibit 21.5; GAB 733; Primary reasons [519].

## Findings and reasons of the trial judge

73 The learned trial judge structured his reasons in the following way:

(a) first his Honour addressed the question as to whether the respondent in fact had sepsis on 10 December 2005 or thereafter;[137]

(b) secondly, having concluded that the respondent did have sepsis by the evening of 10 December 2005, his Honour considered whether the appellant was in breach of its duty of care in failing to detect and treat the sepsis with antibiotics;[138] and

(c) finally, his Honour addressed whether the breach of the duty of care caused the respondent's injuries.[139]

74 In relation to each of these issues, his Honour separately set out the evidence relevant to the issue, before setting out his Honour's analysis and decision.

### Reasons as to whether the respondent had sepsis

75 In the course of setting out the evidence of each of the expert witnesses in relation to the question as to whether the respondent had sepsis, the learned trial judge made express findings concerning his assessment of each witness. In that regard, his Honour referred to the quality of their reports, the extent to which their opinions were supported by relevant research articles, the manner and clarity with which they gave their evidence and the extent to which they demonstrated an understanding of their areas of specialisation.

76 By way of two examples, his Honour made the following findings in relation to Dr Numa and Dr Prebble respectively:[140]

> Dr Numa was, in my respectful view, a very impressive witness. His reports, subject to one qualification, clearly exposed his process of reasoning and the bases for reaching the conclusions that he did, including by reference to articles and publications. The fact that the articles and publications cited by him supported the propositions for which they were cited was at no stage challenged. Further, Dr Numa also gave his oral evidence in a clear and unambiguous way. He

[137] Primary reasons [96] - [696].
[138] Primary reasons [697] - [805].
[139] Primary reasons [806] - [845].
[140] Primary reasons [199], [350]. The 'one qualification' referred to by his Honour is referred to at [192] below.

> demonstrated an intimate understanding of his areas of specialisation. He was able to explain clearly without any difficulty whatsoever the bases for the opinions and conclusions that he had expressed in both his reports and during the course of his evidence even when these opinions and conclusions were subjected to challenge in cross-examination.
>
> …
>
> Dr Prebble was a measured, cautious and conscientious witness. I have no doubt, having read his reports and having watched and listened to him give evidence, that the opinions that he expressed are the result of careful and detailed consideration by him of the circumstances of the plaintiff's case. Nonetheless, the weight that can appropriately be placed on his evidence is, in my view, reduced by reason of the fact that the opinions that he expressed were not based on any practical experience in the management or treatment of ARDS or burns in paediatric patients. That this is the case is reflected, in my view, by the fact that the defendant's counsel did not in his closing submissions endeavour to place any great reliance on the evidence of Dr Prebble, at least in relation to the question of whether or not the plaintiff had sepsis.

77 When it came to his analysis and decision, his Honour commenced by recognising that the respondent bore the onus of proving, on the balance of probabilities, that she had sepsis on 10 December 2015 and that it was necessary that his Honour feel an actual persuasion of the existence of that fact.[141]

78 The learned trial judge then recorded his critical finding: [142]

> Ultimately, I have come to the conclusion that I am satisfied that the plaintiff did by the evening of 10 December 2005 have sepsis and that her sepsis continued to evolve up until the time that she was transferred to and treated in the ICU. I am persuaded of this fact.

79 His Honour then proceeded to give 13 reasons which, in combination, satisfied him of that fact. Most of those reasons are the subject of individual grounds of appeal. In summary, the 13 reasons given by his Honour were:

1. the respondent suffered superficial partial thickness and deep dermal burns to approximately 18% of her TBSA and, in any event, to less than 20% of her TBSA;[143]

[141] Primary reasons [666], referring to ***Briginshaw v Briginshaw*** [1938] HCA 34; (1938) 60 CLR 336 361 - 362.
[142] Primary reasons [667].
[143] Primary reasons [668].

2. the evidence established that although SIRS and ARDS can develop in the absence of sepsis, the more common, if not most common, cause of SIRS, and consequentially ARDS, in paediatric patients is sepsis;[144]

3. the evidence established that where a child of around the respondent's age suffers partial thickness and deep dermal burns to less than 20% of her body, the chance of the child developing ARDS as a result of SIRS in the absence of sepsis is, if not 'vanishingly small', very low indeed;[145]

4. out of the three expert witnesses who were, in his Honour's view, best positioned and best qualified to express an opinion as to whether the respondent did have sepsis on the evening of 10 December 2005 or at any time thereafter (namely Dr Numa, Professor Kesson and Professor Starr) two of them, namely Dr Numa and Professor Kesson, were of the opinion that it was more probable than not that the respondent did have sepsis;[146]

5. out of all the highly qualified and generally impressive expert witnesses who gave evidence, Dr Numa was, in his Honour's opinion, for reasons he had given, the most impressive;[147]

6. none of the appellant's expert witnesses expressed the opinion that the respondent definitely did not have sepsis;[148]

7. on the night of her admission the respondent did have an 'Enterobacter +++' infection in her right knee burn wound. His Honour found that this infection was more than a mere skin contaminant and was capable of contributing to, or causing, the respondent's ARDS in one of two ways: either by the bacteria making its way into the bloodstream or, as explained by Dr Numa, by the infection resulting in the release of cytokines into the bloodstream, which caused the respondent's SIRS (which in turn caused the ARDS);[149]

[144] Primary reasons [669].
[145] Primary reasons [670].
[146] Primary reasons [672]. In this context, his Honour expressly recognised that the relevant question was not to be decided solely on the basis of the numbers of experts favouring one view or the other.
[147] Primary reasons [675].
[148] Primary reasons [676].
[149] Primary reasons [677].

8. by the evening of 11 December 2005 there were several features of the respondent's case which, viewed collectively, were not only consistent with, but also suggestive of, the respondent having a bacterial infection and sepsis rather than simply a severe inflammatory response to sterile burns. His Honour elaborated on this reason by reference to the respondent's vital signs and the evidence of the expert witnesses;[150]

9. the respondent's coagulation profile taken on 12 December 2005 was indicative of sepsis;[151]

10. within a relatively short time of the respondent being placed on antibiotics her white blood cell count and neutrophil count began to rise. This, as was explained by Dr Numa, was consistent with the antibiotics killing the bacteria, with the result that the respondent's bone marrow was able to start replenishing the respondent's neutrophils;[152]

11. although the single blood culture produced from the sample of blood taken from the respondent returned a negative result for bacteria, the evidence adduced revealed, in his Honour's view, credible explanations for the result;[153]

12. although the blood cultures produced from samples taken from the respondent after she had been admitted to the ICU returned negative results for bacteria his Honour was satisfied, on the basis of the evidence given by Dr Numa and Professor Kesson, that there was a credible explanation for those results;[154] and

13. although the samples of fluid taken from various regions of the respondent's body on 13 December 2005 returned negative results for bacteria, his Honour was satisfied on the basis of the evidence given by Dr Numa that those samples were unlikely to return positive results even in the presence of infection and sepsis.[155]

80 Having identified these reasons for the sepsis finding, the learned trial judge went on to make a number of additional points, including that he did not accept the evidence of the radiologist Dr Murray to the

[150] Primary reasons [678].
[151] Primary reasons [684].
[152] Primary reasons [685].
[153] Primary reasons [687].
[154] Primary reasons [690].
[155] Primary reasons [691].

effect that that the nature, tempo and persistence of the respondent's pulmonary opacification were typical of a sterile inflammatory process.[156]

## Reasons as to breach of duty of care

81 In relation to the issue of breach, the learned trial judge commenced by identifying the relevant standard of care that was required of the individual doctors who were responsible for the care and treatment of the respondent while she was a patient in the Hospital's burns ward, and hence the standard of care that was required of the appellant. His Honour found that that was the standard of the ordinary skilled practitioner working within the specialist field of paediatric burns.[157]

82 The learned trial judge identified the relevant provisions of the *Civil Liability Act*, including s 5B and s 5PB. In relation to the latter section, his Honour indicated that he would proceed on the basis that, in order to prove the alleged breach of duty, the respondent was required to prove that the conduct of the doctors who were responsible for the care and treatment of the respondent was not in accordance with a practice which is widely accepted by their peers as competent professional practice. [158]

83 After reviewing the expert evidence in relation to the care provided to the respondent, the learned trial judge concluded:[159]

> Having reviewed the PMH medical records relating to the plaintiff, I am satisfied beyond any doubt that prior to the plaintiff being admitted to the ICU no consideration was given by any of the doctors who, to that point, had been responsible for her care and treatment to the possibility that her deterioration was due to sepsis as opposed to fluid overload.[160] …
>
> In addition, the doctors responsible for the care and treatment of the plaintiff while she was in the burns ward did not commence the plaintiff on antibiotics. The fact that they did not do so is, of course, consistent with the fact that they all considered the cause of the plaintiff's problems to be fluid overload.

[156] Primary reasons [696].
[157] Primary reasons [704].
[158] Primary reasons [714].
[159] Primary reasons [794] - [795].
[160] Primary reasons [794].

84 In light of those findings, the learned trial judge identified the relevant question under s 5PB(1) as being whether the failure to recognise that the respondent might be suffering from sepsis, the failure to test for sepsis and the failure to commence her on antibiotics, was conduct that was in accordance with a practice that was widely accepted by peers of the doctors responsible for the care and treatment of the respondent in the burns ward as competent professional practice.[161]

85 His Honour answered that question 'no', observing that unsurprisingly, none of the medical witnesses who gave evidence suggested that it was widely accepted by peers of doctors working in a paediatric burns unit as competent professional practice to fail to recognise that a patient is, or might be, suffering from sepsis and in those circumstances to fail to test for sepsis and to fail to administer antibiotics.[162]

86 Having concluded that the respondent had discharged the onus of excluding the operation of s 5PB(1) of the *Civil Liability Act*, the learned trial judge turned to the general issue of breach (i.e. under s 5B of the *Civil Liability Act*); namely, whether the burns ward doctors, in failing to recognise by 10 December 2005, or at any time thereafter, that the respondent was, or might be, suffering from sepsis, and from that time in failing to test for sepsis and commence administering antibiotics to the respondent, fell below the standard of care required of them. [163]

87 In that regard, his Honour stated that he was satisfied that:[164]

(a) in burns cases antibiotics should not be given prophylactically;

(b) infection and sepsis is always a concern in burns cases, even in cases involving burns of the nature and extent suffered by the respondent;

(c) sepsis can progress to severe sepsis and ARDS quickly;

(d) where infection and sepsis in a paediatric burns patient is suspected on clinical grounds, the appropriate course of action is, given the potential severity of the consequences of sepsis and

[161] Primary reasons [799].
[162] Primary reasons [800].
[163] Primary reasons [800].
[164] Primary reasons [803] - [804].

the speed with which it can evolve, to take a sample of the patient's blood for culturing and to commence the patient on broad spectrum antibiotics as quickly as possible without waiting for the blood culture results;

(e) the downside of not administering antibiotics to a paediatric burns patient who on clinical grounds is suspected to be suffering from sepsis far outweighs the potential benefits of adopting such a course of action; and

(f) the signs and symptoms which the respondent had exhibited on 10 December 2005 should, by no later than around 2.00 am on 11 December 2005, have caused the team of doctors responsible for the respondent's care and treatment in the burns ward to recognise the possibility (that is, the foreseeable and not insignificant risk) that she did have evolving sepsis and, in light of this possibility, to take a sample of her blood for culturing and to commence administering to her as quickly as was reasonably possible (say, within an hour) broad spectrum antibiotics pending the receipt of the results of the blood culture.

88 Ultimately, the learned trial judge was satisfied that by failing to recognise, by around 2.00 am on 11 December 2005, the possibility that the respondent had sepsis and by failing to administer antibiotics as quickly as was reasonably possible, the team of doctors responsible for the care and treatment of the respondent in the burns ward did not meet the standard of care that was required of the ordinary skilled practitioner working within the specialist field of paediatric burns.

89 These being the findings that are the subject of the appeal, we turn to the grounds of appeal.

## Grounds of appeal

90 The grounds of appeal that were maintained at the hearing of the appeal are as follows (particulars omitted):

> 1. The trial judge erred in law by failing to consider and determine an essential issue, being that for a child of the Respondent's… age who suffers partial thickness and deep dermal burns to less than 20% of her body, the chance of developing [ARDS] as a result of [SIRS] in the absence of sepsis is very low; Judgment [670]. The trial judge ought to have found that the fact that [the respondent] had partial thickness and deep dermal burns to less

than 20% of her body, and had developed ARDS as a result of SIRS, was not relevant to whether on the evening of 10 December 2005 [the respondent] had sepsis.

…

2. The trial judge erred in fact in finding that for a child of [the respondent's] age who suffers partial thickness and deep dermal burns to less than 20% of her body, the chance of developing ARDS as a result of SIRS in the absence of sepsis is very low, was evidenced by the absence of such cases reported in the academic literature; Judgment [670]. The trial judge ought to have found that the fact that the absence of such cases being reported in academic literature was irrelevant to whether on the evening of 10 December 2005 [the respondent] had sepsis.

…

3. The trial judge erred in law and in fact in finding that that the expert medical paediatric burns specialists, Professors Harvey and Kimble, were not qualified to express an opinion as to whether it was more probable than not that [the respondent] suffered from sepsis on the evening of 10 December 2005 or less qualified than other expert medical witnesses, Dr Numa, Professor Kesson and Professor Starr; Judgment [672] - [673]. The trial judge ought to have found that the expert witnesses with the greatest relevant expertise to express an opinion as to whether [the respondent], with partial thickness and deep dermal burns to less than 20% of her body, had developed ARDS as a result of SIRS caused by sepsis, on the evening of 10 December 2005 were Professors Harvey and Kimble.

…

4. The trial judge erred in law in finding that Dr Numa's opinions were to be preferred to those of all other expert witnesses whose opinions differed; Judgment [675]. The trial judge ought to have found that Dr Numa's opinions were of no greater probative value in determining whether on the evening of 10 December 2005 [the respondent] had sepsis, than those of Professor Starr and of less probative value than those of Professors Kimble and Harvey.

…

5. The trial judge erred in fact in finding that the probative value of Professor Harvey's opinion as to the cause of [the respondent] suffering ARDS was adversely affected by Professor Harvey incorrectly recalling details of a case about which he gave evidence to support his opinion; Judgment [588] - [596]. The

trial judge ought to have found that there was no basis to question any opinion of Professor Harvey.

…

6. The trial judge erred in law and in fact in finding that the Enterobacter infection of [the respondent's] right knee burn wound caused the release of cytokines into her bloodstream which caused SIRS which caused ARDS prior to the evening of 10 December 2005; Judgment [677]. The trial judge ought to have found that the respondent had not proved that Enterobacter infection of [the respondent's] right knee burn wound caused [the respondent's] ARDS prior to the evening of 10 December 2005.

…

7. The trial judge erred in law and in fact in finding that by the evening of 11 December 2005 there were several features which collectively were suggestive of [the respondent] having by the evening of 10 December 2005 a bacterial infection and sepsis and not SIRS without infection; Judgment [678]- [682]. The trial judge ought to have found that the respondent had not proved that by the evening of 11 December 2005 there were several features which collectively were suggestive of [the respondent] having by the evening of 10 December 2005 a bacterial infection and sepsis and not SIRS without infection.

…

8. [Not pursued]

9. The trial judge erred in law and fact in finding that changes to [the respondent's] white blood cell count and neutrophil count on 12 and 13 December 2005 was indicative of her having sepsis on the evening of 10 December 2005; Judgment [685]. The trial judge ought to have found that changes to [the respondent's] white blood cell count and neutrophil count on 12 and 13 December 2005 were irrelevant.

…

10. The trial judge erred in law and fact in rejecting the evidence of Professor Starr that bacterial infection of the bloodstream caused by infection on the surface of the skin does not usually occur within the first 24 to 48 hours of infection on the surface of the skin; Judgment [694] - [695]. The trial judge ought to have found that bacterial infection of the bloodstream caused by infection on the surface of the skin does not usually occur within the first 24 to 48 hours of infection on the surface of the skin

and that thereby it was unlikely that Enterobacter infection of [the respondent's] right knee burn wound caused [the respondent's] ARDS prior to the evening of 10 December 2005.

…

11. The trial judge erred in law and fact in finding that by the evening of 10 December 2005 or by 3.00am on 11 December 2005 [the respondent] had sepsis; Judgment [692]. The trial judge ought to have found that the respondent had not proved that that by these times [the respondent] had sepsis.

…

12. The trial judge erred in law in construing s 5PB(l) of the *Civil Liability Act 2002* as requiring that the Appellant prove the existence of a practice that was widely accepted by the burns ward doctors' peers as competent professional practice, in accordance with which the Appellant's officers acted; Judgment [796].

13. The trial judge erred in law and fact in finding that for the purpose of s 5PB(l) of the *Civil Liability Act 2002*, the burns ward doctors peers included Dr Numa and Professor Kesson; Judgment [715], [717] - [743].

…

14. The trial judge erred in law and fact in finding that the alleged breaches of duty by the burns ward doctors prior to the evening of 10 December 2005 were negligent in that the treatment of [the respondent] prior to the evening of 10 December 2005 was contrary to practice widely accepted by the burns ward doctors' peers as competent professional practice; Judgment [793], [801]. The trial judge ought to have found that [the respondent's] treatment by the burns ward doctors prior to the evening of 10 December 2005 was not negligent.

…

15. The trial judge erred in law and fact in finding that the alleged breaches of duty by the burns ward doctors prior to 3.00am on 11 December 2005 were negligent in that the treatment of [the respondent] prior to 3.00am on 11 December 2005 was contrary to practice widely accepted by the burns ward doctors' peers as competent professional practice; Judgment [804]-[805]. The trial judge ought to have found that [the respondent's] treatment by the burns ward doctors to 3.00am on 11 December 2005 was not negligent.

…

> 16. The trial judge erred in fact in finding that prior to the respondent being admitted to the ICU no consideration was given by any of the doctors who had been responsible for her care and treatment to the possibility that deterioration in the respondents condition was due to sepsis as opposed to fluid overload; Judgment [794].

## Grounds 1 to 11 - challenge to the finding of fact that the respondent had sepsis

91 As noted above, grounds 1 to 11 are all directed towards the learned trial judge's finding that, by the evening of 10 December 2005, the respondent had sepsis and that her sepsis continued to evolve up until the time she was transferred to, and treated, in the ICU at the Hospital (the **sepsis finding**). As can be seen from those grounds, many of them challenge the individual reasons identified by his Honour for reaching that finding.

92 Before turning to the individual grounds, it is appropriate to make three general observations in relation to the challenge to the learned trial judge's finding of fact in this regard.

93 First, the sepsis finding was to a substantial extent informed by the learned trial judge's assessment of the credibility and reliability of the parties' expert witnesses. In particular, the learned trial judge was impressed with the credibility and reliability of Dr Numa's testimony. The general principles relevant to appellate restraint in this context were summarised most recently by this court in ***Smart v Power***.[165] The principles have also more recently been restated by the High Court in ***Lee v Lee***:[166]

> A court of appeal is bound to conduct a "real review" of the evidence given at first instance and of the judge's reasons for judgment to determine whether the trial judge has erred in fact or law. Appellate restraint with respect to interference with a trial judge's findings unless they are "glaringly improbable" or "contrary to compelling inferences" is as to factual findings which are likely to have been affected by impressions about the credibility and reliability of witnesses formed by the trial judge as a result of seeing and hearing them give their evidence. It includes findings of secondary facts which are based on a combination of these impressions and other inferences from primary facts. Thereafter, "in general an appellate court is in as good a position as the trial judge to decide on the proper inference to be drawn from facts which are undisputed or which, having been disputed, are established by the findings of the trial judge". (footnotes omitted)

[165] ***Smart v Power*** [2019] WASCA 106 [100] - [105] (Murphy, Beech, Pritchard JJA).
[166] ***Lee v Lee*** [2019] HCA 28 [55] (Bell, Gageler, Nettle, Edelman JJ).

94 These principles apply equally to the testimony of expert witnesses.[167]

95 In this context, due regard must be had to the advantages of the learned trial judge. In that regard, it would:[168]

> be wrong to limit 'the advantages which the primary decision-maker has' to demeanour as a guide to credibility assessment and to ignore the 'feeling of a case' that usually emerges from running a trial. The primary decision-maker is able to assess testimony against the entirety of the evidence and in a situation in which she or he has an appreciation of the way the trial was run. There may, for example, be subtleties in the way questions were asked (or avoided) that are apparent in the heat of battle but which are not quite as clear in a more clinical examination of a transcript. Similarly, the effect of evidentiary rulings or rulings about the pleadings made at one stage of a trial may have a greater impact at another point in the proceedings than will be apparent from the record. In carrying out its duty to decide for itself on the proper inference to be drawn from facts an appellate court must be alive to the entire context in which findings were made.

96 In the present case, a number of the grounds of appeal, in substance (if not in form) proceeded upon an analysis of the competing evidence, and choices made by the learned trial between competing evidence, without clearly articulating why his Honour was wrong to have made those choices. In that regard, it is not sufficient, for example, to submit that his Honour *should* have accepted certain evidence without demonstrating why his Honour was in error not to have done so.

97 Secondly, in relation to a number of the learned trial judge's findings of fact it is apparent that his Honour preferred the evidence of some experts over others. This was particularly the case in relation to the learned trial judge's fifth reason for the sepsis finding, namely that Dr Numa was, in his Honour's assessment, and for reasons he had given, the most impressive witness.[169] This finding and the adequacy of the reasons for it, in particular, are challenged by ground 4, which we will deal with in more detail later.

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[167] ***Abalos v Australian Postal Commission*** [1990] HCA 47; (1990) 171 CLR 167, 178 - 179; ***Kakavas v Crown Melbourne Ltd*** [2013] HCA 25; (2013) 250 CLR 392 [131]; ***DRA Industries Pty Ltd v Kuredale Pty Ltd*** [2018] WASCA 17 [52]; ***Christos v Curtin University of Technology*** [2017] WASCA 110 [104]; ***Western Australia v Watson*** [1990] WAR 248, 305; ***X and Y (by her tutor X) v Pal*** (1992) 23 NSWLR 26, 34, 49; ***Davids Holdings Pty Ltd v Attorney-General (Cth)*** (1994) 49 FCR 211, 243 - 244.

[168] ***Brett v Rees*** [2009] WASCA 159 [69] (Owen JA, Martin CJ & Miller JA agreeing).

[169] Primary reasons [675].

98 For present purposes, however, it is sufficient to observe that, in a number of respects, the appellant's complaint that the learned trial judge gave inadequate reasons for preferring one expert's evidence over another was that his Honour did not give reasons for aspects of (or for the use of particular words in) those reasons. The fact that Dr Numa was found by his Honour to be the most 'impressive' witness, for example, was said to be inadequate because 'impressive was not a reason',[170] notwithstanding that his Honour identified a number of matters arising from Dr Numa's reports, and the manner in which he gave his evidence, for expressing confidence in his evidence.[171]

99 The principles relevant to an evaluation of the adequacy of reasons, are well known and were recently summarised in ***Chief Executive Officer, Department for Child Protection and Family Support v IGR***.[172] They include the need to disclose the intellectual process that led to the decision in sufficient detail and with sufficient certainty to enable the litigant to know why they were unsuccessful and to enable an appeal court to determine whether the decision involved appellable error.

100 Nevertheless, where a primary court does set out that intellectual process, including by reference to the demeanour of witnesses,[173] the obligation to give adequate reasons does not involve, or require, an elaborate explanation of each expression used to describe that evidence, as if by an infinite regression of 'reasons for reasons'. At some point a trial judge reaches bedrock and his or her spade is turned.[174]

101 Finally, in relation to the grounds challenging the sepsis finding, it is to be recalled that the learned trial judge expressly stated that the reasons he set out persuaded him of that fact 'considered not in isolation from each other but rather in combination'.[175]

102 In considering the individual challenges made to the 13 reasons given by his Honour, therefore, it is important to remember that the extent to which a particular reason might be said to have supported the

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[170] Appeal ts 47-50.
[171] See, for example, at [76] above.
[172] ***Chief Executive Officer, Department for Child Protection and Family Support v IGR*** [2019] WASCA 20 [112] (Quinlan CJ, Murphy & Beech JJA).
[173] Which, in the case of experts, will include the clarity of their opinions, their evident familiarity with the subject matter (including any relevant academic or professional literature) in the course of giving evidence, their responsiveness to cross-examination, their apparent ease of explaining their opinions, and the apparent care taken in expressing their opinions.
[174] Wittgenstein L, *Philosophical Investigations* (1953), §217.
[175] Primary reasons [692].

overall sepsis finding is not to be judged in isolation. It may be that an individual matter might not, on its own, support a finding to the required standard that the respondent had sepsis but, nevertheless, provide support for the ultimate finding (in combination with all of the other matters).

103 We turn, then, to the individual grounds of appeal in relation to the sepsis finding.

**Ground 1 - The relevance of the extent of the respondent's burns**

104 A significant issue at trial was the likelihood that a child around the respondent's age with partial thickness and deep dermal burns to less than 20% TBSA, would develop ARDS as a result of SIRS in the *absence* of sepsis. The issue was relevant because, to the extent that the chance of that occurring (i.e. ARDS developing without sepsis) was very low, it supported (albeit did not compel) the inference that the respondent, who did develop ARDS, had sepsis.

105 This consideration was clearly identified by Dr Numa in his first report, in which he said that 'where the [respondent] had no full thickness burns and burns to no more than 18% of her TBSA, ARDS is unlikely to have developed as a result of the burn itself'.[176]

106 The rarity of that occurrence was contested by the appellant at trial, including by reference to articles and case studies referred to by its expert witnesses (the relevance of which were rejected by the learned trial judge).[177]

107 In the appeal the appellant did not challenge the learned trial judge's finding that the chance of a child of the respondent's age with partial thickness and deep dermal burns to less than 20% TBSA developing ARDS as a result of SIRS in the absence of sepsis was, if not 'vanishingly small', very low indeed.[178]

108 Rather, by ground 1, the appellant submits that the learned trial judge erred in taking into account the very low probability of ARDS in the absence of sepsis, because the probability of a child in the respondent's position 'developing ARDS in the relevant circumstances by reason of sepsis and in the absence of sepsis were equally low'.[179]

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[176] See [30] above.
[177] Primary reasons [595], [671].
[178] Primary reasons [670].
[179] Appellant's amended submissions [15]; WAB 14.

In essence, the appellant alleged that, despite the significant contest at trial in relation to the likelihood of developing ARDS in the absence of sepsis, that contest was irrelevant because the likelihood of a child in the respondent's circumstances developing ARDS *with* sepsis, was the same as it developing *without* sepsis.

109 There was no finding by the learned trial judge that the likelihood of those two outcomes were the same. The respondent submits that this is not surprising as that assertion was not explored in the evidence at trial;[180] that is, the suggestion of equal probabilities was not raised in the expert reports, put to the expert witnesses at trial or put in submissions.[181]

110 The respondent's submission in this regard should be accepted. There was no suggestion at trial that the likelihood of a child with burns to less than 20% TBSA developing ARDS in the presence of sepsis, was the same as the likelihood of ARDS developing in the absence of sepsis.

111 Indeed, the very premise of the debate between the expert witnesses as to whether there were any reported cases of a child developing ARDS with burns to less than 20% TBSA in the absence of sepsis, was that any such cases (if they existed) would be a subset of all cases in which ARDS developed in the case of burns to less than 20% TBSA. And in that context his Honour found, contrary to the evidence of Dr Prebble and Professor Harvey, that there were **no** cases reported in the literature of a child with burns to less than 20% TBSA developing ARDS in the absence of infection.[182] Those reported cases in which persons with burns to less than 20% TBSA **did** develop ARDS did not support that proposition, because those cases were concerned with either infection or direct injury to the lungs (which the respondent did not have).[183]

112 The fact that there were reported cases of ARDS developing in persons with burns to less than 20% TBSA *with* infection (such as that referred to by Professor Harvey),[184] rather supports the notion, implicit in the debate between the experts, that the presence of infection *increases* the likelihood of ARDS in a patient with such burns, not that the likelihood of ARDS is the same whether infection is present or not.

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[180] Respondent's amended submissions [18]; WAB 76.
[181] Respondent's amended submissions [20]; WAB 78.
[182] Primary reasons [670] - [671].
[183] Primary reasons [671].
[184] Primary reasons [595].

113 In any event, the evidence at trial did not support the conclusion that the probabilities of developing ARDS in the respondent's circumstances were the same, with or without sepsis. Professor Starr for example, expressly agreed that it was likely to be true that 'it was far more common for ARDS and SIRS to develop if the burns are less than 20% if there is also sepsis present'.[185]

114 The evidence relied upon by the appellant in support of the proposition that the probabilities were the same, with or without infection, was the following evidence of Professor Kimble:[186]

> We treat 1,100 children with new burns every year. So over the last two years, that would be 2,200 children, new burns.
>
> And are you personally involved with each patient?---I'm director of the - the service, and so I am not involved with every single patient. But I will know about them all because they're all discussed. There's only five paediatric burns surgeons here and we have a meeting every week where we discuss all the significant patients and that we - you know, so although I'm not the permanent clinician for all these patients, I make myself aware of them all. And I am personally involved with all the significant burns. That's something which I've done for the last - years that - but for kids with larger burns, you always require two consultant burns surgeons to - to deal with them. And so I can fairly happily say that I've been involved with all the major burns patients really since 1999 in this institution.
>
> And you gave a statistic that only half a dozen of those patients have developed serious ARDS. Is that right?---Yes. Every year, we - we'll only have maybe a half a dozen patients who we'd have enough respiratory compromise to - to require intensive care treatment.
>
> And of those half a dozen, you say that three had problems with infection?---This is over the last two years that we've had three who we felt had significant infections. And - and sometimes you've got to presume these things because cultures don't always - they're not always as helpful as you would think. But we presumed that three of them had the problems partially due, or totally due, to the presence of sepsis: two of them bacterial, one of them viral.
>
> All right. And of the - the other three of that half dozen you say was unexpected ARDS and you didn't find evidence of infection. Is that what you're saying?---Yeah. A number of the infections over the last two years were - we have half a dozen who will require intensive care support. So, you know, all I can say about the others, we have not identified positive organisms. And, you know, these - these are

[185] Trial ts 221; Primary reasons [434].
[186] Trial ts 313-314 (see Appeal ts 21-22).

> estimates I'm giving that - I was never asked to provide accurate statistical data. This is just going from my experience of treating major burns in this hospital.

115 His Honour clarified this evidence, with the agreement of the witness:[187]

> DERRICK DCJ: No. But, Mr Lampropoulos, that's the point. The doctor hasn't given - the doctor is not saying that three of the six didn't have infection. What he's saying is over the past 12 months, six had developed respiratory compromise, and over the last two years, three had presumed infection. He's not saying three of the six.
>
> LAMPROPOULOS, MR: All right.
>
> DERRICK DCJ: At least, that's not what he's said thus far?---That's - that's absolutely correct.
>
> LAMPROPOULOS, MR: Well, if I can just get my mind on that, how many in the last one or two years do you say did not have infection?---As I said, I don't have accurate numbers.

116 This evidence does not support an equivalence of the likelihood of developing ARDS with burns to less than 20% TBSA with infection, with the likelihood of developing ARDS in such a case without infection. On the contrary, it is not addressing cases of ARDS in patients with burns to less than 20% TBSA at all. The evidence was dealing with the incidence, in Professor Kimble's hospital, of respiratory distress and infection generally, without reference to the size of the burns. Indeed, as he stated, Professor Kimble's own personal involvement was with 'kids with larger burns'.

117 For these reasons, the learned trial judge did not err in taking into account the very low probability of ARDS in the absence of sepsis, or in failing to find that probability of a child in the respondent's position developing ARDS by reason of sepsis, and in the absence of sepsis, were equally low.

118 Ground 1 is not made out.

**Ground 2 - The absence of reported cases of ARDS in the absence of sepsis**

119 Ground 2 is related to ground 1, and concerns the learned trial judge's conclusion that the chance of a child of the respondent's age with partial thickness and deep dermal burns to less than 20% TBSA

[187] Trial ts 315.

developing ARDS as a result of SIRS in the absence of sepsis was, if not 'vanishingly small', very low indeed.[188]

120 In that context, the appellant challenges the learned trial judge's reference to, and apparent reliance upon, the 'fact that there are no such cases reported in the literature'. The appellant does not appear to challenge the finding that the relevant chance was 'very low' (which it earlier stated was expressly not challenged)[189] but rather that it was low 'in comparison' to those cases in which there is an infective process involved.[190]

121 The ground of appeal, accordingly, raises essentially the same issue as ground 1, namely that the learned trial judge erred in not finding that the probabilities of a child in the respondent's position developing ARDS by reason of sepsis, and in the absence of sepsis, were equally low. For the reasons set out in relation to ground 1, his Honour did not err in this regard.

122 The additional feature raised by ground 2, is the submission that the learned trial judge did not refer to the 'plainly credible and probative' explanation given by Professor Kimble for there being an absence of reported cases in the literature.

123 The evidence of Professor Kimble relied upon by the appellant in this regard was as follows:[191]

> Have you ever published in relation to ARDS in burns patients in any peer-reviewed publication?---No, and the reason why we don't is because we publish on areas where we have that critical mass of patients, and so the ARDS area in burns is best done by the large - very large paediatric institutes, such as Shriners in the USA and the major burns institutes in China that really, our contribution to the world literature for ARDS in burns, you know that you're not going to get that critical mass to make meaningful studies from this country. And that's why - you know, I'm one of the most published people in the world in paediatric burns, but I will not publish on areas that we don't have that critical mass. You know, we will generally talk about the numbers of our patients and what happened to them, but I do not know any specific publications on ARDS.

[188] Primary reasons [670].
[189] Appellant's amended submissions [12]; WAB 13 ('This finding is not challenged').
[190] Appeal ts 28.
[191] Trial ts 317 (see Appellant's amended submissions [19]; WAB 14).

124 This evidence, with respect, is not an explanation as to why there are no reported cases of a child of the respondent's age with burns to less than 20% TBSA developing ARDS in the absence of sepsis. It is simply an explanation as to why Professor Kimble had not published in the field. It says nothing of the extent of the research literature generally or what that literature reveals.

125 Plainly there **is** research literature in the field of ARDS and burns patients. A number of the expert witnesses referred to it, and various articles were produced by those witnesses. Dr Numa's initial report, for example, specifically referred to literature in support of his opinion that the chance of developing ARDS with burns to less than 20% TBSA in the absence of sepsis was 'vanishingly small'.[192] The fact that Professor Kimble expressed a reason for not publishing such literature was therefore irrelevant to that issue.

126 In addition, in the context of this ground of appeal, the appellant submitted that the articles relied upon by the appellant at trial were to the effect that the pathological mechanisms involved in the occurrence of ARDS are likely to be multifactorial.[193] That may well be so. Those articles do not, however, detract from the learned trial judge's finding as to the very low probability of ARDS developing in a case such as the respondent's in the absence of sepsis. The fact that the aetiology of ARDS may be multifactorial, is not inconsistent with the proposition that TBSA percentage is one relevant factor. Indeed, it was the presence of another factor (sepsis) that was the issue in the present case.[194]

127 In any event the learned trial judge directly addressed those articles in his reasons for decision.[195] No error has been demonstrated in the manner in which his Honour dealt with them.

128 Ground 2 is not made out.

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[192] Exhibit 19.4; GAB 588; Primary reasons [120].
[193] Appellant's amended submissions [20]; WAB 14; See GAB 54.
[194] The appellant also submitted that one article stated that ARDS seems to be not, or only partially, related to TBSA% (Appellant's amended submissions [21]; WAB 14; See GAB 46). The learned trial judge's conclusion was, of course, entirely consistent with the notion that the incidence of ARDS was 'only partially related to TBSA': his Honour held that it was also related to other factors, including infection.
[195] Primary reasons [671].

## Ground 3 - Experts best qualified to given an opinion on sepsis

129 Ground 3 challenges the learned trial judge's conclusion that Dr Numa, Professor Kesson and Professor Starr were 'best qualified to express an opinion as to whether or not the [respondent] had sepsis'.[196]

130 The ground alleges that his Honour erred in finding that Professors Harvey and Kimble were not qualified to express an opinion as to whether it was more probable than not that the respondent suffered from sepsis, or less qualified than other expert medical witnesses. This was submitted to be on the basis of 'incontrovertible facts' as to the expertise of Professors Harvey and Kimble.[197]

131 Ground 3 misses its mark inasmuch as the appellant submitted that the learned trial judge concluded that Professors Harvey and Kimble 'were not qualified to express an opinion',[198] 'did not have relevant expertise',[199] 'did not have relevant expertise to express an opinion'[200] and that his Honour 'completely disregarded their views'.[201]

132 His Honour reached no such conclusions and did not completely disregard the views of Professors Harvey and Kimble. In the impugned passage in his Honour's reasons for decision, the learned trial judge was not concerned with whether Professors Harvey and Kimble possessed sufficient expertise to express an opinion, but with the *weight* that his Honour accorded to the various opinions, in relation to the issue then under consideration. This is made clear by the learned trial judge's repeated use of the expression '*best* qualified'.

133 It was in this context that the learned trial judge drew a distinction between those experts who were respiratory physicians, infectious diseases specialists and experts in ARDS (Dr Numa, Professor Kesson and Professor Starr) and the paediatric burns surgeons (Professors Kimble and Harvey). In that regard, his Honour concluded that the former were better qualified to answer the particular question: did the respondent in fact have sepsis?

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[196] Primary reasons [673].
[197] Appellant's amended submissions [40].
[198] Appellant's amended submissions [22].
[199] Appellant's amended submissions [22].
[200] Appellant's amended submissions [50].
[201] Appeal ts 39.

134 His Honour's reasons for that conclusion included the following passage:[202]

> As is apparent from my above general observations in relation to their evidence, I accept the proposition that Professor Kimble and Professor Harvey are experts who are qualified, based on their study and experience, to express an opinion as to the likelihood or probability of a child of the plaintiff's approximate age who has suffered burns of the type suffered by the plaintiff to approximately 18% of her TBSA, developing SIRS and consequential ARDS in the absence of sepsis (although I note that Professor Harvey's evidence on this issue was, for reasons I have already stated, impacted upon negatively by his apparent incorrect understanding and use of the case the subject of the case report). What I do not accept, however, contrary to the view apparently held by Professor Kimble, is that he and Professor Harvey are the experts who are best qualified to express an opinion as to whether the plaintiff in fact had sepsis. Diseases caused by bacterial infections obviously fall within the sphere of expertise of an infectious diseases physician. Moreover, ARDS, as the name suggests, is a respiratory condition which is treated in intensive care units. Intensivists will always be concerned to identify the cause of a patient's ARDS. In these circumstances, whether or not the plaintiff had sepsis (in the form of a bacterial infection) which caused her to suffer ARDS is a question which, in my view, falls more squarely within the fields of expertise of infectious diseases physicians, respiratory physicians and intensivists rather than paediatric burns surgeons.

135 A number of matters are apparent from this passage:

(a) the learned trial judge does not suggest that Professors Kimble and Harvey lack expertise. Indeed, his Honour recognised that they were qualified to express an opinion as to the likelihood of a child in the respondent's circumstances developing ARDS in the absence of sepsis (one of the matters relevant to the sepsis finding);[203]

(b) his Honour clearly identified the particular matter with which he was concerned (whether the respondent in fact had sepsis); and

(c) his Honour gave reasons *why that particular question* fell more squarely within the fields of expertise of Dr Numa, Professor Kesson and Professor Starr.

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[202] Primary reasons [673].

[203] Contrary to the appellants submissions (Appellant's amended submissions [23]; WAB 15), this recognition did not amount to an inverse finding that the professors had no expertise as to the likelihood of that occurring with sepsis.

136 The passage discloses no error on the part of the learned trial judge. Indeed, it provides a logical and sensible reason for his Honour's preference for the evidence of Dr Numa, Professor Kesson and Professor Starr in relation to that question.

137 The appellant's complaint, in this regard, relied heavily upon the fact that, as burns surgeons, Professors Kimble and Harvey were the experts who treated patients with burns and that the other experts had no such expertise.[204]

138 However, as the learned trial judge made clear, his Honour was not, at this point in his reasons, dealing with issues of treatment, or the standard of treatment of the respondent - or even whether those treating the respondent in the Hospital's burns ward did, or should have, considered the possibility that the respondent had sepsis.

139 At this point in his Honour's reasons, the learned trial judge was concerned with a pure question of historical fact: i.e. whether the respondent *in fact* had sepsis.

140 Unlike the question of the standard of care (which is concerned with issues of foreseeability and risk, based on the evidence available to the treating doctors at the time that the relevant events were unfolding), the factual question as to whether the respondent had sepsis was to be answered, in retrospect, in light of all of the evidence. This included the evidence as to what occurred on the burns ward, but also what occurred after the respondent was admitted to the ICU.[205]

141 It was in that context that his Honour considered that the question whether the respondent had sepsis fell more squarely within the expertise of respiratory physicians, infectious diseases specialists and intensivists. Intensivists, for example, are the practitioners to whom a patient with ARDS will ultimately be referred for treatment and who will, as Professor Harvey agreed, 'have the greatest expertise in managing ARDS'.[206] As his Honour, with respect, quite correctly observed, those practitioners will always be concerned to identify the particular cause of a patient's ARDS. It is not, as the appellant submitted, that other practitioners would be 'unconcerned about such matters'.[207] It is simply a matter of recognising which field of expertise

[204] Appellant's amended submissions [36] - [37], [47]; WAB 18, 19; Appeal ts 40.
[205] See for example the matters referred to by his Honour from 12 December 2005 and 13 December 2005 (at Primary reasons [684], [685]).
[206] Trial ts 458 (Harvey)
[207] Appellant's amended submissions [39]; WAB 18; Appeal ts 40.

assumes ultimate responsibility for the determining the aetiology of the particular condition.

142 His Honour has not been shown to be in error in his assessment of the expert witnesses in this regard.

143 Ground 3 is not made out.

**Ground 4 - Assessment of the evidence of Dr Numa**

144 Ground 4 alleges an error of law in the learned trial judge's finding that Dr Numa was the most impressive of the expert witnesses, and accordingly his Honour's preference for Dr Numa's opinions in making the sepsis finding.

145 The alleged error of law, as set out in the particulars to ground 4, is that the learned trial judge 'did not give any or any adequate reasons' for the finding.[208] The appellant submits that the learned trial judge's reasoning, at Primary reasons [675], was 'wholly' inadequate and 'does not constitute a reasoned basis'.[209]

146 We disagree.

147 On the contrary, in our view the learned trial judge's reasons for preferring the evidence of Dr Numa (and indeed his Honour's reasons more generally) more than adequately discharged his Honour's obligation to disclose the intellectual process that led to that decision.

148 In that regard, the impugned passage of his Honour's reasons must be read in the context of the Primary reasons as a whole. The learned trial judge did not simply declare that Dr Numa was 'the most impressive' expert witness. His Honour expressly incorporated his earlier observations in relation to Dr Numa's evidence (and his reports) in making that assessment.[210]

149 Those earlier observations included the passage reproduced at [76] above. They included his Honour's observations that:

(a) Dr Numa's reports clearly exposed his process of reasoning and the bases for reaching his conclusions;

[208] Particulars to ground 4.
[209] Appellant's amended submissions [56]; WAB 20.
[210] Primary reasons [675].

(b) Dr Numa's opinions were supported by reference to articles and publications, in relation to which there was no challenge to the fact that those articles supported the propositions for which they were cited;

(c) Dr Numa gave his evidence clearly and unambiguously, and demonstrated an intimate understanding of his areas of specialty; and

(d) Dr Numa had no difficulty explaining the bases for his opinion, even when subjected to challenge under cross-examination.

150 These matters, with respect, address the very kind of issues identified as critical to the assessment of expert evidence in decisions such as ***Makita (Aust) Pty Ltd v Sprowles***:[211] namely, clearly demonstrated reasoning, intelligibility, and the identification of the necessary scientific criteria against which to test the accuracy of the expert witness's conclusions.

151 It is apparent, from a reading of the learned trial judge's reasons as a whole, how, in his Honour's assessment, Dr Numa compared favourably to the other experts. In relation to some witnesses, that preference was necessarily more subtle than others. The learned trial judge, for example, also regarded Professor Starr as an impressive witness, although his Honour found that his report was not of the same quality as those prepared by Dr Numa.

152 In other respects, the comparison was more pronounced.

153 Take, for example, the extent to which the experts' opinions were supported by scientific research. As the learned trial judge observed, Dr Numa supported his opinions with research literature and, significantly, the fact that that literature supported the propositions for which they were cited was at no stage challenged.[212]

154 By contrast, Dr Prebble also cited research articles, but was cross-examined as to whether they supported the propositions for which he contended.[213] The learned trial judge found that they did not.[214]

[211] ***Makita (Aust) Pty Ltd v Sprowles*** (2001) 52 NSWLR 705 [59] et seq (Heydon JA).
[212] Primary reasons [199].
[213] Primary reasons [349].
[214] Primary reasons [671].

155 Professor Kimble, on the other hand, cited no scientific literature at all. The learned trial judge inferred that he was unable to point to any literature which supported his evidence.[215] While this did not cause the learned trial judge to discount his evidence, it nevertheless was a relevant factor in his Honour's ultimate assessment as to which evidence he regarded as most persuasive.

156 Similarly, Professor Harvey did not cite scientific literature in his reports, although he referred to reports in his oral evidence. For example, in examination-in-chief, Professor Harvey referred to a case study in which an 18-month-old child with burns to 15% TBSA suffered ARDS in the absence of infection.[216]

157 As the learned trial judge found, it was clear from that case report (which was identified subsequent to Professor Harvey's evidence) that the diagnosis of ARDS in that case was due to infection (albeit a viral infection).[217] The learned trial judge considered that Professor Harvey's reliance upon the case report 'impacted adversely on the credibility of his opinion' as to the cause of the respondent's ARDS. While it did not cause the learned trial judge to dismiss Professor Harvey's evidence completely, it did cause his Honour to give his opinion less weight.[218]

158 His Honour's assessment of Professor Harvey's evidence in this regard is the subject of ground 5. That ground remains to be considered on its merits.

159 In relation to the adequacy of the learned trial judge's reasons, however (the concern of ground 4), his Honour's reasons provide an ample explanation for why he preferred the evidence of Dr Numa to that of the other expert witnesses. No error has been demonstrated in that regard.

160 While ground 4, strictly, is concerned with the adequacy of the learned trial judge's reasons, certain of the appellant's submissions tended to go further and suggest that his Honour erred, in fact, by preferring the evidence of Dr Numa. It will be apparent from the brief summary above that there is no basis for such a contention.

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[215] Primary reasons [492].
[216] Trial ts 407, 449, 451.
[217] Primary reasons [595].
[218] Primary reasons [596].

161 As noted earlier, the advantages of a trial judge, to which an appellate court must give due regard, apply equally to the assessment of expert witnesses as they do to any other evidence. In the present case, the learned trial judge was required to make findings of fact based on his assessment of a number of expert witnesses all of whom he found were 'highly qualified and generally impressive'.[219] In assessing the respective opinions proffered by individual witnesses, and making the findings that he did the learned trial judge, would inevitably have to have had regard to the manner in which the experts gave their evidence, including any subtle differences between them. These matters were referred to by his Honour, with examples, in the reasons that his Honour gave. There is no basis to conclude that his Honour misused his advantage in this regard.

162 Ground 4 is not made out.

## Ground 5 - The case report relied upon by Professor Harvey

163 Ground 5 challenges the learned trial judge's conclusion, referred to at [157] above, that Professor Harvey's reliance upon the case report 'impacted adversely on the credibility of his opinion' in relation to the cause of the respondent's ARDS. That conclusion followed from the fact that Professor Harvey had 'incorrectly recalled the details of the case the subject of the case report and consequently erroneously relied upon it in arriving at his opinion'.[220]

164 The learned trial judge summarised the case report at Primary reasons [591] - [593]. Relevantly, his Honour observed that the case report stated that the patient (an 18-month-old child) had a viral infection, specifically Respiratory Syncytial Virus (RSV) and that the ultimate diagnosis of the patient's condition was 'ARDS *due to RSV + ve*' (emphasis added). Professor Harvey made no mention of this infection or its causative effect in his evidence.

165 The appellant submits that there was no error on the part of Professor Harvey and that the learned trial judge ought to have found that there was no basis to question any opinion of Professor Harvey.[221] The appellant makes that submission on the basis that the case report relied upon by Professor Harvey involved a *viral* infection and not a

[219] Primary reasons [596].
[220] Primary reasons [596].
[221] Appellant's amended submissions [66] - [67]; WAB 22.

*bacterial* infection and that, as the appellant submitted, 'antibiotic treatment has no effect on viral infection'.[222]

166 To properly understand the learned trial judge's conclusion in this regard, some context is necessary.

167 As is apparent from grounds 1 and 2, one of the issues at trial was the likelihood of the respondent developing ARDS as a result of the burns alone, given that she had no full thickness burns and burns to no more than 18% of her TBSA.[223] Dr Numa, who proffered the opinion that the chance of that occurring was 'vanishingly small', observed that there was no case report of 'ARDS secondary to burns of this nature and area … unless accompanied by sepsis'.[224]

168 It therefore became an issue in the trial whether there was any case report in the literature of a child with burns such as those suffered by the respondent, developing ARDS in the absence of sepsis.

169 Dr Prebble, for example, referred to four articles in his report of August 2017 in support of the proposition that there were several reports of ARDS in burns of less than 20% TBSA. [225] Dr Prebble, who was the first expert witness to give evidence, was cross-examined in relation to those articles.[226] The learned trial judge found that those articles did not reveal a case of a child with burns to less than 20% TBSA developing ARDS in the absence of infection.[227] There is no challenge to that finding.

170 Professor Harvey was the final expert witness to give evidence at trial. While he had not referred to published literature in his reports, in examination-in-chief he gave the following answer in relation to the proposition that ARDS may occur in minor burns of less than 20% TBSA:[228]

> Have you seen that in your patients?---Yes, I have. I have an anecdotal case. It's very difficult from the literature but there's certainly some evidence, the paper that we can refer to, that the conclusion may draw that ARDS is not related to body surface area burn. There's another

[222] Appellant's amended submissions [65]; WAB 22.
[223] See [30] above.
[224] Exhibit 19.6; GAB 629 - 630; Primary reasons [123].
[225] Exhibit 6; GAB 41; Primary reasons [306].
[226] Primary reasons [349].
[227] Primary reasons [670] - [671].
[228] Trial ts 407.

> paper from [our] institution[229] when they looked at all their children with ARDS they found that 30 per cent of those patients had burns less than 25 per cent. There's an anecdotal case that was reported at our annual scientific meeting last month, at one of the other major burns units in Australia, that was I believe an 18 month old child with a 15 per cent burn that suffered a very similar outcome, a major systemic inflammatory response, ARDS.

171 The first paper referred to by Professor Harvey (i.e. from 'our institution') was the first of the articles referred to by Dr Prebble: 'Crystalloids, colloids and kids: a review of paediatric burns in intensive care', Burns 24 (1998) 771-724.[230] Ground 5 is not concerned with that reference.

172 The second article referred to by Professor Harvey was the 'anecdotal case' involving an 18 month old child with a 15% burn. This is the relevant case report for the purposes of ground 5 and the judge's findings referred to in [164] above. The case report had not previously been referred to in the evidence of any witness (including Professor Kimble, who, as it later transpired, was the author of the report). Nor, it seems, had it been discovered.

173 The context in which the case report was referred to by Professor Harvey was clearly concerned with the issue of the likelihood of a child with burns of less than 20% of their TBSA developing ARDS in the absence of sepsis (and whether there was any such case reported in the literature).

174 This was made clear in cross-examination, in which Professor Harvey returned to the case report:[231]

> So apart from that, the Swedish article and this report from your hospital you weren't able to locate any other literature that suggested that you could get ARDS from less than 20 per cent burns?---Well, other than the fact that during the Australia New Zealand Burns Association annual general meeting in October there was another case report, as I mentioned, of a child with 15 per cent burn that suffered severe ARDS *in the absence of sepsis*.

---

[229] The trial transcript records this reference as 'AREA (?) institution'. In context, the reference was clearly to 'our institution' (that is, the Royal Alexandria Children's Hospital for children at Westmead, where both Professor Harvey and the authors of the article worked). See also Trial ts 449, 451, where Professor Harvey refers to the article coming from 'our institution' and 'my unit' respectively.

[230] See Primary reasons [349] and Trial ts 449, 451.

[231] Trial ts 452.

> Where was that, sorry, AGM, October, what, this year?---Annual Scientific Meeting, yes, in Adelaide this year. There was a case report presented at that meeting.
>
> Do you have a copy of the case report?---No.
>
> Did you provide it to the solicitors?---No.
>
> All right?---It informed my opinion. It helped to form my opinion, let's put it that way.
>
> Your opinion contained in the reports or your opinion somewhere else?---No, no. *The opinion that I hold that ARDS can occur in patients with burns less than 20 per cent total body surface area*. (emphasis added)

175 Following cross-examination in relation to the other articles referred to by Dr Prebble, the cross-examiner returned in detail to the subject matter of the case report relied on by Professor Harvey:[232]

> This AGM case study you said it was, what, 15 per cent?---Yes.
>
> How old was the child?---It was 18 months old.
>
> *Any direct inhalational injuries?---No inhalational injury, it was a scald burn.*
>
> *Any infection?---No*.
>
> And who presented this?---Who presented it? Professor Kimble from the Brisbane Burns Unit, Lady Cilento Hospital.
>
> This is the - the same Dr Kimble that's given evidence in this case?---Yes. (emphasis added)

176 In the preceding passage of evidence, the fourth question posed by senior counsel for the respondent was not expressly put on the basis that a distinction was being drawn between bacterial and viral infections. Nor did Professor Harvey seek to distinguish between the two when giving his answer to the question of whether there was 'any infection'. When asked if there was 'any infection', his answer was in the negative. He was not, on the face of this evidence, distinguishing between the *types* of infection (bacterial and viral) and their modes of treatment. The question of whether his evidence was impliedly intended to be limited to the presence of bacterial infection is one for which the learned trial judge would have had a better 'feel' than this Court. That qualification to Professor Harvey's evidence does not appear from the transcript.

[232] Trial ts 455.

177 It was open to the learned trial judge, who heard the evidence as it unfolded, to take the view that Professor Harvey was not intending to confine his answer to the presence of bacterial infection only, whilst leaving open, but unstated, the proposition that the child in that case had a respiratory viral infection. That conclusion is confirmed by, but not dependent upon, a consideration of the way in which the parties subsequently dealt with this issue at trial, referred to below.

178 As noted at [157] above, after the learned trial judge reserved his decision, the respondent identified the case report and sought leave to reopen her case to tender the case report. The parties consented to the tender of the case report, which was titled 'The need for ECMO in a paediatric burns patient',[233] and filed supplementary submissions in relation to it.

179 In its supplementary submissions to the learned trial judge, the appellant appears to have accepted that Professor Harvey's evidence in relation to the case report was incorrect, at least by omission. It submitted:[234]

> It is therefore not surprising that the viral infection identified on Day 8 of the patient's treatment, and post the commencement of ECMO, may not have been readily observed by Professor Harvey. The Defendant submits that no criticism of Professor Harvey should be made in respect of this oversight.

180 The conclusion that Professor Harvey incorrectly recalled the details of the case report was, objectively, correct. In any event, the conclusion was one plainly open to the learned trial judge having regard to the advantages he enjoyed as a trial judge. Professor Harvey's evidence was either directly inconsistent with the case report insofar as he said that the child in that case had no 'infection', or was at least incomplete insofar as he was suggesting that the child in that case, with burns to only 15% of their TBSA, had gone on to develop an acute diffuse and inflammatory lung injury (ARDS)[235] without mentioning that the injury was 'due to' the presence of a (viral) respiratory infection.

181 Moreover, the fact that the case report was ultimately revealed to involve a respiratory viral infection, rather than a bacterial infection, was not to the point. The question for the judge, ultimately, was what

[233] Exhibit 27; BAB 217.
[234] Defendants Supplementary Submissions dated 22 January 2018 [19]; BAB 227-231.
[235] Primary reasons [83].

weight ought to be given to Professor Harvey's evidence as to the cause of the respondent's ARDS, when assessing his evidence in light of other competing expert opinions and the evidence as a whole. As part of this broader assessment, it was open to the learned trial judge to have regard to Professor Harvey's apparent lack of command of the detail of the one anecdotal report which he effectively cited as evidence for the proposition that a child with burns to less than 20% of the TBSA could develop ARDS in the absence of sepsis (a serious complication of, and a systemic response to, infection).[236]

182 Ground 5 is not made out.

## Ground 6 - Enterobacter and cytokines

183 Ground 6 focusses on the seventh of his Honour's reasons for the sepsis finding, namely that the Enterobacter infection on the respondent's right knee was capable of causing, or contributing to, the respondent's ARDS in one of two ways: either by the bacteria making its way into the bloodstream or, as explained by Dr Numa, by the infection resulting in the release of cytokines into the bloodstream causing the respondent's SIRS which in turn caused the ARDS.[237]

184 The appellant, by ground 6, alleges that the learned trial judge erred in finding that the Enterobacter infection caused the release of cytokines into her bloodstream, which caused SIRS, which caused ARDS prior to the evening of 10 December 2005.

185 The appellant, in its submissions, called this the 'cytokine hypothesis' or the 'cytokine thesis'.[238] The 'cytokine hypothesis', the appellant submits, was solely based upon the evidence of Dr Numa.

186 Ground 6 alleges, essentially, that the learned trial judge erred by accepting the 'cytokine hypothesis' on the basis that it was not referred to in the expert reports exchanged prior to trial and not put to the appellant's expert witnesses (who were called prior to Dr Numa). The ground also alleges that, insofar as the 'cytokine hypothesis' was rejected by Professor Harvey (who was called after Dr Numa), the learned trial judge erred in not preferring the evidence of Professor Harvey.

187 Two preliminary points may be made in relation to ground 6.

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[236] Primary reasons [75].
[237] Primary reasons [677].
[238] Appellant's amended submissions [71]; Appeal ts 78, 83, 86.

188 First, as a matter of the interpretation of the Primary reasons, we do not accept, as ground 6 alleges, that the learned trial judge actually found that the Enterobacter infection caused the release of cytokines, ultimately leading to ARDS. That is, the relevant finding was that the infection could cause, or contribute to, the development of ARDS **either** by the bacteria itself moving through the bloodstream **or** by the release of cytokines into the bloodstream. It was a finding identifying the various mechanisms by which a localised infection can lead to a systemic response to the infection (sepsis).

189 It was not necessary for the learned trial judge, in the context of the issues in the trial, to determine the precise mechanism by which the Enterobacter infection gave rise to the systemic response (sepsis). Nor did his Honour do so. This is confirmed by the next passage in his Honour's reasons:[239]

> I note in this context that I have, in accepting Dr Numa's evidence that the Enterobacter infection *was capable of* causing the plaintiff's ARDS in the second of the ways mentioned, borne in mind that this aspect of his evidence was not directly put to Professor Starr and Professor Kimble during cross-examination. (emphasis added)

190 As this passage makes clear, his Honour found that the cytokine mechanism *was capable of* leading to the systemic response to the infection (i.e. sepsis), not that it did so lead. That systemic response was also capable of arising from the first of the mechanisms identified by his Honour, namely passage of bacteria through the bloodstream.

191 This particular passage also raises the second preliminary point in relation to ground 6, namely that the fact that the 'cytokine hypothesis' was not referred to in Dr Numa's reports and not put to the appellant's expert witnesses (the appellant's complaint on appeal) was expressly recognised and taken into account by the learned trial judge.

192 In this respect, the learned trial judge acknowledged that Dr Numa did not clearly disclose in his reports that infection can still cause the systemic response that the respondent had by reason of the effect of cytokines, rather than the infection being in the bloodstream.[240] This omission was, as his Honour expressly recognised, the one qualification to the otherwise entirely complimentary observations made in relation to Dr Numa's evidence.

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[239] Primary reasons [677].
[240] Primary reasons [200].

193 His Honour, therefore, clearly recognised and had regard to the fact that this aspect of Dr Numa's evidence was not specifically put to Professor Starr and Professor Kimble in cross-examination. The evidence was, nevertheless, not the subject of objection by the appellant at trial, nor was there an application to recall any of the appellant's witnesses to address the issue.[241]

194 The evidence was, therefore, before the learned trial judge and, subject to his Honour giving due regard to the fact that it had not been specifically addressed by a number of the appellant's expert witnesses, it was a matter for his Honour whether his Honour accepted the evidence. As with the rule in ***Browne v Dunne***[242] generally, the learned trial judge was not *bound* to reject Dr Numa's evidence on the basis that it had not been put to other witnesses.[243] His Honour *was* bound to have regard to the circumstances in which the evidence was given. And that is what he did.

195 This was not a case in which it was, in all of the circumstances, unfair or an error for the learned trial judge to accept Dr Numa's evidence that infection can cause the systemic response that the respondent had by reason of the effect of cytokines.

196 The reasons for this are as follows.

197 First, as we have said, the relevant evidence related to the mechanisms by which a localised infection can lead to a systemic response to the infection (sepsis). Acceptance of the 'cytokine hypothesis' was not an essential finding for the success of the respondent's case. In that regard, we reject the appellant's submission that it 'ended up being the central issue in the plaintiff's case'.[244]

198 Rather, in our view, the significance of the 'cytokine hypothesis' to the issues in the case was somewhat overstated by the appellant on appeal. We should explain why that is so.

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[241] Indeed trial counsel for the appellant referred, without objection, to the evidence in relation to cytokines in closing (Trial ts 481).

[242] ***Browne v Dunne*** (1894) 6 R 67. The analogy with the rule in ***Browne v Dunne*** in the present case is particularly apt by reason of the fact that (while a witness for the plaintiff) Dr Numa was called after all of the defendant's witnesses (other than Professor Harvey).

[243] ***Allied Pastoral Holdings Pty Ltd v Commissioner of Taxation*** [1983] 1 NSWLR 1, 26 (Hunt J).

[244] Appeal ts, 82.

199 It arises from an important distinction, found by his Honour and reflected in the evidence, between 'sepsis' and 'septicaemia'.

200 As noted at [43] above, 'septicaemia' refers to the presence of replicating bacteria or their products in the bloodstream. The learned trial judge used that definition.[245]

201 No one suggested, at trial, that the respondent ever suffered from septicaemia. That is, there was no suggestion at trial that the respondent had replicating bacteria in the bloodstream.

202 'Sepsis', on the other hand, is a systemic response to the presence of infection. This is precisely the sense in which the learned trial judge defined, and used, the term.[246] It is a clinical syndrome caused by infection.

203 As his Honour also recognised, the systemic response involved in sepsis occurs 'when chemicals released into the bloodstream to fight the infection trigger inflammatory responses throughout the body'.[247] Those chemicals that trigger the inflammatory response are cytokines. It may clearly be seen, then, that the effect of cytokines was not referred to by Dr Numa (or his Honour) as an independent cause of ARDS that was separate and distinct from sepsis (i.e. as an alternative to it). Rather, the effect was referred to as an explanation of the physiological processes involved in sepsis itself.

204 This was made clear in the evidence of the appellant's own experts. Professor Allen, when explaining the process involved in the development of a systemic response to burn, identified cytokines as part of the mechanism in the systemic response to burns and infection:[248]

> All right, now you've said a word there that I am not familiar with cytofines or something?---Cytokines. So the cytokine - so basically what happens is the body in a -in a burn situation produces an intense response to the burn itself, not dissimilar to that if there was infection. The body then has a - what's called an inflammatory cascade, which then most of the time the body's able to cope with but if the body's unable to cope with it you get - you get into trouble.
>
> And can you spell that word for the purpose of the transcript, please?---C-y-t-o-k-i-n-e-s. And there are those and the inflammatory - inflammatory mediators.

[245] Primary reasons [90].
[246] Primary reasons [86].
[247] Primary reasons [87].
[248] Trial ts 229.

205 Indeed, similar evidence appeared in Professor Allen's reports provided prior to trial (see [58] above). For example, in commenting upon Dr Numa's report of 26 December 2012, Professor Allen said:[249]

> The other authors have discussed an inflammatory cytokine cascade like response as a cause for [the respondent's] deterioration. There is no doubt that infection can cause the deterioration and one should then see my comments about Prof Kesson's report [which he had earlier described as 'well written and thought out' but in relation to which he referred to the risk of hindsight].

206 Essentially the same mechanisms were described in the evidence of Professor Starr, the infectious diseases expert called by the appellant. Professor Starr said:[250]

> All right. Can you explain, please, what you mean by ARDS?---So the acute ARDS, it's had various meanings for the acronym, but it's a respiratory distress syndrome that ensues from an insult which might be infection but is often a - for example in this case, burns, where there is a - an initial insult or trauma and - and what ensues is the release of a number of cytokines, or inflammatory mediators which then set off a cascade of events where there are a number of changes within the immune regulatory system. And that whole cascade leads to shifts of fluid across blood vessels. The fluid goes across the blood vessels into particular certain spaces and - and one in particular is the lung. There are often four shifts into tissues as well. In the lungs in particular the alveoli, the small areas of the airways of the lung, are then inflamed and more permeable and there is - are now filled with fluid and as a result of that there is difficulty with gas exchange and resultant raised respiratory rate and hypoxia, low oxygen.
>
> And ARDS isn't just caused by burns?---No, ARDS is a - a response to a number of - we see children who have generalised trauma who in road - road accidents children with infection, so over - have overwhelming sepsis and associated with that they may have ARDS. And a head injury can be associated with ARDS as well so there are a number of different triggers.
>
> All right. Now, would you accept that infection is a - is a major cause of ARDS?---Yes.
>
> …
>
> Yes?---And is a - and often - often unfettered but there is then a companion response that is a counter sort of response, CARS, which is a counter immune response where then there is immune suppression in

[249] Exhibit 13.5; GAB 474 - 475.
[250] Trial ts 191 - 192.

> response to that as well. So there's a - a cascade of events that occurs in the - again, that might be caused by sepsis burns or other - other events, other traumatic events and there is an overwhelming sort of inflammatory response.
>
> And once again that inflammatory response can be caused by infection?---Yes, it can.

207 As is apparent from this passage Professor Starr also identified an 'inflammatory cytokine cascade' as a systemic response to both burns and infection (amongst other things).

208 Both Professor Starr and Professor Allen gave evidence prior to Dr Numa. Accordingly, while the learned trial judge was (appropriately) cautious in relation to, and bore in mind, the fact that cytokines were not referred to in Dr Numa's reports, it is not the case that Dr Numa was the only, or indeed the first, witness to refer to cytokines as a mechanism by which sepsis occurs. The only additional feature arising from Dr Numa's evidence appears to be the specific reference to the fact that cytokines may cause those effects without bacteria being in the bloodstream.

209 Even then, the evidence of Professor Kesson, also given prior to Dr Numa, was clear in this regard:[251]

> So sepsis is a bacterial infection?---Correct.
>
> That is not within the bloodstream?---Correct.

210 When Dr Numa did come to give evidence, as in the case of the evidence of Professors Starr and Allen, his first reference to cytokines was in the course of describing the mechanism by which infection and sepsis affect the body generally, and in particular by reference to the heart rate. That first reference was in the following passage:[252]

> [C]an you explain how sepsis causes the faster heart rate, please?---There's a variety of mechanisms. One of the - one of the key pathologies in sepsis is a disruption of the integrity of the capillary endothelium, such that the capillaries become leaky. So fluid that's normally contained within the vascular space, within the circulation, tends to leak through the capillaries into interstitial tissues. This results in depletion of the intravascular volume in the same way that dehydration depletes the intravascular volume. So that the heart's got less - less volume to work with. So that's the - that's the first

[251] Trial ts 163.
[252] Trial ts 332.

> mechanism. Metabolic rate is increased in sepsis and so tissues require more oxygen. *Part of sepsis is - part of sepsis involves release of inflammatory chemicals broadly characterised as - broadly called cytokines, some of which depress cardiac function*. (emphasis added).

211 Indeed, the following passage from the cross-examination of Dr Numa confirmed that his evidence in this regard was, in substance, no different to that of Professors Starr and Allen:[253]

> And how then did it spread to affect her lungs?---Well, the - in the process of fighting infection, chemicals which we broadly refer to as cytokines are produced. The cytokines will circulate throughout the body and they will disrupt the function of organs distant to the site of infection. This was well described.
>
> These cytokines are the same ones that are released in systemic inflammatory response syndrome? Is that it?---The same ones indeed.

212 In our view, there is no doubt that Dr Numa's references to cytokines were references to a mechanism involved in sepsis.[254] They were not, and were not found by his Honour to be, an alternative independent cause of ARDS to that of sepsis. The appellant's submission that it appears that the learned trial judge 'construed Dr Numa's evidence as being that release of cytokines into the bloodstream does not cause sepsis',[255] is simply a misconstruction of the evidence and his Honour's reasons.

213 For these reasons, reference to cytokines by Dr Numa (in a manner not dissimilar to references by the appellant's own witnesses) did not change or alter the nature of the respondent's case at trial or lead to a position where it would be unfair or in error for the learned trial judge to accept that evidence.

214 It is appropriate to ask, in this context: why then was the 'cytokine hypothesis' relevant to the respondent's case at all?

215 The answer to that is that the cytokine mechanism was one of the explanations for the absence of bacteria in the blood culture taken from the respondent on 11 December 2005.[256] This can be found in the eleventh reason given by the learned trial judge for the

[253] Trial ts 357.
[254] See also the answers to questions asked by the learned trial judge at Trial ts 352.
[255] Appellant's amended submissions [70]; WAB 23.
[256] Primary reasons [42].

sepsis finding:[257]

> Eleventh, although the single blood culture produced from the sample of blood taken from the plaintiff at 11.15 pm on 11 December 2005 returned a negative result for bacteria, the evidence adduced reveals, in my view, credible explanations for the result. One such explanation is that the bacteria was in the bloodstream on an intermittent basis only and that the blood sample was taken during one of the intervals when the bacteria was not circulating in the bloodstream. A second explanation is that although the bacteria was in the bloodstream at the time the sample was taken, given the small amount of the sample it simply did not 'capture' an amount of blood that had the bacteria in it. The third explanation is that sepsis does not necessarily involve bacteria being in the bloodstream. As was stated by Dr Numa, the infection can be localised and produce cytokines which spread, cause a severe inflammatory response, and ARDS.

216 What Dr Numa said in relation to the negative blood culture, included the following:[258]

> [A]n infection anywhere in the body can cause all the catastrophic complications that we see in [the respondent]. We don't necessarily have to identify the bacteria in the blood. We don't have to have the bacteria in the blood. We - we have to have the inflammatory mediators, the cytokines, which are what make you unwell. They have to be in the bloodstream. But we don't actually test for those ... And so an infection - so for example, you know, we may see patients with meningitis who have a severe infection in the brain, and we never recover the bacteria from the blood, and yet they have multi organ failure, they have the ARDS, and the renal failure, and the liver impairment, and - and marrow suppression, and so on and so forth, all because there's a severe infection going on in the brain which is setting up a marked inflammatory response, releasing chemical mediators into the bloodstream, which are circulating in the body, and causing all sorts of pathology.

217 The eleventh reason given by the learned trial judge for the sepsis finding (at [215] above) is not the subject of the appeal by the appellant.

218 Again, as can be seen from the passage at [215] above, his Honour identified multiple explanations for the negative culture result, one of which referred to the action of cytokines. As with the earlier passage referred to at [188]-[189] above, his Honour did not, nor did he need to, settle upon which explanation (or explanations) were present or

[257] Primary reasons [687].
[258] Trial ts 351.

operative in the respondent's case. It was sufficient for his Honour to find that there *were* explanations for what might otherwise have been evidence tending against the sepsis finding.

219 That was the significance, and the only significance, of the 'cytokine hypothesis' for the issues before the learned trial judge. It was open to his Honour to accept Dr Numa's evidence for the purposes for which it was relied upon.

220 Finally in relation to this ground of appeal, it is necessary to deal with the appellant's submission that the learned trial judge ought to have rejected the 'cytokine hypothesis' because it had been rejected by Professor Harvey.

221 Professor Harvey's evidence in this regard was in response to the following question from the learned trial judge:[259]

> [C]an I ask you to just consider this scenario, professor. So you've got the situation where you have the [Enterobacter] in the right knee wound but that infection remains localised, it remains in the wound; so the bacteria itself doesn't get into the bloodstream but what does happen is that the infection results in inflammatory markers, the cytokines, being released into the bloodstream and those cytokines, while they're circulating in the blood then cause all sorts of pathologies and cause the parts of the body - affect parts of the body and result in a multi-organ failure. What do you say to that scenario?---I think almost always a systemic - it's a little bit difficult, I'm not a bacteriologist, but my understanding of the pathophysiology is that in order for a systemic inflammatory response, in other words, this is the cytokine response of the body to an invasive sepsis but it has to be invasive sepsis. In other words, the bacteria has to invade the bloodstream. A localised infection - we quite frequently have contaminated wounds, in other words, bacteria grown from the wounds. Unless there's evidence locally of invasion, in other words, there's signs of swelling, redness, pain and inflammation, we do not regard that as being an entity that requires treatment. When, and if there are signs of local redness, inflammation, swelling, then that we generally treat but that has to be obvious and then if a patient becomes unwell with a systemic inflammatory response, my understanding is that that only results - I mean again it's difficult to - usually results from invasion of the bloodstream and dissemination of the organs - of the bacteria to the rest of the body. So I think it, honestly, is unlikely to have a major systemic reaction, a systemic inflammatory response to a localised colonisation of a wound without obvious signs of infection in that area, I would find it - - -

[259] Trial ts 417 - 418.

> Without the bacteria itself actually spreading into the bloodstream?---Yes. Yes. That's the criteria that we use. I mean it - again, I mean, because of the intermittent nature of the spread of the bacteria through the bloodstream it is a problem but we didn't find this in this case. It's not say it wasn't there but I think it's unlikely to have been there in the presence of another explanation of the symptoms. So there's no positive finding of invasive sepsis and its invasive sepsis that I would suggest is responsible for the systemic inflammatory response; not a localised colonisation of the wound.

222 It is correct, as the appellant submits, that Professor Harvey did express the understanding that 'invasive sepsis' (as he referred to it) 'usually results from invasion of the bloodstream … of the bacteria to the rest of the body'.[260] He did not, however, directly address the effect of cytokines from localised infections causing sepsis (beyond the reference at the beginning of the answer to the cytokine response being from invasive sepsis).

223 Professor Harvey's evidence in relation to sepsis generally, however, must be understood in the context of the fact that, as the learned trial judge recognised, Professor Harvey did not draw a distinction between sepsis and septicaemia.[261] He used the expressions interchangeably.[262]

224 When first asked to describe how bacteria could cause sepsis, for example, Professor Harvey referred to septicaemia, saying 'it's a direct invasion of bacteria into the bloodstream from an infected wound'.[263]

225 Of course, as set out at [200] above, 'septicaemia' **does** necessarily involve bacteria in the bloodstream: that particular condition involves the presence of replicating bacteria or their products in the bloodstream. However, as also noted above, it was not alleged at trial that the respondent had septicaemia. Rather, the allegation at trial, and in relation to which the other witnesses gave evidence, was whether the respondent had the systemic response to infection (sepsis).

226 And in that regard, as the evidence of Professors Allen and Starr also confirmed, the mechanism which sets off the cascade of adverse effects (from both infection and other trauma) is the release of cytokines (the inflammatory markers).

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[260] Trial ts 418.
[261] Primary reasons [91], [552].
[262] Trial ts 427 ('So when you're speaking of sepsis, you're speaking of septicaemia?---Yes.')
[263] Trial ts 417.

227 In light of this evidence, and Professor Harvey not having distinguished between sepsis and septicaemia, it was clearly open to the learned trial judge to accept the evidence of Dr Numa in relation to the effect of cytokines, including the additional feature that cytokines may cause those effects without bacteria being in the bloodstream. Professor Harvey's evidence did not compel a different conclusion.

228 No error has been demonstrated in the learned trial judge's fact finding in this regard.

229 Ground 6 is not made out.

## Ground 7 - The clinical signs of sepsis

230 Ground 7 challenges the findings by the learned trial judge that together constituted the eight reason his Honour gave for the sepsis finding: namely, that there were several clinical features of the respondent's case which, viewed collectively, were not only consistent with, but also suggestive of, the respondent having a bacterial infection and sepsis rather than simply a severe inflammatory response to sterile burns.[264]

231 In that regard, in summary his Honour referred to:

(a) the respondent's abnormally high temperature, heart rate and respiratory rate, particularly her temperature which, in Dr Numa's opinion was more likely the result of infection than burns alone;[265]

(b) the respondent's C-reactive protein levels which again, in Dr Numa's opinion, was far more likely the result of infection than inflammation alone;[266] and

(c) the respondent's sudden and dramatic drop in neutrophils (neutropenia). [267]

232 His Honour's overall conclusion in this regard was:[268]

> In short, although the symptoms experienced by the plaintiff up until the late evening of 11 December 2005 are symptoms seen in patients suffering from SIRS in the absence of infection, the severity and

[264] Primary reasons [678] - [683].
[265] Primary reasons [679].
[266] Primary reasons [680].
[267] Primary reasons [681].
[268] Primary reasons [682].

> breadth of the symptoms, and the catastrophic and rapid nature of the plaintiff's decline were, when one has regard to the evidence of Dr Numa and Professor Kesson, more suggestive of sepsis than SIRS in the absence of infection. To the extent that the evidence of the other expert witnesses suggested that the symptoms, to which I have referred, were no more suggestive of sepsis than SIRS in the absence of infection, I prefer, as is in any event apparent from what I have said, the evidence of Dr Numa and Professor Kesson.

233 It will be apparent that these particular findings cannot be divorced from the learned trial judge's preference for the evidence of Dr Numa and Professor Kesson in relation to this question, including his overall preference for Dr Numa addressed in relation to ground 3. As we have concluded, his Honour was entitled to reach such a preference and gave adequate reasons for doing so.

234 For this reason, ground 7 can be largely answered by reference to whether the findings reached by his Honour as to the specific matters were supported by the evidence of the witnesses that he preferred (i.e. that of Dr Numa and Professor Kesson). Put another way, while the appellant submits, in support of ground 7, that the learned trial judge's conclusions were contrary to 'the weight of the evidence',[269] the 'weight of the evidence' cannot be divorced from the weight to which the learned trial judge gave to particular parts of it.

235 In this regard, the appellant's submissions in relation to ground 7, were, with respect, not attentive to the advantages of the learned trial judge and were more akin to submissions that might be made in closing at trial.

236 We have, nevertheless, considered the evidence in relation to the three matters set out at [231] above.

### *Elevated temperature*

237 In relation to the respondent's temperature, Dr Numa gave evidence (which his Honour accepted) that a fever of 39.4º C was very high. Normally 38.5º C was used as the cut-off for significant fever. In that regard, Dr Numa noted that the *American Burns Association Consensus Conference to Define Sepsis and Infection in Burns*[270] (the *Consensus statement*) had chosen 39º as 'as a slightly more stringent benchmark'. [271]

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[269] Appellant's amended submissions [98]; WAB 27.
[270] Exhibit 19.4; GAB 612 - 626.
[271] Trial ts 156.

238 Professor Kesson also described the respondent's fever of 39.4º C as 'markedly elevated'.[272] While Professor Kesson did, as the appellant submitted, say that the respondent could have been febrile because of her burns,[273] she ultimately regarded the respondent's high temperature and tachycardia as clinical indicators of infection.[274]

239 Recognising, of course, that it was only one of the clinical factors to be considered in combination with the other signs, the learned trial judge was entitled to accept Dr Numa's evidence that a temperature of 39.4º C was more suggestive of infection. In this regard, Dr Numa's evidence was supported by the research cited by him. The *Consensus statement*, for example, stated:[275]

> The burn sepsis definition must … distinguish changes in patient status as a result of infection due to a microbial entity from the alterations secondary to the burn injury itself or associated events (such as inhalation injury).
>
> … As a manifestation of the hypermetabolic state, patients with large burns "reset" their baseline temperature to around 38.5º C. Therefore, fevers are not considered a sign of sepsis until they reach 39º C.

### *C-reactive protein*

240 Similarly, in relation to the C-reactive protein, all of the expert witnesses agreed that an elevated C-reactive protein may be present in both sterile burns and infection. The significance, for the learned trial judge's finding was the *extent* of the elevation.

241 The particular evidence relied upon by the learned trial judge was the following evidence from Dr Numa:

> [W]e now have serological markers such as C-reactive protein, and procalcitonin, which again, you know, none of these tests are 100 per cent definitive, but the C-reactive protein is a good marker of serious infection. Normal range less than 10; if we see a CRP of more than 30, we are certainly concerned, and a CRP of 100 is significantly elevated.
>
> And you say there that:
>
> > The CRP is a nonspecific marker of infection/inflation - inflammation.

[272] Trial ts 156.
[273] Trial ts 161.
[274] Trial ts 164.
[275] Exhibit 19.4; GAB 612 - 626.

> …
>
> So it can be, what, either one or the other, or a combination, is that what - - -?---Correct, and - and there's some - there's some sort of quantitative analysis of that. So you know, a CRP of 100, you know, starts to look more like infection than inflammation to me. A CRP of - I think it went up to 200 subsequently, 242, is very high indeed, and to my mind is far more suggestive of severe infection than anything else.
>
> We heard yesterday again from Dr Kimble that with burns you'll always get some level of inflammation?---Correct.
>
> Okay. How does that fit in with this then?---Yeah, as I - look, burns patients will have elevated CRP. If you look at the literature, the - the typical burns patient will have a CRP up to around about 100, 120. Now, of course there's a - there's a range, that's a - that's a - that's a - a typical or a mean value. Some will be lower, some will be higher. But - but CRP in - in burns is - is usually around the 100 value, rather than higher.

242 Again, the learned trial judge was entitled to accept this evidence. It was supported by the study Dr Numa identified in his report of 26 December 2012,[276] reliance upon which was not challenged in cross-examination.

### *Neutropenia*

243 Thirdly, his Honour referred to the respondent's sudden and dramatic drop in neutrophils (neutropenia).

244 In this regard, the evidence of both Dr Numa and Professor Kesson, which his Honour preferred, provided strong support for his Honour's finding.

245 Professor Kesson, for example, gave the following evidence in examination in chief:[277]

> All right. And then on 10 December the white cell count has gone down to 4.9, so it's below the - the normal range, and the neutrophils have gone down from 6 to 0.7. Can you explain, first of all, the significance of the drop in the white cell count to 4.9 from 10.7 24 hours earlier?---Well, the drop in the white cell count is largely due to the drop in the neutrophils because the lymphocytes are much the same and the monocytes have dropped a little bit. And the significance of the drop of neutrophils would suggest that this child is being overwhelmed by infection to the extent that she is running out of white cells to fight the

[276] Exhibit 19.6; GAB 627 - 630.
[277] Trial ts 160.

> infection. It is actually a very, very significant finding to go from neutrophils of whatever they were before - - -
>
> Six, I think?--- - - - 6 to 0.7 is a very profound and significant drop and should immediately raise alarms. This child is at significant risk of overwhelming infection at this point.

246 Then, referring to blood samples taken at 11.15 pm on 11 December 2017, Professor Kesson said:[278]

> And the blood sample taken at 2315 hours by which time she was in the paediatric intensive care unit had dropped for the white blood cell count of 1.3 with the neutrophil count down to 0.1 and the comment in the report is marked neutropenia. Can you tell us about the significance of the drop down to that level, please?---Well, the process has been continuing with the drop in neutrophil count so her bone marrow is exhausted and she's unable to generate enough neutrophils any more to counter the infectious process. She is in grave danger. This is a very, very alarming and concerning situation. This is life-threatening.
>
> So there was an evolving process reflected in the deterioration in the neutrophil count during her admission to the hospital over the first 48 hours?---That is correct.

247 Following cross-examination, in response to questions from the learned trial judge, Professor Kesson's evidence in this regard was equally robust:[279]

> [W]hy do you say that [the respondent] had sepsis as opposed to systemic inflammatory response syndrome in the absence of infection?---Because of the neutropenia and the left shift and toxic changes on her blood film. They're in - the left shift and toxic changes are commonly seen in patients who have bacterial infections. Neutropenia is not, and neutropenia in someone who is infected is a sign of impending doom. It's a very, very serious event. …
>
> It's the neutropenia, which I think in fact is very unusual.

248 Dr Numa's evidence was to the same effect. In relation to the neutrophil count (of 0.1) taken at 11.15 pm on 11 December 2017 he said:[280]

> It had dropped to 0.1?---Yes.
>
> So you - - -?---Appallingly low. It just - just - and again, rarely seen. You know, this is - this is - and regardless of how you get there, if you

[278] Trial ts 161.
[279] Trial ts 181 - 182.
[280] Trial ts 336.

> have .1 neutrophils, your ability to fight infection is severely compromised, and this - this is a medical emergency, because we've - we just - as I say, you know, even if - if you've got .1 neutrophils because you've had chemotherapy because you've got leukaemia, or some other childhood cancer and your neutrophil count drops to .1 people are very concerned about that. It's a - it's a very dangerous place to be.
>
> And in terms of differentiating between sepsis being the cause or the trigger or inflammation or a combination of the two?---I've never seen neutropenia of that severity purely due to inflammation. The only - the only time I've seen neutropenia in this - of that severity is related to sepsis or to some other pre-existing condition, such as a child who's having chemotherapy and so on. But putting those to one side, neutropenia is sepsis rather than SIRS.

249 It will be readily apparent that there was ample evidence upon which the learned trial judge could be satisfied that:

(a) the respondent's neutrophil count had dropped to as low as it could possibly be; and

(b) a neutrophil count of this figure was inconsistent with sterile inflammation in the absence of infection.

250 Indeed, in its submissions, the appellant did not seek to challenge these propositions. It submitted:[281]

> Even if the second and third propositions [i.e. those identified at [249] above] are accepted, it means little. As with his Honour's reasoning at Judgement [680], it is not explained how a neutrophil count at 11.15pm on 11 December 2005 says much about something on the evening of 10 December 2005.

251 Apart from observing the lack of any challenge to the two propositions set out in [249] above, two points be made in relation to this submission.

252 First, both Professor Kesson and Dr Numa readily explained the relevance of the 11 December 2005 blood count to the diagnosis of the respondent's condition. Both experts confirmed that the respondent's condition was an 'evolving' one in which the various clinical features worsened over time.[282] In those circumstances the *pattern* of the changes to the respondent's white blood cell count was relevant.

[281] Appellant's amended submissions [97]; WAB 27.
[282] Trial ts 160 - 161, 180 (Professor Kesson), 334 - 335 (Dr Numa).

253 Secondly, as stated at [139] to [140] above, this portion of the learned trial judge's reasons was concerned with a pure question of historical fact: i.e. whether the respondent *in fact* had sepsis. Unlike the question of the standard of care, that factual question was to be answered, in retrospect, in light of all of the evidence.

254 No error has been demonstrated in the manner in which the learned trial judge dealt with these issues.

255 Ground 7 is not made out.

**Ground 9 - The relevance of the post antibiotic neutrophil count**

256 Ground 9 alleges that the learned trial judge erred in finding (at Primary reasons [685]) that changes to the respondent's white blood cell count and neutrophil count on 12 and 13 December 2005 was indicative of her having sepsis on the evening of 10 December 2005. The ground goes on to allege that his Honour ought to have found that changes to the respondent's white blood cell count and neutrophil count on 12 and 13 December 2005 were irrelevant.

257 In its written submissions, save for submitting that Dr Numa was not an infectious diseases specialist, the submissions in support of ground 9 were identical to those in relation to ground 7.[283]

258 The appellant's case initially suggested that the learned trial judge was in error in having regard to the respondent's improvement when ultimately put on antibiotics on the basis that it was irrelevant.

259 That suggestion must be rejected. Dr Numa's evidence clearly supported the relevance of the respondent's improvement:[284]

> And similarly with the neutrophils, which was 0.7 on the 10th gone down to 0.1 antibiotics were administered and the next time they tested it's 0.5?---Yes.
>
> Any significance to that?---Yeah, I think - I think so. As we - as we discussed, the - the neutrophils are depleted by - by having to fight the bacteria, so they're - they're - they're little, you know, kamikaze pilots the neutrophils, they - they kill the bacteria but - but die themselves in the - in the process. And when we see that neutrophil count falling what it means is that the neutrophils are being used up faster than they can be replenished by the bone marrow. Antibiotics also kill bacteria,

[283] The submissions at Appellant's amended submission [107]; WAB 28 (in relation to ground 9) are identical to the submissions at Appellant's amended submission [96]; WAB 27 (in relation to ground 7).
[284] Trial ts 354.

> so in the presence of antibiotics there's less work for the neutrophils to do because some of the bacteria will be - will be (indistinct) by the antibiotics. And - and so now the balance shifts, so instead of all the neutrophils being used up in killing the bacteria we find that the antibiotics are now killing a significant number of the bacteria and so the - the - the use rate of the neutrophils drops, the bone marrow's still churning neutrophils out and so the numbers start to replenish again. So we're - we're seeing some recovery as - as a result of - of therapy.

260 Ultimately, the appellant proffered no submissions in support of the proposition that this evidence was irrelevant.

261 Rather, in oral submissions on the appeal,[285] the appellant submitted that ground 9 was in fact directed to the learned trial judge's reasons at Primary reasons [686] in which his Honour rejected Professor Harvey's evidence that the increase in the respondent's white blood cell count could have been due to her receiving a blood transfusion.

262 Professor Harvey's evidence was to the following effect:[286]

> What do you say to the - to this proposition, that the increase in the level of neutrophils and the white cells is consistent with the antibiotics that had been administered killing off the bacteria in the bloodstream with the result that this was enabling the neutrophil level, the production of the neutrophil level to increase and the neutrophil levels in the bloodstream begin to increase?---I think that's one of the possibilities that the blood test I believe that we're referring to, your Honour, were all taken following a blood transfusion, so both the haemoglobin and the white cell count would respond to a transfusion of blood. I think also that the effect of appropriate management of ARDS with ventilation circulatory support as well as the antibiotics all contributed. So I agree, I can't discount the fact that antibiotics have had an effect in improving the white cell count.
>
> But would they only have that effect if there was bacteria in the bloodstream?---Well, I think it's - in - in isolation of any other treatment, I think it would be reasonable to say that, you know, the antibiotics have had an effect in improving the white cell count. But in the setting where more appropriate treatment was given for the ARDS and a blood transfusion was given, it's difficult to be sure of the effect of the antibiotics. But I certainly would not discount the fact that they have had an effect in improving the white cell count.

[285] Appeal ts 117 - 121.
[286] Trial ts 459 - 460.

263 It may at once be observed from this passage, that Professor Harvey did not discount the explanation given by Dr Numa for the improvement in the respondent's neutrophil count. He simply identified that a blood transfusion may be an alternative explanation, such that it was 'difficult to be sure'.

264 It was in this context that his Honour observed that the transfusion was of 'Packed RBC', that is, packed red blood cells and, for this reason, he did not accept this aspect of Professor Harvey's evidence.

265 At trial, the appellant accepted that the reference to 'Packed RBC' in the Hospital records was a reference to packed red blood cells. It is also the case that, when Professor Harvey raised the suggestion that the transfusion might account for the respondent's improved neutrophil count, he was not taken to the reference to 'Packed RBC' in the Hospital records.[287]

266 The appellant, nevertheless, now submits that there was no evidence that 'necessarily excluded the existence of white blood cells' in the 'packed red blood cells' and that his Honour erred by rejecting this aspect of Professor Harvey's evidence.[288]

267 This challenge to the learned trial judge's findings (which was raised for the first time on the second day of the appeal) must be rejected.

268 While it is correct that there was no direct evidence, in as many words, that 'packed red blood cells' do not contain white blood cells, it was open to the learned trial judge to infer that that was the case. On the face of it, the reference to 'packed red blood cells' is a reference to a blood product that has been produced from whole blood and that contains 'red' blood cells. There was certainly no evidence to the contrary.

269 In this regard Professor Harvey, as we have noted, did not refer to the reference to 'Packed RBC' in the Hospital records. On the contrary he referred to a different entry that referred to 'packed cells in progress'.[289] Indeed, he gave the following evidence in relation to that entry: [290]

> You assume from that, that that included white blood cells?---Yes.

[287] Appeal ts 120 - 121.
[288] Appeal ts 119.
[289] Trial ts 461.
[290] Trial ts 461.

> It also had been simply red blood cells?---No.
>
> What are you saying?---Because packed blood cells are - is blood without plasma, the fluid component.
>
> Yes?---That's what it means.

270 Professor Harvey's evidence in this respect was, therefore, based upon his assumption as to the type of transfusion referred to in the reference to 'packed cells in progress'. He did not suggest that all transfusions (including those specifically of 'packed red blood cells') contained white blood cells. Nor was he taken to the entry referring to 'Packed RBC'.

271 No doubt the inference drawn by the learned trial judge would have been more easily reached had Professor Harvey been taken to that entry. Nevertheless, given the evidence that was available, that inference was open.

272 In any event, it must be remembered that this particular finding by the learned trial judge was very much a second or third order finding, in the sense that his Honour was providing an explanation for why he did not accept Professor Harvey's explanation for why Dr Numa's reliance upon the respondent's improved neutrophil count might be misplaced.

273 In that regard, it was sufficient, to support the learned trial judge's acceptance of Dr Numa's evidence in that regard, that there was good reason for his Honour not to accept Professor Harvey's evidence. The fact that Professor Harvey's explanation was based on an assumption from one entry in the Hospital records, without reference to another (more specific) reference in those records, provided a sound basis for rejecting that evidence.

274 Ground 9 has not been made out.

## Ground 10 - The timing of bacterial infection

275 Ground 10 alleges that the learned trial judge erred in rejecting the evidence of Professor Starr that bacterial infection of the bloodstream caused by infection on the surface of the skin does not usually occur within the first 24 to 48 hours of infection on the surface of the skin.

276 That finding was not, strictly, one of the thirteen reasons identified by the learned trial judge for the sepsis finding. It was one of the 'additional points' noted by his Honour in addressing the evidence in

relation to the issue.[291]

277 The particular finding challenged by the appellant is as follows:[292]

> The evidence of Professor Starr was that although germs that live on the skin can permeate the skin and cause infection, this takes some time and does not usually occur within the first 24 to 48 hours. This aspect of Professor Starr's evidence was something that the defendant's counsel placed particular reliance on during his closing address. However, Professor Starr's evidence on this point was contradicted by Dr Numa. Dr Numa's evidence was that sepsis can evolve very rapidly, that it is possible to go from being completely well to being critically unwell (even dead) within 12 to 24 hours with certain bacteria, and that gram negative bacteria (such as Enterobacter) are particularly notorious for causing a rapid decline. Given my favourable assessment of Dr Numa in comparison to all of the other expert witnesses, including Professor Starr, I prefer the evidence given by Dr Numa on this point to the evidence given by Professor Starr.

278 It will immediately be apparent that the learned trial judge's finding in this regard was based upon his favourable assessment of Dr Numa. We have already concluded, in relation to ground 4, that the learned trial judge was entitled to accept the evidence of Dr Numa and that there is no basis to conclude that his Honour misused his advantage in this regard.

279 In those circumstances, the fact that the learned trial judge preferred the evidence of Dr Numa to that of Professor Starr in relation to this particular issue reveals no error.

280 In any event, having regard to the reasons given by the learned trial judge for the sepsis finding, the evidence of Professor Starr that the appellant submits ought to have been accepted was only of relatively minor significance. That evidence was as follows:[293]

> Germs that live on all of our skin will live on the skin of - of that child as well, and they may then permeate the skin and cause infection. But that takes some time. It doesn't happen within the first - usually, within the first 24 to 48 hours. And so that children presenting like this, in the first day or so, one doesn't normally think that infection is the likely cause.

[291] See [80] above.
[292] Primary reasons [695].
[293] Trial ts 186.

281 As is apparent from the evidence itself, Professor Starr was expressing a view, firstly, as to what 'usually' happened, and secondly, as to what one would 'normally think' in the first day or so following a burn.[294]

282 Clearly, that evidence was describing the 'usual' course of infection and did not suggest that the course of infection could not occur with the rapidity described by Dr Numa.

283 This was, indeed, confirmed in Professor Starr's evidence in cross-examination:[295]

> Are you there saying that, in fact, she did have signs consistent with possible infection in those investigations?---Yes. I - as I have said, the symptoms, signs and investigation results can overlap and they do overlap and it makes it very difficult. But as - as I've said, in the first 48 hours many of those symptoms, signs and investigation results are more likely secondary just to an inflammatory response than to sepsis.
>
> And is the case that infection can progress to severe - I beg your pardon - infection can progress to severe life-threatening sepsis fairly quickly?---Yes. Depending on the germ and the setup, yes, it can.

284 Even accepting Professor Starr's evidence in this regard, it was not necessarily inconsistent with the finding reached by the learned trial judge; namely that, in the respondent's particular case, the symptoms and signs were due to infection.

285 Ground 10 is not made out.

## Ground 11 - The sepsis finding

286 Ground 11 challenges the ultimate finding of the learned trial judge that by the evening of 10 December 2005 or by 3.00 am on 11 December 2005 the respondent had sepsis.[296]

287 The ground concerns the consequences of what the appellant alleges were the errors of the learned trial judge leading to that ultimate finding (namely the errors alleged in in grounds 1 to 10).[297] Ground 11

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[294] As an aside, we note, contrary to the manner in which this ground of appeal and the submissions in support of it were formulated, Professor Starr does not expressly refer to bacterial infection 'of the bloodstream' (see Appellant's amended submissions [108], [112]; WAB 29). In this respect the appellant's submissions fail to clearly recognise the distinction between 'sepsis' and 'septicaemia' discussed at [199] to [202] above.

[295] Trial ts 204.

[296] Primary reasons [692].

[297] Appeal ts 126 - 127.

was necessary because, depending upon which of the 'strands' leading to the ultimate finding were overturned, this Court might need to consider whether the remaining strands supported the ultimate finding or, alternatively, whether a retrial was necessary.

288 Ground 11 is, nevertheless, dependent upon the success of some or all of those preceding grounds.

289 As we have not upheld any of the grounds 1 to 10, ground 11 must necessarily fail.

290 For these reasons, the appellant's challenge to the sepsis finding must fail.

291 We turn, then, to the grounds of appeal concerned with the finding of breach of duty of care.

## Grounds 12 to 16 - breach of duty and s 5PB of the *Civil Liability Act*

292 As noted at the beginning of these reasons, grounds 12 to 16 concern the learned trial judge's finding that the appellant was in breach of its duty of care to the respondent. The relevant breach was, in summary, the failure of the doctors treating the respondent to recognise, by around 2.00 am on 11 December 2005, the possibility that the respondent had sepsis and by failing to administer antibiotics as quickly as was reasonably possible.[298]

293 Much of the appellant's focus in relation to these grounds was concerned with s 5PB of the *Civil Liability Act*. Before turning to consider grounds 12 to 16 individually, it is appropriate to set out our views as to the proper construction of s 5PB.

### Section 5PB - legislative history

294 Section 5PB appears in Part 1A of the *Civil Liability Act*, which, subject to a number of presently irrelevant exceptions, applies to 'any claim for damages for harm caused by the fault of a person'. In a number of respects, Part 1A makes provision in relation to liability in the tort of negligence, including in relation to breach of duty of care.

[298] Primary reasons [804].

295 In that regard, s 5B, which sets out the preconditions for establishing a breach of duty, rather than the existence of a duty,[299] provides:

> **5B. General principles**
>
> (1) A person is not liable for harm caused by that person's fault in failing to take precautions against a risk of harm unless -
>
> (a) the risk was foreseeable (that is, it is a risk of which the person knew or ought to have known); and
>
> (b) the risk was not insignificant; and
>
> (c) in the circumstances, a reasonable person in the person's position would have taken those precautions.
>
> (2) In determining whether a reasonable person would have taken precautions against a risk of harm, the court is to consider the following (amongst other relevant things) -
>
> (a) the probability that the harm would occur if care were not taken;
>
> (b) the likely seriousness of the harm;
>
> (c) the burden of taking precautions to avoid the risk of harm;
>
> (d) the social utility of the activity that creates the risk of harm.

296 It will be apparent that many of the issues relevant to determining the response of a reasonable person to a particular risk under s 5B are similar to (although not identical to)[300] the factors identified by Mason J in ***Wyong Shire Council v Shirt***[301] as relevant to a consideration of the response of a reasonable person according to the common law.

297 Prima facie, s 5B applies to all cases for breach of duty to which Part 1A of the *Civil Liability Act* applies, including claims against health professionals.

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[299] ***CGU Insurance Limited v Coote*** [2018] WASCA 117 [77]; ***Mamo v Surace*** [2014] NSWCA 58; (2014) 86 NSWLR 275 [48]; ***Department of Housing and Works v Smith (No 2)*** [2010] WASCA 25; (2010) 41 WAR 217 [70] - [83]; ***Adeels Palace Pty Ltd v Moubarak*** [2009] HCA 48 (2009) 239 CLR 420 [27].

[300] See e.g. the difference between a 'foreseeable' risk and a 'not insignificant' risk: ***Lightfoot v Rockingham Wild Encounters Pty Ltd*** [2018] WASCA 205 [54] - [55] (Buss P, Murphy & Beech JJA).

[301] ***Wyong Shire Council v Shirt*** [1980] HCA 12; (1980) 146 CLR 40, 47 - 48 (Mason J).

298 It is in that context that s 5PB makes specific provision with respect to 'health professionals' (which relevantly includes medical practitioners).[302] Section 5PB, in full, provides:

> **5PB. Standard of care for health professionals**
>
> (1) An act or omission of a health professional is not a negligent act or omission if it is in accordance with a practice that, at the time of the act or omission, is widely accepted by the health professional's peers as competent professional practice.
>
> (2) Subsection (1) does not apply to an act or omission of a health professional in relation to informing a person of a risk of injury or death associated with -
>
> (a) the treatment proposed for a patient or a foetus being carried by a pregnant patient; or
>
> (b) a procedure proposed to be conducted for the purpose of diagnosing a condition of a patient or a foetus being carried by a pregnant patient.
>
> (3) Subsection (1) applies even if another practice that is widely accepted by the health professional's peers as competent professional practice differs from or conflicts with the practice in accordance with which the health professional acted or omitted to do something.
>
> (4) Nothing in subsection (1) prevents a health professional from being liable for negligence if the practice in accordance with which the health professional acted or omitted to do something is, in the circumstances of the particular case, so unreasonable that no reasonable health professional in the health professional's position could have acted or omitted to do something in accordance with that practice.
>
> (5) A practice does not have to be universally accepted as competent professional practice to be considered widely accepted as competent professional practice.
>
> (6) In determining liability for damages for harm caused by the fault of a health professional, the plaintiff always bears the onus of proving, on the balance of probabilities, that the applicable standard of care (whether under this section or any other law) was breached by the defendant.

[302] *Civil Liability Act* s 5PA.

299 It is clear, as a matter of history, that s 5PB (which was introduced into the *Civil Liability Act* by the *Civil Liability Amendment Act 2004* (WA)) had its genesis in a report to the federal Minister for Revenue and Assistant Treasurer prepared by a Panel chaired by the Hon David Ipp entitled *Review of the Law of Negligence Final Report dated September 2002* (the Ipp Report).

300 Equally, it is clear, both from the Ipp Report and the legislative history of s 5PB, that it is intended, in part, to introduce a standard of care akin to that which applies in the United Kingdom; namely what is known as the Bolam standard (deriving from ***Bolam v Friern Hospital Management Committee*** [1957] 1 WLR 582 (***Bolam***)). So much is reflected in the Explanatory Memorandum to the *Civil Liability Amendment Act 2004* (WA).[303]

301 This legislative history is relevant to the construction of s 5PB, revealing as it does the context in which the provision was enacted. It is important to recognise, however, that the Court's duty, in interpreting the provision, is to give meaning to the statutory text, not to give effect to what the Court might conclude is the underlying intention of the Parliament detached from that statutory text.[304]

302 As the High Court stated in ***Thiess v Collector of Customs***:[305]

> Statutory construction involves attribution of meaning to statutory text. As recently reiterated:
>
> > 'This Court has stated on many occasions that the task of statutory construction must begin with a consideration of the [statutory] text'. So must the task of statutory construction end. The statutory text must be considered in its context. That context includes legislative history and extrinsic materials. Understanding context has utility if, and in so far as, it assists in fixing the meaning of the statutory text.'

303 It would be a mistake, therefore, to reduce the task of interpreting s 5PB to a matter of identifying 'what does ***Bolam*** require?' or 'what did the Ipp Report propose?'[306]

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[303] Explanatory Memorandum, Civil Liability Amendment Bill 2004 (WA) pt 2.

[304] See ***Taylor v The Owners - Strata Plan No 11564*** [2014] HCA 9; (2014) 253 CLR 531 [40] (French CJ, Crennan & Bell JJ).

[305] ***Thiess v Collector of Customs*** [2014] HCA 12; (2014) 250 CLR 664 [22] (French CJ and Hayne, Keifel, Gageler and Keane JJ).

[306] Cf Appellant's amended submissions [137] - [138]; WAB 34 - 35.

304 The task of construing s 5PB must begin, and end, with the statutory text.

305 Indeed, insofar as the Ipp Report is concerned, it is apparent that the text of s 5PB of the *Civil Liability Act* departs quite markedly from the recommendation in that report. The relevant recommendation in the Ipp Report was:

> **Recommendation 3**
>
> In the Proposed Act, the test for determining the standard of care in cases in which a medical practitioner is alleged to have been negligent in providing treatment to a patient should be:
>
> > 'A medical practitioner is not negligent if the treatment provided was in accordance with an opinion widely held by a significant number of respected practitioners in the field, unless the court considers that the opinion was irrational.'

306 As will become apparent, the difference in language between 'an opinion' (in the Ipp Report) and 'a practice' (in s 5PB) is relevant to ground 12.

307 Similarly, it is important to recognise that s 5PB differs, in significant respects, from provisions in other Australian States whose progenitors are also the Ipp Report and which may broadly be described as having the same legislative purpose.

308 Section 5O of the *Civil Liability Act* 2002 (NSW) (the ***NSW Act***), for example, provides:

> (1) A person practising a profession (**a professional**) does not incur a liability in negligence arising from the provision of a professional service if it is established that the professional acted in a manner that (at the time the service was provided) was widely accepted in Australia by peer professional opinion as competent professional practice.
>
> (2) However, peer professional opinion cannot be relied on for the purposes of this section if the court considers that the opinion is irrational.
>
> (3) The fact that there are differing peer professional opinions widely accepted in Australia concerning a matter does not prevent any one or more (or all) of those opinions being relied on for the purposes of this section.

> (4) Peer professional opinion does not have to be universally accepted to be considered widely accepted.

309 This provision, which will be considered further in the context of ground 12, has been interpreted as providing a defence, in relation to which the onus of proof lies on the defendant.[307] That represents a significant difference from s 5PB. Where s 5PB is engaged, the onus of proof lies on the plaintiff to establish that the relevant act or omission of the health professional was **not** in accordance with a practice that, at the time of the act or omission, is widely accepted by the health professional's peers as competent professional practice.[308]

310 The relevant provision in the *Civil Liability Act 2003* (Qld), which also creates a defence, is different again.[309]

311 Accordingly, while assistance may be derived from the consideration of the provisions of civil liability legislation in other States, it is important that the Court is attentive to the differences between them.

312 Against that background, it is now appropriate to address some preliminary matters of interpretation in relation to s 5PB that are relevant in the present case.

## Section 5PB - preliminary matters

313 At the outset, two related issues arise in relation to the application of s 5PB concerning:

(a) the relationship between s 5PB and the general provision in relation to breach of duty in s 5B; and

(b) whether a defendant who seeks to rely upon s 5PB is required to plead the applicability of the provision in the particular circumstances of the case.

314 In relation to the first issue, as noted above, as a matter of construction, prima facie, s 5B would appear to apply to all cases of breach of duty to which Part 1A of the *Civil Liability Act* applies, including claims against health professionals. That is, an allegation that

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[307] ***Sydney South West Area Health Service v MD*** [2009] NSWCA 343; (2009) 260 ALR 702 [21] (Hodgson JA), [51] (Allsop P), [58] (Sackville AJA); ***Dobler v Halverson*** [2007] NSWCA 335; (2007) 70 NSWLR 151 (***Dobler v Halverson***) [54] - [61] (Giles JA).
[308] *Civil Liability Act* s 5PB(6).
[309] *Civil Liability Act 2003* (Qld) s 22.

a health professional is liable for harm caused by some particular omission by the health professional is perfectly capable of being analysed against the standard set out in s 5B, without reference to whether there is a practice that, at the time of the act or omission, is widely accepted by the health professional's peers as competent professional practice.

315 That observation tends to be confirmed by the reference to a 'negligent act or omission' in s 5PB(1). Against the background of s 5B, the reference to 'negligent act or omission' in s 5PB(1) appears to indicate that even if a finding of breach of duty of care might otherwise be available pursuant to s 5B, the act or omission in question is 'not … negligent' if it is 'in accordance with the practice that … is widely accepted by the health professional's peers as competent professional practice' at the time. In other words, s 5PB(1) appears to assume that, but for the circumstances of its operation, negligence may be found where a reasonable person would have taken precautions in accordance with s 5B.

316 Moreover, as a matter of statutory construction, there must be circumstances in which the conduct of a health professional is to be assessed in accordance with s 5B.

317 In that regard, s 5PB does not create a code in relation to the conduct of health professionals. In some respects this is reflected in the express exceptions to s 5PB(1), such as an act or omission in relation to informing a person of a risk of injury in relation to a proposed course of treatment.[310] In those circumstances, the determination as to whether the health professional is in breach of his or her duty of care (or negligent) is to be assessed in accordance with the provisions of s 5B.

318 More broadly however, in our view, in the absence of evidence that there is, in fact, some practice that is 'widely accepted by the health professional's peers as competent professional practice', whether a health professional is in breach of his or her duty of care to a person is to be assessed by reference to the standard identified in s 5B.

319 Most obviously, there may be no widely accepted practice in relation to a novel or emerging medical issue.

[310] *Civil Liability Act* s 5PB(2).

320 Put another way, and at the risk of sounding trite, the standard in s 5PB(1) only applies if, and when, it is the 'applicable' standard of care.[311] If, on the pleaded case and the evidence, the act or omission in question is not one in respect of which there is 'a practice that … is widely accepted by the health professional's peers as competent professional practice' at the time, an allegation that a health professional is liable for harm caused by their fault in failing to take a particular precaution will fall to be determined in accordance with the standard set out in s 5B. Indeed, s 5PB(6) expressly contemplates that the standard referred to in s 5PB(1) may not be the 'applicable' standard by reference to which the relevant act or omission is to be assessed.

321 This may be illustrated by reference to elements of a cause of action as they would appear in a pleaded case disclosing a reasonable cause of action.

322 It would be sufficient, for example, for a plaintiff who alleges that a health professional defendant was negligent, in order to establish the cause of action, to plead, and prove, that in all of the circumstances a reasonable person in the defendant's position would have taken the particular precautions that the plaintiff alleged ought to have been taken against the risk of harm (as per s 5B). A statement of claim making such an allegation would disclose a reasonable cause of action and would not be liable to be struck out.[312] In that regard, the plaintiff is not required to plead, or allege, that the defendant's conduct was 'not in accordance with a practice that is widely accepted by their peers as competent' professional practice (as per s 5PB).[313] A pleading that failed to do so would not be liable to be struck out as not disclosing a reasonable cause of action.

323 Before s 5PB is potentially engaged, something more is required. In our view, there must at least be some evidence as to the practice widely accepted by peers as competent professional practice before s 5PB is engaged. Of course, the nature of such evidence may be disclosed in the defendant's pleaded defence and so, in discharging his or her onus of proof, the plaintiff will need to address the putative competent professional practice. The plaintiff would, in such a case, fail if the court accepted that, in relation to the act or omission in question, there was a practice that is widely accepted by their peers as

[311] Cf *Civil Liability Act* s 5PB(6).
[312] *Rules of the Supreme Court 1971* (WA) O 20 r 19(1)(a).
[313] As this point, we are not concerned with the distinction, raised by ground 12, between 'practice' and 'a practice'.

competent professional practice', and the plaintiff did not prove that the defendant failed to act in accordance with that practice.[314]

324 Notwithstanding that s 5PB(1) does not create a defence, the following remarks of Basten JA in ***Sparks v Hobson*** are apt to describe the manner in which the issue may be expected to unfold in this State:[315]

> There can only be one standard against which to judge the conduct of a professional defendant, although that standard may depend upon the resolution of conflicting evidence called by the plaintiff and the defendant. It is only if one takes the plaintiff's evidence in isolation that a two-stage process, involving the assessment of the plaintiff's claim followed by assessment of an affirmative defence, will arise. However, in a practical sense, that is not how the dispute should be determined. Rather, a judgment will be given based on all of the evidence. Nor is the exercise helpfully clarified by speaking of shifting burdens of proof. The question for the trial judge is ultimately whether the plaintiff has established that the conduct of the defendant failed to comply with the relevant standard of care. This approach is consistent with ***Dobler*** and is not to say that a plaintiff must seek out and negative opinions inconsistent with those of the experts on whom he or she relies. Beyond that proposition, ***Dobler*** did not turn upon the onus of proof.
>
> …
>
> It is true that s 5O [s 5PB(1)] will not be engaged unless there is evidence of a widely accepted professional practice supporting the defendant's conduct, but where there is such evidence, unless it can be rejected by the trial judge, it will fix the relevant standard; there cannot be two legally supportable standards operating in the one case.

325 Similarly, in Western Australia, once there is evidence that there is a widely accepted practice supporting the defendant's conduct, that practice will generally provide the standard against which the conduct of the defendant is to be judged – unless the plaintiff proves otherwise.

326 In such a case, if the plaintiff does not disprove the application of s 5PB(1), for example by establishing that the putative practice is not widely accepted by competent peers, or that the defendant's act or omission was not in accordance with that practice, or that the practice is 'so unreasonable that no reasonable health professional in the health professional's position could have acted or omitted to do something in

[314] Subject always to the operation of s 5PB(2) and (4).
[315] ***Sparks v Hobson*** [2018] NSWCA 29; (2018) 361 ALR 115 (***Sparks v Hobson***) [18], [24] (Basten JA) referring to ***Dobler v Halverson*** [60] - [61] (Giles JA).

accordance with that practice' (s 5PB(3)), that will be the end of the matter. The defendant is not liable.

327 If, however, if there is no evidence of a widely accepted practice, or the plaintiff does disprove the application of s 5PB(1) in one of these ways, then the case remains to be determined in accordance with s 5B. Section 5PB(1), after all, only provides for circumstances in which a health professional is ***not*** negligent. It does not deal with the converse; i.e. the circumstances in which a health professional ***is*** negligent. To answer that question it is necessary to apply s 5B.

328 Again, save for the references to onus of proof, the following remarks of Simpson JA in ***Sparks v Hobson*** are apposite to the position in this State: [316]

> Failure by a defendant to prove the s 5O [s 5PB(1)] circumstances (however that section is construed) does not conclude the matter. The onus remains on the plaintiff to establish that the defendant failed to provide the professional service in accordance with the standard of the ordinary skilled person practising that profession.
>
> The claim is then to be determined by reference to s 5B of the CLA: see ***Adeels Palace Pty Ltd v Moubarak; Adeels Palace Pty Ltd v Bou Najem*** (2009) 239 CLR 420; [2009] HCA 48 at [27]. The relevant questions are:
>
> - was there a foreseeable risk of harm arising from the conduct the subject of the claim?;
> - was the risk not insignificant?;
> - would a reasonable person in the position of the defendant have taken precautions to safeguard against the risk? (It is necessary for the plaintiff to identify the precautions it is alleged ought to have been taken.)

329 It will be apparent from the above discussion that depending upon the circumstances in each particular case, s 5PB of the *Civil Liability Act*, may, or may not, be relevant to the determination of liability in a claim for negligence against a health professional. If, at the close of the evidence, for example, there is no evidence as to 'a practice … widely accepted by the health professional's peers as competent professional practice' the plaintiff will be required to establish his or her claim by reference to s 5B. If on the other hand, there were such evidence, which the court ultimately accepted, the plaintiff would not establish

[316] ***Sparks v Hobson*** [334] - [335] (Simpson JA).

liability by merely proving what might otherwise be a breach of duty of care by reference to s 5B.

330 It follows from this that, while the onus is on the plaintiff in relation to whether the defendant breached any applicable standard of care, there must be some evidence as to the kind of practice referred to in s 5PB(1) before it arises for consideration. This is not to raise any issue of evidential onus; the evidence might arise as part of the plaintiff's case. It is simply to recognise that s 5PB(1) serves to identify particular circumstances in which an act or omission is not a negligent one and that those circumstances might not arise at all on the facts of a particular case.

331 This leads to the second preliminary issue: namely, whether a defendant, seeking to contest liability on the basis that he or she acted in accordance with a practice that was widely accepted by the health professional's peers as competent professional practice at the time, is required to plead material facts concerning the applicability of s 5PB(1).

332 The answer to that question is, generally, 'yes'.

333 The reason for this is (clearly) not because s 5PB is a defence that must be specifically pleaded. Rather, the obligation to plead the circumstances engaging s 5PB will generally arise as a matter of fairness to ensure that the plaintiff is not taken by surprise as to the case that they are required to meet.[317] In that regard, we would adopt the remarks of Allsop P in ***Sydney South West Area Health Service v MD*** that s 5PB:[318]

> transforms what would otherwise be relevant evidence as to negligence to be weighed by a judge in the familiar calculus into evidence that may be determinative of the appeal. It also may raise, in other cases, although it did not here, issues as to schools of medical practice, the geographical or other areas in which those schools might obtain and other matters requiring specificity and particularisation. In my view, for the reasons Hodgson JA has given and for the reasons in *Dobler v Halverson* [2007] NSWCA 335; 70 NSWLR 151 as well, it is a matter that needs to be pleaded. There is also the question of the surprise rule and precise terms of the relevant rule, *Uniform Civil Procedure Rules* 2005 (NSW) r 14.14.

[317] *Rules of the Supreme Court 1971* (WA) O 20 r 9(1)(b).
[318] ***Sydney South West Area Health Service v MD*** [2009] NSWCA 343; (2009) 260 ALR 702 [51] (Allsop P).

334 The remarks were, of course, made in the context of s 5O of the *NSW Act*, which transfers the onus of proof onto the defendant. Nevertheless, they are equally applicable in a case under the *Civil Liability Act*. Indeed, they may be more so. Given that the plaintiff is required to negative s 5PB(1) where it is successfully invoked, it is important, in order to avoid surprise, that a plaintiff is on notice that the defendant relies upon s 5PB(1), and upon what basis.

335 This is particularly the case given that, as we have concluded in relation to ground 12, for s 5PB(1) to operate it is necessary to identify 'a practice' that existed at the time of the events in question, rather than some more generic reference to 'professional practice'. In those circumstances, as a matter of fairness, the plaintiff will need to address the 'practice' upon which the defendant relies.

336 Nevertheless, as we have stated at [332], we have qualified the answer to this question with the word 'generally'. That is because, there may well be circumstances in which, by reason of the exchange of expert evidence (for example), the particular basis upon which s 5PB is invoked will be readily apparent without the need for it to be specifically pleaded.

337 Much, of course, will depend upon the particular circumstances of each case.

338 We raise the issue of pleading, however, because the appellant in the present case did plead s 5PB. Paragraph [30] of the Re-Amended Defence provided:[319]

> Further, the Defendant says that, pursuant to section 5PB of the *Civil Liability Act 2002* (WA), at all material times it acted in accordance with a practice that was widely accepted as competent professional practice.

339 The respondent sought further and better particulars of the relevant 'competent professional practice' which request was answered as follows:[320]

> 1. The relevant competent professional practice includes:
>
> 1.1 Assessment of total body surface area burnt using a Lund and Browder Burns Chart;

[319] BAB 168.
[320] BAB 170 - 171.

> 1.2 Provision of initial intravenous fluid replacement in accordance with the Parkland Formula;
>
> 1.3 Provision of maintenance fluids in accordance with the Modified Parkland Formula and by assessment of urine output and adjustment of the rate of fluid replacement to maintain a urine output of approximately 1ml/kg/hour; and
>
> 1.4 Regular observation and monitoring of clinical signs including hydration, urinary output, urine specific gravity, haemodilution, and for the presence of peripheral oedema; and
>
> 1.5 Adjustment of intravenous infusion rates in response to signs of fluid retention and increased urinary output.

340 These particulars, it should be emphasised, were given at a time when the respondent's case included an allegation that the appellant was in breach as a consequence of the respondent being administered with too much fluid in the course of her treatment (fluid overload). That allegation was abandoned prior to trial.[321] In those circumstances, the learned trial judge was correct to observe that the practice referred to in the particulars provided by the appellant were not really to the point.[322] That is because the relevant practice, for the purposes of s 5PB, must relate to the act or omission that is alleged to be negligent. It is *that* act or omission that must be in accordance with the relevant practice.

341 In the present case, there was (by the time of trial) no allegation of a negligent act or omission of the appellant in relation to the administration of fluid to the respondent. Widely accepted practice in relation to the administration of fluid was therefore irrelevant.

342 At trial, the relevant acts or omissions were the failure to recognise that the respondent might be suffering from sepsis; the failure to test for sepsis; and the failure to commence her on antibiotics. It was, accordingly, those acts or omissions that the learned trial judge focussed on:[323]

> The question that must be considered under s 5PB(1) in the present case is whether the alleged negligent conduct (that is, the failure to recognise that the plaintiff might be suffering from sepsis, the failure to test for sepsis and the failure to commence her on antibiotics) was conduct that

[321] Primary reasons [66]. See Appeal ts 150 - 151.
[322] Primary reasons [799].
[323] Primary reasons [799].

> was in accordance with a practice that was widely accepted by peers of the doctors responsible for the care and treatment of the plaintiff in the burns ward as competent professional practice.

343 This leads us to a consideration of ground 12.

## Ground 12 - 'a practice' vs 'practice'

344 Ground 12 alleges that the learned trial judge erred in law in construing s 5PB(l) of the *Civil Liability Act* as requiring that the appellant prove the existence of a practice that was widely accepted by the burns ward doctors' peers as competent professional practice, in accordance with which the appellant's officers acted.

345 This ground of appeal refers in particular to the passage at Primary reasons [796], in which, consistent with the passage reproduced at [342] above, his Honour said: [324]

> [T]he first question that must be considered in deciding if the plaintiff has established the alleged breach of duty is whether the plaintiff has proved that the conduct of doctors in the burns ward in failing to recognise that she might be suffering from sepsis, in failing to test for sepsis and in not commencing her on antibiotics, was not in accordance with a practice that was widely accepted by their peers as competent professional practice.

346 In its terms, ground 12 is clearly misconceived. It suggests that the learned trial judge required the *appellant* to prove the existence of the relevant practice. The learned trial judge did no such thing. It is clear that his Honour, correctly, identified that the onus was on the *respondent* to prove that the conduct was **not** in accordance with any such a practice.

347 As developed in the appellant's submissions, however, it is clear that ground 12 is not concerned with the onus of proof but rather with whether, for the purposes of s 5PB, it is necessary to identify 'a practice', in the sense of a regular course of conduct in particular circumstances or, alternatively, whether s 5PB operates where the defendant's conduct accords with 'practice' in a general sense, i.e. 'practice that is widely accepted by peers as competent professional practice'.[325]

[324] Primary reasons [796].
[325] Appellant's amended submissions [128]; WAB 33.

348 The appellant submits that s 5PB does not require the identification of 'a practice'.

349 By way of shorthand, then, the issue raised by ground 12 is whether, properly construed, s 5PB is concerned with 'a practice' or simply with 'practice'?

350 The distinction between the two was usefully articulated by Basten JA in ***Sparks v Hobson***:[326]

> To speak of 'a practice' adopted by a group of professional persons suggests a regular course of conduct adopted in particular circumstances. By contrast, the phrase 'competent professional practice' is apt to cover the whole gamut of professional services provided by the practitioner, whether or not the particular circumstances have arisen sufficiently often to result in an established practice.

351 In this regard the current state of authority in New South Wales, in relation s 5O of the *NSW Act*, albeit with some controversy, favours the requirement for 'a practice'. The appellant submits that that is 'plainly wrong' and should not be followed.

352 The requirement for 'a practice' in New South Wales may be traced to the decision of the Court of Appeal in ***McKenna v Hunter & New England Local Health District***.[327] In ***McKenna***, Macfarlan JA (with whom Beazley P agreed) stated:[328]

> To establish a defence under s 5O a medical practitioner needs to demonstrate, first, that what he or she did conformed with a *practice* that was in existence at the time the medical service was provided and, secondly, to establish that that *practice* was widely, although not necessarily universally, accepted by peer professional opinion as competent professional *practice*. (original emphasis)

353 The decision in ***McKenna*** was overturned by the High Court on grounds unrelated to s 5O.[329]

354 Following the High Court's decision, the authority of ***McKenna*** in relation to the construction of s 5O was considered by the Court of Appeal in ***Sparks v Hobson***.

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[326] ***Sparks v Hobson*** [31] (Basten JA).
[327] ***McKenna v New England & Hunter Local Health District*** [2013] NSWCA 476 (***McKenna***).
[328] ***McKenna*** [160] (Macfarlan JA).
[329] ***New England & Hunter Local Health District v McKenna*** [2014] HCA 44; (2014) 253 CLR 270.

355 In ***Sparks v Hobson***, Macfarlan JA adhered to the construction he had reached in ***McKenna***:[330]

> Section 5O uses the past tense ('at the relevant time ... was widely accepted') to refer to the relevant peer professional opinion. Thus, the opinion about the manner in which the defendant acted must have existed, and been widely accepted, at the time the conduct occurred. It is not enough that experts called to give evidence consider that the conduct was reasonable and that it would have been so regarded by other professionals if they had been asked about it at the time of the conduct.

356 Basten JA disagreed. His Honour concluded that:[331]

> although the language used in *McKenna* may well sufficiently describe many circumstances in which s 5O is invoked, I would not understand it as a general proposition as to the constraints imposed by s 5O(1).

357 The third member of the court in ***Sparks v Hobson***, Simpson JA, disagreed with the reasoning in ***McKenna*** but considered that she was obliged to accept it as stating the proper construction of s 5O. Her Honour said:[332]

> I consider that I am obliged to accept ***McKenna*** (in this Court) as stating the prevailing construction of s 5O: ***Gett v Tabet*** (2009) 254 ALR 504; [2009] NSWCA 76 at [277] - [279]. But for that constraint, I would have considered that the language of s 5O makes it plain that 'competent professional [here, medical] practice' is intended to denote 'the practice of a profession [here, medicine]', and not a specific practice or method of providing the professional services in question. That construction is supported by the absence of the indefinite article in conjunction with 'professional practice', and the use instead of the adjective 'competent', which to my mind signifies professional practice in a general sense, rather than an identifiable, specific, and discrete aspect of the profession or method of providing the professional service.

358 It will be apparent, therefore, that there is a divergence, in ***Sparks v Hobson***, between the *reasoning* of the members of the Court and its *authority* for the prevailing construction of s 5O of the *NSW Act*.

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[330] ***Sparks v Hobson*** [211] (Macfarlan JA).
[331] ***Sparks v Hobson*** [34] (Basten JA).
[332] ***Sparks v Hobson*** [332] (Simpson JA).

359 Following ***Sparks v Hobson***, Leeming JA described this divergence in ***South Western Sydney Local Health District v Gould*** as:[333]

> [W]hether the reference to 'practice' is a reference to the practice of the relevant profession, or more narrowly to a particular specific practice or method of providing the services.

360 As Leeming JA went on to note, in ***South Western Sydney Local Health District v Gould***, the divisive issue could be entirely put to one side for the purpose of that appeal.

361 The division of opinion in New South Wales, therefore, remains unresolved.

362 Whether the reasoning in ***McKenna*** ultimately prevails in New South Wales is, of course, not the concern of this Court. That reasoning, and the division in relation to it, concerns the particular statutory text of s 5O of the *NSW Act.*

363 The text of s 5PB of the *Civil Liability Act* is different. However, fortuitously (in the sense that there is no necessity for this Court to enter the sphere of debate in New South Wales), it is different in a manner that makes the reasoning in ***McKenna*** even more applicable to s 5PB of the *Civil Liability Act* than it is to its New South Wales counterpart.

364 Put another way, whether the approach in ***McKenna*** is ultimately held to reflect the proper construction of s 5O of the *NSW Act*, it is apparent that **all** of the different approaches in New South Wales support the approach in ***McKenna*** as the proper construction of s 5PB of the *Civil Liability Act*.

365 That is because, unlike s 5O of the *NSW Act*, s 5PB of the *Civil Liability Act* expressly refers to '**a** practice' widely accepted by peers as competent professional practice.

366 The controversy in relation to s 5O of the *NSW Act* in fact only exists because the provision does not refer to 'a practice' but rather refers to the professional having acted 'in a manner' that was widely accepted in Australia by peer professional opinion as competent professional practice.

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[333] ***South Western Sydney Local Health District v Gould*** [2018] NSWCA 69; (2018) 97 NSWLR 513 [114].

367 The members of the New South Wales Court of Appeal who have expressed disagreement with the approach in ***McKenna*** have expressly done so for the reason that, unlike s 5PB of the *Civil Liability Act*, the *NSW Act* provision does not refer to 'a practice'.

368 Basten JA, for example, having set out the central holding in ***McKenna*** (reproduced at [352] above) commenced his discussion of the issue by noting that 'there is a risk in reformulating the statutory language'.[334] As the passage immediately following (reproduced at [350] above) makes clear, the 'reformulation' Basten JA was referring to was use of the expression 'a practice'.

369 In s 5PB of the *Civil Liability Act*, to refer to 'a practice' is not to reformulate the statutory language. It **is** the statutory language.

370 Indeed, Basten JA went on in ***Sparks v Hobson*** to say:[335]

> There are other reasons for thinking that the reference to 'competent professional practice' does not require evidence of 'a practice'. First, it is the 'manner' in which the defendant acted which must be the focus of the opinion. Secondly, if it were necessary to establish a practice, one might expect subs (3) to refer to 'opinions ... concerning that practice', rather than 'opinions ... concerning a matter'.

371 The very language that Basten JA said 'one would expect' to find in s 5O(3) of the *NSW Act* if it required 'a practice' is precisely what one finds in s 5PB. Section 5PB(3) (the equivalent provision to s 5O(3) of the *NSW Act*), rather than referring to 'opinions … concerning a matter', refers to 'the practice' differing from or conflicting with 'another practice'.

372 The language of s 5PB, as a whole, is therefore more consistent with 'a regular course of conduct adopted in particular circumstances'.[336]

373 Similarly, the reasoning of Simpson JA in ***Sparks v Hobson*** (reproduced at [357] above), while contrary to the application of the reasoning in ***McKenna*** to s 5O of the *NSW Act* in fact supports the application of that reasoning to s 5PB of the *Civil Liability Act*. As her Honour noted, the 'absence of the indefinite article' militated against a

[334] ***Sparks v Hobson*** [31] (Basten JA).
[335] ***Sparks v Hobson*** [32] (Basten JA).
[336] ***Sparks v Hobson*** [31] (Basten JA).

construction that contemplated a specific practice or method of providing the professional services in question.[337]

374 In s 5PB, not only does the section refer to 'a practice' using the indefinite article, it refers later to 'the practice' and 'another practice'. Such language, in our view, contemplates 'a specific practice' (per Simpson JA) or 'a regular course of conduct adopted in particular circumstances' (per Basten JA).

375 Accordingly, whether or not the reasoning in ***McKenna*** is clearly wrong in relation to s 5O of the *NSW Act*, in our view, it is directly applicable to s 5PB of the *Civil Liability Act*. In that regard, s 5PB expressly refers to '**a** practice that, at the time … is widely accepted'. That requires the identification of an existing practice whose wide acceptance can be the subject of evidence.

376 What s 5PB, in its reference to 'a practice' is referring to, in our view, is an established course of conduct adopted in particular circumstances. Both the reference to 'a practice' and the backwards looking nature of the enquiry ('at the time of the act or omission') are such that (leaving aside the question of onus, for the moment), the practice must *as a matter of actual fact* have existed, and been widely accepted, at the time. It is not a matter of asking whether a particular practice, in the sense of the course of conduct, *would have* been widely accepted were competent peers asked about it at the time.

377 Expert evidence as to whether a particular practice or course of conduct *would have* been widely accepted at the time (if competent peers were asked about it), is of course, not irrelevant. It would remain relevant to the assessment of breach generally (under s 5B).

378 In that regard, the position under s 5B is consistent with the approach at common law described by Gleeson CJ in ***Rosenberg v Percival***, namely that:[338]

> [T]he *relevance* of professional practice and opinion was not denied; what was denied was its *conclusiveness*. In many cases, professional practice and opinion will be the primary, and in some cases it may be the only, basis upon which a court may reasonably act. But, in an action brought by a patient, the responsibility for deciding the content of the doctor's duty of care rests with the court, not with his or her professional colleagues. (original emphasis)

[337] ***Sparks v Hobson*** [332] (Simpson JA).
[338] ***Rosenberg v Percival*** [2001] HCA 18; (2001) 205 CLR 434 [7] (Gleeson CJ).

379 Of course, the change made by s 5PB is that, where there is evidence of 'a practice that, at the time of the act or omission, is widely accepted by the health professional's peers as competent professional practice' then (subject to the express exceptions) acting in accordance with that practice will be *conclusive* (the onus of negativing which lies on the plaintiff).

380 In that context, requiring evidence of an actual practice is not inconsistent with the policy underlying s 5PB. It simply reflects its terms.

381 Nor does the section require that the 'practice' to which s 5PB applies have any particular formality, in the form of a code of practice or prescribed method. It does not require, as the appellant characterised the approach in ***McKenna***, 'some pre-existing library of practices that are dipped into from time to time'.[339]

382 A widely accepted practice may well be a course of conduct widely followed, and observed, by competent peers, that is not to be found in a 'library of practices', but in the actual practice of competent practitioners.

383 Indeed, that was precisely the position in ***Bolam*** itself, which was concerned with 'a practice' (or 'technique' as McNair J also described it).[340]

384 In that regard, the practitioner whose conduct was in issue in ***Bolam***, Dr Allfrey, had applied a technique of restraint in the application of electro-convulsive therapy (**ECT**) which involved 'arranging for the [patient's] shoulders to be held, the chin supported, a gag used, and a pillow put under his back'.[341] There was also evidence at the trial that, in addition to this practice, other practitioners used relaxant drugs or restraining sheets. There was therefore evidence of a variety of specific 'practices'.

385 It was in this context that McNair J directed the jury:[342]

> [I]t is not essential for you to decide which of two practices is the better practice, as long as you accept that what the defendants did was in accordance with a practice accepted by responsible persons.

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[339] Appeal ts 131.
[340] There was also a 'failure to warn' case in ***Bolam***, which is, of course, wholly excluded from the scope of s 5PB by s 5PB(2).
[341] ***Bolam*** 586 (McNair J).
[342] ***Bolam*** 587 - 588 (McNair J).

386 It can, therefore, be seen that ***Bolam*** involved 'a practice' of the kind contemplated by the approach in ***McKenna***.

387 To take another example, in the present case there was evidence (which the learned trial judge accepted) that there was a widely accepted practice, namely that in 'burns cases antibiotics should not be given prophylactically'.[343] Accordingly, if there had been an allegation by the respondent that the appellant was negligent because reasonable care required that all burns patients should be administered prophylactic antibiotics, s 5PB(1) would have been fatal to such an allegation (unless the respondent could establish that s 5PB(3) applied).

388 But that was not the negligent act or omission alleged in the present case. In the present case the alleged negligent act or omission was 'in failing to recognise that [the respondent] might be suffering from sepsis, in failing to test for sepsis and in not commencing her on antibiotics'.[344]

389 Returning, then, to the passage in the Primary reasons impugned by ground 12 (at [344] above), it will be apparent that, far from that passage reflecting an erroneous construction of s 5PB(1), the learned trial judge simply reproduced the terms of the statutory text itself in the context of the particular conduct that his Honour had found to have occurred.

390 That is, to pose the question as being whether the respondent had proved that that conduct 'was not in accordance with a practice that was widely accepted by [the doctors in the burns ward's] peers as competent professional practice' was precisely what s 5PB of the *Civil Liability Act* required.

391 It discloses no error.

392 Ground 12 has not been made out.

393 In any event, before leaving this ground, we would observe that the distinction drawn by the appellant between 'a practice' and 'practice' is not one that would have affected the ultimate conclusion that his Honour reached in relation to the applicability of s 5PB to the respondent's claim.

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[343] Primary reasons [803].
[344] Primary reasons [796].

394 In that regard it is to be recalled that the fundamental starting point for his Honour's findings on breach was his conclusion that it was beyond any doubt that, as a matter of fact, prior to the respondent's admission to the ICU 'no consideration was given by any of the doctors … responsible for her care and treatment to the possibility that her deterioration was due to sepsis'.[345]

395 The failure to recognise that possibility was, in the circumstances of the case, sufficient for the respondent to discharge her onus of proof under s 5PB because, as his Honour found: [346]

> [N]one of the medical witnesses who gave evidence suggested that it was widely accepted by peers of doctors working in a paediatric burns unit as competent professional practice to fail to recognise that a patient is, or might be, suffering from sepsis and in those circumstances to fail to test for sepsis and to fail to administer antibiotics.

396 It may be observed that this conclusion holds true whether the failure to recognise that the respondent might be suffering from sepsis was 'a practice' or, simply, 'practice'.

## Ground 13 - were Dr Numa and Professor Kesson relevantly 'peers'?

397 Ground 13 alleges that the learned trial judge erred in finding that for the purpose of s 5PB(l) of the *Civil Liability Act*, the relevant professional 'peers' included Dr Numa and Professor Kesson.

398 The 'finding' challenged by the appellant by ground 13 does not appear explicitly in the Primary reasons. That is, while the learned trial judge did express the relevant question as being whether the conduct of the doctors 'in the PMH burns ward' was in accordance with a practice which is widely accepted by 'their peers',[347] he did not expressly characterise Dr Numa and Professor Kesson as 'peers' in that sense.

399 It may be that his Honour implicitly found this to be the case, given that his Honour expressly adopted the following views expressed by Sweeney DCJ in ***Wright v Minister for Health***:[348]

> As to who constitutes the medical professional's peers, I would construe that to mean members of a group of professionals who are charged with, or generally responsible for, making such decisions as the professional under scrutiny made. Part of a case for alleged negligence

[345] Primary reasons [794].
[346] Primary reasons [800].
[347] Primary reasons [714].
[348] ***Wright v Minister for Health*** [2016] WADC 93 [87] (Sweeney DCJ). See Primary reasons [715].

> may include an allegation that a professional acted above their experience and level of responsibility, so the peers should not be confined to those at a level of the professional whose actions are being scrutinised, but should include those who are generally regarded as being the appropriate professionals to make such decisions.

400 In this regard, again, in relation to the question whether the respondent exhibited signs and symptoms sufficiently suggestive of infection such as to require that infection be considered by the treating doctors, the learned trial judge may have implicitly found that *all* of the relevant experts were persons who (in varying ways) were generally responsible for assessing such signs and symptoms - and could give relevant evidence in that regard.

401 If that were the case, in our view it would have been a finding that was open to the learned trial judge.

402 It is not necessary, however, to resolve that issue for the purposes of ground 13.

403 In that regard, again, the act or omission identified by the learned trial judge, and upon which the issue of breach was analysed, was the failure by the treating practitioners to consider the possibility that she might have sepsis. His Honour found that none of the expert witnesses suggested that it was competent professional practice to fail to do so. That finding itself is not challenged.

404 That it was not competent professional practice to fail to consider the possibility of infection was therefore supported by all of the expert evidence. There is no basis for challenging that finding.

405 Insofar as ground 13 makes a different submission and suggests that Dr Numa and Professor Kesson were not qualified to give evidence relevant to breach of duty of care, that submission must be rejected.

406 There was no challenge at trial to the expertise of each of Dr Numa and Professor Kesson to express the opinions that they did in relation to the treatment of the respondent and the signs exhibited by her which raised a suspicion of sepsis. As the learned trial judge found, all of the expert witnesses called by the parties were generally impressive and provided opinion evidence on the basis of which the learned trial judge was entitled to make findings, both for the purposes of s 5PB and, more broadly, for the purposes of s 5B.

407 Ground 13 has not been made out.

## Ground 14 - application of s 5PB

408 Ground 14 maintains that, '[h]aving misconstrued s 5PB(1), his Honour did not consider the evidence that was relevant to the correct application of the provision'.[349] The ground of appeal alleges, in the particulars to the ground, that the only evidence of burns doctors at trial was that of Professors Kimble and Harvey and that they gave evidence that the treatment of the respondent was competent professional practice.

409 We have already concluded that the learned trial judge did not misconstrue s 5PB(1) and as such, ground 14 does not strictly arise.

410 For completeness, however, it is appropriate to address it in further detail.

411 The submissions in support of this ground of appeal appeared to characterise the relevant finding of breach as being the following conclusion at Primary reasons [801]:

> In summary, in my opinion, the plaintiff has discharged her burden of proving that the alleged negligent conduct of the doctors who were responsible for her care and treatment while she was a patient in PMH's burns ward was not conduct that was in accordance with a practice that was, in December 2005, widely accepted by their peers as competent professional practice.

412 This was **not** the learned trial judge's finding of breach. Rather, this particular finding was his Honour's conclusion as to why s 5PB(1) was not applicable to the respondent's case. Indeed, his Honour immediately went on to say that the question remained whether the conduct of the doctors fell below the standard of care required of them.[350]

413 That is, in the manner described in [327] to [329] above, the learned trial judge, having concluded that the respondent had disproved the application of s 5PB, went on to consider whether, in accordance with s 5B, the appellant was nevertheless in breach of its duty to take reasonable care. For the reasons given at in [327] to [329] above, his Honour was correct to proceed in this way.

[349] Appellant's amended submissions [154]; WAB 38.
[350] Primary reasons [802].

414 The finding of breach, therefore, is that which follows at Primary reasons [803] to [805].[351] In those paragraphs it is clear that his Honour was applying the provisions of s 5B of the *Civil Liability Act*. The critical findings of fact leading to that finding of breach were:

(a) the signs and symptoms which the respondent had exhibited on 10 December 2005 should, by no later than around 2.00 am on 11 December 2005, have caused the team of doctors responsible for the respondent's care and treatment in the burns ward to recognise the possibility (that is, the foreseeable and not insignificant risk) that she did have evolving sepsis;

(b) in light of this possibility, the doctors should have taken a sample of her blood for culturing and to commence administering to her as quickly as was reasonably possible (say, within an hour) broad spectrum antibiotics pending the receipt of the results of the blood culture; and

(c) the downside of not administering antibiotics to a paediatric burns patient who on clinical grounds is suspected to be suffering from sepsis far outweighs the potential benefits of adopting such a course of action.

415 In making these findings of fact, which were relevant to the application of s 5B, the learned trial judge was entitled to have regard to all of the evidence before him. It is apparent from the Primary reasons that his Honour had regard to all of the expert evidence. He was entitled to do so.

416 To the extent that the appellant submitted that the learned trial judge could *only* have regard to the evidence of Professors Kimble and Harvey,[352] in making an assessment of the reasonable person's response to the risk of harm under s 5B, that submission must, again, be rejected. The identification of signs and symptoms of infection was a matter in relation to which experts from a variety of specialist fields were able to express opinions and which, without objection, they did.

417 The learned trial judge was not obliged to accept the evidence of Professors Kimble and Harvey. The findings that he made were not otherwise demonstrated to be ones that were made in error.

418 Ground 14 is not made out.

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[351] See [87] to [88] above.

[352] See the particulars to Ground 14; WAB 9; Appellant's amended submissions [156]; WAB 38.

## Ground 15 - finding of breach at 3.00 am on 11 December 2005

419 Ground 15 is expressed to be same challenge as ground 14, the difference only being 'as to the time of breach'.[353]

420 Unlike ground 14, the particulars and submissions in support of this ground do refer to the finding of breach found at Primary reasons [803] - [805].

421 As with ground 14, however, they suffer from the same misconception as that ground, namely that the learned trial judge was in those paragraphs dealing with s 5PB, and the identification of a practice within the meaning of that section. His Honour was not. Rather, as has been explained and as was the proper course, his Honour was, in that part of the Primary reasons (having been satisfied that s 5PB was negatived by the respondent), applying the provisions of s 5B of the *Civil Liability Act*.

422 Ground 15 is not made out.

## Ground 16 - challenge to the critical finding of fact on breach

423 Ground 16 challenges the following finding of fact:[354]

> Having reviewed the PMH medical records relating to the plaintiff, I am satisfied beyond any doubt that prior to the plaintiff being admitted to the ICU no consideration was given by any of the doctors who, to that point, had been responsible for her care and treatment to the possibility that her deterioration was due to sepsis as opposed to fluid overload. All the references in the notes refer, in effect, to fluid overload as being the suspected cause of the plaintiff's deteriorating condition. There is no reference in the notes made by the doctors responsible for the plaintiff's care and treatment in the burns ward to sepsis as a possible differential diagnosis or indeed any reference to sepsis at all. I note in this regard that it was not suggested on behalf of the defendant that I could find on the evidence that the doctors in question did at any stage consider that the plaintiff's deterioration was due to sepsis as opposed to fluid overload.

424 The finding is purely a finding of fact. It is the factual finding upon which the learned trial judge's conclusion in relation to s 5PB was based.[355]

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[353] Appellant's amended submissions [162]; WAB 38.
[354] Primary reasons [794].
[355] See [395] above.

425 The particular challenge brought by ground 16 is that the learned trial judge's finding was against 'the weight of the evidence'.[356] In oral submissions, the appellant went so far as to submit that it was 'plainly absurd'.[357]

426 As his Honour recorded, this was not the appellant's position at trial. On the contrary, at trial, the appellant did not suggest that the learned trial judge could find that the doctors did consider that the respondent's deterioration was 'due to sepsis as opposed to fluid overload'.[358]

427 This final reference ('as opposed to fluid overload') is significant because, as his Honour found, the medical records *did* make reference to '? fluid overload' and 'Imp: fluid overload' as the possible explanation for the respondent's deterioration.[359] It is not the case, therefore, that the medical records were silent as to the matters considered by the staff in the burns ward: they specifically recorded one possibility and did not record the other.

428 The appellant's expert witnesses confirmed in cross-examination that the medical records revealed no consideration of the possibility of sepsis until the respondent was transferred to the ICU.[360]

429 This was in contrast with the contents of medical records following the respondent's admission to ICU. The doctors at ICU identified and recorded infection and sepsis as a possible cause of the respondent's condition.[361] It could not, therefore, be said that sepsis was so obvious a differential diagnosis as to require no record.

430 There was, accordingly, a rational basis for the learned trial judge to place significance on the absence of any reference to sepsis in the respondent's medical records while she was being treated in the burns unit, in contrast to the references to fluid overload. It was not 'plainly absurd' to infer from the absence of any such record that sepsis was not actively considered at that time.

431 As for the 'weight of the evidence', there was in fact no other evidence to suggest that the doctors in the burns ward considered the possibility of sepsis as the cause for the respondent's deterioration.

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[356] Particular (a) to Ground 16; WAB 10.
[357] Appeal ts 136.
[358] Primary reasons [794].
[359] See e.g. GAB 101 - 103.
[360] Trial ts 107 - 108 (Dr Prebble), 205 - 206 (Professor Starr), 430 - 432 (Professor Harvey).
[361] Trial ts 388; GAB 115.

432 It is not without significance that none of the doctors treating the respondent were called to give evidence that they had given consideration to the possibility that the respondent's deterioration was due to sepsis.

433 In its submissions, the appellant submitted that the respondent 'at trial did not invite any inference due to the [appellant] not calling any of the treating doctors in the burns unit'.[362] That is not correct. The respondent expressly made a submission to that effect, referring to ***Jones v Dunkel***.[363]

434 In any event, as we have said, there was no evidence that those doctors had considered the possibility of sepsis.

435 The only evidence that the appellant points to in this regard is the evidence of Professors Kimble and Harvey that infection is 'something which we always have to consider'.[364] That is not, however, evidence in relation to what the practitioners treating the respondent considered on 10 and 11 December 2005, or whether their attention was unreasonably focussed only on the possibility of fluid overload.

436 In this regard, the appellant's submissions sought to create something of a straw man, by submitting that his Honour had found that those treating the respondent had 'simply not known' or were 'unaware of the risk of infection'.[365] The learned trial judge's finding was not a finding of some generalised deficiency in the knowledge of those treating the respondent. It was a finding that, in the particular circumstances of the respondent's case, those treating doctors had not considered sepsis as a possible cause of deterioration on 10 and 11 December 2005.

437 The inference drawn by the learned trial judge was open to him and, in our view, was correct.

438 Ground 16 has not been made out.

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[362] Appellant's amended submissions [171]; WAB 40.

[363] ***Jones v Dunkel*** (1959) 101 CLR 298; See Plaintiff's Outline of Closing Submissions dated December 2017 [8]; BAB 199 - 200. The respondent, on appeal, repeated the submission in support of a ***Jones v Dunkel*** inference in her notice of contention; WAB 108.

[364] Trial ts 310 - 311 (Professor Kimble). The Appellant's amended submissions [166]; WAB 39 also refer to Professor Harvey's evidence that differentiating inflammation from infection is a 'perennial problem faced by physicians'. This was in fact Dr Numa's evidence (see GAB 589 - 590) and one of the reasons for Dr Numa expressing the opinion that the signs and symptoms should not be looked at in isolation.

[365] Appellant's amended submissions [166] - [167]; WAB 39 - 40.

## Notice of contention

439 The respondent filed an amended notice of contention (the **notice of contention**) raising two issues, namely that:

(a) even if the appellant's construction of s 5PB was accepted, the result would not be different as the failure to recognise that the respondent might be suffering from sepsis would not be in accordance with widely accepted competent practice; and

(b) the inference that the doctors in the burns ward had not considered the possibility of sepsis as the cause for the respondent's deterioration was strengthened by the failure of the appellant to call any of those doctors.

440 We have already addressed (to an extent) the first contention, at [393] to [396] above and, in relation to the second contention, we are satisfied that the learned trial judge's finding at Primary reasons [794] was correct in any event.

441 Beyond that, it is not necessary for the purposes of determining this appeal to deal with the notice of contention.

## Conclusion

442 For the above reasons, the appeal must be dismissed.

I certify that the preceding paragraph(s) comprise the reasons for decision of the Supreme Court of Western Australia.

JS
Research Associate to the Honourable Chief Justice Quinlan

27 SEPTEMBER 2019