A study into the liability in the tort of negligence of the governing bodies of Australian contact sport for failing to warn their athletes of the risk of the brain disorder chronic traumatic encephalopathy (CTE) prior to CTE being declared an 'obvious risk' of contact sport

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Abstract

This thesis considers the question of the liability in negligence of the governing bodies of major contact sport in Australia in failing to take reasonable steps to inform themselves of the risk of the debilitating brain disorder chronic traumatic encephalopathy (CTE), to warn their premier-level athletes of that risk. Collateral consideration is given to failure to introduce ameliorating protocols to reduce the risk of contracting CTE. The thesis concentrates on the years and decades prior to 2010, a year when it is roughly estimated that CTE will have become an 'obvious risk' of contact sport at premier level for purposes of state civil liability legislation and common law. As argued, from 2010 forward those who play contact sport will be deemed in law to have volunteered to accept the risk of CTE. Athletes who played premier-level contact sport in the decades prior to 2010, ex-players now in their 40s, 50s, 60s and 70s, are those who almost exclusively, at the time of writing this thesis, claim to be suffering the symptoms of CTE.

CTE is caused by repetitive concussive and sub-concussive head trauma experienced over a period of years playing contact sport. As trauma to the head accumulates, a point is reached where a protein, tau, is released from neurones of the brain to form phosphorated tau, that clumps around small blood vessels and cells disrupting normal cognitive function. Over time, the condition spreads throughout the brain causing a sufferer to experience increasingly severe symptoms such as poor concentration, headaches, short-term memory loss, suicidality, aggression, explosivity, dementia and parkinsonism. There is no known cure, and sufferers ultimately die of, or with, the disease.

Three medical studies spanning more than 80 years are argued in this thesis to provide points in time from which to consider the negligent liability of the governing bodies of major contact sports in Australia in negligently causing their athletes to contract CTE. In 2005 Dr Bennet Omalu et al found at autopsy that National Football League (NFL) player Mike Webster was affected by CTE. In 2009 Dr Ann McKee et al implicated repetitive head trauma (RHT) in the playing of several contact sports common to Australia as a cause of CTE. CTE associated with RHT in boxing was revealed as far back as 1928 by Dr Harrison Martland in an article published in the *Journal of the American Medical Association*. Most publications on CTE were concerned with boxing up until the 1970s when, increasingly, a range of contact sports were revealed as presenting a risk of CTE.

The fundamental question explored by the thesis is: At a time when a premier-level athlete was exposed to the risk of CTE, did his or her sport governing body (SGB) know, or should it have known, of the link between RHT in playing contact sport and CTE and taken reasonable steps to ameliorate the risk of harm by warning its premier players of that risk?

There are now hundreds of athletes in Australia and globally who are diagnosed by their treating physician as suffering the symptoms of CTE, or who were diagnosed definitively at autopsy as having been affected by CTE. This number is predicted to rise to several thousand

over the coming decades.[1] Many premier-level athletes have indicated a willingness to sue in negligence the SGB that administered the premier-level competition in which they. Ex-players of the Australian Football League (AFL) filed a representative action (class action) in the Supreme Court of Victoria against the AFL in 2023. Premier-level rugby league players have threatened similar action against the National Rugby League in Australia. More than 180 British rugby players have reportedly begun a suit in negligence against World Rugby, the Rugby Football Union and the Welsh Rugby Union. A suit against the NFL in the United States was settled in 2014 for around US$1 billion after it had been revealed that the NFL had conducted its own research into the damaging effects of RHT in American football but had decided not to reveal the results of the study to its players.

A case in negligence associated with the contraction of CTE in Australia and across the common law jurisdictions is yet to be considered in a court of law. This thesis addresses this gap in our knowledge of the law of negligence as it applies to liability for the causation of CTE specifically, and more broadly to the scope of the legal obligations of sporting organisations to those who play under their authority.

Statement of originality

This is to certify that to the best of my knowledge the content of this thesis is my own work. This thesis has not been submitted for any degree or other purposes.

I certify that the intellectual content of this thesis is the product of my own work and that all the assistance received in preparing this thesis and sources have been acknowledged.

David Thorpe, 18 August 2025.

[1] A reference made by neurologist and CTE specialist medical practitioner Dr Rowena Mobb: see Jessica Halloran and Wendy Carlisle, 'Wally Lewis: The Tragic Cost of a Glorious Rugby League Career', *The Australian* (Sydney, 31 July 2023).

Chapters published as papers/edited book chapters

These publications were completed as part of the thesis, put before supervisors and then submitted for publication, as encouraged by the School of Law, and accepted for publication. They are part of the thesis.

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Attestation

As supervisor for the candidature upon which this thesis is based, I can confirm that the authorship attribution statements above are correct.

Professor Cameron Stewart

18 August 2025

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Contents

Abstract 2
Statement of originality 3
Chapters published as papers/edited book chapters 4
Attestation 5
Acknowledgments 6
Chapter 1: Introduction 19
1.1 Introduction 19
1.1.2 Development of the thesis 20
1.2 Research objectives and the thesis argument 20
1.2.1 The length of this thesis 20
1.3 The significance of this thesis topic 21
1.4 Research methodology 22
1.4.1 Concerns of law and doctrine 22
1.4.2 Concerns of fact 24
1.5 Rationales to the approaches taken in this thesis 25
1.5.1 Which party in litigation? 25
1.5.2 The sports where CTE is a risk of harm 26
1.5.3 Collision, contact and combat sport 27
1.5.4 Why the SGB is the 'defendant of choice' 27
1.5.5 Why 'premier-level' athletes of contact sports? 29
1.5.6 Why evidence that contact sport can cause CTE and that an individual athlete is afflicted by CTE is necessary 32
1.5.7 Pending and threatened litigation 32
1.5.8 Time periods to provide a structure 34
1.5.9 Why players of an era prior to *circa* 2010? 35
1.5.10 Why breach of a duty by failure to 'be informed' and 'to warn' of the risk of CTE? 36
1.5.11 Sources of information on athletes said to be suffering with CTE 37
1.6 Glossary of terms used in this thesis 38
1.6.1 Nomenclature and CTE 38
1.6.2 Sport governing body (SGB) 38
1.6.3 Premier-level athletes 38
1.6.4 Chronic traumatic encephalopathy (CTE) 39
1.6.5 Concussion (symptomatic) 39
1.6.6 Asymptomatic concussion (sub-concussion) 39

1.6.7 Neuropathology 40
1.6.8 Tau protein 40
1.6.9 Neurofibrillary tangles 40
1.6.10 Repetitive head trauma 40
1.7 Chapter descriptions in this thesis 40
Chapter 2: The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments 43
2.1 Introduction 43
2.1.1 This chapter proceeds as follows 44
2.2 What is CTE? 45
2.2.1 The tau protein and tauopathy 47
2.3 Brief overview of reports of Australian athletes suffering CTE 47
2.4 The importance of the timeline of revelations of CTE 49
2.4.1 Three landmark medical studies 50
2.4.2 Two other influential periods 53
2.5 Head trauma (symptomatic concussion) and sub-concussive head trauma (asymptomatic concussion) 55
2.5.1 The significance of sub-concussive head trauma 57
2.5.2 Severe head trauma and CTE 62
2.6 Conclusion 63
Chapter 3: A Duty Relationship Between an SGB and the Athletes Who Play Contact Sport 65
3.1 Introduction 65
3.1.1 This chapter proceeds as follows 67
3.2 Duty of care owed by an SGB to its premier players 70
3.2.1 Foreseeability not sufficient: Absence of neighbourhood 71
3.2.2 A 'recognised category' in limited circumstances 72
3.2.3 What of an employer–employee relationship? 73
3.3 Salient features and the SGB's duty of care 74
3.3.1 The development of a 'salient features' approach 75
3.3.2 Scope and content also applicable under a salient features approach 80
3.3.3 Salient features and cases below 80
3.4 *Agar v Hyde*: Distinguishing a sport case of 'no duty' 80
3.4.1 *Agar v Hyde*: A factual distinction with the SGBs of Australian sport 81
3.4.2 The fact of an absence of 'salient features' underpinning the decision in *Agar v Hyde* 82
A Insufficient control and relationship 82

B Insufficient assumption of responsibility 82
C Insufficient reliance and vulnerability 83
D A general duty of care under different circumstances? 83
3.4.3 *Agar v Hyde*: Contrasts with the major SGBs in Australia 84
3.4.4 Is a 'governing body' case an 'accepted category'? 84
3.4.5 Was the IRFB 'really' a governing body? 85
A What is a 'governing body'? 86
B A suit against individuals 86
C Absence of control 87
D Control over the game, its administration, rules and protocols 87
3.5 Sport cases that establish a duty of care in the SGB 88
3.5.1 *Watson v British Boxing Board of Control*: Express and implied obligations of care 88
A Assuming responsibility for medical care of boxers 89
B The fact of 'salient features' 91
3.5.2 *Green v Country Rugby Football League* 92
A Why did the CRL owe a general duty of care? 93
3.6 SGBs and athletes: A 'duty' relationship 93
3.6.1 A matrix of 'salient' facts 94
A *Agar* and *Watson*: A 'salient features' approach? 95
3.6.2 Control over the hazards of play 95
A Limitations on 'control'? 96
3.6.3 The base contractual means of 'control' 96
3.6.4 Contractual terms and general control 97
A An ongoing history of control over the game and its players 97
B Contractual terms of control 99
3.6.5 Reliance and expectations of care for player health and welfare 101
3.7 The vulnerable athlete: Reliance and control 103
3.8 Reliance, vulnerability and the violent nature of the sport itself 106
3.8.1 The examples of *Watson* and *Green* 107
3.8.2 Vulnerability and reliance on the sole decider of policy 108
3.9 Foreseeability as a salient feature 109
3.9.1 Analogous cases 110
3.9.2 Using the examples above: Fact and foreseeability 114
3.9.3 CTE, foreseeability and a 'known uncertainty' 114
3.9.4 Foreseeability and access to knowledge of CTE 115

3.9.5 Foreseeability and the boxing studies 117
3.9.6 Precise risk of harm need not be foreseeable, but … 117
3.10 Questions of policy 119
3.10.1 Indeterminant liability 120
3.11 Conclusion 121
Chapter 4: CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care 124
4.1 Introduction 124
4.1.1 This chapter proceeds as follows 126
4.2 The link between the SGB and medicos 128
4.2.1 Applying the 'medical model' as the standard of care 129
4.2.2 The SGB's obligations 132
4.2.3 Distinct duties to be informed and to warn 134
4.3 The law: Breach of duty 135
4.3.1 The statutory provisions 135
4.3.2 The pre-required conditions: 'Foreseeability' and 'not insignificant' 137
A 'Ought' the SGB have informed itself of the risk of CTE? 138
B 'Ought' the SGB to have known of a risk of playing if not warned? 139
C Was the risk of CTE 'not insignificant'? 140
4.3.3 The 'calculus of negligence': Would a reasonable SGB have taken precautions against the risk of CTE? 141
4.3.4 Accessing information on the risk of CTE 142
4.3.5 The probability that the harm would occur if care was not taken 142
4.3.6 The likely seriousness of the harm 144
4.3.7 The burden of taking precautions to avoid the risk of harm 145
4.3.8 The social utility of the activity that creates the risk of harm 146
4.3.9 The burden of taking precaution against similar risks 148
4.4 The content of the standard of care 149
4.4.1 Three principal cases establishing the standard of care 150
4.4.2 The standard of care: The obligation to be informed 151
4.4.3 Standard of care: Medical developments, judgment and proactive obligations 154
4.4.4 Standard of care: The obligation to inquire and discern 156
A The issue of 'non-peer reviewed' information 158
B Keeping informed — novelty and foreseeable complications 159
C Practical limits 159

D The standard for delegation 160
E A proactive 'personal' duty to keep informed 160
F A positive obligation to search and consider? 161
G The obligation to inquire where potential harm is discerned 162
H Breach by failure to take steps to be informed 163
4.5 Rule changes and the standard of care: Safety measures — voluntary or duty? 163
4.5.1 Is there a legal obligation to change the rules of play to make a sport safer? 164
A The *Ipp Report*: Object and recommendations 164
B Precedent 164
C Obvious risk and assumption of risk 165
D Judicial recognition of athlete and spectator acceptance 165
E Autonomy and parliamentary interference 166
F Who is to decide what level of risk is acceptable? 166
G Consistency of mandated adjustments across all dangerous sports 167
H Legal tolerance of violent sport 168
4.5.2 The (collateral) rules that SGBs have volunteered to adjust 170
A A possible exception 174
4.5.3 Conclusion to this section 175
4.6 Conclusion 175
Chapter 5: Accessible Knowledge of CTE in Australia at Relevant Times 178
5.1 Introduction 178
5.1.1 This chapter proceeds as follows 180
5.2 The fundamental legal question: Available information indicating foreseeability 182
5.2.1 Drawing an inference 183
5.3 Martland's enduring warning of 'traumatic encephalitis' 183
5.4 Extrapolating from the boxing studies 185
5.5 Non-sport concussive trauma as a source of information on CTE 186
5.6 'Pre-Omalu' reports of CTE in sports *other* than boxing 188
5.7 The Australian studies relevant to knowledge of CTE prior to 2010 190
5.7.1 National Health and Medical Research Council (1994) 191
A Doubts expressed 191
5.7.2 Nathan Gibbs (1994) 192
5.7.3 Australian Law Reform Commission (2003) 192
5.7.4 The combined significance of the Australian studies: Are they sufficient on their own? 193
5.8 Extrapolating from American football 194

5.9 Conclusion 195
Chapter 6: Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of 'Individual Agency' 198
6.1 Introduction 198
6.1.1 This chapter proceeds as follows 200
6.2 Of law and fact: Two broad problems of proof 203
6.2.1 Doubts expressed as to CTE as a 'real' disease 203
6.3 At what stage is CTE compensable at law? 205
6.4 Diagnosis of CTE post-mortem and *in vivo* 207
6.5 The standard of proof and inference of CTE *in vivo* 208
6.5.1 Drawing an inference 210
6.5.2 Inference through multiple facts 211
6.6 CTE as a unique disease: Fact and inference 212
6.6.1 Diagnosis and inference of CTE *in vivo*: Proof of affliction through multiple indicators of harm 214
6.7 Traumatic encephalopathy syndrome (TES) and inference of harm in the form of CTE 215
6.7.1 The NINDS TES criteria 217
A The primary criteria 217
B The supportive criteria 219
6.7.2 Provisional levels of certainty for CTE pathology 220
A Suggestive of CTE 220
B Possible CTE 220
C Probable CTE 220
D Definite CTE with TES 220
6.7.3 A TES case study example 220
6.7.4 TES and the question of *in vivo* diagnosis of CTE 221
6.7.5 An Australian application of the NINDS TES criteria 222
6.7.6 TES diagnosis and treatment as a fact in inferring contraction of CTE 222
6.8 Other methodologies in diagnosing CTE *in vivo* 224
6.8.1 Pierre et al: The spectrum of indicators and inference of harm in the form of CTE 224
6.8.2 Clinicopathological evaluation and inference of harm in the form of CTE 225
6.8.3 Turk: Cerebrospinal fluid as biomarker and inference of harm in the form of CTE 226
6.9 Two notable core indicia of CTE 226
6.9.1 Athlete history of RHT: CTE 226
6.10 The Bradford Hill criteria 229

6.11 Application: Multiple *in vivo* indicators to infer CTE 230
6.11.1 Application: Points of consideration 231
6.11.2 Analytical illustration: Inference of harm, the Danny Frawley case in proxy 232
A Provisional certainty for CTE pathology 234
6.12 Conclusion 236
Chapter 7: Factual Causation of Harm: Failure to Warn and the Athlete's Response Had a Warning Been Given 239
7.1 Introduction 239
7.1.1 This chapter proceeds as follows 240
7.2 The law and the athlete's response to warnings 241
7.3 The materiality of CTE as a subject of warning 243
7.4 Context: Head trauma yet to be experienced 244
7.5 Means of inferring an athlete's response 246
7.5.1 The circumstances of the 'reasonable' person 246
7.5.2 Inferring the athlete's response through objective indicators 248
7.5.3 The question of credit 249
7.6 Further indicators from which an inference may be formed 249
7.6.1 A 'reluctance to report' 250
7.6.2 Responses of those experiencing multiple incidents of head trauma 252
7.7 'Running the risk' or not? 254
7.8 Conclusion 256
Chapter 8: The Question of 'Generic Capacity': Does the SGB's Contact Sport Cause CTE? 258
8.1 Introduction 258
8.1.1 This chapter proceeds as follows 260
8.2 Failure to warn and the 'but for' test of causation (the generic capacity to cause harm) 261
8.2.1 Proving generic capacity: The 'but for' test of harm 262
8.2.2 The equivalence of 'but for': 'Necessary condition' 263
8.2.3 Generic capacity to cause harm is foundational to failure to warn of CTE 263
8.2.4 The High Court's use of the 'but for' test in failure to warn cases 264
8.3 The implications should causation of physical harm *not* have to be proven in a failure to warn case 267
8.4 Multiple causes of CTE and 'material contribution' to harm 268
8.5 Conclusion 270
Chapter 9: CTE and the Characterisation of 'Risk of Harm': Dangerous Recreational Activity, Assumption of Risk and Other Defences 271
9.1 Introduction 271

9.1.1 This chapter proceeds as follows 272
9.2 Legislative provisions: Obvious risk, dangerous recreational activity and assumption of risk of harm 274
9.3 An emphasis on 'obvious risk' 278
9.4 Fundamental questions in contention 278
9.5 Dating when the risk of CTE became obvious 279
9.6 Defining harm: General and specific applications 281
9.6.1 CTE: A general or specific characterisation of risk? 282
9.6.2 Obvious risk: Conduct and harm 283
A The relevance of the statutory 'general principles' establishing breach 288
B Borrowing from the medical cases 289
9.6.3 A methodology to characterise the 'risk of harm' 291
9.6.4 Characterising CTE as the risk of harm: How general is too general? 294
9.6.5 CTE: The necessity for specification 295
A Obvious risk and breach to be characterised at the same level of generality 296
B Obvious risk and causal mechanism and the level of generality 297
C The subject matter of the failure to warn of CTE 297
D Causal connection between harm and risk 297
E Specifying the causal mechanism 298
F The harm for which the plaintiff seeks damages 298
G In summary 301
9.6.6 The inefficacy of concussion as a descriptor of the risk of harm 301
9.6.7 The bind: Foreseeable risk of harm and obvious risk 303
9.7 Obvious to a reasonable person in the position of that person (the victim) 304
9.7.1 The voluntary element of assumption of risk 307
9.7.2 Discerning an assumption of risk 308
9.8 CTE as a *not* 'obvious risk of harm' to a reasonable premier-level contact sport athlete 309
9.8.1 Subjective perception of the obvious risk 309
9.8.2 Awareness of the type or kind of risk is sufficient 309
9.8.3 Full appreciation of the danger 310
9.8.4 Freely and voluntarily agreed to accept the risk 311
9.9 Contributory negligence 311
9.9.1 What the plaintiff athlete ought to have known 313
9.10 Conclusion 315
Chapter 10: Conclusion 317

10.1 Conclusion to thesis 317
10.2 A review of key arguments and findings 317
10.3 Recommendations 323
10.3.1 Evaluation of existing measures 324
10.3.2 The unavoidability of head trauma in contact sport 324
10.3.3 The ongoing obligation to be informed and to warn 325
10.3.4 Changing the collateral rules 325
10.3.5 Forced retirement at the threshold of CTE-like harm 326
10.3.6 Further legal and medical research 329
10.4 Concluding remarks 332
Bibliography 335
A Articles/Books 335
B Cases 346
C Legislation 349
D Media/Other 349
Appendix 1: The Science of Concussion and CTE in Sport 352
1.1 Introduction 352
1.1.1 This appendix proceeds as follows 352
1.2 The US NFL and NHL litigations 353
1.3 Financial arrangements between SGBs and district clubs 356
1.4 Tau accumulation and contact sport 358
1.5 Contact sport and the bio-mechanism of damage 362
1.5.1 Damage to axons 364
1.6 The progression of CTE 364
1.6.1 The onset of CTE 364
1.6.2 The stages of CTE 365
A Stage I CTE 366
B Stage II CTE 367
C Stage III CTE 367
D Stage IV CTE 367
1.6.3 Death and CTE: Footballers and boxers 368
1.7 Second impact syndrome 369
1.8 Conclusion 372
Appendix 2: CTE and 'Negativing Influences': Do They Determine Liability? 373
2.1 Introduction 373

2.1.1 This appendix proceeds as follows 373
2.2 CTE 'denialism' 374
2.3 The historical record countering an overreaction to doubt 376
2.4 The CISG Consensus Statements 376
2.4.1 What Consensus Statements were published within the critical time frame? 377
2.4.2 As against the historical record, are the Consensus Statements truly influential in law? 377
2.4.3 The Consensus Statements' use of qualifying words 378
2.4.4 Is there really disagreement? 378
2.5 McCrory: Expressing doubt as to CTE 382
2.5.1 Are McCrory's comments sufficiently substantive to dismiss CTE a 'real risk' and excuse an SGB's failure to be informed and to warn? 383
2.5.2 What did McCrory mean by 'neuromythology'? 385
2.6 The 'medicalisation' of CTE: A sociological view 386
2.6.1 To what extent can medicalisation be taken to have contributed to the 'friction' between the relevant groups of medicos? 389
2.6.2 Can 'medicalisation' apply to the disease of CTE — a real medical problem? 390
2.6.3 'Conflict' regarding the 'cause-and-effect' of CTE — is there an obligation to warn? 391
2.6.4 A 'scientific application' 394
2.7 Conclusion 394
Appendix 3: A Brief Consideration of the Scope of Liability 396
3.1 Introduction 396
3.1.1 This appendix proceeds as follows 396
3.1.2 Outline of 'scope of liability' 397
3.1.3 The relevance of the common law 398
3.2 The commonality of foreseeability in duty and scope of liability 398
3.2.1 The rationale of 'scope of liability' 399
3.3 The content of the scope of liability 399
3.3.1 Common sense and value judgments 400
3.3.2 Remoteness and reasonable foreseeability of harm 401
A Foreseeability of harm as a 'type' or 'class' 401
B The advantage of foreseeability by 'type' of harm for plaintiff athletes 402
C Can CTE be incorporated into a 'type'? 403
D Why foreseeability of CTE as a 'type' of cognitive harm/ concussion is unlikely 405
E CTE as a foreseeable risk of harm 405
F Notification and foreseeability 406

3.3.3 Other considerations 406
A The scope of the risk of harm protected by the duty and created by the breach was foreseeable 407
B The injury was not coincidental to the breach but the direct and immediate consequence of the negligence 407
C There was no intervening act or abnormal event 407
D Harm was from the same cause 407
3.4 An established class and the question of 'why?' 407
3.5 Genetic predisposition and the 'egg-shell skull' rule 409
3.5.1 On genetic risk and CTE 409
3.5.2 On steroid use and CTE 410
3.6 Conclusion 411
Appendix 4: Factual Means of Assessing Generic Capacity to Cause CTE 413
4.1 Introduction 413
4.1.1 Two interrelated questions 414
4.1.2 This appendix proceeds as follows 414
4.2 The general body of medical knowledge 415
4.2.1 Causal connection and a known risk 416
4.2.2 Cases of similar application 416
4.2.3 Inference and expert evidence 417
4.3 The Bradford Hill criteria: Determining the cause of CTE 418
4.4 Nowinski's application of the Bradford Hill criteria to CTE 420
4.4.1 The criteria 420
A Criterion 1: Strength of association 420
B Criterion 2: Consistency 421
C Criterion 3: Specificity 422
D Criterion 4: Temporality 422
E Criterion 5: Biological gradient 423
F Criterion 6: Plausibility 423
G Criterion 7: Coherence 424
H Criterion 8: Experiment 424
I Criterion 9: Analogy 425
4.4.2 Nowinski's conclusions 425
4.4.3 Is the Nowinski study factually and legally acceptable? 427
4.5 Other means of proving generic capacity 431

4.6 Conclusion 432
Appendix 5: Multiple Causes of CTE and 'Material Contribution' to Harm 434
5.1 Introduction 434
5.1.1 This appendix proceeds as follows 435
5.2 The concept and operation of 'material contribution' in law 437
5.3 'Material contribution' and the joinder of other sporting organisations 440
5.3.1 Possible combinations 440
5.4 The fact of multiple tortfeasors and cumulative head trauma 441
5.4.1 The scientific fact of head trauma in junior and senior contact sport 441
5.5 Evaluation of 'material contribution' 447
5.6 Multiple tortfeasors and the 'exceptional case' 449
5.6.1 The statutory approach to causation as an 'exceptional case' 449
5.6.2 The advantage of an 'exceptional case' 451
5.6.3 The application of the exceptional case 451
5.6.4 Can the 'exceptional case' apply to multiple contributors to CTE? 452
A The classic exceptional case 453
5.7 Conclusion 456

Chapter 1:
Introduction

1.1 Introduction

In January 2017, the US National Football League (NFL) settled a class action in negligence brought by ex-NFL players suffering from chronic traumatic encephalopathy (CTE) and ex-players fearful of developing CTE. The settlement, at finality, approached US$1 billion, and the settlement recipients are estimated to number as many as 20,000 ex-players and relatives of deceased ex-players.[1] In 2018, the National Hockey League (NHL) in North America settled a class action in negligence with 318 ice hockey players concerned with their exposure to CTE.[2] Both cases were settled by these sporting organisations leaving questions of policy, law and liability unresolved in respect of the sufferers of CTE in the United States and more broadly in other common law countries.

Media outlets report hundreds of Australian athletes as suffering symptoms of CTE and several have been confirmed at autopsy as afflicted with the disease. Although not scientific studies many are affirmed by treating medical practitioners. Most sufferers are in their 40s, 50s, and 60s some — disturbingly — are in their 20s and 30s. As CTE causes the loss of cognitive function years prior to death, a sufferer will face a significant period of disability and dependence on others, with consequent financial and personal costs.

This thesis considers the question of the liability in negligence of the sports governing bodies (SGBs) of Australian contact sports where the athletes who played in their premier competitions, perhaps years after ceasing to play, have displayed the symptoms of CTE.

Research and discussion are directed mainly to the contact or collision sports of rugby league, rugby union, soccer (football) and Australian football.[3] In addition, semi-contact sports such as netball, cricket and equestrian sports are also considered. What these sports and many others have in common is repetitive head trauma (RHT), the cause of CTE.

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1 *In Re National Football League Players' Concussion Injury Litigation*, No 2:12-md-02323-AB, MDL No 2323, Plaintiffs' Amended Master Administrative Long-Form Complaint, [6]–[7]–[8]–[9]; District Court of the Eastern District of Pennsylvania.

2 Settlement Agreement between the NHL and Plaintiffs' Counsel (NHL/NHL Retired Player Settlement Agreement), 5 November 2018.

3 From here the wording 'contact sport' will be used to encompass both contact and collision sports, except where reference to 'collision sport' is specifically required. 'Collision sports' are those involving heavy contact person to person or object to person, such as rugby, AFL, boxing and football (soccer) and any other sport where players make hard contact with each other. At times it may also encompass sports such as cricket or baseball, should a ball strike the head of a batter.

1.1.2 Development of the thesis

Two things strike the researcher who begins an examination of negligence in law associated with CTE: the vast quantity of factual material, and the realisation that the application of the various elements and defences in the law of negligence in this context is far from settled. There is no 'free ride' — there is no mere adaptation of previous writings and judgments.

This thesis began as a research article exploring the emerging issue of CTE in contact sport. It was soon apparent that the ground to be covered was vast, and the legal issues complex and intricate. A thesis seemed the best method of examination. And yet, again, the number of words available to explore the interrelationship of fact and law meant that a doctoral thesis, even with its relatively generous length of text, cannot hope to cover each and every issue relevant to CTE. Where a legal issue associated with a particular fact is thought to be relatively straightforward, a less comprehensive approach is taken to afford greater analysis of more difficult areas of fact and law.

1.2 Research objectives and the thesis argument

The object of this thesis is to examine the proposition that the governing bodies of contact sport in Australia owed a duty of care to the athletes who played in their premier-level competitions, which was breached by a failure to take reasonable steps to be informed of the risk of CTE and to warn those athletes of that risk. The thrust of argument is directed towards the affirmation of that proposition.

1.2.1 The length of this thesis

It is not intended to consider all matters that touch upon the tort of negligence in respect to CTE and the potential liability of SGBs. The thesis is not the presentation of a 'case in negligence'; rather, it seeks to elucidate the law and facts that are not only the key elements of an athlete's claim in negligence but also the most complex and difficult to resolve from a plaintiff's perspective.

Supplementary information concerned primarily with medical and factual matters relevant to the thesis are provided in an appendix. It is hoped that these materials and any associated discussion will be available for those who wish greater clarification of matters and issues that, given limitations in available word length, are not included within the body of the thesis and are considered collateral. Is the decision to provide a comprehensive or broad study of the topic of sport-related CTE unwarranted — to the contrary, new applications of law were explored, facts relevant to law and lawyers revealed and contextualised within a common profession.

1.3 The significance of this thesis topic

Contact sport at premier level has been played by thousands of Australians. These athletes have been exposed to the risk of contracting the debilitating brain disorder of CTE. They were not warned of the risk of CTE, and mitigating protocols were not introduced by the governing organisations of contact sport until *circa* 2011 — decades after scientific and medical research had revealed a risk of long-term cognitive harm in the playing of contact sport.

A factual base to the thesis question is revealed through a 2018 newspaper article regarding the development of CTE-like symptoms in ex-rugby league player Ian Roberts, who was then 52 years old:

> Ian Roberts is used to hard hits. He reckons he was knocked out or heavily concussed more than a dozen times during his 12-year rugby league career. He suffered his first concussion when he was about 13 years old. But the biggest blow came years after the league legend hung up his boots when he was told he had 'irreversible brain damage'. …
>
> 'People don't realise concussion is brain damage. It's literally causing damage to your brain,' Roberts said. …
>
> 'We all thought we were 25, 10-foot and bulletproof,' he said. 'Young people coming in to the sport need to be made aware of the long-term consequences [of concussion].'...
>
> [A study conducted by concussion expert Dr Alan Pearce at La Trobe University found] clear evidence that players with a history of concussion experienced significant and lingering deficits to their brain function.[4]

The emotional and financial costs of CTE are visited upon athletes, their families and friends, and the community. Given the increasing debilitative effects of CTE over a period of years, the expense of care may exceed that of an athlete made quadriplegic through a sporting accident — around $20 million over a lifetime. Of those who play contact sport in Australia, a significant percentage have developed and will develop the sequelae of CTE. Almost all will die of the disease or with the disease.

A mounting number of Australian contact sport athletes have been diagnosed at autopsy with CTE or are reported as showing *in vivo* symptoms of the disease. Some are famous; others are journeymen. Of the former, rugby league 'legend' Wally Lewis, 63 years old in 1923, 'revealed he has been diagnosed with the early signs of chronic traumatic encephalopathy'.[5] Furthermore, CTE is not confined to male athletes. Australian Football League (AFL) Women's player Jacinda Barclay took her own life in 2021 at the age of 29, an autopsy

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4 K Aubusson, 'The Effects of Concussion Linger Decades after NRL Stars Hang Up their Boots', *Sydney Morning Herald* (Sydney, 7 February 2018).

5 'Rugby League Legend Wally Lewis Diagnosed with Early Signs of Brain Disease CTE', *The Australian* (online, 31 July 2023).

revealing ‘degradation to her cerebral white matter’ that is believed to be associated with CTE.[6] An Australia study by Suter found at autopsy that six out of twelve donors to the Australian Sports Brain Bank with CTE had died by suicide.[7]

That CTE is associated with head contact in sport not only presents the prospect of compensation in law but also threatens individual participation in contact sport and the financial viability of some of Australia’s major commercial enterprises. Unlike the United States, where a population of 350 million drives sport revenue into the billions of dollars, the earnings of Australian sport are relatively modest, suggesting an existential threat to contact sports and their associated industries should sport organisations, as a matter of law, be deemed liable. Despite the wide publicity of the threats of CTE-related legal action, questions of negligence associated with CTE are yet to be considered and applied. This represents a gap in our knowledge of the law of negligence as it applies to liability for the causation of CTE specifically, and more broadly to the scope of the legal obligations of sporting organisations to those who play under their authority.

1.4 Research methodology

The research methodology undertaken in this thesis is interdisciplinary, integrating legal analysis with scientific and medical evidence. At the most basic level, the thesis presents a unique set of facts to which the law has not yet been applied. The object of this thesis is to apply the most relevant elements of law to a novel cause of action, the failure of Australian contact sport SGBs to warn their premier players of the risk of CTE. Legislation and case law are construed, the legal operation is considered and the law applied to the case of SGB negligence.

1.4.1 Concerns of law and doctrine

Law is the framework that permits facts in contention to be analysed and judged. This thesis presents a unique range of facts to which the law has not yet been applied. To so apply, as in this thesis, requires the relevant law to be identified, analysed and applied. Analysis involves detailed consideration of the meaning of the law through the primary sources of legislation and case law, along with authoritative legal commentary.

Some sections of discussion involve detailed examination of the law and its analysis and application to a unique fact. In other sections, the law is settled and requires little analysis.

Applications of law directed to the unique question of CTE in this thesis include the following:

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[6] Stephanie Convery, ‘Damage Found after Late AFLW Player Jacinda Barclay’s Brain Donated for Concussion Research’, *The Guardian* (London, 30 April 2021). The death was described by police as ‘not suspicious’.

[7] CM Suter et al, ‘Chronic Traumatic Encephalopathy in Australia: The First Three Years of the Australian Sports Brain Bank’ (2022) 216 *Medical Journal of Australia* 530, 530.

- *Establishing duty of care using the 'salient features' approach*: The thesis engages in analysis by exploring the legal requirements for establishing a duty of care, particularly in novel cases where no established precedent exists. It involves a close analysis of facts and circumstances relevant to the relationship between the plaintiff and the defendant. The thesis considers 'salient features' such as 'control', 'reliance' and 'vulnerability' to determine whether an SGB owes a duty of care to its premier-level athletes.

- *Evaluating the standard of care*: The thesis addresses what a reasonable SGB would do in response to the risk of CTE, and considers the 'calculus of negligence' as indicated in civil liability legislation.

- *Analysing civil liability legislation*: The thesis interprets and applies relevant sections of civil liability legislation to a context applicable to CTE cases. It examines provisions related to negligence, causation and the scope of liability to determine how they apply to claims against SGBs for failure to warn.

- *Foreseeability and knowledge*: The thesis considers a core legal principle: that an SGB can be liable in negligence if it 'knew or ought to have known' about the risk of CTE before it became obvious to players.

- *The obligation to be informed*: The thesis looks at legal principles associated with the base obligation of SGBs to be informed of the scientific knowledge of the day about the risk of injury to premier players.

- *Legal standards of causation*: The thesis analyses the legal standards of causation, including the 'but for' test and its statutory equivalents ('necessary condition' or 'factual causation'). It examines how these tests are applied in cases where there are multiple potential causes of harm, and considers the concept of 'material contribution'.

- *Generic causation and High Court precedent*: The thesis examines the unique utilisation of the 'but for' test in legislative form as a method to approach 'actual harm' in a long latency case. It considers the application of the Bradford Hill criteria as a matter of law to generic capacity, as well as other relevant methodologies.

- *Role and interpretation of case law*: The thesis analyses and synthesises relevant case law to extract legal principles and precedents. For example, it discusses cases such as *Wallace v Kam*, *Rogers v Whitaker* and *Chappel v Hart* in discerning the application of legal principles related to causation and the admissibility of evidence.

- *Consideration of 'material contribution'*: The thesis considers circumstances where the plaintiff has been exposed to head trauma from multiple sources, and therefore the aetiology of CTE is not fully agreed on; and considers whether this exception to the 'but for' test applies.

- *Defence of obvious risk*: The thesis considers how state civil liability legislation treats obvious risks in the context of dangerous recreational activities, and analyses whether SGBs can rely on this defence to avoid liability for CTE-related claims.

1.4.2 Concerns of fact

The application of law to fact is how a case of negligence is determined and unavoidably directs much of the discussion of the chapters of this thesis. As said in the High Court:

> [A]ll cases generally consist more of matters of fact, than of law …[8]
>
> If the notions of meaning and construction are independent [of law] … then it is difficult to see how meaning is a question of law …[9]

The law is applied to facts. In this thesis, the facts most material are questions of science and medicine. The thesis is primarily concerned with whether an SGB was negligent in failing to warn its premier-level athletes of the risk of CTE. The law is noted, construed and applied to the facts relevant to: when CTE was discovered, how it is caused, is it connected to contact sport, is it a material risk of contact sport, could Australian SGBs know of the risk, when was the risk obvious to premier-level athletes, among many other questions.

The application of law to fact is, by the very nature of the subject under consideration, an essential and unavoidable component of this thesis. Consider the following in brief compass:

- *Duty of care*: Each element of the law must be proven by factual evidence. For example, in establishing a novel case through a 'salient features' approach, the thesis highlights 'control', 'reliance' and 'vulnerability'. Several facts are relied on to prove these features, including the relationship between the SGB and its players, and indications of contractual control over aspects of the players' professional, medical and private lives.

- *Foreseeability*: Foreseeability of harm through a failure to warn of the risk of CTE is a question of fact, discerned through the question whether the defendant SGB 'ought' to have known of the risks associated with CTE in playing contact sport through its associated medicos and other scientific sources.

- *Breach of duty*: Falling below the standard of care is a question of fact. Did the SGB take reasonable steps to inform itself of the risk of CTE? Were reasonable steps taken to warn players of the risk of CTE? Both are questions of fact.

- *Factual causation*: Proving 'factual causation' — how an athlete would have responded if warned — is a question of fact that is discerned through inference based on

[8] *State Rail Authority of NSW v Earthline Constructions Pty Ltd* (1999) 160 ALR 588, 72, quoting *R v Earl of Banbury* (1694) Skinner 517, 532.

[9] *Collector of Customs v Possolanic* (1993) 43 FCR 280, 3967.

considerations such as reasonableness and credibility: 'but for' the failure to warn, the plaintiff athlete would not have played in the SGB's competition and sustained CTE.

- *Proof of 'generic causation'*: This is a question of fact proven by the presentation of medical and scientific research showing that RHT in the playing of a defendant SGB's sport has the capacity to cause CTE.
- *Proof of 'individual agency'*: Whether a person suffers personal injury is a question of fact. CTE is proven by autopsy, or traumatic encephalopathy syndrome, or several possible medical methodologies referencing a plaintiff athlete's symptoms, sequelae and other manifestations.
- *Access to information*: Whether information on the risk of CTE through RHT in the playing of contact sport was accessible to SGBs in Australia before or during the time a plaintiff athlete competed in their competitions is a question of fact. This is proven through the presentation of evidence in scientific and medical journal accounts, through conveyance by an SGB's medical associates or, at times, through personal experience and judgment.
- *Material contribution*: Whether an SGB made a material contribution to a plaintiff athlete's contraction of CTE is a question determined by factual evidence.
- *Characterisation of CTE as an obvious risk*: Whether CTE is characterised broadly or narrowly for the purposes of 'obvious risk' in playing contact sport is a question of fact.

1.5 Rationales to the approaches taken in this thesis

1.5.1 Which party in litigation?

An athlete struck down with CTE in, say, middle or late middle age, will have experienced symptomatic and asymptomatic head trauma from his or her first junior game of contact sport until retirement from premier competition years or decades later.[10] As a plaintiff must be able to show a 'foundational duty of care in the circumstances existing between the [defendants] and the plaintiffs',[11] who, then, is a premier-level athlete suffering from CTE to sue for the negligent failure to care for his or her well-being? Is it the individuals who harmed the athlete during play, the junior club that first exposed him or her to risk of harm, the athlete's senior club, or the governing organisation of the sport? There is little scope to sue the many opponents who, over an entire career, may have inflicted concussive or sub-concussive head trauma on an individual athlete. Even where offending opponents can be identified, the plaintiff athlete

[10] This is discussed fully in Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"'.

[11] *Hyde v Agar* (1998) 45 NSWLR 487, 500 (Court of Appeal).

must prove in each case that contact was negligent rather than an ordinary and objectively accepted incident of the sport.[12]

While the entity most directly responsible for a premier-level professional athlete's well-being is his or her employer club, the district clubs in Australia (as discussed above) are often not profitable, or are only marginally so, and rely on subsidies from their SGB or their sponsors, or through government grants, in the absence of which they would be impecunious. Although all tortfeasors are jointly and severally liable, as the main purpose of a negligence suit is to acquire damages to compensate for loss, the 'deep pockets' of the major sporting organisation offer the most practical — or indeed the only realistic — target of litigation. [13]

1.5.2 The sports where CTE is a risk of harm

Dr Ann McKee, neurologist and neuropathologist with the Center for the Study of Traumatic attached to the Boston University School of Medicine, published a report in 2009 with colleagues, implicating 'a wide variety of contact sports, including football, boxing, wrestling, rugby, hockey, lacrosse, soccer, and skiing', as well as 'karate, horse riding, and parachuting' in causing CTE.[14] McKee et al revisited the concern stating that although 'CTE has been primarily associated with boxing, CTE may also occur as a consequence of American football, hockey, wrestling, rugby and exposure to blast or concussive injury associated with military service'.[15]

What these sports share is RHT. As such, in this thesis reference a relevant sport is one sport that has been reported in medical scholarship as a cause or potential cause of CTE through repetitive concussive and sub-concussive head trauma. At times, rather than identifying a sport as the cause of CTE, discussion will centre on the mechanism by which CTE is caused, such as tackling, being tackled, heading the ball or being punched. Again, it is the 'high prevalence of CTE among former contact sports athletes with a history of [RHT], particularly in high-intensity sports and elite- level competition', that is relevant.[16]

Nonetheless, as this thesis is concerned with claims of negligence against Australian SBGs, the types or categories of sport discussed tends towards contact sports common to Australia. These, predominantly, are Rugby League, Rugby and AFL. Football (soccer) also features in

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[12] *Hilton v Wallace* [1989] ATortsRep 68,488.

[13] *Sienkiewicz v Grief* [2011] 2 AC 229, [141]–[142]. See further Appendix 5: 'Multiple Causes of CTE and "Material Contribution" to Harm'.

[14] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 709. Of passing interest, *Time Magazine* in 2018 named McKee as one of the 100 most influential people. The recognition 'spotlights 100 rising stars who are shaping the future of business, entertainment, sports, politics, science, health and more'.

[15] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43.

[16] Bote Qi et al, 'Prevalence of Chronic Traumatic Encephalopathy in Athletes With Repetitive Head Impacts: A Systematic Review and Meta-Analysis' (2025) 35 *Scandinavian Journal of Medicine & Science in Sports* 11.

discussion because, while not a collision sport, repetitive heading of the soccer ball introduces similar brain trauma to the tackling of an opponent in rugby. Other non-collisions sports, such as cricket and field hockey are noted occasionally.

1.5.3 Collision, contact and combat sport

In this thesis, to avoid repetition, the term 'contact sport' is used to refer to both collision sports and contact sports and, depending on context, combat sports.

A collision sport is, as the name implies, a sport where players collide with each other. Collision sports can be defined as a sport that permits deliberate collision between players, including tackling, body checking, or striking. The most common collision sports in Australia are AFL, rugby union, and rugby league. Collision sports also include American football (NFL) and ice hockey.

Combat sports are those where the object of the sport is to punch, kick or wrestle an opponent with the purpose of securing a win by knockout, or points earned for accuracy and strength of delivery.[17]

Contact sports are those where contact is made between players person to person or object (such as a ball) to person, which is often incidental to the sport. Examples, include football (soccer), basketball, netball, cricket, baseball.

1.5.4 Why the SGB is the 'defendant of choice'

Major sport in Australia is organised vertically or hierarchically, in what is sometimes called a 'pyramid' structure. An SGB — such as the National Rugby League (NRL), the AFL or Netball Australia — conducts a premier-level competition comprised of district or regional clubs and is usually responsible for the selection of international representative teams. Hallmarks of a premier-level governing organisation are the power to alter the rules and protocols of the game, and significant control of the game's revenue. As said by Healey,

> [t]he top tier professional sports such as the football codes (rugby, rugby league, Australian football and soccer (football)), cricket, golf, and tennis are organisers and managers and provide lucrative careers for their best participants. Their professional teams may be privately or community owned. Broadcast rights and sponsorships provide substantial funds to develop and organise all levels of these sports. In addition to their commercial arrangements, most of these top tier sports receive government funding for athlete development and to support community participation.[18]

An SGB may delegate or mandate duties, provide oversight, and support a secondary level of organisation at regional level or to conduct specific tournaments. For example, Netball

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[17] GF Symons et al, 'The Neurological Consequences of Engaging in Australian Collision Sports' (2020) 37 *Journal of Neurotrauma,* 792, 792.

[18] Deborah Healey, 'Governance in Sport: Outside the Box?' (2009) 23(3) *Economic and Labour Relations Review* 39, 40.

Australia 'provides support and service to netball participants and providers through membership [of] the association in each State/Territory'.[19]

The source of an SGB's power lies in its contractual dominance over the district clubs and the players that participate in its premier competition. In addition, as the national body controlling a major sport, the governing body negotiates media rights and is the prime financial beneficiary of those rights along with commercial sponsorship and endorsement. District clubs of a major sport in Australia are at best minimally profitable and may in fact experience a financial loss over the course of a season. For this reason an SGB is the 'defendant of choice' in matters where damages payments are likely to run into multiple millions of dollars. The financial arrangements between SGBs and district clubs is discussed in detail below.[20]

For purposes of clarity, the term 'sport governing body' is a convenient eponymous title. The analysis undertaken in this thesis does not depend on the use of the term 'sport governing body'. The material question is whether a particular organisation meets the factual basis that defines the organisation as one that governs a contact sport, and whether the elements that secure a claim of negligence operate to identify the relationship between plaintiff and defendant.

The arguments presented in this thesis reflect the complex contractual terms that permit an SGB to control the professional life and much of the off-field activities of a premier player. Further, the thesis asserts that a 'neighbour relationship' exists, whereby an SGB is duty-bound to care for the health and well-being of its premier players. Some sports, however, are not controlled by a sophisticated, complex and well-ordered governing body but, rather, are organised though an association of interested parties with limited resources and no expectation that emerging risks of harm will be sought out. Neither do these associations possess the means to direct medical personnel to monitor for developing risks of harm in their sports and will not necessarily make decisions with the health of the player in mind. Any claim that such associations are liable for harm caused in playing its sport is likely to be dismissed under the ruling in *Agar v Hyde*.[21]

A history of the reporting of CTE in medical journals dating back to 1928 implicating boxing — and from the 1970s implicating sports like rugby and football — as causative of CTE through RHT, is crucial to a plaintiff athlete's argument that the risk of CTE ought to have been known by an SGB. More recently, non-person-on-person sports, such as surfing and skiing, have reported cases of CTE.[22] Although head contact with the ground or objects is not

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[19] 'Member Organisations', *Netball Australia* (Web Page) <http://netball.com.au/about-netball-australia/member-organisations/>.

[20] See Appendix 1 at **[1.3]**: 'Financial arrangements between SGBs and district clubs'.

[21] *Agar v Hyde* (2000) 201 CLR 552. See the discussion in Chapter 3 at **[3.4]**: '*Agar v Hyde*: Distinguishing a sport case of "no duty"'.

[22] Maxwell N, et al, 'Ski racers' understanding of sports- related concussion and its management: are contemporary findings and clinical recommendations reaching the target audience, the racers themselves?' (2020) 54 (17) *Br J Sports Med*, 4.

uncommon and a known cause of concussion, skiing has not been associated with CTE in the pre-2010 era, nor at that time is there a history in legal or medical journals to support the argument that the risk of CTE ought to have been known by administrators. Harmon et al noted that in the United States, where most CTE research has been conducted, 'Injury surveillance systems … primarily study a small sample of organised college or high school sports to estimate concussion rates. Numbers are limited or not available for recreational or club sports or for activities such as bicycling, skiing, snowboarding, skateboarding, the fighting arts or for youth/early adolescent athletes.'[23] McKee reports analysing the brains of 'cognitively intact subjects without a history of brain injury' and found a skier negative for CTE.[24] Nor do there appear to be any autopsy studies reporting (p)tau accumulating in the brains of ex-skiers. These facts do not mean that individuals afflicted with CTE in playing sports that do not involve person-on-person violence, are necessarily without a claim of negligence against an organising body. It does mean, however, that the basis to such a claim will be formulated on different legal avenues and different factual circumstances to those presented in this thesis. Consideration of these sports is, therefore, outside the scope of this thesis.

1.5.5 Why 'premier-level' athletes of contact sports?

The object of this thesis is to examine the question of whether SGBs of major contact sport in Australia were negligent in failing to inform themselves of the risk of CTE and to warn their premier-level players of that risk. The period under consideration is that prior to *circa*-2010, a date argued in this thesis to approximate when CTE would have become an 'obvious risk' to premier athletes playing contact sport. An athlete will, all things being equal, be taken to have volunteered to accept obvious risks of harm.[25]

Deliberation directed to premier-level athletes raises the question of whether an SGB owed a duty of care to those who played its sport at a lower level; say, as a casual suburban club player or junior player. In addition to introducing an extra layer of complexity to a crowded thesis topic, there are good reasons of law and fact as to why premier-level contact sport athletes are considered rather than suburban or casual participants.[26]

Any notion that non-premier players (casual players) would come under an SGB's duty of care appears to be based solely on the element of foreseeability of harm; ignoring, however, the necessary element of neighbourhood or proximity, which requires that a plaintiff establish a relationship that extends beyond mere foreseeability of harm to include a 'neighbour

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[23] Harmon KG et al, American Medical Society for Sports Medicine position statement on concussion in sports, (2019) 53 (16) *Br J Sports Med*, 215.

[24] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43, 48.

[25] See, eg, *Roads and Traffic Authority of NSW v Dederer* (2007) 234 CLR 330; *Woods v Multi-Sport Holdings* (2002) 186 ALR 145.

[26] The reasoning guiding the selection of premier-level athletes is explained in detail in Chapter 3: 'A Duty Relationship Between an SGB and the Athletes Who Play Contact Sport'.

relationship'. In *Agar v Hyde*,[27] the High Court of Australia determined that an international body setting the rules of rugby did not owe a duty of care to two junior suburban players who suffered quadriplegia in separate scrum collapses. Gleeson CJ found that because the rules of the game expose players to the risk of harm, injury was foreseeable and, indeed, that members of the rule-making body 'ought to have [the plaintiffs] in contemplation'. Nonetheless, 'it does not follow that the members of the Board were under a legal duty of care to the players of the game'.[28]

In contrast, a viable case may be argued that the proximity of relationship between premier players and their SGB is sufficient to establish an obligation on the part of the SGB to care for the health and well-being of those players. The foundation of High Court's decision in *Agar v Hyde* was an absence of 'neighbourhood' and the 'indeterminacy of liability' that would attach to the rule-making body should a duty of care be established. The absence of neighbourhood, along with indeterminant liability, would likely work to exclude a duty attaching to an SGB with respect to suburban, casual players. Furthermore, owing a duty of care to all casual players of their sports would expose sporting organisations such as the AFL and NRL to potential claims numbering into the hundreds of thousands, and include many forms of athlete harm.[29]

In addition, several factors bearing upon the question of negligence are argued to apply to the relationship between an SGB and its premier players that are not indicated with respect to suburban or casual players. Most particularly, proximity is argued in this thesis to arise in part from a contractual relationship where, through express and implied terms, the SGB undertakes to care for the health and well-being of its premier players.[30] In contrast, SGBs in Australia have no contractual arrangement with suburban club players or those who play their sport on a casual basis. Having said that, most games in suburban competitions are played under the same general rules as those applying to the game at national level. Nonetheless, it is doubtful that a duty exists to mitigate harm through the modification of rules that all suburban players are taken to have willingly accepted.[31]

Further, as argued in this thesis, a claim made against an SGB by a premier player will be determined as a 'novel case' requiring consideration of various 'salient features' such as those

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[27] (2000) 201 CLR 552.

[28] Ibid [20]–[21].

[29] For example, according to the AFL, '[p]articipation in Australian Rules Football has exceeded more than 528,000 in 2024': 'Football Community Sets New Participation Record in 2024', *AFL News* (Web Page) <https://www.afl.com.au/news/1180673/football-community-sets-new-participation-record-in-2024>.This figure includes all forms of Australian football.

[30] This is discussed extensively in Chapter 3 at **[3.6.4]**: 'Contractual terms and general control'.

[31] See *Hilton v Wallace* [1989] ATortsRep 68,448: 'The [dangerous] contact could be regarded as being within the "spirit and intendment of the rules of the game" as they are applied in practice … [and of which] the plaintiff could be regarded as having consented to take the risk', quoting *Pallante v Stadiums Pty Ltd* [1976] VR 331, 339.

collated by Allsop P in *Caltex Refineries (Qld) Pty Ltd v Stavar*,[32] principally the salient features of 'control', 'vulnerability' and 'reliance'. The basis of a salient features approach has its foundation on the 'relationship between the plaintiff and the defendant'.[33] In considering the relevant salient features, there is no relationship reasonably justifying a casual athlete to realistically believe that the level of control exercised by the SGB over their well-being, or their vulnerability and their reliance on the actions of the SGB, is sufficient to establish a new category of duty.

Given, as argued, that casual or suburban club players lack proximity to an SGB sufficient to establish a duty of care, could it be said that a better description of the relevant duty of care is 'any athlete impacted by decisions of SGBs', with the qualifier that this category is 'highly likely to be professional/premier players'? As explained by Gleeson CJ in *Agar v Hyde* (above),[34] to be 'impacted' by a decision of an SGB is not to the point. To be 'impacted' or affected by a decision of a defendant does not describe the establishment of a duty of care, nor does it identify the risk of harm to which a duty attaches. Dating back to *Donoghue v Stevenson*, Lord Atkin pointing out that 'in English law there must be, and is, some general conception of relations giving rise to a duty of care' A neighbour, in the legal sense, is someone 'so closely and directly affected by my act that I ought reasonably to have them in contemplation'.[35] Without a close and direct relationship, the defendant 'is entitled to be as negligent as he pleases towards the whole world if he owes no duty to them'.[36]

Could a more general categorisation of duty be based on the capacity or 'any decision' made by an SGB that affects any athlete; for example, a suburban junior player? Such a class of potential plaintiffs is non-specific and, unless a particular plaintiff can point to a particular fact that places the athlete within the SGB's locus of proximity, a claim is likely to be stuck down under the rationale of *Agar v Hyde*. Could it be said that such a class of plaintiff would work to exclude non-premier players, given an absence of proximity? Such a description, while on some level perhaps useable, is a category based on a negative definition and fails to identify with precision those to whom a duty is owed. In terms of *Donoghue v Stevenson*, the classification of duty would not identify those who use the manufacturer's product, but would rather state that 'no duty is owed to those who do not use the manufacturer's product'. Moreover, there is no commonality of class between those who play in an SGB's professional premier competition and those who play in a competition organised by a district club or casual community group. Unlike the premier-level athlete, a casual player cannot point to the most

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[32] (2009) 75 NSWLR 649, [102].

[33] Ibid.

[34] (2000) 201 CLR 552.

[35] *Donoghue v Stevenson* [1932] AC 562, 580 (House of Lords).

[36] *Mallonland Pty Ltd v Advanta Seeds Pty Ltd* [2024] HCA 25, [29] (Gageler CJ, Gordon, Edelman, Steward, Gleeson, Jagot and Beech-Jones JJ), referencing *Sullivan v Moody* (2001) 207 CLR 562 at 576 [42] and quoting *Le Lievre v Gould* [1893] 1 QB 491, 497; *Vairy v Wyong Shire Council* (2005) 223 CLR 422, 431 [23].

basic identifier of 'neighbourhood' in a sport context: a contractual relationship with the SGB. In other words, as said by the High Court in *Sullivan v Moody*, 'different classes of case give rise to different problems in determining the existence and nature or scope, of a duty of care'.[37] Gummow J observed similarly in *Perre v Apand Pty Ltd* that 'in determining whether the relationship is so close that a duty of care arises, attention must be paid to the particular connections between the parties … There is no simple formula which can mask the necessity for examination of the particular facts.'[38]

1.5.6 Why evidence that contact sport can cause CTE and that an individual athlete is afflicted by CTE is necessary

The form of negligence considered in this thesis is the failure of an SGB to inform itself of the risk of CTE and to warn its premier players of that risk. That is, 'but for' the breach of these obligations, the athlete would not have contracted CTE. This is known as 'factual causation'. Two other forms of causation of medical-based harm must also be proven. These are: proof that playing the SGB's type of contact sport, or that RHT similar to that endured in playing the SGB's sport, can cause CTE (referred to as 'generic capacity'); and proof that the symptoms a plaintiff athlete displays are those of CTE (referred to as 'individual agency').[39]

Overcoming these evidential burdens is essential to a plaintiff athlete's case. It is not sufficient to establish merely a duty of care and breach of that duty. As said in the High Court, it is not the 'impairment of the … right to choose' or the 'exposure … to an undisclosed risk' for which compensation is awarded, but rather 'the occurrence and consequences of physical injury'.[40] In other words, an integral and necessary component of an athlete's case is that he or she prove affliction with CTE and, further, that the CTE was likely caused in playing the SGB's sport at premier level.

1.5.7 Pending and threatened litigation

CTE and concussion in sport are now well-publicised subjects of claims in negligence against major sporting organisations in Australia. In 2017 James McManus, who played for the Newcastle Knights in the NRL competition, sought a subpoena for documents pursuant to a statement of claim filed against the corporate wing of his club claiming that 'the company owed him a duty of care to exercise reasonable care to prevent him from suffering permanent brain damage as a result of head injuries sustained by him … when playing games of rugby league

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[37] *Sullivan v Moody* (2001) 207 CLR 562, [49].

[38] *Perre v Apand Ltd* (1999) 198 CLR 180, [198]–[199].

[39] See Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"'; Chapter 8: 'The Question of "Generic Capacity": Does the SGB's Contact Sport Cause CTE?'

[40] *Wallace v Kam* (2013) 250 CLR 375, [9].

and while training'.[41] McManus listed a series of concussions and head injuries suffered by him during his career with the Knights and requested the subpoena for the 'legitimate forensic purpose' of establishing that 'his position is not unique';[42] that is, that other players had also suffered such effects of negligence by the Knights organisation. In 2019, several ex-players of the AFL were reported as 'threatening to sue the AFL for memory loss and other cognitive issues they say stem from on-field concussion', claiming that

> the AFL breached its duty of care by allowing players to routinely return to the field on the same day after suffering a serious hit, and almost always play again the week after such hits, leading to the devastating maladies they now endure.[43]

The threat became reality in 2023 when a representative action (class action) was filed by retired AFL player Max Rooke in the Supreme Court of Victoria against the AFL.[44] The 'Group Proceeding Summary Statement' recorded that '[t]he lead plaintiff brings the claim on his own behalf and on behalf of other claimants who have claims against the defendant arising from similar or related circumstances'.[45] In the writ, the group plaintiffs 'were listed players (Players) with affiliated AFL Clubs subject to and bound by the rules and regulations of the AFL as determined by the defendant', and 'suffered concussion in the course of training for or playing an AFL game in the AFL Competition'.[46] The writ in part contended that the AFL was negligent as it 'knew or ought to have known' of the 'potential long-term consequences of concussion suffered by AFL Players while training for and playing AFL football', failed to make policies that 'reduce the incidence of concussion' and failed to inform players 'regarding the symptoms and risks of long-term injury arising from concussion'.[47]

Similar legal proceedings are reportedly being brought by rugby players in the United Kingdom. Steve Thompson, a 45-year-old ex-England rugby representative who was reportedly 'diagnosed with early onset dementia' in 2020, is claiming that 'the governing bodies have failed in their duty of care, having not acted upon the known risks of head injury,

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41 *McManus v Knights Rugby League Pty Ltd* [2017] NSWSC 1101, [3]. The case did not proceed to trial, and although the terms of the settlement were not released, media sources reported that the case was settled in the Knights' favour. Nonetheless, the claims remain relevant and are common to other CTE cases.

42 Ibid [16].

43 Konrad Marshal, 'The Concussion Class Action that Could Punch a Hole in the AFL', *The Age* (online, 16 March 2019) <https://www.theage.com.au/sport/afl/the-concussion-class-action-that-could-punch-a-hole-in-the-afl-20190311-p513ay.htm>.

44 *Rooke v Australian Football League* (Supreme Court of Victoria, S ECI 2023 00969, commenced 14 March 2023) Writ.

45 *Rooke v Australian Football League* (Supreme Court of Victoria, S ECI 2023 00969, commenced 14 March 2023) Group Proceeding Summary Statement, cl 2.

46 *Rooke v Australian Football League* (Supreme Court of Victoria, S ECI 2023 00969, commenced 14 March 2023) Writ, cl 1.

47 Ibid cll 10, 15.

particularly after professionalism led to a clear intensification in the sport's dynamics.'[48] Thompson is joined by more than 180 other British rugby players in their suit against World Rugby, the Rugby Football Union and the Welsh Rugby Union.[49]

1.5.8 Time periods to provide a structure

Reports of CTE-like harm from the late 1920s until the present date number in the hundreds. At what point after the 1920s can it be said that an SGB ought to have known of the risk of harm in the form of CTE? Three landmark studies emanating from the United States are commonly cited in medical journal reports as drawing a link between contact sport and CTE. These studies provide a structure in time by which to consider the question of available knowledge and a concomitant obligation to warn. These are the 1928 article by Dr Harrison Martland on 'punch drunk syndrome' (CTE) in boxing;[50] the 2005 report of Dr Bennet Omalu et al after Omalu autopsied a retired American footballer and first identified CTE histologically;[51] and the 2009 study of Dr Ann McKee et al confirming at autopsy CTE in athletes who participated in a range of contact sports.[52]

What links these three studies together? The 1928 report is confined to boxing but nonetheless reveals a common link to other sports in RHT. The 2005 report concerned American football, a different game to the common contact sports played in Australia, but again similarities in heavy contact and RHT suggest a uniformity of medical cause. The 2009 study linked many contact sports played in Australia with the risk of CTE.

These reports consider the medical issue of CTE in depth and were published in well regraded medical journals thereby providing a high level of authority. The recognition of CTE as a disease identified by deposits of the protein phosphorated tau in the depths of the sulci of the brain in Omalu et al's report gave a concrete histological explanation of medical cause moving the question beyond that of conjecture.

Two further time periods by which the risk of CTE was arguably revealed and should, thereby have been acted upon by Australian SGBs. From the 1970s on, CTE was increasingly reported as a risk across a range of contact sports or compared to the risk of CTE in boxing. In the mid-1990s two reports suggested a link between RHT in the contact sports of rugby league

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[48] Michael Alwyn and Andy Bull, 'Rugby World Cup Winner Steve Thompson Reveals He Has Dementia and Joins Landmark Legal Case', *The Guardian* (London, 8 December 2020). The article adds that Thompson's legal counsel 'claims there is a "ticking timebomb" of players who are developing symptoms as they reach their 40s and 50s'.

[49] 'Dementia and Sport: Rugby Players Launch Legal Action against Governing Bodies', *BBC Sport*, *BBC* (online, 25 July 2022) <https://www.bbc.com/sport/rugby-union/62290045>.

[50] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

[51] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[52] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

and Australian football (AFL). In 1994 Dr Nathan Gibbs, ex-rugby league player and sports physician, suggested that CTE might be associated with head trauma in rugby league.[53] A report by the National Health and Medical Research Council suggested some evidence of 'long term problems' caused by a 'single head injury' in contact sport and noted possible cumulative effects.[54] The relevance of these reports is discussed extensively below.

These Australian medical reports, while suggestive of CTE as a possible consequence of playing contact sport, are unlikely to have provided sufficient factual or medical detail to oblige an SGB to warn of the risk of CTE. The value of these reports lies, however, in alerting the governing bodies of contact sport to a possible risk of CTE from which further inquiry and research would reasonably ensue. Further inquiry would inevitably take the researcher to the hundreds of articles in medical journals documenting the risk of CTE across multiple sports; initially boxing and then contact sports where concussion and head trauma are common. And while some sports, such as American football, differ from the common Australian contact sports such as AFL, rugby and soccer, what is shared in common, and what each medico and sports official should be aware of, is concussion and RHT. In some instances, the common cause of CTE is described simply as RHT, by being 'bumped' in AFL, tackled in rugby and heading the ball in soccer. At other times, as between, say, American football and rugby, the act in common is more specific, such as tackling and being tackled.

1.5.9 Why players of an era prior to *circa* 2010?

At some point after the publication in 2005 of Dr Omalu et al's report of an American footballer afflicted with CTE, or after Dr McKee et al's 2009 study revealing a risk of CTE across several contact sports, the argument that CTE is an 'obvious risk' of a 'dangerous recreational activity' will be accepted by a court and liability will not attach to an SGB irrespective of negligence.[55] An obvious risk is one that would be obvious to a reasonable person in the position of the injured party. Hence, a risk of harm that may be obvious to a defendant need not be an obvious risk to a plaintiff.

It is a contention of this thesis that from *circa* 2010 the risk of CTE is likely to be deemed an 'obvious risk'. The year 2010 is a convenient approximation for purposes of discussion and certainly the argument could be made that the risk of CTE was obvious in 2005, or 2009, or perhaps a few years later. What is important is that at some point in time CTE will have been declared an obvious risk. Beyond these putative times, litigation for failure to know and to warn is not likely to be successful and claims by a CTE sufferer against sporting organisations will turn to an argument that the sporting organisation failed to enforce concussion ameliorating protocols.

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[53] Nathan Gibbs, 'Common Rugby League Injuries' (1994) 18(6) *Sports Medicine* 438, 445.

[54] National Health and Medical Research Council (NHMRC), *Football Injuries of the Head and Neck* (1994) 18, 19.

[55] This is a different outcome from that which would ensue should an SGB indicate that it will initiate certain concussion mitigation protocols and fail to do so.

The significance of the period *circa* 2010 was recognised by the AFL inquiry into accusations of plagiarism by neurologist Dr McCrory, where it stated that 'during the period 2011 to 2021 … there has been extremely rapid escalation in stakeholder and public scrutiny of, and interest in, the incidence, prevention and appropriate management of concussion in contact sports' and that 'over that same period, the understanding of the relationship between head injuries and neurodegenerative diseases, including chronic traumatic encephalopathy, has significantly evolved'.[56]

1.5.10 Why breach of a duty by failure to 'be informed' and 'to warn' of the risk of CTE?

This thesis is concerned primarily with the duty of an SGB to know of the risk of CTE and to warn its premier players of that risk. Axiomatically, one cannot warn of a risk of harm unless one knows of that risk. That is, a duty of an SGB to inform itself of the risk of CTE precedes the duty to warn.[57] Cases commonly express a failure to warn as an adjunct to a failure to take reasonable steps to be informed.[58]

Consequently, this thesis considers in detail the legal obligation of a duty holder to take reasonable steps to know of the risk of harm, the legal steps required to fulfil that obligation, and the law pertinent to the obligation to warn.

Similarly, claims that an SGB did not, for example, remove a player from the field of play following a concussion, or permitted an athlete to return to play too soon after experiencing a concussion, also depend first on information on the medical need to introduce such protocols.

Moreover, although both begin with the obligation to acquire information on the risk of harm, the duty to warn presents a different legal pathway to that of a duty to introduce post-concussion protocols and with differing applications regarding causation in law. Warning of a risk of harm to afford an athlete the opportunity to consider his or her own mitigation strategies, including retirement from contact sport, is substantively different to introducing post-concussion protocols such as medical treatment and reducing the risk of a second concussion.

For example, failure to enforce a protocol requiring a player to be removed from the field when concussed necessitates that the player prove that remaining on the field in this one instance caused, or materially contributed to, the CTE with which he or she later suffered. Nor, where enforced, does a protocol mandating a player sit-out for several weeks prevent subsequent concussions — albeit it may prevent a second concussion during a period of argued vulnerability. It is perhaps inevitable that a CTE sufferer from the pre-2010 era will ultimately,

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[56] *Independent Review of the Work Undertaken by Associate Professor Paul McCrory For or At the Request of the Australian Football League* (17 October 2022) 6–7 ('*Quinn Inquiry*').

[57] The duty to be informed, it may be added, also precedes a duty introduce concussion amelioration protocols.

[58] *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2.

or as an alternative, come to rely on the obligation to warn in furthering a claim in negligence against an SGB.

1.5.11 Sources of information on athletes said to be suffering with CTE

While efforts are made to present all cases of Australian athletes medically diagnosed with CTE, discussion in this thesis of individual examples of symptoms and behaviour traits associated with CTE is somewhat and unavoidably dependent on media reports or personal statements of those who claim to be afflicted with CTE. This is for obvious reasons — personal medical records of living patients are rarely made publicly available. In fact, medical research papers occasionally note the paucity of official medical reports confirming or asserting an individual's affliction with CTE. This paucity necessitates the gathering of information through other means. For example, Bieniek's 2020 study of CTE used 'google' to search online obituaries to match against high school yearbooks for participation in contact sports.[59] Of course, should a case of negligence go to trial there is no reason why ex-players or their relatives, friends and medicos who had furnished reports to the media will not be called upon to provide documents, give evidence, to be cross examined and their accounts put to expert witnesses for assessment.

Casper appears to accept that journalists have a role to play in transmitting knowledge of CTE, stating that '[m]any disclosures that we and investigative journalists have evaluated are far from complete'.[60] Indeed, the authors criticised some medical reports, particularly the CISG Consensus Statements, as succumbing to the undue influence of the sports industry, to suggest greater of due diligence to investigate the veracity of claims by 'industrial sport' and the disclosure of grants and relationships with funders of their research.

A similar paucity is found in presenting evidence of CTE confined to the playing of Australian contact sports — a difficulty recorded in medical journals irrespective of jurisdiction. For example, Katz noted that clinicopathologic data relevant to a diagnosis of CTE was limited largely to American tackle football and boxing such that future studies would benefit from research on the risk from 'other contact sports' such as 'ice hockey, soccer, rugby, mixed martial arts, professional wrestling, motocross, rodeo, bull riding, and race car driving.' The authors went on to note, though, a common link: 'the preponderance of research indicates that CTE pathology … is found only in individuals with a history of repetitive mild brain trauma.'[61] A comment confirmed in countless medical studies of CTE. Despite Katz' lamentation, sports commonly played in Australia, as well as contact sports other than

[59] K Bieniek et al, 'Association Between Contact Sports Participation and Chronic Traumatic Encephalopathy: A Retrospective Cohort Study' (2020 30 *Brain Pathology* 63.

[60] S Casper et al, 'Toward Complete, Candid, and Unbiased International Consensus Statements on Concussion in Sport' (2021) 49(3) *Journal of Law, Medicine & Ethics* 372, 372.

[61] DI Katz et al, 'National Institute of Neurological Disorders and Stroke Consensus Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2021) 96 *Neurology* 848, 859.

American football, are well represented in medical journal reports from the mid-1970s on as causative of CTE and are referred to extensively throughout this thesis.[62]

1.6 Glossary of terms used in this thesis

1.6.1 Nomenclature and CTE

Over many decades what is now known as chronic traumatic encephalopathy has been referred by several different names, most commonly *dementia pugilistica*, punch drunk syndrome and traumatic encephalopathy.

In this thesis, the primary nomenclature used is 'chronic traumatic encephalopathy'; though usually in the abbreviated form 'CTE'. However, the name of the disease is maintained in full in the titles of journal articles, in quotations from journal articles, media reports, and other documents.

At times, the term 'CTE-like harm' is used to describe the harm and symptoms associated with CTE. At other times, words such as 'long-term cognitive harm' or similar descriptions are used for the same purpose, depending on context.

1.6.2 Sport governing body (SGB)

In this thesis, a sport governing body is the entity empowered to exercise governance over an organisation, the basic feature of which is 'control' of the sport as the ultimate decider of rules and policies at the premier, provincial and national level. The Australian Sports Commission said of the somewhat broader, yet informing, term 'governance' that '[a]t its most basic, governance is the system by which organisations are directed and controlled'.[63]

The abbreviation 'SGB' is commonly used in this thesis for a sport governing body.

1.6.3 Premier-level athletes

A 'premier-level athlete' describes an athlete who has played in the first-division competition run by an SGB, such as the AFL, the NRL, Rugby Australia and the Football Association. Such competitions generally comprise the grades that make up this competition, such as 'first grade', 'second grade' or an age-specific grade such as 'under-23s'. Those who played for an SGB at premier level may also be referred to along the lines of 'premier players', or merely the SGB's 'players'. A 'representative player' is one who has gained representative honours at provincial or national level through participation in a premier-level competition of an SGB.

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[62] See, eg, soccer: H Ling, 'Mixed Pathologies including Chronic Traumatic Encephalopathy Account for Dementia in Retired Association Football (Soccer) Players' (2017) 133 *Acta Neuropathologica* 337; rugby: EB Lee et al, 'Chronic Traumatic Encephalopathy Is a Common Co-morbidity, But Less Frequent Primary Dementia in Former Soccer and Rugby Players' (2019) 138 *Acta Neuropathologica* 389; wrestling: B Omalu, 'Chronic Traumatic Encephalopathy in a Professional American Wrestler' (2010) 6 *Journal of Forensic Nursing* 130.

[63] 'The Startline', *Australian Sports Commission* (Web Page) <https://www.sportaus.gov.au/governance/principles/the-startline>.

1.6.4 Chronic traumatic encephalopathy (CTE)

CTE is 'a progressive degenerative disease of the brain found in people with a history of repetitive head impacts (RHI) often incurred during contact sport play, military service, and other activities that involve repeated blows to the head. These may include symptomatic concussions as well as sub-concussive hits that do not cause symptoms.'[64] That is, it is a type of neurodegenerative disease associated with repeated head injuries, concussions or sub-concussions.

CTE causes a wide range of symptoms, often relating to thinking, mood and behaviour. However, symptoms can begin much earlier than a disease like Alzheimer's disease. Cognitive and behavioural (mental health) symptoms might begin in a person's 20s or 30s. Comparatively, Alzheimer's disease might not be symptomatic until a person's 80s.

While CTE is most commonly associated with contact sports, it has been recognised in a variety of other athletes and individuals who have experienced repeated head traumas.

1.6.5 Concussion (symptomatic)

Concussion is 'a disturbance in the brain's ability to acquire and process information. The reduced function of the brain represents damage to nerve cells (neurons). The neurons can be damaged by a direct blow to the head, which causes the brain to rotate and/or move forward and backward.'[65]

Concussion is a common type of mild traumatic brain injury that occurs when the brain is shaken or jolted inside the skull. The impact causes brain strain, with resultant inflammation, damage to nerve fibres, and a change in the person's metabolic state. The brain suddenly releases neurotransmitters that can either overstimulate or inhibit brain function, and there may be microscopic bleeding with a loss of brain cells.

1.6.6 Asymptomatic concussion (sub-concussion)

A sub-concussion refers to head injuries that do not result in the clinical symptoms of a concussion, but can carry less, equal or greater force than a concussion. Some researchers now use the term 'non-concussive impacts' to refer to these mild brain injuries. Individually, these injuries seem minor because the person will not show any noticeable symptoms. However, repeated sub-concussions can compound over time and cause cumulative injury to the brain.

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[64] 'Frequently Asked Questions about CTE', *Boston University, Center for the Study of Traumatic Encephalopathy* (Web Page) <https://www.bu.edu/cte/about/frequently-asked-questions/>. Research by CA Tagge et al — 'Concussion, Microvascular Injury, and Early Tauopathy in Young Athletes after Impact Head Injury and an Impact Concussion Mouse Model' (2018) 141 *Brain* 422 — indicates that CTE occurs independently of concussion; the principle cause, according to Tagge et al, is asymptomatic RHT.

[65] 'Concussion Management Policy', *Brisbane Netball Association*, adopted 9 September 2013 (Web Page) <https://www.pdhpe.net/wp-content/uploads/2015/12/Brisbane-Netball-Association-Concussion-Management-Policy.pdf>.

1.6.7 Neuropathology

Neuropathology is a specialised type of pathology focused on diseases of the nervous system. Neuropathology is relevant to this thesis because researchers analyse brain tissue to diagnose CTE.

1.6.8 Tau protein

Tau is a natural protein of the human central nervous system. Under normal conditions, it is 'primarily associated with microtubules in axons, where it is neither toxic nor associated with neurofibrillary pathology'.[66] Tau protein is most commonly found in brain cells (neurons). Tau is important because it helps stabilise the microtubules or 'internal skeleton' of the neuron. The disfunction of tau proteins is associated with many different brain diseases.

1.6.9 Neurofibrillary tangles

Neurofibrillary tangles are abnormal accumulations of tau proteins. Rather than sticking to the microtubules, tau proteins clump together and form tangles, which prevents the neuron from functioning properly.

1.6.10 Repetitive head trauma

When the term 'repetitive head trauma' (RHT) is used in this thesis, it should be read (unless otherwise indicated) as a reference to RHT in the playing of contact sport, and may include both symptomatic concussion and, though less commonly, asymptomatic concussion.

> [CTE] is a progressive degenerative disease of the brain found in people with a history of repetitive head impacts (RHI) often incurred during contact sport play, military service, employment as a first responder, and other activities that involve repeated blows to the head. These may include symptomatic concussions as well as non-concussive hits that do not cause symptoms.[67]

1.7 Chapter descriptions in this thesis

The thesis is divided into chapters. Several chapters are augmented with an appendix that expands on medical or certain factual matters that may supplement an understanding of the issue under consideration.

- *Chapter 1: Introduction:* This chapter introduces concepts and rationales that are important in understanding the nature of CTE as a disease and the application of the law of negligence as a means of redress by athletes afflicted with CTE. The chapter explains

[66] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43, 60.

[67] 'Frequently Asked Questions: What Is CTE?', *Boston University, Center for the Study of Traumatic Encephalopathy* (Web Page) <https://www.bu.edu/cte/about/frequently-asked-questions/>.

the significance of CTE as a topic worthy of deep research, and why particular avenues of research have been undertaken, along with foundational concerns of law and fact.

- *Chapter 2: 'The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments'*: This chapter highlights the medico-scientific characteristics of CTE that are argued as essential to understanding the application of the legal elements to a case of an SGB's failure to be informed of extant and emerging risks of CTE. This chapter is augmented by Appendix 1, which further assesses medical features of CTE and the scientific evolution of information relevant to a disease caused in playing contact sport.

- *Chapter 3: 'A Duty Relationship Between an SGB and the Athletes Who Play Contact Sport'*: This chapter argues that Australian SGBs of major contact sports possess a duty to take reasonable steps to care for the health and well-being of the players in their premier competitions. This is argued to be established through a 'salient features' approach.

- *Chapter 4: 'CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care'*: This chapter considers the standard of care owed by an SGB to its premier players. The chapter argues that this standard was breached by SGBs failing to take reasonable steps to be informed of risks of harm to their premier players in playing contact sport, and failing to take reasonable steps to warn of the risks of CTE-like harm.

- *Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times'*: This chapter considers whether information on the risk of CTE through RHT in the playing of contact sport was available to Australian SGBs during the period when those who today suffer with apparent symptoms of CTE, or who were diagnosed with CTE at autopsy, played in their premier competitions. Appendix 2 considers the argument of some commentators that the risk of CTE had been downplayed through certain publications to such an extent that CTE could not be considered a real risk, thereby excusing SGBs from failure to warn their players of that risk.

- *Chapter 6: 'Proof* In Vivo *of Personal Damage in the Form of CTE: The Question of "Individual Agency"'*: This chapter acknowledges the difficulties a plaintiff athlete faces in proving that the symptoms they suffer are those of CTE, given that definitive diagnosis is possible only at autopsy. The chapter presents recently developed methods of medical diagnosis, and argues that these methods are sufficient to allow a living athlete to prove that he or she is afflicted with CTE on the balance of probabilities — the legal standard.

- *Chapter 7: 'Factual Causation of Harm: Failure to Warn and the Athlete's Response Had a Warning Been Given'*: This chapter considers the fundamental claim in negligence of Australian SGBs' failure to warn their premier players of the risk of CTE. Emphasised is a court's assessment of a player's response had a warning been provided by an SGB. Appendix 3 supplements this chapter through discussion of the 'scope of liability' in

respect to an obligation of a SGB to be informed and to warn. Appendix 3 briefly considers the question of an SGB's 'scope of liability'.

- *Chapter 8: 'The Question of "Generic Capacity": Does the SGB's Contact Sport Cause CTE?'*: A plaintiff athlete must be able to show that the particular sport accused of causing CTE through RHT is, as a matter of fact, 'generically capable' of doing so. The object of this chapter is to show by reference to High Court precedent and scholarly commentary that in a 'failure to warn' case, such as that debated in this thesis, the law pertinent to proving generic capacity is the 'but for' test of causation of harm. Appendix 4 supplements the legal discussion in this chapter by reviewing the factual means by which the generic capacity of contact sport to cause CTE may be proven. Appendix 5 also augments this chapter in discussing 'material contribution' to CTE from more than one source.

- *Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences'*: This chapter considers the state civil liability defences associated with 'obvious risk' of harm. Emphasised in discussion is the correct characterisation of CTE as a risk. A risk characterised broadly, such as 'concussion', in general terms is more likely to be considered by a court as obvious and thereby 'liability defeating'. A narrow risk, in general terms, would be less likely to be obvious to a premier-level athlete.

- *Chapter 10: 'Conclusion':* As a conclusion to the thesis, this chapter reviews the key arguments and findings in respect to the duty of SGBs in Australia to take reasonable steps to be informed and to warn their premier-level athletes of the risk of CTE posed through RHT in the playing of its contact sport. Certain law-based recommendations are made regarding present and future approaches and actions to be taken by SGBs and contact sport athletes in respect to the risk of CTE.

Chapter 2:
The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments

2.1 Introduction

The previous chapter, Chapter 1, provided a foundational outline of the subject of this thesis. It also explained the fundamental legal and factual approaches taken in this thesis and, most importantly, emphasised why those specific approaches were thought necessary within the context of this thesis.

Chapter 2 contends that the application of law relevant to a claim of negligence by a plaintiff athlete against an SGB due to failure to warn cannot be properly understood unless 'the disease processes' of CTE are comprehended. This chapter introduces the medical condition CTE relevant to claims in law by premier players of contact and collision sports in Australia that their SGBs were negligent in failing to take reasonable steps to inform themselves of the risk of CTE and to warn their premier players of that risk.

The next chapter, Chapter 3, concerns the establishment an SGB's a duty to care for the health and well-being of its premier players through a 'salient features' approach.

This chapter provides a basis for understanding the factual foundation of claims of a breach of duty within a broad legal context. Indeed, an understanding of the discovery or CTE as a disease, its identification as a condition associated primarily with the sport of boxing in the earlier part of the twentieth century to its recognition from the 1970s through RHT across a range of contact sports — some common to Australia, the discovery of its histological cause in 2005 and the quest for an *in vivo* means of diagnosis up to the present time, its reporting in medical and scientific journals that informed SGBs of the threat posed to their athletes, are all essential to a plaintiff athlete's claim of negligence and without which the present-day legal issues and their resolution cannot be appreciated or comprehended.

The law states that liability attaches only where knowledge of a risk of harm is known or ought to be known by a duty-holder. When knowledge of when CTE ought to have been known by an SGB is a question of fact. Should it have been known, say, in 1928 with the medical revelation of *dementia pugilistica* in boxers, in the 1970s when a range of non-pugilistic contact sports were implicated, or a time from 2005 with the discovery of the histological basis of CTE?

The law excludes a duty holder from liability where a risk of harm is or should have been 'obvious' to a reasonable plaintiff. When a risk is obvious is a question of fact. Should an SGB be excused because a reasonable premier-level athlete would have known of the risk of CTE in 1928, or in the 1970s and 1980s when general contact sport was implicated, or in 2010 when SGBs began to inform their players of that risk?

This chapter aims to provide the factual background to the disease of CTE, how it is caused and its development against, for purposes of context and perspective, a legal backdrop.

2.1.1 This chapter proceeds as follows

Section 2.2: What is CTE?

This section explains the medical and scientific basis of CTE, as it applies to matters of negligence of an SGB by failure to be informed of the risk of CTE and to warn its premier players of that risk. This section is relevant to: Chapter 3: 'A Duty Relationship'; Chapter 4: 'CTE and the Obligation to be Informed and to Warn'; Chapter 7: 'Factual Causation'; Chapter 8: 'The Question of "Generic Capacity"'; and Appendix 5: 'Multiple Causes and "Material Contribution" to Harm'.[1]

Section 2.3: Brief overview of reports of Australian athletes suffering CTE

This section gives an overview of reports of Australian athletes suffering CTE, parties to litigation and the NFL Litigation. This section is relevant to: Chapter 3: 'A Duty Relationship'; Chapter 4: 'CTE and the Obligation to be Informed and to Warn'; Chapter 7: 'Factual Causation'; and Appendix 5: 'Multiple Causes and "Material Contribution" to Harm'.

Section 2.4: The importance of the timeline of revelations of CTE

On the basis that a duty-holder is only duty-bound to inform itself and to warn of a risk of harm that is known or ought to be known, foundational to an understanding of when the obligation of an SGB to warn of the risk of CTE arose is the date of publication and the influence of three landmark medical journal articles notifying of the risk of CTE in contact sport. This section is relevant to: Chapter 3: 'A Duty Relationship'; Chapter 4: 'CTE and the Obligation to be Informed and to Warn'; Chapter 5: "Accessible Knowledge of CTE'; and Chapter 9: 'CTE and the Characterisation of "Risk of Harm"'.

Section 2.5: Head trauma (symptomatic concussion) and sub-concussive head trauma (asymptomatic concussion)

This section explains that CTE through the playing of contact sports is associated with two broad forms of head trauma: concussive (symptomatic concussion) and sub-concussive (asymptomatic concussion). It notes, however, that those suffering with CTE, or where it is confirmed at autopsy, are reported to have virtually all endured multiple concussive blows,

[1] Chapter titles are truncated.

often numbering well into the dozens and, in many cases, had been knocked unconscious several times. This section is relevant to: Chapter 4: 'CTE and the Obligation to be Informed and to Warn'; Chapter 7: 'Factual Causation'; Chapter 8: 'The Question of "Generic Capacity"'; and Appendix 5: 'Multiple Causes and "Material Contribution" to Harm'.

Section 2.6: Conclusion

This chapter provided an introduction to the disease of CTE, its cause and aetiology which is argued as essential to understanding the basis to claims of negligence made by athletes who played in the premier competitions of SGBs in Australia. The 'timeline of discovery' of CTE-like harm was also considered and argued as relevant to considerations of when an SGB ought to have known of the risk of CTE.

Appendix 1: The Science of Concussion and Chronic Traumatic Encephalopathy in Sport.

This appendix supplements this chapter by providing extended information on the cause of CTE through damage to the axons of the brain, and it progression as a disease

2.2 What is CTE?

CTE is characterised by cognitive disfunction, irritability, aggression, depression, short-term memory loss, heightened suicidality and ultimately death, which may, in advanced forms, be preceded by parkinsonism. CTE is a tauopathy, a neurodegenerative disease characterised by abnormal neuronal deposits of tau-positive inclusions or expressed generally, CTE is a form of dementia.[2] CTE is a disease and must not be confused with concussion, a condition, not a disease, that usually resolves within a week or two. By its nature CTE is a disease of latency where decades separate the stimulus of causation from the initial symptoms, characteristics that have a profound influence on the application of law in a claim of negligent failure to warn.

In medical terms, CTE is

> a progressive degenerative disease of the brain found in people with a history of repetitive head impacts (RHI) often incurred during contact sport play, military service, and other activities that involve repeated blows to the head. These may include symptomatic concussions as well as subconcussive hits that do not cause symptoms.[3]

[2] Y Zhang et al, 'Tauopathies: New Perspectives and Challenges' (2022) 17(1) *Molecular Neurodegeneration* 28.

[3] 'Frequently Asked Questions about CTE', *Boston University, Center for the Study of Traumatic Encephalopathy* (Web Page) <https://www.bu.edu/cte/about/frequently-asked-questions/>. Research by CA Tagge et al — 'Concussion, Microvascular Injury, and Early Tauopathy in Young Athletes after Impact Head Injury and an Impact Concussion Mouse Model' (2018) 141 *Brain* 422 — indicates that CTE occurs independently of concussion; the principle cause, according to Tagge et al, is asymptomatic RHT.

RHT may lead to an accumulation of a protein known as 'tau', which, when leaked from the axons[4] of neurones, is transformed into toxic 'hyperphosphorylated tau-positive aggregates' ('p-tau'). This form of tau[5] forms clumps to entangle small blood vessels and cells of the brain, disrupting neuronal function. Excessive tau entanglements are associated with 'memory loss, confusion, impaired judgment, impulse control problems, aggression, depression, suicidality, parkinsonism, and eventually progressive dementia'.[6] Histologically, CTE is 'defined as a tauopathy with a distribution of tau-positive neurofibrillary tangles (NFTs) that is distinct from other tauopathies, and usually shows an absence of beta-amyloid deposits, in contrast to Alzheimer's disease'.[7] CTE is also characterised as a form of dementia, a condition entirely different to symptomatic concussion.[8] In a healthy brain, 'tau is found in the axons, the transmission lines of neurons … In conditions such as Alzheimer's and CTE, tau instead forms tangles that clump together to disrupt the cells' transport system.'[9]

Given that CTE shares common clinical symptoms with other forms of cognitive impairment such as Alzheimer's disease or parkinsonism, a definitive diagnosis can only be made post-mortem.[10] Nonetheless, although CTE shares with Alzheimer's disease an abnormal accumulation of tau, each disease is unique: with CTE tau accumulations are found 'around the cortical vessels in the depths of the cortical sulci, and predominantly in the deeper neocortex', while in Alzheimer's disease tau is 'initially localized in the deeper neocortical layers'.[11] For a living athlete suffering the debilitating effects of CTE, a definitive diagnosis at autopsy is of little temporal value. However, recent research combining clinical indicators,

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[4] An axon is 'the process of a nerve cell along which impulses travel away from the cell body. It branches at its termination, forming synapses at other nerve cells or effector organs. Many axons are covered by a myelin sheath formed from the cell membrane of a glial cell': 'Axon', *Medical Dictionary* (Web Page) <https://medical-dictionary.thefreedictionary.com/axon>.

[5] While the full term for this form of tau is 'hyperphosphorylated tau-positive aggregates', or 'p-tau', the simplified term 'tau' will generally be used as the operative descriptor in this text.

[6] 'Frequently Asked Questions about CTE', *Boston University, Center for the Study of Traumatic Encephalopathy* (Web Page) <https://www.bu.edu/cte/about/frequently-asked-questions/>.

[7] LN Hazrati et al, 'Absence of Chronic Traumatic Encephalopathy in Retired Football Players with Multiple Concussions and Neurological Symptomatology' (May 2013) *Frontiers in Human Neuroscience* 1.

[8] 'Chronic Traumatic Encephalopathy (CTE) Dementia', Dementia Australia (Web Page) <https://www.dementia.org.au/about-dementia/chronic-traumatic-encephalopathy-cte-dementia >.

[9] 'How Long Does Concussion Last: Long-Term Effects', *Queensland Brain Institute* (Web Page) <https://qbi.uq.edu.au/concussion/how-long-does-concussion-last-long-term-effects>.

[10] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 709. *In vivo* tests for CTE are being sought. For full discussion, see Chapter 6. Although 'CTE shares many microscopic similarities with Alzheimer's disease (AD) and other tauopathies, it has several distinguishing features': BE Gavett et al, 'Chronic Traumatic Encephalopathy: A Potential Late Effect of Sport-related Concussive and Sub-concussive Head Trauma' (2011) 30 *Clinical Sports Medicine* 179, 181.

[11] D Iacono, 'Chronic Traumatic Encephalopathy: Known Causes, Unknown Effects' (2017) 28 *Physical Medicine and Rehabilitation Clinics of North America* 301, 308.

scans and behavioural manifestations are argued as sufficient to prove by inference on the balance of probabilities the contraction of CTE *in vivo*.[12]

2.2.1 The tau protein and tauopathy

Tau is a natural protein of the human central nervous system, where, under normal conditions, it is 'primarily associated with microtubules in axons, where it is neither toxic nor associated with neurofibrillary pathology'.[13] The microtubules could be described as small passageways within the axon through which impulses are transmitted from one cell to another. In its natural location, tau surrounds the microtubule and acts to stabilise it in shape and form. Phosphorylation changes the structure of the normal tau proteins 'to cause them to clump together'.[14]

At its most simple, CTE results from brain trauma that causes tau to dislodge from the microtubules within the axon of a neurone (or, as argued by some, from minute tears in micro-blood vessels)[15] where, for not fully known reasons, it becomes abnormally phosphorylated (p-tau), forms into clumps and entangles micro-blood vessels, and disrupts the normal operation of neurones.

2.3 Brief overview of reports of Australian athletes suffering CTE

Although it was 'unclear what severity or recurrence of head injury is required to initiate CTE', neurologist Dr Ann McKee et al implicated 'a wide variety of contact sports, including football, boxing, wrestling, rugby, hockey, lacrosse, soccer, and skiing' as well as 'karate, horse riding, and parachuting.'[16] In 2013 McKee et al reiterated the concern, stating that although historically 'CTE has been primarily associated with boxing, CTE may also occur as a consequence of American football, hockey, wrestling, rugby and exposure to blast or concussive injury associated with military service'.[17]

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[12] See Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"'.

[13] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43, 60.

[14] 'How Long Does Concussion Last: Long-Term Effects', *Queensland Brain Institute* (Web Page) <https://qbi.uq.edu.au/concussion/how-long-does-concussion-last-long-term-effects>.

[15] See the references to CA Tagge et al, 'Concussion, Microvascular Injury, and Early Tauopathy in Young Athletes after Impact Head Injury and an Impact Concussion Mouse Model' (2018) 141 *Brain* 422.

[16] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 709.

[17] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43.

CTE manifests clinically in a range of debilitating conditions. McKee et al state that

> [t]he symptoms of CTE are insidious, first manifested by deteriorations in attention, concentration, and memory, as well as disorientation and confusion, and occasionally accompanied by dizziness and headaches. With progressive deterioration, additional symptoms, such as lack of insight, poor judgment, and overt dementia, become manifest. Severe cases are accompanied by a progressive slowing of muscular movements, a staggered propulsive gait, masked facies, impeded speech, tremors, vertigo, and deafness.[18]

Other symptoms include

> irritability, impulsivity, aggression, depression, short-term memory loss and heightened suicidality … With advancing disease, more severe neurological changes develop that include dementia, gait and speech abnormalities and parkinsonism. In late stages, CTE may be mistaken clinically for Alzheimer's disease and frontotemporal dementia.[19]

CTE, which can only be definitively diagnosed by histological examination of brain tissue at death, has been confirmed by autopsy in several cases reported in Australia including at coroners' inquiry and in hundreds of cases of similar reports overseas. Some medical specialists say they are treating dozens or hundreds of ex-contact sport athletes displaying classic symptoms of CTE. To illustrate, neurologist Dr Rowena Mobbs, specialising in CTE, stated in a submission to the Australian Senate inquiry in concussion in sport that 'I have experience in managing a cohort of 180 patients with suspected traumatic encephalopathy syndrome (TES), 52 confirmed with probable TES on cognitive testing'.[20] Dr Mobbs revisited her experience in media commentary confirming that she is

> treating more than 40 rugby league players for brain injuries [and] I have seen over 100 in trouble, another 100 patients under watch, mainly through playing footy for 20–30 years.

Dr Mobbs extrapolated from that figure to estimate that there are as many as 'tens of thousands of Australians with CTE'.[21]

While media reports of CTE are not definitive proof that an individual has contracted CTE, which can only be supplied by an histological examination of brain matter at autopsy, several media reports of ex-contact sport athletes said to be suffering from CTE are listed below to indicate, somewhat imperfectly, the breadth of the medical and legal problem of CTE. Many of these reports are, however, affirmed by comments of the patient's treating physician, some

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[18] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 710.

[19] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43, 44.

[20] Rowena Mobbs, Submission No 1 to Senate, Community Affairs References Committee, Parliament of Australia *Concussions and Repeated Head Trauma in Contact Sports* (September 2023) 3. Because, like most neurodegenerative diseases, CTE can only be diagnosed definitively at autopsy, 'traumatic encephalopathy syndrome' (TES) is the term used to describe the *in vivo* clinical manifestation of CTE.

[21] Jessica Halloran and Wendy Carlisle, 'Wally Lewis: The Tragic Cost of a Glorious Rugby League Career', *The Australian* (Sydney, 31 July 2023).

of whom have referred to *in vivo* diagnostic tests for CTE.[22] Understandably, sufferers of disease do not usually wish their actual medical records released publicly such that some reliance on information flowing from the patient to the media is essential to understanding the apparent impact of CTE. The symptoms reported in the media are, it may be said, consistent with those recognised in medical journal reports of *in vivo* indicators of CTE.[23]

In fact, media reports of athletes with symptoms consistent with CTE are regular and extensive. And while not definitive diagnoses, the athletes who furnish these reports are those who are likely bring a suit in negligence against their SGB for failing to take reasonable steps to ameliorate the risk of CTE. Indeed, some of those listed below have been joined to the AFL players group proceedings against the AFL.[24]

2.4 The importance of the timeline of revelations of CTE

Any litigation against an Australian sporting organisation, be it a governing body or a club, will, in common with the NFL litigation, likely focus on several propositions — the organisation's failure to warn players of the risk of CTE; failure to have informed itself of the risk of CTE when information was reasonably available; failure to implement adequate concussion protocols; and, in the case of a governing body, failure to adjust the rules of play to minimise the risk of CTE. In the now settled McManus suit, for example, the player claimed in part that 'Knights Rugby League Pty Ltd breached its duty to him by continually exposing him to the risk of concussive injury' and that he 'should have been warned of the risks of playing when concussed'.[25] Almost identical claims have been made in the AFL class action filed by Max Rooke in 2023: that the AFL 'knew or ought to have known' of the 'potential long-term consequences of concussion suffered by AFL players', failed to inform players of those risks, and failed to introduce and enforce mitigating protocols.[26] These legal claims hinged foremost on the relevant governing organisations failure to take reasonable steps to inform themselves of available knowledge that RHT in contact sports such as rugby league or AFL may cause CTE and, consequently, failing to warn their premier players of that risk.[27]

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[22] See Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"'.

[23] See Chapter 8: 'The Question of "Generic Capacity": Does the SGB's Contact Sport Cause CTE?'; Chapter 9 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences'.

[24] *Rooke v Australian Football League* (Supreme Court of Victoria, S ECI 2023 00969, commenced 14 March 2023).

[25] *McManus v Knights Rugby League Pty Ltd* [2017] NSWSC 1101, [10].

[26] *Rooke v Australian Football League* (Supreme Court of Victoria, S ECI 2023 00969, commenced 14 March 2023) Writ, cll 10, 15.

[27] It is worth noting that, as considered in subsequent chapters, SGBs may have been informed of the risk of CTE by, say, their medical associates, but for reasons not disclosed decided not to warn their players of that risk.

A negligent failure to be informed and to warn is referenced to the knowledge of the day. That is, a reasonable defendant cannot be expected to know of a risk of harm that has not emerged, been revealed or discovered at the time of the purported tort. In *Roe v Minister of Health*[28] two patients suffered paralysis below the waist after their anaesthetist injected into their spines nupercaine that had been contaminated with the acid phenol. Having been sterilised through immersion in phenol, the glass ampoules of nupercaine collided one with the other, causing imperceptible cracks that facilitated the transport of phenol into the anaesthetic. The harm to the patients occurred in 1947, but the risk of that harm was not widely realised until 1951. Morris LJ commented: '[C]are has to be exercised to ensure that conduct in 1947 is only judged in the light of knowledge which then was or ought reasonably to have been possessed.'[29] Denning LJ stated: 'There is no duty of care owed to a person when you could not reasonably foresee that he might be injured by your conduct',[30] concluding that once a risk could be reasonably known, a duty arose: 'Nowadays it would be negligence not to realize the danger, but it was not then.'[31]

2.4.1 Three landmark medical studies

Three landmark studies spanning more than 80 years are commonly credited with drawing a link between head trauma in sport and the onset of symptoms of CTE. These studies provide points in time from which to consider the legal liability of sporting organisations in Australia as regards the occurrence in their athletes of CTE. In short, at the time when a premier-level athlete was exposed to the risk of CTE did the relevant governing body know, or should it have known, of a nexus between RHT in contact sport and long term cognitive disfunction, and taken steps to mitigate the risk of harm — in the main by warning of such a risk and initiating other protective protocols?

In 1928, pathologist Dr Harrison Martland referred to 'punch drunk syndrome' in an article published in the *Journal of the American Medical Association* to describe a condition found in boxers characterised initially by

> an occasional and very slight flopping of one foot or leg in walking, noticeable only at intervals; or a slight unsteadiness in gait or uncertainty in equilibrium …[32]

to be followed later on, in severe cases, with

> a peculiar tilting of the head, a marked dragging of one or both legs, a staggering, propulsive gait with the facial characteristics of the parkinsonian syndrome, or a backward swaying of the body,

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[28] [1954] 2 QB 66. This case is discussed at length in Chapter 4: 'CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care'.

[29] Ibid 92.

[30] Ibid 94.

[31] Ibid 96.

[32] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103, 1103.

> tremors, vertigo and deafness. Finally, marked mental deterioration may set in necessitating commitment to an asylum.[33]

Although recognising that similar markers presented in other forms of brain injury, Martland drew the conclusion that 'the occurrence of the symptoms in almost 50 per cent of fighters … seems to be good evidence that some special brain injury due to their occupation exists'.[34]

In 2002, medical examiner for the San Joaquin County in California Dr Bennet Omalu performed a post-mortem dissection on the brain of 50-year-old Mike Webster who retired from the NFL age 40, stating in 2005 in the journal *Neurosurgery*: 'We herein report the first documented case of long-term neurodegenerative changes in a retired professional NFL player consistent with chronic traumatic encephalopathy.'[35] When stained sections of Webster's brain were examined microscopically, 'neurofibrillary tangles' — neurones entangled with the protein 'tau' — were detected. Tau is a naturally occurring brain protein that supports the structure of a neurone, but which, following repeated head trauma, may be produced in excess leading to CTE.[36]

A link between playing American football and cognitive impairment was largely ignored by the NFL at this time — a position that must have found some comfort in dismissive medical commentary at the end of Omalu et al's article that described his findings as 'presumptuous', given that the subject was 'a single individual' who played in a position 'associated with the lowest frequency of concussions in the NFL'.[37]

A meta-study in 2009 conducted by Dr Ann McKee et al and published in the *Journal of Neuropathology and Experimental Neurology* reported five players of American football as

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[33] Ibid.

[34] Ibid.

[35] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128, 129. Of interest, a psychologist, Dr Krieg, had examined Webster in November 1998 with respect to his application for NFL disability benefits, finding him 'with brain damage — that is, dementia resulting from his football related head traumas'. On 22 June 1999, a psychiatrist, Dr Himmelhoch, stated that 'Webster suffered from "traumatic or punch drunk encephalopathy" resulting from football, leaving him totally and permanently disabled': *Sunny Jani, Administrator of the Estate of Michael L Webster, deceased v The Bert Bell/Pete Rozelle NFL Player Retirement Plan; the NFL Player Supplemental Disability Plan*, Unpublished Judgment of the United States Court of Appeals for the Fourth Circuit, December 13, 2006, at 13 and 14 respectively.

[36] See 'The tau protein and tauopathy' at **[2.2.1]** above.

[37] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128, 133: 'Comments', Daniel F Kelly, Los Angeles, California. It may be noted here that the fact that Webster's NFL playing position was not associated with a high level of reported concussions is in itself instructive, as focus later turned to the risks of CTE from what were termed 'sub-concussive' blows.

suffering from 'neuropathologically verified CTE'.[38] More concerning, however, McKee et al's study stated that

> although most athletes who have a concussion recover within a few days or weeks, a small number of individuals develop long lasting or progressive symptoms. This is especially true of repetitive concussion or mild TBI [traumatic brain injury], in which at least 17% of individuals develop chronic traumatic encephalopathy.[39]

The publication of McKee et al's study made denial of an association between American football and CTE implausible, prompting the NFL to implement head injury protocols. Contact sport in Australia was slower to respond. Only in 2011 did the NRL consider the question of CTE as worthy of specific research, its Chief Medical Officer Ron Muratore stating as follows: 'The idea is to do some research into former players … At the moment, we don't have any information specific to rugby league at all.'[40] Furthermore, it was not until 2014 that the NRL fully implemented concussion protocols,[41] a delay reflected in the 2019 statement of its CEO, Todd Greenberg: 'We have made huge changes going back to 2014 and I'm very confident those rules are in place for the primary reason for the care of our players.'[42] There was also, perhaps understandably, a reluctance to fully accept the findings of the American studies, Muratore commenting as follows: 'I think that we need to be pretty careful how we interpret the (US research) because their game is completely different.'[43]

The AFL appeared to be more proactive, at least initially. An information paper published in May 2012 stated:

> The AFL, in conjunction with medical experts in the field, has been active for approximately 25 years in monitoring and researching concussion in our game … [The AFL's] concerns relate to a possible increased likelihood of the development of depression, dementia and other forms of cognitive impairment later in life, as a result of impacts to the head during games or practice, and not just those hits resulting in concussion.[44]

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[38] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 710. Specifically: 'Of the 51 neuropathologically confirmed cases of CTE, 46 (90%) occurred in athletes. The athletes included 39 boxers (85%) who fought as amateurs and as professionals for varying lengths of time (range, 4Y25 years; mean, 14.4 years), 5 football players (11%) whose playing time ranged between 14 and 23 years (mean, 18.4 years; SD, 3.9), 1 professional wrestler, and 1 soccer player.'

[39] Ibid 709.

[40] B Read, 'NRL Wants to Widen Research into Concussion', *The Australian* (Sydney, 3 August 2011).

[41] See T Webeck, 'Concussion Guidelines Explained', *National Rugby League* (Web Page, 14 March 2014) <https://www.nrl.com/news/2014/03/14/concussion-guidelines-explained/>.

[42] M Woods, 'NRL Taking Concussion Seriously: Greenberg', *ESPN* (Web Page, 8 July 2019) <https://www.espn.com/nrl/story/_/id/27143857/greenberg>.

[43] P Kent, 'NRL Should Heed US Lessons over Dangers of Repeated Head Knocks', *news.com.au* (online, 8 September 2012) <https://www.news.com.au/sport/nrl/nrl-should-heed-us-lessons-over-dangers-of-repeated-head-knocks/news-story/e9a1778b5e51bad6a0e6b5f999c807da>.

[44] AFL, 'Responsible Approach to Concussion in the AFL', Information Paper, May 2012, 1.

The information paper claimed that research 'has been ongoing since 1985'; it acknowledged that 'our game is a contact sport where concussion is a relatively common injury' and stated that '[f]or this reason we are committed to enhancing our understanding of concussion'.[45] As discussed in succeeding chapters, such statements — rather than exonerating the AFL — suggest a negligent failure to investigate reports of CTE-like harm caused by RHT that extend back decades. To illustrate, a 1976 article by Adams in *The Lancet* advised that there are 'reasonable grounds for advising patients to stop collision sports if they have had two episodes of head injury associated with loss of consciousness and/or amnesia'.[46] In short, a 'an active monitoring' of concussion and perusal of available studies could not have overlooked the risk of long-term cognitive disfunction associated with contact sport, and from which a reasonable governing body is, as argued below, duty-bound to warn its premier-level athletes.

Of local interest, and while not a recognised landmark international study, Australian medico Dr CG McDonald described 'traumatic encephalopathy' or 'punch drunk' in a 1936 *Medical Journal of Australia* article as 'a condition found in professional boxers whose ability to take punishment is greater than their skill to avoid it'. McDonald stated further, in terms relatable to today's research:

> 'Punch drunk' was well known to habitues of the ring for years before its grudging recognition by neurologists … [W]hatever the pathology, no doubt can exist of the clinical entity of this condition. Punch drunk fighters commonly suffer from slurring speech with considerable difficulty in pronouncing consonants, from mild ataxia, from tremor of head or limbs, and from a stiffness and clumsiness of one or both feet … The clinical manifestations are progressive and the suggestion has been made that a slow gliosis of the brain develops in the areas where the vascular damage is greatest … The significance from the neurological point of view of 'punch drunk' (now glorified with the long and ugly name of 'traumatic encephalopathy') is that we have here *definite organic stigmata* of repeated but apparently mild and transitory attacks of concussion without fracture of the skull.'[47]

2.4.2 Two other influential periods

Although the studies quoted above elevated the issue of CTE to public prominence, medical journals had reported on a link between contact sport — principally boxing, though from the 1970s increasingly implicating other sports — and CTE-like symptoms since the time of Martland's 1928 article,[48] raising the question of whether a reasonable sporting organisation

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[45] Ibid.

[46] ID Adams, 'Brain Damage in Sport' (13 March 1976) *The Lancet* 585.

[47] CG McDonald, 'The Sequelae of Head Injuries' (11 January 1936) *Medical Journal of Australia* 45, 47. Emphasis added. McDonald observed interestingly (at 47): 'It would seem that there is some virtue in a fractured skull, for a boxer who sustains a fracture may take the hint and abandon what [American boxer] Gene Tunney described as a dangerous occupation.'

[48] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

should have drawn from these publications knowledge sufficient to oblige it to warn its athletes prior to Omalu et al's 2005 article.[49]

Medical research articles published from the 1970s onward, move any debate as to foreseeability of CTE beyond what may be described as the extreme violence of boxing to a clearly foreseeable risk of CTE in contact sports such as rugby and AFL; see, for example, Adams' article published in *The Lancet* in 1976 (quoted in the previous section).[50] Further, in 1980, Lindsay drew a parallel between the cumulative effects of minor head injury and traumatic encephalopathy in boxing and other sports, including Association football, rugby football, and wrestling.[51]

Others have suggested a period beginning with the publication of the 1994 the National Health and Medical Research Council (NHMRC) convened a panel of medical experts 'to inquire into head and neck injuries resulting from participation in the sport of football (all codes) and to suggest ways of minimising injuries'.[52] Greenhow proposed that the report 'indicates a point in time when concerns were sufficient to warrant recommendations to mitigate the risks associated with the potential harm associated with concussive injury management.'[53] Curiously, Greenhow does not explain in law or engage in legal analysis as to why it would be that the report warrants recommendations to 'mitigate the risks' noting only that a 'court is likely to be interested in expert views on what the "some evidence" meant'. This reference and also to a 1996 publication by sport medico Dr Nathan Gibbs is discussed at length in Chapter 5, where caution is advised in over-estimating the impact of these studies on the legal obligation to warn.[54]

Over time, more detailed research continued. For example, a 2001 article by Rabadi published in the *Clinical Journal of Sports Medicine* informed physicians of the 'important public health concern' of 'chronic traumatic brain injury' and the 'long-term neurologic consequences of repetitive concussive and sub-concussive blows to the brain', as found primarily in boxing, but that can 'be anticipated in other contact sports such as soccer, football, ice hockey, and the martial arts'.[55]

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[49] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[50] ID Adams, 'Brain Damage in Sport' (13 March 1976) *The Lancet* 585.

[51] KW Lindsay et al, 'Serious Head Injury in Sport' (1980) 281 *British Medical Journal* 789, 790.

[52] NHMRC, *Football Injuries of the Head and Neck* (Report, 1994) iii.

[53] A Greenhow, 'Mitigating the Concussion Crunch: League Governance, Passive Actors and Regulators of Risk' (2021) 14(1) *Australian and New Zealand Sports Law* 36, 73.

[54] See **[5.7.2]**.

[55] MH Rabadi and BD Jordan, 'The Cumulative Effect of Repetitive Concussion in Sports' (2001) 11 *Clinical Journal of Sport Medicine* 194, 194.

Given recent media reports of proposed litigation by NRL and AFL players thought to be suffering the effects of CTE, the question of whether a reasonable sporting organisation in Australia should have warned its athletes of the risk of a CTE-like harm, and introduced head trauma mitigatory protocols, such as the issuance of warnings, is prominent. As liability is determined by the state of knowledge existing at the time of a tort, should the reasonable organisation have acted on the findings of Omalu et al's 2005 study? Or is it acceptable in law that sporting organisations did not act until after the publication of McKee et al's 2009 findings?[56] Or should a sports organisation have been cognisant of, and acted upon, the hundreds of peer-reviewed journal reports of CTE-like harm associated with contact sport that have been published since Martland's 1928 study?

In any case, it is suggested that a period of time perhaps spanning no more than a year following the publication of McKee et al's research in 2009 represents an endpoint by which any reasonable organisation should have warned their players and introduced effective concussion-treatment protocols. Moreover, from around this point in time, say, from *circa* 2010, players of contact sport must be adjudged to have fully accepted the 'obvious' risk of CTE.[57]

This thesis considers in detail the failure of SGBs to take reasonable steps to be informed of the risk of CTE and to warn the athletes who play in their premier competitions of that risk. A claim against an opposition player who negligently contacted the head of the plaintiff is unlikely to be successful as CTE is associated with repeated concussive and sub-concussive head trauma over a time period rather than a single event and, in addition, non-intentional head contact is taken in law to be a risk consented to in the playing contact sport.[58]

2.5 Head trauma (symptomatic concussion) and sub-concussive head trauma (asymptomatic concussion)

Before beginning this section, it is important to again note that most medical and scientific journal articles concerned with CTE refer most commonly to 'repetitive head trauma' as a source of harm rather than drawing a distinction between concussive or sub-concussive trauma. The point being a governing body does not avoid liability by claiming that it could not know

[56] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

[57] See the pertinent provisions of the various state Civil Liability Acts relating to 'dangerous recreational activity', 'presumption of awareness of obvious risk', 'inherent risk', 'risk warnings' and 'contributory negligence', several of which are discussed in Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences'.

[58] *Re Jewell* [1987] 1 VAR 370, 372.

of the contribution of sub-concussive head trauma to CTE.[59] It is also clear that CTE is a disease known to be associated with sport and head trauma in the playing of sport, as reported for nearly a century.

CTE through the playing of contact sports is associated with two broad forms of head trauma: concussive (symptomatic concussion, at times referred to as mild traumatic brain injury, or MTBI) and sub-concussive (asymptomatic concussion). With concussive head trauma, the player suffers symptoms such as headaches, blurred vision, dizziness and vomiting.[60] In comparison, Symons defined a sub-concussive impact 'as brain trauma that does not induce any of the clinically observable symptoms associated with SRC (sports related concussion)'.[61] The athlete who experiences sub-concussive head trauma is without apparent symptoms during and after the game, but may, where blows to the head are repetitive over a long period of time, develop CTE. The nomenclature 'sub-concussive head trauma' or 'asymptomatic concussion' does not necessarily imply light contact. The emphasis remains on the word 'concussive' or 'concussion', and although severe symptoms may not be displayed, hits to the head are often far from benign. For example, two boxers may fight 10 rounds and neither may experience symptomatic concussion but each punch, delivered with intent to hurt, will carry hundreds of kilograms of force directed to the head of the opponent and which disturbs the structure and cellular integrity of the brain.[62]

Players of contact sport in Australia who suffer symptoms of CTE, almost invariably describe symptomatic head trauma.[63] For example, as noted above,[64] Ian Roberts was knocked out or heavily concussed 'more than a dozen times' during his 12 years as a rugby league player.[65] The AFL's John Platter is reported to have 'suffered a concussion in at least 30 games of his illustrious 12-year, 258-game career'.[66] NRL player James Graham reports being

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59 The characterisation of CTE as a form of harm is discussed in Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences'.

60 J Kohler, 'An Introduction to Concussion in Sport' (2013) 31(1) *Sport Health (Australasian College of Sports Physicians)* 15.

61 Georgia Symons et al, 'The Neurological Consequences of Engaging in Australian Collision Sport' (2020) 37 *Journal of Neurotrauma* 792, 793.

62 The blog *Physics Network* applied Newtonian physics to the question of a skilled boxer's punching power, multiplying acceleration of 30 metres/second squared by a boxer's weight of 80 kilograms to arrive at a figure of 2400 Newtons or '540 pounds-force': 'What is the Force of a Human Punch?', *Physics Network* (Blog Post, December 2022) <https://physics-network.org/what-is-the-force-of-a-human-punch/>.

63 See Appendix 1: 'The Science of Concussion and CTE in Sport'.

64 Ibid.

65 K Aubusson, 'The Effects of Concussion Linger Decades after NRL Stars Hang Up their Boots', *Sydney Morning Herald* (Sydney, 7 February 2018).

66 Ebony Weston, 'Love and Pain: An AFL Legend Looks Back at the Damage Done', *The Standard* (online, 20 October 2020 <https://www.theswinstandard.net/2020/10/20/love-and-pain-an-afl-legend-looks-back-at-the-damage-done/>.

concussed 'around 100' times.[67] Neurologist Dr Rowena Mobbs, in a submission to the Australian Senate inquiry into 'Concussions and Repeated Head Trauma in Contact Sports', commented from her clinical experience that '[f]ootball players seem to develop later Parkinsonism, but nonetheless carry a typical burden for CTE of at least 20–100 concussions, some with more than 100 concussions and over 30 years of exposure in their risk profile'.[68]

As singular events, concussive and sub-concussive head trauma are different from each other — to such an extent that, as a matter of legal causation, the accepted aetiology of one may not necessarily provide evidence with respect to the other. Of course, symptomatic concussion does not exclude the occurrence of asymptomatic concussion and may in fact be seen as a proxy of its occurrence. As considered below, the most likely scenario for the contraction of CTE is several, or many, concussive blows accompanied perhaps by thousands of sub-concussive blows.

2.5.1 The significance of sub-concussive head trauma

As noted above, with sub-concussive head trauma the athlete does not exhibit discernible symptoms of concussion; however, repetitive smaller shocks — perhaps numbering into the hundreds or even thousands over an athlete's career — may exceed Lipton's[69] unknown 'degree of exposure' beyond which the brain is unable to cope and the protein tau is liberated from the axons of the brain, leading to a progressive build-up of abnormal p-tau that over many years spreads cell to cell over many years to cause the neurodegenerative disease of CTE.

NFL player Mike Webster, the subject of Omalu et al's first reported case of CTE,[70] was an 'offensive center' — the player in the middle of a scrimmage line who snaps the ball between his legs to the quarterback to begin an attacking play. Those in the offensive scrimmage line are tasked to protect the quarterback from charging opponents. This often requires heavy head-on-head (or, more accurately, helmet-on-helmet) contact, such that some head trauma is all but inevitable. Although the number of symptomatic concussions experienced by Webster was not recorded, on some estimates he sustained more than 20,000 contacts to the head over his career. When Webster's estate sued the NFL, Duncan J commented on the exposure of those who play in such positions:

> Defensive players are permitted to rush at the quarterback as soon as the football is snapped. The center is particularly vulnerable because he must right himself after the snap to protect the quarterback from the oncoming defensive rush. Of particular danger to offensive linemen in

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67 James Graham, 'Medication Helps Me Live in My Own Head', *The Australian* (Sydney, 26 September 2022).

68 Rowena Mobbs, Submission No 1 to Senate, Community Affairs References Committee, Parliament of Australia *Concussions and Repeated Head Trauma in Contact Sports* (September 2023) 6.

69 ML Lipton et al, 'Soccer Heading Is Associated with White Matter Microstructural and Cognitive Abnormalities' (2013) 268(3) *Neuroradiology* 850, 854.

70 Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

> Webster's era was the 'head slap' technique, in which defensive linemen would begin their quarterback rush by striking the offensive linemen on the sides of the helmet to daze them.[71]

A study in the *Journal of Neurosurgery* confirmed that American footballers endure a significant number of sub-concussive blows in a season. Data on head impacts recorded by helmet-mounted accelerometers was collected from eight collegiate football teams in the United States between 2005 and 2010. Of 1,833 subject footballers, 'a total of 1,281,444 head impacts were recorded, from which 64 concussions were diagnosed'.[72] While the number of diagnosed concussions — at 3.34 per cent — appears relatively low, the concern with sub-concussive trauma relates to the number of head impacts accumulated by an athlete over his or her playing career; in this example, there were an average of 699 head impacts per player over the five-year period of review.

Sub-concussive head trauma leading to CTE occurs in both contact/collision sports and in non-contact sport where blows to the head are common. For example, a study of cognitive disfunction in soccer (football) players revealed that

> [p]layers head the ball, on average, 6–12 times during competitive games, where balls travel at high velocities (up to 80 km per hour or more in nonprofessional matches …). During practice sessions, however, heading drills, where the ball, often tossed at low velocity, is headed repeatedly (up to 30 or more closely spaced repetitions), are commonplace.[73]

The New York Times in 2014 reported on the death of US collegiate soccer player Patrick Grange at the age of 29, stating that he was 'the first named soccer player found to have CTE'.[74] Grange's brain was examined by Dr McKee who, though less than definitive as to whether CTE was present, said that

> [h]e had very extensive frontal lobe damage ... We have seen other athletes in their 20s with this level of pathology, but they've usually been [American] football players ... We can't say for certain that heading the ball caused his condition in this case ... But it is noteworthy that he was a frequent header of the ball, and he did develop this disease.[75]

While Grange may have been the first 'named' soccer player to suffer CTE, Duru reports that England representative Jeff Astle died in 2002 age 59 from 'brain damage suffered during

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[71] *Sunny Jani, Administrator of the Estate of Michael L Webster, deceased v The Bert Bell/Pete Rozelle NFL Player Retirement Plan; the NFL Player Supplemental Disability Plan*, Unpublished Judgment of the United States Court of Appeals for the Fourth Circuit, December 13, 2006, 5.

[72] S Rowson et al, 'Can Helmet Design Reduce the Risk of Concussion in Football?' (2014) 129 *Journal of Neurosurgery* 919, 920.

[73] ML Lipton et al, 'Soccer Heading Is Associated with White Matter Microstructural and Cognitive Abnormalities' (2013) 268(3) *Neuroradiology* 850, 854.

[74] J Branch, 'Brain Trauma Extends to Soccer Field', *The New York Times* (New York, 26 February 2014).

[75] Ibid.

his career'.[76] Astle played in the era of heavy leather soccer balls. The coroner concluded that 'it was heading the soccer ball that had killed him'.[77]

Both concussive and sub-concussive head trauma, either separately or in combination, are thought to contribute to CTE. 'To date', said Russell, 'virtually all individuals in whom CTE neuropathologic change is described have prior history of TBI or repetitive head impact exposure.'[78] Sancar opined that CTE 'is likely caused in part by repetitive traumatic brain injury (TBI), including concussion, which accounts for more than 80% of all TBI cases', stating further that 'mounting evidence suggests that asymptomatic sub-concussive head injuries can produce neurophysiological changes associated with CTE'.[79]

In addition to the degree of force, the means and direction from a blow to the head is delivered influences the occurrence of symptomatic and or asymptomatic concussion. According to McKee et al, sudden rotational movements and 'lateral (side-to-side) injuries produce the most injury', while '[s]agittal (front-to-back) injuries result in relatively good recovery'.[80] Of consequence, head trauma in sport is most often that of the 'rotational acceleration and deceleration' type, caused when a force such as an attacker's knee strikes a tackler's head.[81] The athlete's head accelerates in rotation, causing the brain — which is 'floating' in cerebral fluid — to be pushed against the hard, rotating sides of the skull and deformed, shearing tissue and small blood vessels.

The linear shock waves of 'blast exposure' experienced by artillery soldiers are also a reported cause of CTE, and a major reason for accepting the premise of asymptomatic concussion as a mechanism of CTE causation. In this case, a linear force impacts on the skull and brain in one direction, as opposed to the rotational forces experienced by a blow to the head that causes the brain to deform as it contacts several surfaces of the internal skull.

Tagge et al investigated the relative effects of both mechanisms of head trauma on the brains of mice. Some subjects were exposed to physical impacts to the head and others to simulated blast exposure in the laboratory. 'Computational modelling showed that *impact injury* generated focal point loading on the head and seven-fold greater peak shear stress in the brain

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[76] N Duru, 'In Search of the Final Head Ball: The Case for Eliminating Heading from Soccer' (2018) 83(3) *Missouri Law Review* 559, 574.

[77] Ibid.

[78] Emma Russell, 'Association of Field Position and Career Length with Risk of Neurodegenerative Disease in Male Former Professional Soccer Players' (2021) 78(9) *JAMA Neurology* 1057, 1058.

[79] Feysa Sancar, 'Subconcussive Football Hits May Leave a Telltale Signature of Brain Damage' (2019) 322(16) *Journal of the American Medical Association* 1537, 1537.

[80] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 731.

[81] Ibid 731.

compared to blast exposure. Moreover, intracerebral shear stress peaked before onset of gross head motion.'[82]

Hirad reported similar findings regarding the differential forces of symptomatic and asymptomatic concussion:

> Inertial loading from a concussive impact includes both linear and rotational acceleration. While linear acceleration is correlated with increased intracranial pressure and accounts for some neurological dysfunction, it is not associated with acute mental state alterations — the pathognomonic feature of mTBI. In contrast, rotational acceleration is found to account for 78% of the variance in shear stress, which plays an important role in the occurrence of DAI [diffuse axonal injury]. Head impacts, such as those that typically occur in the setting of American football, are associated with a rapid onset of rotational forces, which cause shear waves that are transmitted throughout the brain.[83]

Do legal implications arise where sub-concussive trauma is determined to be a cause of CTE? One argument is that, unlike the obvious effects of symptomatic concussion, asymptomatic concussion is less identifiable, such that a sporting organisation could not be expected to know of an associated risk of harm. Hence, if asymptomatic concussive trauma is shown to be the only cause of CTE, a sporting organisation has a defensive argument that a causative connection was not, and could not, be discernible given the state of medical science at the relevant time. However, such an argument, as discussed extensively below,[84] is unlikely to gain traction. While both concussive and sub-concussive head trauma have been identified as causes of CTE expressly and impliedly for decades, causation in law requires no more than a plaintiff prove on the balance of probabilities that symptomatic concussion is a 'not negligible' cause of CTE, irrespective of whether asymptomatic trauma is also partly causative.[85] On the facts presented throughout this thesis, there can be no doubt of concussive trauma being a causative factor in the contraction of CTE.

It is also important to note that many of the studies of CTE-like harm refer to causation through 'head trauma', 'low grade concussive brain injury', 'impulsive forces to the brain', 'single or repeated blows to the head or jaw', 'sub-lethal blows to the head', 'blows to the head', 'repetitive head injury' rather than symptomatic concussion.

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[82] CA Tagge et al, 'Concussion, Microvascular Injury, and Early Tauopathy in Young Athletes after Impact Head Injury and an Impact Concussion Mouse Model' (2018) 141 *Brain* 422, 422. Emphasis added.

[83] Adnan Hirad et al, 'A Common Neural Signature of Brain Injury in Concussion and Subconcussion' (2019) 5(8) *Science Advances* 1, 1, 2 <https://www.science.org/doi/10.1126/sciadv.aau3460>. Hirad added (at 2): 'It has also been argued that rotational forces have a dose-dependent correlation with DAI in the brainstem in primate and porcine models of mTBI.'

[84] See Chapter 7: 'Factual Causation of Harm: Failure to Warn and the Athlete's Response Had a Warning Been Given'.

[85] *Western Australia v Watson* [1990] WAR 248, 286. See also Appendix 5: 'Multiple Causes of CTE and "Material Contribution" to Harm'.

Of further relevance, where symptomatic concussion is singular or occasional as compared to repetitive the chances of contracting CTE appear remote.[86] Nonetheless, exceptions have been reported, an illustration of which is the case of a former college American footballer who, in a medically 'unusual' event, suffered post-concussion syndrome[87] and possible CTE at age 25, following several symptomatic concussions.[88] The player, whose name is not recorded in the literature, began football at age six and participated for 16 years, including three years in Division 1 college football. He experienced 10 concussions over that period, the first at age eight. In his first year of college he was knocked momentarily unconscious, and then experienced

> ongoing headaches, neck pain, blurry vision, tinnitus, insomnia, anxiety, and difficulty with memory and concentration. When he returned to play after a few days, symptoms persisted … He stopped playing football at the beginning of his junior season owning to ongoing symptoms. He began failing courses despite having above-average grades in high school … [Further symptoms] included apathy, anhedonia, decreased appetite, hypersomnia, feelings of worthlessness and passive suicidal ideations … At age 23 years, he became verbally and physically abusive towards his wife … [Following post-mortem examination, doctors] unanimously supported postconcussive syndrome (PCS) as the primary diagnosis, with *possible* CTE and major depression as contributing diagnoses.[89]

In sport, contact with the 'head, face, neck or *elsewhere on the body* with an "impulsive" force that is transmitted to the head' may cause concussion.[90] The revelation of 'elsewhere on the body' widens the possible application of CTE to semi-contact and supposedly non-contact sports such as netball, basketball and soccer, where, for example, players may be shouldered or tripped off their feet, to fall hard to the ground. Furthermore, athletes who play ball sports that ostensibly involve no contact to the head, such as cricket and field hockey, are known to suffer concussive and sub-concussive trauma when struck by a ball or stick.[91]

Notwithstanding the obvious risk of concussive trauma, the collision sports are also likely to be the most hazardous with respect to sub-concussive blows. In playing 'unhelmeted' sports, such as rugby, rugby league or Australian football, participants avoid blows to the head for the

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[86] Nonetheless, some research indicates cognitive defects or the formation of tau following single or a few episodes of concussive head trauma.

[87] Post-concussion syndrome 'occurs when symptoms of a mild traumatic brain injury last longer than expected after an injury. These symptoms may include headaches, dizziness, and problems with concentration and memory. They can last weeks to months': 'Persistent Post-concussive Symptoms (Post-concussion Syndrome)', *Mayo Clinic* (Web Page) <https://www.mayoclinic.org/diseases-conditions/post-concussion-syndrome/symptoms-causes/syc-20353352>.

[88] J Mez et al, 'Pathologically Confirmed Chronic Traumatic Encephalopathy in a 25-Year-Old Former College Football Player' (2016) 73(3) *JAMA Neurology* 353.

[89] Ibid 353, 354. Emphasis added. There is an inconsistency in the article in that it refers to 'confirmed CTE', but places the primary diagnosis as 'postconcussive syndrome': 'Although CTE was considered, the lack of delay in symptom onset, [the player's] young age, and his family history of depression reasoned against CTE as the primary diagnosis' (at 354). The player died of cardiac arrest related to a congenital heart defect.

[90] P McCrory et al, 'Consensus Statement on Concussion in Sport' (Conference Paper, International Conference on Concussion in Sport, Zurich, November 2008) 37. Emphasis added.

[91] Sean Ingle, 'Steve Smith's Test Concussion Reignites Debate about Safety, *The Guardian* (London, 19 August 2019).

obvious reason of avoiding pain. Tacklers in the rugby codes are taught to place their head to the side and to contact the opponent's body with their shoulder. Inevitably, however, given the pace of play, a player's head will be jarred, and the brain accelerated and decelerated — perhaps imperceptibly — in some proportion of tackles. To illustrate just how many tackles a rugby league player may execute, in 28 games of the 2022 NRL season, Reed Mahony of the Parramatta Eels performed 1,207 tackles, the most of any player. The player with the second-most tackles performed 1,127 and the third-most, 1,101. Even the player with the 50th-most tackles, Tyson Frizell, performed 654.[92] Noting that an unsuccessful tackle also exposes a player to body contact — or to being 'palmed off' to the face, or to a heavy fall to the ground — the player with the most 'missed tackles' was Ben Hunt who missed 115.[93] Head jarring also occurs when a player is tackled or attempts to escape a tackle. In this regard, NRL statistics for 2022 show that after 24 games, the highest number of 'tackle breaks' was 169 (by James Tedesco of the Sydney Roosters).[94] Post-NRL match photos and videos, in common with other contact sports, reveal faces bruised, swollen or cut — each wound the product of head contact and clearly reflective of the quoted statistics.

The information above indicates that both concussive and sub-concussive head trauma are implicated as causes of CTE, a point reinforced by comments of researcher Cantu that is worth quoting in full:

> In our experience at Boston University with hundreds of cases of CTE, it is total brain trauma at the concussive and sub-concussive level that best correlates with risk for CTE. In approximately 20% of CTE cases, we find no history of concussion but many years of RTBI [repetitive traumatic brain injury] from sub-concussive hits.[95]

In addition, as noted, journal articles on the cause of CTE most often use broad terms such as 'repetitive head trauma' to describe the risk contact sport athlete's face. That is, an SGB does not avoid liability on the mere claim that words such as 'chronic traumatic encephalopathy' had to be used to establish knowledge of the relevant risk of harm.

2.5.2 Severe head trauma and CTE

Tagge et al emphasised that a number of impacts 'independent of concussive signs' can induce brain injury associated with CTE.[96] In contrast it is worth noting that severe head trauma alone has not, according to Iacono, been associated with the development of CTE on the basis that

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[92] 'Stats: Players', *National Rugby League* (Web Page) <https://www.nrl.com/stats/players/?competition=111&season=2022&stat=4>.

[93] Ibid.

[94] Ibid.

[95] R Cantu and A Budson, 'Management of Chronic Traumatic Encephalopathy' (2019) 19(10) *Expert Review of Neurotherapeutics* 1015, 1017.

[96] CA Tagge et al, 'Concussion, Microvascular Injury, and Early Tauopathy in Young Athletes after Impact Head Injury and an Impact Concussion Mouse Model' (2018) 141 *Brain* 422, 423.

severe injuries cause 'death within a time interval usually too short to develop any evident macroscopic lesions'.[97]

2.6 Conclusion

This chapter gave an introduction to the medical condition known as CTE, the aetiology and indicators of which are relevant to claims of negligence brought by athletes who play contact and collision sports in their governing organisation's premier competitions, and who, later in life come to suffer cognitive impairment.

Trauma to the brain over several years — perhaps decades, extending through junior competitions into the senior premier grades — may cause tau proteins to accumulate as toxic 'hyperphosphorylated tau-positive aggregates' that entangle small blood vessels and brain cells to disrupt neuronal function.

With the publication of Dr Harrison Martland's study of 'punch drunk syndrome' in the *Journal of the American Medical Association* in 1928,[98] a causative connection was drawn between head trauma in sport and cognitive disfunction. Although Martland's study concerned the sport of boxing, later medical journal reports broadened the association to include other sports: Dr Bennet Omalu et al in 2005[99] linked CTE with American football and Dr Ann McKee et al in 2009[100] implicated a wide range of contact sports. While Omalu et al's and McKee et al's studies caused media headlines, reports of CTE-like harm have been published regularly since the time of Martland's 1928 research. The availability of research implicating boxing and — increasingly from the 1970s — other sports such as rugby and soccer in the contraction of CTE.

With confirmation through brain dissection and revelations of cognitive disfunction in players of contact sport, CTE is 'a fact' of Australian sport. Many Australian athletes who claim to be affected by CTE, or who present symptoms of CTE, report several or even dozens of symptomatic concussions across their professional careers. As noted above, CTE is linked to both multiple concussive and sub-concussive events. Of course, a high count of symptomatic concussions does not exclude a high count of asymptomatic concussions; indeed, it may be indicative thereof. Each tackle or collision in rugby and Australian football, and each heading

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[97] D Iacono, 'Chronic Traumatic Encephalopathy: Known Causes, Unknown Effects' (2017) 28 *Physical Medicine and Rehabilitation Clinics of North America* 301, 307.

[98] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

[99] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[100] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

of the ball in soccer,[101] is likely to cause jarring of the head and consequential, though perhaps imperceptible, minor trauma to the brain.

For the litigating Australian athlete there remain several hurdles in proving that head contact in sports such as rugby, Australian football and soccer may result in CTE. Notably, not all who play contact sport, or who are exposed to head trauma in sport, experience CTE. Nor is it known what a threshold level of head trauma must be exceeded before an athlete will suffer from CTE. These are concerns directed largely to the scientific inability to describe the cellular 'mechanisms' by which CTE occurs following repeated head trauma rather than the existence of a known disease and its root cause. Indeed, such concerns are not uncommon. As neuroscientist Nowinski points out, '[t]he evidence on the link between [RHT] and CTE is imperfect, and like all similar research, it will remain imperfect in perpetuity'.[102]

Despite threats of litigation in many contact sports around the world, at the time of writing no case has ever proceeded to trial. In the NFL and NHL settlements, neither organisation admitted liability. On one level this is understandable. Unlike, say, the recognised liability in negligence of a food manufacturer supplying contaminated food, no elements that make up a negligence claim against a governing body following the contraction of CTE by a premier-level athlete is established or legally secure — there is no 'easy ride'.

[101] See **[2.5.1]** above.

[102] Christopher Nowinski et al, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13(938163) *Frontiers in Neurology* 1, 14.

Chapter 3:
A Duty Relationship Between an SGB and the Athletes Who Play Contact Sport

3.1 Introduction

The previous chapter, Chapter 2, explored CTE as a disease, examining how it is caused, and its symptoms, sequelae and progression through stages of cognitive disfunction leading to death. The chapter also considered the developmental recognition of CTE as a medical concern and how it became categorised as a disease of RHT in the playing of contact sport. These factors are argued in this thesis as necessary to understanding the legal implications associated with an SGB's failure to be informed and to warn of the risk of CTE.

Chapter 3 considers whether the SGB of an Australian contact sport possesses a legal duty to take reasonable steps to care for the health and well-being of the athletes who participate in its premier-level competition. To date, no appeal court in Australia has found such a duty to exist. Consequently, as argued, the claimed duty of care must be established as a novel case through a 'salient features' approach.

The next chapter, Chapter 4, concerns the standard of care expected of an SGB in informing itself of risks of harm to its premier players and in warning of those risks.

An essential element of liability in negligence is that the defendant owed the plaintiff a duty of reasonable care. This is a question of law. Its answer, like all questions of law, largely depends on precedent, and where there is no relevant precedent — such as an established category of duty — to guide the court, on the general principles of negligence concerned with how a duty may be established in a novel case. This chapter will consider whether the SGBs of a contact sport owe a duty of care to the athletes who play in its premier competition?[1] Doubt has been expressed that there is such a duty for two main reasons.

First, in *Agar v Hyde*,[2] the High Court of Australia found that the International Rugby Football Board (IRFB), which was responsible for formulating rules of play, did not owe a duty of care to two rugby players injured in a scrum collapse to introduce safety protocols to minimise the risk of harm in scrummaging. This chapter argues that the court's determination

[1] The negating of a duty of care through state civil liability legislation concerned with 'dangerous recreational activity', 'assumption of risk' and 'obvious risk of harm' are considered in Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences'.

[2] (2000) 201 CLR 552.

in *Agar v Hyde* does not apply to the governing organisations of contact sport in Australia for two reasons: (1) the IRFB was not a 'governing body' in a sense relevant to the SGBs of Australian contact sport; and (2) the High Court decision is distinguishable on the facts that go to establish a duty relationship — pertinently facts that establish a novel case under a salient features approach.

A second argument proposes that an SGB, such as the National Rugby League (NRL) or the Australian Football League (AFL), merely provides a competition in which players voluntarily engage, thereby isolating the governing organisation from a duty to care for the health and well-being of its participant athletes. In *Green v Country Rugby Football League of New South Wales Pty Ltd*, for example, the defendant league claimed it bore no responsibility for the plaintiff's harm because it 'had a somewhat passive role, and was a mere information conduit'.[3] In *Watson v British Boxing Board of Control; World Boxing Organisation Inc* the sporting organisation attempted to defend a negligence suit by claiming that a duty of care does not reside in just 'laying down Rules for the benefit of boxers generally'.[4] Moreover, professional athletes in Australia are not usually the employees of their SGB but, rather, serve their district clubs under contracts of employment; clubs that direct the athletes' day-to-day workplace activities, as the argument goes, to insulate the SGB from claims of negligence.[5]

Both 'defensive' arguments are resolved largely through proof that the SGBs of Australian contact sport exercise almost total control over the professional lives of their premier players, as well as the rules and protocols under which the sport is conducted and mandate the terms governing the relationship between the premier district clubs and their players. On this basis, the chapter argues that the general principles of negligence required to establish a novel case support the finding of a legal duty of care owed by an SGB to its premier players.

A novel case is established through facts or 'salient features' that are potentially relevant to the existence of a duty.[6] Allsop P in *Caltex Refineries (Qld) Pty Ltd v Stavar*[7] drew upon several High Court cases to formulate a list of 'salient features'. This chapter argues that the salient feature of 'control' over the contact sport it organises is a material factor in establishing a duty of care owed by such a body. Importantly, 'control', it is argued, underpins several other salient features listed by Allsop P, notably those of 'reliance' and 'vulnerability'. Other salient features are also considered and the importance of foreseeability as a salient feature is highlighted in a separate section.

The question of an SGB's duty to ameliorate the risk of harm to athletes who play in elite contact sport is somewhat vexed, and relevant commentary is rare. Standard texts do not appear

3 *Green v Country Rugby Football League of New South Wales Pty Ltd* [2008] NSWSC 26, [98].

4 *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [82].

5 An exception is a contract of employment by Rugby Australia upon a player's selection into the national team.

6 *Mallonland Pty Ltd v Advanta Seeds Pty Ltd* [2024] HCA 25.

7 (2009) 75 NSWLR 649.

to have addressed the issue in any depth.[8] Nonetheless, the utilisation of a salient features approach to CTE has been noted in some publications and commentary.[9]

3.1.1 This chapter proceeds as follows

Section 3.1: Introduction

This section gives a brief overview of the law of negligence applicable to a plaintiff athlete's claim that his or her SGB breached its duty of care by failing to take reasonable steps to inform itself of the risk of CTE and to warn its premier players of that risk. Emphasis is directed to cases heard in Australia and Britain where courts found that no duty was owed by an SGB to take reasonable steps to safeguard the health and safety of athletes who play its sport, contrasted with those cases where courts did find an extant duty. The difference between these judicial determinations are due, as argued, to facts and circumstances relevant to either the absence or presence of several 'salient features' identified by Allsop P in *Caltex Refineries (Qld) Pty Ltd v Stavar*[10] as relevant to establishing a duty of care. As no appeal court in Australia has made a determination relevant to the duty as proposed in this thesis, the chapter argues that the matter is to be decided according to a salient features approach.

Section 3.2: Duty of care owed by an SGB to its premier players

This section explains that while a risk of harm from a failure to take reasonable steps to care for the safety and well-being of premier players is reasonably foreseeable, the other required limb, that of 'neighbourhood' as established in *Donoghue v Stevenson*,[11] is absent in those sport cases where a duty of care has been denied by a court. This absence, it is proposed, is why courts have not recognised a duty of SGBs in Australia to care for the health and well-being of those who play the sport it administers. In contrast, in the sections below, neighbourhood is argued as established through the facts, or salient features, that characterise the relationship between the SGBs that administer major contact sports in Australia and their premier players. The section also points out that, as a general proposition, there is no employer–employee relationship between an SGB and its premier players from which a duty of care is recognised or can be established.

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8 See, eg, D Rolph, *Balkin & Davis Law of Torts* (LexisNexis Australia, 6th ed, 2021); P Stewart and A Stuhmcke, *Australian Principles of Tort Law* (The Federation Press, 5th ed, 2022).

9 See David Thorpe et al, *Sports Law* (Oxford University Press, 3rd ed, 2017, p284): 'An athlete may be able to point to a "salient feature" by which liability attaches to the governing body', referring to emphasis placed by Kirby J on 'vulnerability', 'special control' and 'knowledge' expressed in *Graham Barclay Oysters Pty Ltd v Ryan* [2002] HCA 54, [236]; A Greenhow, 'Mitigating the Concussion Crunch: League Governance, Passive Actors and Regulators of Risk' (2021) 14(1) *ANZSLJ* 36, 41. Although 'actual or constructive' knowledge of what Greenhow referred to as 'concussive injury mismanagement' was acknowledged as something 'useful to examine', no position was taken by Greenhow in respect of a legal outcome at that time.

10 (2009) 75 NSWLR 649.

11 *Donoghue v Stevenson* [1932] AC 562, 580 (House of Lords).

Section 3.3: Salient features and the SGB's duty of care

Judicial reasoning about whether a duty of care exists first looks at decided precedents, both for and against a duty. This section explains that no appeal-level court in Australia has found that an SGB owed its premier players a duty to care for their health and well-being. The section proposes, therefore, that the potential liability of an SGB to its premier players does not fall within a recognised category and, as such, is a novel case to be resolved through a 'salient features' approach under Australian law.

Section 3.4: *Agar v Hyde*: Distinguishing a sport case of 'no duty'

This section argues that *Agar v Hyde*,[12] although involving an international rule-making sporting body (the IRFB) as defendant, did not establish an 'SGB category' of case in Australia. In addition, the section argues that several of the salient features formulated by Allsop P in the case of *Caltex Refineries (Qld) Pty Ltd v Stavar*,[13] which assist a court in the recognition of a new category of case, were absent or lacking in *Agar v Hyde*, thereby precluding recognition.

Section 3.5: Sport cases that establish a duty of care in the SGB

This section considers two sport cases where a controlling body was found to have possessed a duty to care for the health and welfare of its players: *Watson v British Boxing Board of Control*[14] and *Green v Country Rugby Football League of New South Wales Pty Ltd.*[15] The section then argues that a duty of care was established because what were later recognised as several salient features — by Allsop P in *Stavar* — were present. Note, however, that these factors were not referred to in *Watson* and *Green* as 'salient features', but were nonetheless facts considered by the court in reaching a determination. The remainder of the section argues that, as in *Watson* and *Green*, the SGBs of premier-level contact sports in Australia are characterised by these same salient features and, as such, are argued to owe a duty of care to their premier players.

Section 3.6: SGBs and athletes: A 'duty' relationship

This section considers and reveals the matrix of salient features characterising the relationship between the major SGBs of contact sport in Australia and their premier players. Emphasis is placed on the salient feature 'control' — the forms it takes, and how it is gained and exercised by an SGB to potentially influence a premier player. 'Control' is then argued to be a progenitor of several other salient features, especially those of 'reliance' and 'vulnerability'.

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12 (2000) 201 CLR 552.

13 (2009) 75 NSWLR 649.

14 [2001] QB 1134.

15 [2008] NSWSC 26.

Section 3.7: The vulnerable athlete: Reliance and control

This section emphasises the salient feature of 'vulnerability' and how it flows from the salient features of 'control' and 'reliance'. The section considers how an athlete's reliance on the actions of an SGB to be mindful of his or her health and welfare, along with the control exercised by an SGB over most aspect of an athlete's professional life, creates vulnerability.

Section 3.8: Reliance, vulnerability and the violent nature of the sport itself

This section explores the exceptional level of vulnerability that is experienced when a single organisation alone, an SGB, has sole control and influence over an individual's athlete's health and well-being in the playing of a violent contact sport.

Section 3.9: Foreseeability as a salient feature

A defendant cannot be liable unless a reasonable person in the position of the defendant would have foreseen the risk of harm. This section considers 'foreseeability' as a salient feature, which is to say that it considers foreseeability as an element of negligence. The application of foreseeability is reviewed through analogous cases, particularly the mesothelioma cases. An important distinction is drawn between the foreseeability of symptomatic concussion and CTE.

Section 3.10: Questions of policy

This section briefly discusses questions of policy in the establishment of a duty relationship between an SGB and its premier players.

Section 3.11: Conclusion

This section is the conclusion to the chapter, and puts forward the proposition that the High Court determination in *Agar v Hyde* did not establish a duty relationship between SGBs and their premier players, thereby requiring claims of negligence against an SGB by players to be considered as a novel case through a salient features approach. The conclusion is reached that the salient features underpinning the relationship between SGBs in Australia and their premier players are sufficient to establish a duty residing in an SGB to care for the health and well-being of its players.

Appendix 2: CTE and 'Negativing Influences': Do They Determine Liability?

The appendix supplements this chapter by informing of commentary that questioned the reality of CTE as a disease, or as a disease caused by RHT in playing contact sport. It considers the accusation of 'CTE denialism' and questions whether those said to have engaged in this practice were in fact doing so or merely expressing reasonable doubt. Rhetorically, could it be said that certain publications and commentary so discounted the threat of CTE to the point where it was not a foreseeable risk in playing contact sport.

3.2 Duty of care owed by an SGB to its premier players

Plaintiff athletes (who were players in the premier competition of an SGB) suffering symptoms of CTE must prove that the SGB administering the contact sport they played possessed a legal duty that extended to taking reasonable steps to care for their health and well-being.[16] In simple terms Lord Atkin's well-known dictum in *Donoghue v Stevenson* requires a plaintiff to prove two foundational elements to establish a duty of care — 'foreseeability' of harm and 'neighbourhood':

> The rule that you are to love your neighbour becomes in law, you must not injure your neighbour; and the lawyer's question, Who, then is my neighbour? receives a restricted reply. You must take reasonable care to avoid acts or omissions which you can reasonably foresee would be likely to injure your neighbour. Who, then, in law is my neighbour? The answer seems to be — persons who are so closely and directly affected by my act that I ought reasonably to have them in contemplation as being so affected when I am directing my mind to the acts or omissions which are called in question.[17]

This dictum was reiterated recently by the High Court, as it has many times before, in the case of *Mallonland v Advanta Seeds*:

> An essential element of the tort of negligence is that the defendant owes the plaintiff a duty to take reasonable care when engaging in an activity to avoid causing the plaintiff a particular type of damage or loss that is reasonably foreseeable. 'A man is entitled to be as negligent as he pleases towards the whole world if he owes no duty to them.'[18]

As proof of foreseeability at the duty stage is said to be 'undemanding',[19] and given an SGB's access to research stretching back decades on the risk of CTE in contact sports that it 'ought to have known', this limb is likely to be of little concern to a plaintiff athlete. Presenting greater difficulty is proof of neighbourhood to be evidenced through the relationship between the athlete plaintiff and the defendant SGB.

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[16] Chapter 4 argues that an SGB is obliged to be informed of the scientific knowledge of the day and to warn its athletes of the risk of long-term cognitive harm in the playing of contact sport. That is, an SGB, as a singular entity or through its associated medical staff, is obliged to investigate and understand the risks of harm in playing contact sport and to warn of any 'material risk' of which it is aware.

[17] *Donoghue v Stevenson* [1932] AC 562, 580 (House of Lords).

[18] *Mallonland Pty Ltd v Advanta Seeds Pty Ltd* [2024] HCA 25, [29] (Gageler CJ, Gordon, Edelman, Steward, Gleeson, Jagot and Beech-Jones JJ), referencing *Sullivan v Moody* (2001) 207 CLR 562 at 576 [42] and quoting *Le Lievre v Gould* [1893] 1 QB 491, 497; *Vairy v Wyong Shire Council* (2005) 223 CLR 422, 431 [23].

[19] *Crimmins v Stevedoring Industry Finance Committee* (1999) 200 CLR 1, 81. Per Kirby J.

3.2.1 Foreseeability not sufficient: Absence of neighbourhood

It is unremarkable that the playing of contact sport involves a foreseeable risk of physical harm in general and head trauma in particular.[20] However, 'foreseeability' alone is not sufficient under the *Donoghue v Stevenson* formulation. The plaintiff must also prove a 'neighbour' relationship. A 'neighbour' in the legal sense, explained Lord Atkin, is someone 'so closely and directly affected by my act that I ought reasonably to have them in contemplation'.[21]

While harm in the form of CTE is, with little debate, foreseeable by an SGB that had dutifully informed itself of the risk of CTE, the difficulty the plaintiff athlete faces is the absence of 'neighbourhood', foreseeability alone being inadequate in the absence of a judicially recognised category of relationship.[22]

As explained by Gleeson CJ in *Tame v New South Wales*, '[a] necessary, although not sufficient, condition of the existence of a legal duty of care is reasonable foreseeability of the kind of injury that has been suffered by the person to whom the duty is owed'.[23] Hayne J said similarly that 'foresight of harm does *not* suffice to establish the existence of a duty of care'.[24]

Again, this emphasises that the ostensible relationship between an SGB and a plaintiff athlete, being 'neither direct, nor remote',[25] reveals a relationship based on what was often referred to as 'proximity'.

Although a case of economic loss rather than personal injury, Glass JA explained in general terms in *Minister Administering the Environmental Planning and Assessment Act 1979 v San Sebastian Pty Ltd* that establishing a duty of care

> rests upon proof that the defendant and the plaintiff are so placed in relation to each other that it is reasonably foreseeable as a possibility that careless conduct of *any kind* on the part of the former may result in damage of *some kind* to the person or property of the latter.[26]

The test of reasonable foreseeability is general in nature — a test described by Kirby J as 'undemanding'.[27] In *Chapman v Hearse* the majority of the High Court found it correct 'to ask

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[20] These factors are discussed extensively in Chapters 2, 4 and 5.

[21] *Donoghue v Stevenson* [1932] AC 562, 580 (House of Lords).

[22] The term 'neighbourhood' will be used as a general descriptor in this chapter.

[23] *Tame v New South Wales* (2002) 211 CLR 317, [12]. Note that the case concerned a psychiatric condition, where foreseeability of harm has never been recognised as sufficient.

[24] Ibid [250]. Original emphasis.

[25] Ibid [185].

[26] *Minister Administering the Environmental Planning and Assessment Act 1979 v San Sebastian Pty Ltd* [1983] 2 NSWLR 268, 295–6. Emphasis added.

[27] *Crimmins v Stevedoring Industry Finance Committee* (1999) 200 CLR 1, 81.

whether a consequence of the same general character as that which followed was reasonably foreseeable as one not unlikely', adding that '[i]t is sufficient if it appears that the injury to a class of person of which [the plaintiff] was one might reasonably have been foreseen as a consequence'.[28] The class of person was a person who negligently creates a dangerous situation and who is therefore 'liable to the rescuer for any injury sustained by him in aiding the person imperilled'.[29]

It is quite foreseeable that athletes, as a class of persons, are exposed to a risk of harm when participating in a premier-level contact sport organised and administered by an SGB. Gleeson CJ provided an apt example of both elements in the sport case of *Agar v Hyde*, stating:

> It is … clear that the rules [of the game] will expose the players to the risk of injury in the sense that they will lay down the conditions of a physical contest in which some people are likely to be hurt. In that respect, harm is foreseeable. … It does *not* follow that a rule-making body is under a legal duty of care to the players of the game of the kind presently alleged.[30]

Deane J said in *Nagle v Rottnest Island Authority*:

> [A]s a prerequisite to the existence of a duty of care, there must be a necessary degree of proximity of relationship and that the touchstone is that, in all the circumstances, there may be a real risk of injury to the visitor or to the class of which the visitor is a member.[31]

While absence of neighbourhood has been the reasons for failure in many sport cases, it is argued below that the facts characterising the association between an SGB controlling a contact sport in Australia and its premier players establish, through a salient features approach, a neighbour relationship recognised at law.

3.2.2 A 'recognised category' in limited circumstances

On the basis that a purported duty of care owed by an SGB to the athletes who compete in its premier competition does not fall within an 'accepted category', a 'salient features' approach is considered in detail below. There are, however, some instances where the relationship between the player and the organisation falls within an 'accepted category'.[32] To illustrate, players representing at state or national level have a direct contractual relationship with their SGB. Upon selection to the national side, rugby players become employees of the SGB, Rugby Australia, and are required to undergo a medical examination prior to participation. In conducting this examination, Rugby Australia is reasonably obliged to inquire into each player's history of head trauma and, irrespective of the degree of trauma, to warn all players of the risk of CTE and cognitive disfunction. Of course, athletes who are not selected for

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[28] *Chapman v Hearse* (1961) 106 CLR 112, 125 (Dixon CJ, Kitto, Taylor and Windeyer JJ).

[29] Ibid.

[30] *Agar v Hyde* (2000) 201 CLR 552, [20]. Emphasis added. This case is discussed in detail below: see **[3.4]**.

[31] *Nagle v Rottnest Island Authority* (1993) 112 ALR 393, 396.

[32] Word limitations place an extensive report on this topic beyond the scope of this thesis.

representative honours far exceed those who are, and many who are selected play only a few games, suggesting a *de minimis* contribution to CTE should contraction occur. In these circumstances a 'salient features' approach to questions of duty remains applicable. In any case, the evaluation of a supposed employee–employer relationship between an athlete and an SGB necessitates an entirely different framework and legal development that is not a feature of this thesis.

3.2.3 What of an employer–employee relationship?

It is perhaps worth considering, nonetheless, some commentary on the possible availability of employment law as a means of establishing a duty of care, even though the relationship between a player and the SGB is not one of employment. Of the spinal injury case of *Agar v Hyde*,[33] legal commentator Opie, writing a case note, stated that the 'High Court left open the possibility [of the exception] … to its view that a sport's governing body owed no duty of care to participants to amend the rules so as to make the sport safer'.[34] This position in support by legal commentator Greenhow, who refers to Opie's article, to state:

> The window left open in *Agar v Hyde*, where a duty might exist based on an employer-employee relationship and based on a sports injury occurring at 'work', is an area worthy of further consideration.[35]

The matter of concern is whether in fact the High Court in *Agar v Hyde* 'left a window open'. No analysis, as opposed to description, of the elements of an employment relationship in case law is provided by either Opie or Greenhow in support of this position.

The injured parties in *Agar v Hyde* were two young hookers who suffered quadriplegia in two separate scrum collapses while playing amateur rugby. They were not employees of their team or the competition organiser.

On one level, the majority judgment was merely contrasting the position of the injured players as volunteers with that of employees who could be directed, with perhaps little choice, to perform certain tasks, including dangerous tasks. The import of the court's statement was that the defendants, members of the IRFB, had no power to compel the players to engage in any activity, including scrummaging. Consider the following statement, which continued the theme of the IRFB's lack of control over local rules that had been initiated in the previous paragraph of the judgment:

> Neither the Board nor [those who attended its meetings as] delegates invited, let alone directed or required, either [plaintiff] to play in the match in which he was injured. In this respect the cases which the respondents seek to make differ fundamentally from the case of an employee injured at work. An

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33 (2000) 201 CLR 552.

34 H Opie, 'The Sport Administrator's Charter: *Agar v Hyde*' (2001) 9 *Torts Law Journal* 131, 143.

35 A Greenhow, 'Concussion Policies of the National Football League: Revisiting the "Sport Administrator's Charter" and the Role of the Australian Football League and National Rugby League in Concussion Management' (2011) *Bond University Sports Law eJournal* 13, 16.

> employer can direct the employee to go in harm's way and to do so in circumstances over which the employer can exercise control.[36]

The majority went on to state that the IRFB's absence of control over the matches was 'important', such that the plaintiffs' contention that the members of the IRFB should have acted to control or influence the conduct of third parties to reduce the risk of injury could not be achieved because the 'short answer … is that [the IRFB] did not have the power to do so'.[37]

Opie's use of the words 'left open' is unclear, at least to the degree that it is said to indicate that the High Court kept the option in reserve for future or special consideration. The more probable meaning is that the court merely drew a distinction between the IRFB and an employer and, in so doing, stated a known and contrasting principle: that an employer possesses an obligation to provide a system of work that is 'as safe as reasonable care can make it'.[38] *Agar v Hyde* was not concerned with that issue, and moreover the purported meaning does not appear to have received further judicial support over a 25-year span, and it is not considered in this thesis. Opie does make worthwhile comments on the IRFB's 'special knowledge' of spinal injury, and how this should have been the subject of warnings and rules changes for the benefit of players. What Opie did not recognise, however, is that spinal injury was sufficiently well known within rugby circles at the time the tragic harm befell the players that today it would be considered, or at least argued, as an 'obvious risk' under several civil liability defences.[39] As a general comment paraphrasing Brennan J *Nagle v Rottnest Island Authority*, by substituting the words 'non-employer' for 'public authority':

> It is one thing to impose an obligation on an employer to safeguard an employee from the risk of casual failure to take care for his own safety in the course of his employment; it is another thing to impose a similarly onerous duty on a 'non-employer'…[40]

3.3 Salient features and the SGB's duty of care

In two well-recognised sport negligence cases, the relationships between the SGB and its athletes were *not* characterised as a 'recognised duty of care category'. Rather the court applied what is now referred to as a 'salient features' approach to determine that there was no duty owed by the defendant organisations. In the spinal injury case of *Agar v Hyde*, Gleeson CJ stated: '*No existing category* of case in which a duty of care has been held, in this court, to

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[36] *Agar v Hyde* (2000) 201 CLR 552, [82].

[37] Ibid [83].

[38] *McLean v Tedman* (1984) 155 CLR 306, [8].

[39] For extensive discussion, see Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences'. See also comments in *Wyong Shire Council v Shirt* (1980) 146 CLR 40; *Rootes v Shelton* (1967) 116 CLR 383; *Woods v Multi-Sport Holdings* (2002) 186 ALR 145.

[40] *Nagle v Rottnest Island Authority* (1993) 112 ALR 393, 405.

exist, covers the present [case].'[41] In *Watson v British Boxing Board of Control; World Boxing Organisation Inc*, a boxing injury case, Phillips MR commented: 'At the outset … I propose to identify some significant features of the present case, which place it outside any *established category* of duty of care in negligence.'[42]

3.3.1 The development of a 'salient features' approach

As Kirby J remarked in *Harriton v Stephens*, '[m]ost tort actions fall within a recognised duty of care category', and, 'of the actions that fall outside, or lie on the boundary of, an established duty category, the test of reasonable foreseeability will ordinarily provide guidance in determining whether a duty is in fact owed'.[43] However, some cases do not fit within either category, in which case 'instruction on the duty issue can be secured from several "salient features" that have been identified as potentially relevant to the existence of a duty'.[44]

Accordingly, a 'salient features' approach focuses on 'novel cases' — those of a type where a duty of care has not previously been determined:

> If the circumstances fall with an accepted category of duty, little or no difficulty arises. If, however, the posited duty is a novel one, the proper approach is to undertake a close analysis of the facts bearing on the relationship between the plaintiff and the putative tortfeasor by reference to the 'salient features' or factors affecting the appropriateness of imputing a 'legal duty' to act to take reasonable care to avoid harm or injury.[45]

From *Gala v Preston*[46] in 1991 through to *Hill v Van Erp*[47] in 1997, the High Court of Australia favoured a test of 'proximity'. In *Pyrenees Shire Council v Day* in 1998, Kirby J said 'there [must] exist between the alleged wrong-doer and [the plaintiff] a relationship characterised by the law as one of "proximity" or "neighbourhood"'.[48] A 'three-stage' test[49]

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41 *Agar v Hyde* (2000) 201 CLR 552, [20]. Emphasis added. For further discussion, see David Thorpe, 'Negligence and Civil Liability' in David Thorpe et al, *Sports Law* (Oxford University Press, 3rd ed, 2017) 284.

42 *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [41]. Emphasis added.

43 *Harriton v Stephens* (2006) 226 CLR 52, [63].

44 Ibid [64].

45 *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649, [102]–[104] (Allsop P; Basten and Simpson JJA agreeing).

46 (1991) 172 CLR 243.

47 (1997) 188 CLR 159.

48 *Pyrenees Shire Council v Day* (1998) 192 CLR 330, 419.

49 This test was outlined as follows in *Graham Barclay Oysters Pty Ltd v Ryan* (2002) 211 CLR 540, [232]: '(1) whether it was reasonably foreseeable to the alleged tortfeasor that the particular conduct or omission would be likely to cause harm to a person such as the claimant; (2) whether between that tortfeasor and the claimant a relationship existed that could be characterised as one of "proximity" or "neighbourhood"; and (3) if so, whether it was fair, just and reasonable that the law should impose a duty of a given scope upon that tortfeasor for the benefit of that person.'

adopted from the English case of *Caparo Industries Plc v Dickman*[50] was favoured by Kirby J in several cases, including *Perre v Apand Pty Ltd*[51] and *Crimmins v Stevedoring Industry Finance Committee.*[52]

Importantly, as regards the absence of neighbourhood, a comment made by Gummow J in *Perre v Apand Pty Ltd* in 1998 foreshadowed a 'salient features' approach and made specific reference to a need to consider the 'connections between the parties':

> In determining whether the relationship is so close that the duty of care arises, attention is to be paid to the particular connections between the parties. ... There is no simple formula which can mask the necessity for examination of the particular facts.[53]

As explained below, the connections between the SGB and its premier-level athletes, such as the control exerted by the SGB over the playing rights of its athletes and reliance placed by those athletes on the SGB to utilise that power of control by taking reasonable steps to protect their well-being within the context of contact sport, that are germane in establishing a duty of care.

The High Court first adopted a 'salient features' methodology in 2001, stating in a joint judgment in *Sullivan v Moody*,

> [t]he relevant problem will … become the focus of attention in a judicial evaluation of the factors which tend for or against a conclusion to be arrived at as a matter of principle ... [C]onduct and relationships which may have been held not to give rise to a duty of care, and the reasons for that holding may provide an important guide to the solution of the problem in a new case.[54]

The methodology was based on several characteristics or features suggestive of a duty of care owed by an alleged tortfeasor to a plaintiff. The approach is one of 'incrementalism' rather than the proclamation of a new category of case. As said in commentary: '[A] court may look to those factors which may be common to the new case and to earlier cases in a similar (but not identical) general category. Where those factors have been influential in the resolution of the duty question in the past they will assist in a new case.'[55]

Although *Sullivan v Moody*, did not define a 'salient feature', several subsequent cases gave body to the concept. In *Graham Barclay Oysters v Ryan*, Kirby J referred to 'a range of factors, sometimes called salient features', which had been 'helpfully collected' by Callinan J in the same case: 'vulnerability, power, control, generality or particularity of the class, the resources

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50 *Caparo Industries Plc v Dickman* [1990] 2 AC 605.

51 (1999) 198 CLR 180, 286–91.

52 (1999) 200 CLR 1, 80–6 [223]–[235].

53 *Perre v Apand Pty Ltd* (1998) 198 CLR 180, 253.

54 *Sullivan v Moody* (2001) 207 CLR 562, 590.

55 P Stewart and A Stuhmcke, *Australian Principles of Tort Law* (The Federation Press, 5th ed, 2022) 198.

of, and demands upon the authority", the "core, or ... non-core" functions or relation to "a matter of policy or executive action" and so on'.[56]

In *Harriton v Stephens* Kirby J, drawing upon previous determinations, listed several factors that support or do not support the identification of a 'salient feature':

> [I]nstruction on the duty issue can be secured from several 'salient features' that have been identified as potentially relevant to the existence of a duty. In *Sullivan v Moody* three particular considerations were identified which will often point against the existence of a duty. These were (1) that finding a duty of care would cut across or undermine other legal rules; (2) that the duty asserted would be incompatible with another duty; and (3) that to recognise a duty would expose the defendant to indeterminate liability.
>
> Elsewhere, factors capable of supporting a duty of care have been identified. These include (1) vulnerability on the part of the plaintiff; (2) special control; or (3) knowledge possessed by the defendant about the circumstances that gave rise to the damage suffered by the plaintiff.[57]

Allsop P in *Caltex Refineries (Qld) Pty Ltd v Stavar* collated an extensive list of 'salient features' drawn from, and thereby carrying the authority of, the High Court of Australia. The 'salient features' included:

> (a) the foreseeability of harm; (b) the nature of the harm alleged; (c) the degree and nature of control able to be exercised by the defendant to avoid harm; (d) the degree of vulnerability of the plaintiff to harm from the defendant's conduct, including the capacity and reasonable expectation of a plaintiff to take steps to protect itself; (e) the degree of reliance by the plaintiff upon the defendant; (f) any assumption of responsibility by the defendant; (g) the proximity or nearness in a physical, temporal or relational sense of the plaintiff to the defendant; (h) the existence or otherwise of a category of relationship between the defendant and the plaintiff or a person closely connected with the plaintiff; (i) the nature of the activity undertaken by the defendant; (j) the nature or the degree of the hazard or danger liable to be caused by the defendant's conduct or the activity or substance controlled by the defendant; (k) knowledge (either actual or constructive) by the defendant that the conduct will cause harm to the plaintiff; (l) any potential indeterminacy of liability; (m) the nature and consequences of any action that can be taken to avoid the harm to the plaintiff; (n) the extent of imposition on the autonomy or freedom of individuals, including the right to pursue one's own interests; (o) the existence of conflicting duties arising from other principles of law or statute; (p) consistency with the terms, scope and purpose of any statute relevant to the existence of a duty; and (q) the desirability of, and in some circumstances, need for conformance and coherence in the structure and fabric of the common law.[58]

It is important to note that, as said by Allsop P, '[i]n essence the court focuses on the relationship between the plaintiff and the defendant identifying the salient features and using

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[56] *Graham Barclay Oysters Pty Ltd v Ryan* [2002] HCA 54, [236], referring to Callinan J at [321].

[57] *Harriton v Stephens* (2006) 226 CLR 52, [64]–[65].

[58] *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649, [103]. Relevant High Court cases include *Sullivan v Moody* (2001) 207 CLR 562; *CAL No 14 Pty Ltd v Motor Accidents Insurance Board* (2009) 239 CLR 390; *Stuart v Kirkland-Veenstra* (2009) 237 CLR 215; *Graham Barclay Oysters Pty Ltd v Ryan* (2002) 211 CLR 540.

those features to determine whether a duty of care should be imposed'.[59] It can be noted that 'knowledge' in reference to the conduct that caused harm is mentioned in both lists above.

On the question of imputing a duty of care in a novel case, Allsop P said the proper approach was to analyse the facts bearing on the relationship between 'the plaintiff and the putative tortfeasor by references to the "salient features" or factors affecting the appropriateness of imputing a legal duty to take reasonable care to avoid harm or injury'.[60]

The salient features approach to identifying a novel duty of care was again recognised recently by the High Court majority in *Mallonland v Advanta Seeds* where the sole issue for consideration was whether the producer of a hybrid grain sorghum seed owed the grower which used the seeds, which had become contaminated with a different seed known as shattercane, a duty to take reasonable care to ensure the growers did not sustain purely economic losses due to a hidden defect in the bags of seeds.[61] A caveat was nonetheless issued in reference to the statement in *Sullivan v Moody* that '"factors ... for or against" recognition of a duty of care in a novel case should not be understood as inviting any form of "instinctive synthesis" of competing considerations "without a chain of reasoning linking these factors with the ultimate conclusion"'.[62]

To illustrate the 'salient features' methodology, in *Laoulach v Ibrahim* the plaintiff, who became quadriplegic after diving into Botany Bay from a boat that had drifted into shallow water, argued that the boat's driver had 'taken on the responsibility of taking reasonable care to ensure that the vessel was securely anchored'.[63] The plaintiff relied 'particularly on salient features (c), (d) and (e) referred to by Allsop P in *Caltex*'.[64] The methodology, at least as expounded in *Laoulach v Ibrahim*, is to nominate the relevant salient features (for example, from those listed by Allsop P) and consider the degree to which the facts of the case align with those features. As said by Tobias AJA:

> [W]here the posited duty was a novel one, the proper approach was to undertake a close analysis of the facts bearing on the relationship between the plaintiff and the imputed tortfeasor by reference to the 'salient features' or factors affecting the appropriateness of imputing a legal duty to take reasonable care to avoid harm or injury.[65]

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[59] *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649, [102].

[60] Ibid.

[61] *Mallonland Pty Ltd v Advanta Seeds Pty Ltd* [2024] HCA 25.

[62] Ibid [37], referring to *Sullivan v Moody* (2001) 207 CLR 562, 579–80 [50].

[63] *Laoulach v Ibrahim* [2011] NSWCA 402, [107].

[64] Ibid [64]. That is, 'the degree and nature of control able to be exercised by the defendant to avoid harm', 'the degree of vulnerability of the plaintiff to harm from the defendant's conduct, including the capacity and reasonable expectation of a plaintiff to take steps to protect itself' and 'the degree of reliance by the plaintiff upon the defendant'.

[65] *Laoulach v Ibrahim* [2011] NSWCA 402, [60] (Giles and Macfarlan JJA agreeing), referring to the 'salient features' application proposed by Allsop P in *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649.

Clearly, more than one feature can operate in establishing a duty of care. In fact, Greenhow points to several active features in the boxing case *Watson v British Boxing Board of Control; World Boxing Organisation Inc*, mentioned above: 'Characteristics such as control, vulnerability, reliance, assumption of responsibility, knowledge, proximity and relationship were considered by the Court and influenced judicial decision-making.'[66]

Having said that, no single factor necessarily establishes a duty, nor is it 'compulsory in any given case to make findings about all these features'.[67] Rather, as Allsop P commented in *Caltex*, 'it provides a non-exhaustive universe of considerations of the kind relevant to the evaluative task of imputation of the duty and the identification of its scope and content'.[68] Clearly, additional facts may also be relevant in imputing a duty of care.

It is worth noting at this point that the High Court of Australia in *Sullivan v Moody* eschewed a test based on 'fair, just and reasonable', the concern being that after foreseeability is determined, 'the succeeding questions will be reduced to a discretionary judgment based upon a sense of what is fair, and reasonable as an outcome of a particular case'.[69] Davies and Malkin point out that '[a]lthough Allsop P's dauntingly long list does not include any explicit reference to policy considerations, it would be naïve to think that policy plays no part in the courts' consideration of duty questions'.[70] Nonetheless, the authors then, relevantly, limit the application as follows: '[T]he increasing importance of the salient features or autonomy and vulnerability can be seen as the expression of a judicial policy favouring increased self-reliance.'[71] They conclude that '[t]he role of policy is no longer as frankly stated as it has been in the past, but it has not disappeared completely under the salient features approach'. To emphasise the point: policy is stated as that relevant to 'self-reliance'. Further consideration of this point is beyond the scope of the chapter given the well-recognised limitations voiced by the learned authors.

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66 Annette Greenhow, 'Mitigating the Concussion Crunch: League Governance, Passive Actors and Regulators of Risk' (2021) 14(1) *Australian and New Zealand Law Journal* 36, 62.

67 *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649, 676 [104].

68 Ibid.

69 *Sullivan v Moody* (2002) 207 CLR 562, 579.

70 M Davies and I Malkin, *Torts* (LexisNexis Butterworths, 8th ed, 2020) 319

71 Ibid. The other 'policy' consideration was the undesirability of reopening cases that have already been resolved.

3.3.2 Scope and content also applicable under a salient features approach

Allsop P made the point that identification of salient features extends to questions of 'scope or content', such that it is 'an approach to be used to assist in drawing the conclusion whether in novel circumstances the law imputes a duty and, if so, in identifying its scope or content'.[72]

3.3.3 Salient features and cases below

Three sport cases concerned with duty of care are examined in detail below. In each case, it is argued, the court applied a 'salient features' approach to resolving the question of duty. In *Agar v Hyde*,[73] the High Court of Australia found that the board recommending rule changes for international rugby did not owe a duty of care to two injured rugby players. In *Watson v British Boxing Board of Control; World Boxing Organisation Inc*,[74] the England and Wales Court of Appeal found a duty of care in the governing organisation's relationship with its constituent boxers. In *Green v Country Rugby Football League of New South Wales Pty Ltd*,[75] the court found that the SGB owed a 'general duty of care' but had not breached that duty.

In each case, the overriding 'salient feature' was 'control'. In *Agar v Hyde*, 'control' was absent. In *Green*, 'control' existed but was limited, given the overriding power of the New South Wales Rugby League, which exercised ultimate authority over the Country Rugby Football League. In *Watson*, 'control' over the profession of boxing and its participants was near total.

3.4 *Agar v Hyde*: Distinguishing a sport case of 'no duty'

In *Agar v Hyde* the High Court of Australia determined that the rule-making body, the IRFB, did not owe a duty 'to take reasonable care in monitoring the operation of the rules of the game to avoid the risk of unnecessary harm to players'.[76] Given the authority of this judgment (and the extent to which it may apply in the present context), a plaintiff athlete suffering the symptoms of CTE must distinguish his or her case from that of *Agar v Hyde*. As argued in this chapter, the relationship between the players and the sporting organisation in *Agar*, in comparison to the players and SGBs in premier-level competitions in Australia, are so factually different as to preclude a relevant precedent. To foreshadow later discussion and for purposes

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72 *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649, 676 [104].

73 (2000) 201 CLR 552: see **[3.4]** below.

74 [2001] QB 1134: see **[3.5.1]** below.

75 [2008] NSWSC 26: see **[3.5.2]** below.

76 *Agar v Hyde* (2000) 201 CLR 552, [5].

of space and expedition, findings concerned with 'salient features' are emphasised in this section.

3.4.1 *Agar v Hyde*: A factual distinction with the SGBs of Australian sport

Agar v Hyde concerned two rugby players, both hookers in the scrum, who, in different competitions, were rendered quadriplegic when the front row of their scrum collided heavily with their opposition front row at engagement. The plaintiff players argued that the IRFB, an unincorporated body responsible for formulating the rules of rugby, was, along with each board member, negligent in failing to alter scrum laws to better protect their safety; or as described in the pleadings, failing to 'depower' the scrum.

The term 'salient features' does not appear in *Agar v Hyde*, however, the factual basis of the judges' determinations is that of a 'salient features' approach. The rationale of a refusal to recognise a duty of care is founded by and large on the absence of the salient features noted in the judgments cited above in **[3.3]**. As Stewart and Stuhmcke said of *Caltex Refineries (Qld) Pty Ltd v Stavar*,[77] '[f]eatures which the High Court regards as salient in determining the duty of care question will be those that link the parties or which operate to prevent the parties being linked'.[78]

In evidence in *Agar v Hyde* was an article from the *Medical Journal of Australia* that reviewed spinal cord injuries to 107 rugby players over a period of several years. The article stated that 'collision at scrum engagement caused the majority of such injuries' and that such injuries 'were largely preventable by appropriate rule changes'.[79] The article, which was published in August 1987, just a few weeks after the plaintiffs were injured, led in 1988 to changes to the scrummaging laws requiring the front rows of opposing scrums to 'crouch, touch, pause' before engagement to avoid head-on-chest contact.

Although the rules as formulated by the IRFB exposed players to the risk of injury, and indeed 'from time to time … are changed with considerations of safety in mind',[80] Gleeson CJ concluded, that '[i]t does not follow that the members of the Board were under a legal duty of care to the players of the game *of the kind presently alleged*'.[81] In essence, the 'control attributed to the members of the Board was remote from the respondents'.[82] The members of the IRFB did not contemplate a legal responsibility to hundreds of thousands of players

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77 (2009) 75 NSWLR 649.

78 P Stewart and A Stuhmcke, *Australian Principles of Tort Law* (The Federation Press, 5th ed, 2022) 201.

79 See *Hyde v Agar; Worsley v Agar* (1998) 45 NSWLR 487, 492 (Court of Appeal).

80 *Agar v Hyde* (2000) 201 CLR 552, [10].

81 Ibid [20]. Emphasis added.

82 Ibid [21].

worldwide with liability 'practically indeterminate in extent'.[83] Given the factual circumstances, it was not reasonable that players should rely on the IRFB to formulate the suggested safety rules.

3.4.2 The fact of an absence of 'salient features' underpinning the decision in *Agar v Hyde*

The circumstances — in fact, 'salient features' — argued here as guiding the court in *Agar v Hyde* to determine that the IRFB did not owe a duty of care to the players are discussed below. These salient features (essentially those cited by Allsop P is *Caltex v Stavar*) were absent in *Agar v Hyde* but, as argued, are present in the relationship between SGBs of major sport in Australia and their premier-level athletes.

A Insufficient control and relationship

According to Gleeson CJ, 'the control attributed to the members of the Board was remote from the respondents, and extended only to participating in, or having the capacity to participate in, a process of laying down the conditions of a sporting contest in which people might voluntarily engage'.[84] On the facts, a duty of care of the type claimed could not be established because

> no individual appellant controlled the game internationally. No individual appellant was a lawgiver. … To speak of persons who were sent once a year to London, as representatives of national unions, as controlling a game of football played in a Sydney suburb, or a country town, by reason of their collective capacity to alter the international rules, is to speak of a remote form of control.[85]

Callinan J commented similarly that while the members were empowered to change the laws for the purposes of the IRFB, they could 'not compel the home unions to adopt them'.[86] His Honour also stated that in the circumstances, including the IRFB's 'answerability to and relationship with their home unions' and 'the distance in time, place, and contemplation between the respondents playing in the games in which they were injured and the appellants', a duty of care did not arise.[87]

B Insufficient assumption of responsibility

The remoteness between the rule-makers and the injured litigants was also emphasised by Gaudron, McHugh, Gummow and Hayne JJ:

> The complaint is that [the IRFB members] failed to alter the status quo, failed to alter the rules under which the respondents voluntarily played the game. In our view, they no more owed a duty of care

[83] Ibid.

[84] Ibid [20].

[85] Ibid [16].

[86] Ibid [123].

[87] Ibid [127].

> to each rugby player to alter the laws of rugby than parliamentarians owe a duty of care to factory workers to amend the factories legislation.[88]

Similarly, Callinan J spoke of 'the distance in time, place, and contemplation between the respondents playing in the games in which they were injured and the appellants'.[89]

C Insufficient reliance and vulnerability

Because the IRFB could not control relevant aspects of the game, it was not reasonable to rely on its members to secure a safer game by altering the rules. Indeed, according to Gleeson CJ, factors beyond 'the rules' may be a more likely cause of harm:

> As to reliance, the sporting contest involved an obvious risk of injury; a risk that would be affected by a number of factors, including the attitudes, capacities, and propensities of individual players, which are beyond the influence of the appellants.[90]

D A general duty of care under different circumstances?

On the question of ameliorating 'unnecessary risks' through an alteration to the rules of play, Gleeson CJ stated:

> Of course, the rules of the game affect the players of the game. It is equally clear that the rules will expose the players to the risk of injury in the sense that they will lay down the conditions of a physical contest in which some people are likely to be hurt. In that respect, harm is *foreseeable*. It does not follow [however] that the members of the Board were under a legal duty of care to the players of the game *of the kind presently alleged*.[91]

Gleeson CJ indicated nonetheless that while harm was foreseeable, 'neighbourhood', given the circumstances of the case, could not be established:

> It cannot be denied that … players of rugby football are so closely and directly affected by what the IRFB does that members of the Board ought to have them in contemplation as being so affected, but neighbourhood in that sense is not the issue in the present case.[92]

While the High Court determined that the IRFB did not owe the plaintiffs a duty of care, it did *not* eschew the possibility of liability altogether or in circumstances different to those experienced by the plaintiffs. The absence of 'neighbourhood' was confined to 'the kind presently alleged': altering the rules of scrummaging to reduce the risk of injury.[93] This is not,

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88 Ibid [69].

89 Ibid [127].

90 Ibid [21].

91 Ibid [20]. Emphasis added.

92 Ibid.

93 It is perhaps arguable on the basis that the IRFB was 'remote' from the players concerned that an Australian SGB, which is not remote, may possess a duty to alter the rules of play for purposes of safety. However, such argument is difficult to sustain given comments in several judgments on the acceptance of 'obvious risk'.

though, the language to establish a category of duty, and may arguably suggest some other 'kind' of neighbourhood could be recognised.

3.4.3 *Agar v Hyde*: Contrasts with the major SGBs in Australia

None of the fact-based reasons by which the defendants in *Agar v Hyde* were found not to owe a duty of care are applicable to the elite competitions administered by the premier SGBs in Australia. Quite the opposite. Consider the facts ('salient features') in *Agar* in comparison to the facts ('salient features') that characterise the relationship between the governing organisations of sport in Australia and their elite players:

- The SGBs of major sporting organisations in Australia control the game at the premier level of competition.
- The governing board meets domestically and regularly.
- Each individual player registers to play with the governing organisation.
- Each elite player's name is almost invariably known to each board member.
- Board members are 'law-makers' with respect to creating the rules of play.
- Players reasonably rely on the SGB to be apprised of relevant threats to their health, investigate these threats and warn of any material risk.

It is also worth noting that board members and other officials controlling the major contact sports in Australia are commonly appointed because of their interest and expertise in the sport they administer. Board members are often ex-players and coaches. For example, in 1994 Dr Nathan Gibbs, ex-player in the NRL competition, expressed concern in the journal *Sports Medicine* of the possible risk of '*dementia pugilistica*' in rugby league.[94] At that time, Ken Arthurson, an ex-Australian and New South Wales representative player and coach of the Manly Club in Sydney, was the Chairman of the Board of the Australian Rugby League. In that same year, the Chairman and CEO of the AFL, Ross Oakley, was a veteran player with the St Kilda Football Club in Melbourne. In most cases, board members are extremely familiar with all aspect of their sport and its elite participants.

3.4.4 Is a 'governing body' case an 'accepted category'?

Given the High Court's finding in *Agar v Hyde*, can it be said that an 'accepted category' is now operative in Australia in respect to a duty of care owed by governing bodies to their

[94] Nathan Gibbs, 'Common Rugby League Injuries' (1994) 18(6) *Sports Medicine* 438, 445.

premier players?[95] Three rationales are considered in arguing that *Agar v Hyde* did not create an 'accepted category' of case in negligence:

1. *Agar v Hyde* did not concern a 'governing body', a hallmark of which is control over all or almost all facets of the sport, but rather a 'board' the power of which in essence did not extend beyond recommending rugby rules of play.

2. Should *Agar v Hyde* be considered to have formed an 'accepted category', it is confined to cases with similar facts — facts that do not apply to 'true' governing bodies and is not relevant to the question of duty discussed in the chapter.

3. Should *Agar v Hyde* not be considered to have created an 'accepted category', a pure 'salient features' methodology concerned with the facts, such as those extracted from High Court determinations by Allsop P in *Caltex Refineries (Qld) Pty Ltd v Stavar*,[96] is applicable.

3.4.5 Was the IRFB 'really' a governing body?

The question of whether *Agar v Hyde* is 'really' a 'governing body' case is briefly considered. If it is not, the implication is clear: a 'governing body' case remains 'novel', to be determined under a salient features methodology.

Some commentary indicates that *Agar v Hyde* established a form of 'accepted category' of case by which an SGB of an Australian sport did not owe a duty of care to relevant players. For example, Opie states:

> In *Agar v Hyde* the High Court of Australia dealt with a novel claim that has important implications for the role of preventive medicine in sport … Sport governing bodies routinely review their sport's rules to assess, inter alia, issues of safety. Some sports have standing committees of scientific and coaching experts for this purpose. Rule changes are often the result and may bring changes in the inherent risks of participation. Notwithstanding the element of responsibility suggested by the foregoing, the High Court has in effect held that this is a moral, not a legal, responsibility of sport governing bodies. … Unfortunately, the High Court in large measure has taken away that prospect and the incentives for safe behaviour …[97]

It seems, according to Opie, that *Agar v Hyde* suggests that governing bodies are not responsible for the well-being of their players. In similar vein, Gelber comments in her article 'Suing the AFL for Negligence' that her main focus is 'on establishing how an AFL class action could proceed, given the denial of the existence of a duty of care owed by sport governing

[95] This section is placed in this position because the question requires familiarity with the facts and rulings in *Agar v Hyde* (2000) 201 CLR 552.

[96] (2009) 75 NSWLR 649.

[97] Haydon Opie, 'Australian Medico-Legal Issues in Sport: The View from the Grandstand' (2002) 13(1) *Marquette Sports Law Review* 113, 118–19.

bodies to participant players regarding the risk of injury that was laid down by the High Court of Australia in *Agar v Hyde*'.[98]

In fact, as argued here, the IRFB was *not* a governing body of rugby. Apart from what might be called the definition of a governing body, this fact is clear in the *Agar v Hyde* judgment itself. Consider the briefly presented arguments below.

A What is a 'governing body'?

There is no definition of a 'governing body' of a sport; rather, it is identified through its traits. Principally, as the name suggests, a governing body is empowered to exercise governance over an organisation, the basic feature of which is to 'control'. The history and methods of 'control' are discussed briefly in **[3.6]** below, which notes several findings of courts disclosing just such a feature.[99] The Australian Sports Commission said of the somewhat broader, yet informing, term 'governance' that '[a]t its most basic, governance is the system by which organisations are directed and controlled'.[100] The Australian Sports Commission to accept a sporting organisation as a governing body requires that it 'demonstrate it represents all forms of the sport from grassroots/community to high performance' and that it 'is accountable for the selection or nomination of teams to represent Australia in events recognised by its International Federation'.[101] These criteria are accurate descriptors of the major governing bodies of contact sport in Australia. For example, the AFL Commission calls itself 'the custodian of Australian Football',[102] while Cricket Australia says it is 'the national governing body for the game of cricket in Australia'.[103] The Australian Rugby League Commission describes itself as the 'single controlling body and administrator of Rugby League in Australia'.[104]

B A suit against individuals

The plaintiffs in *Agar v Hyde* sued the members of the IRFB as 'individuals'; the IRFB itself was not the subject of the High Court appeal — both points indicating that that the IRFB was

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[98] Henrietta Gelber, 'Suing the AFL for Negligence: An Analysis of the Process and Prospects of a Potential Class Action Proceeding Based on Concussion-related Injuries Sustained in AFL Football' (2021) 14(1) *Australian and New Zealand Law Journal* 108, 108.

[99] See, eg, *Buckley v Tutty* (1971) 125 CLR 353; *News Ltd v Australian Rugby Football League* (1996) 139 ALR 193.

[100] 'The Startline', *Australian Sports Commission* (Web Page) <https://www.sportaus.gov.au/governance/principles/the-startline>.

[101] 'Recognition of National Sporting Organisations', *Australian Sports Commission* (Web Page) <https://www.sportaus.gov.au/recognition_of_national_sporting_organisations/_nocache#recognition_criteria_july_2022>.

[102] 'Corporate Governance', *Australian Football League* (Web Document) <https://resources.afl.com.au/afl/document/2019/11/15/e970874a-504d-421d-b6a1-471cc3a303ab/AFL-Corporate-Governance.pdf>.

[103] 'Governing the Game', *Cricket Australia* (Web Page) <https://www.cricket.com.au/governing-the-game>.

[104] 'About', *National Rugby League* (Web Page) <https://www.nrl.com/about-us/>.

not an SGB. As Gleeson CJ pointed out, '[n]o individual appellant could amend the rules. No individual appellant controlled the game internationally. No individual appellant was a lawgiver.'[105] Callinan J said on the same issue: 'The appellants are, with the exception of the [New Zealand Rugby Football Union], all natural persons representing the major rugby union playing nations of the world who attended in a voluntary capacity the annual meeting.'[106]

C Absence of control

A hallmark of an SGB is the capacity to 'control' — either completely or to a large extent — the rules and protocols of the game it administers. The basis of the plaintiffs' case was that the 'members' of the IRFB were negligent in not 'depowering' scrums through rule alteration. On this, Gleeson CJ said:

> To speak of persons who were sent once a year to London, as representatives of national unions, as controlling a game of football played in a Sydney suburb, or a country town, by reason of their collective capacity to alter the international rules, is to speak of a remote form of control.[107]

In fact, as considered above, the members of the IRFB, and the IRFB itself, had no control over — nor could they compel — the implementation of the rules of rugby in Australia. His Honour went further, stating that 'control attributed to the members of the Board was remote from the respondents, and extended only to participating, or having the capacity to participate, in a process of laying down the conditions of a sporting contest in which people might voluntarily engage'.[108]

Callinan J also stated that the 'natural persons who are [the defendants]' could only 'change the laws for the purposes of the IRFB and seek to influence, but not compel the home unions to adopt them'.[109]

D Control over the game, its administration, rules and protocols

The IRFB was not the 'controller' of rugby in Australia, and possessed no control over the game, its administration, or the implementation of its rules, policies and protocols. The facts that establish the salient feature of 'control' are discussed in detail below.[110] The presence of this salient features in the case of an Australian SGB is, as argued, in contrast with the absence of this same features in the IRFB.

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[105] *Agar v Hyde* (2000) 201 CLR 552, [16].

[106] Ibid.

[107] Ibid.

[108] Ibid [21].

[109] Ibid [123].

[110] See **[3.6]**.

3.5 Sport cases that establish a duty of care in the SGB

This section considers cases where a duty of care was found to reside in the SGB of a contact sport and where the courts in so deciding applied a salient features approach.

In *Watson v British Boxing Board of Control; World Boxing Organisation Inc*,[111] the court, although not using the term, applied a 'salient features' approach in deciding that the SGB owed a duty of care to its constituent athletes. Again, the argument is made that the salient features that led to the recognition of a duty of care in *Watson* share a commonality with those characterising the relationship between the SGBs of contact sport in Australia and the athletes who participate in their premier competitions.

3.5.1 *Watson v British Boxing Board of Control*: Express and implied obligations of care

The facts in *Watson v British Boxing Board of Control; World Boxing Organisation Inc*,[112] from which the Court of Appeal of England and Wales found that a duty of care resided in the SGB, are similar — and in some respects identical — to those describing the relationship between the SGBs of contact sport in Australia and the athletes who play in their elite competitions.

The plaintiff boxer, Watson, suffered permanent brain damage, physical and mental disability, and was paralysed down the left side of his body following a World Boxing Organisation Super-Middleweight title bout. The referee stopped the fight in the final round, when the plaintiff was unable to defend himself. On returning to his corner the plaintiff, who had suffered a brain haemorrhage, collapsed unconscious. The plaintiff successfully claimed that the British Boxing Board of Control (BBBC) breached its duty of care to him in failing to provide immediate post-injury medical assistance when it had impliedly and expressly undertaken to do so.[113] The BBBC was a company limited by guarantee and the sole controlling body regulating professional boxing in the United Kingdom; 'There is no statutory basis for [the BBBC].'[114] Not unlike the authority exercised by elite Australian contact sports, the BBBC controlled the rules and protocols pertinent to 'boxers, promoters, managers, referees, timekeepers, trainers, seconds, masters of ceremonies, match-makers, agents for overseas

[111] [2001] QB 1134.

[112] Ibid.

[113] Lord Phillips MR noted: 'Because the facts of this case are so unusual, there is no category in which a duty of care has been established from which one can advance to this case by a small incremental step. In these circumstances the task is to look at the circumstances in which specific factors have given rise to the duty of care and to consider whether, on the facts of this case, they should also give rise to such a duty': *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [86].

[114] *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [12].

boxers, ringmasters and whips', all have whom had 'to be licensed by the board to perform their particular functions and [became], when granted their licences, members of the board'.[115] Contracts between boxer and manager, and boxer and promoter, were in standard form, and expressly provided that the parties will observe the BBBC's rules.

A Assuming responsibility for medical care of boxers

Lord Phillips MR (May and Laws LJJ in agreement) found that a duty of care was established by the BBBC promulgating extensive rules and medical protocols designed to protect the health of the boxers under its administration.[116] The BBBC's concern with well-being was expressed in several sources, both generally and specifically. For example, the Articles of Association stated that an object of the BBBC is to

> promote and safeguard the interests of members of the company in the United Kingdom and throughout the world including members' (being boxers) interests in boxing contests and tournaments … including … the encouragement and promotion of safety standards …[117]

Furthermore, a witness statement of the BBBC's General Secretary stated: 'The Board believes as I do, that the safety of the boxers is of great importance and takes precedence over commercial and other interests.'[118]

In an open letter to the British Medical Association, the Chief Medical Officer to the BBBC, Dr Whiteson, wrote: 'The British Boxing Board of Control is justifiably proud of its reputation of being in the vanguard of the protection of professional boxers.'[119] Some time later, he stated: 'As Chief Medical Officer, my approach has always been that preventative controls are the key to making a physically hazardous sport as safe as possible … [O]ur interest in preventative controls covers the whole gamut of professional boxing.'[120] In addition, Dr Whiteson commented in an article on injuries to professional boxers that his task was to 'control the medical aspects of boxing' and to liaise with 'medical experts which includes neurologists'.[121]

Concerns as to the physical safety of boxers extended to the BBBC's rules and regulations.[122] There were three categories of rules: 'rules designed to ensure that a boxer is not permitted to fight unless he is fit', 'rules designed to restrict the physical injuries that may

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[115] Ibid [13].

[116] A boxing promoter 'is responsible for organising all aspects of the fight from the date to the venue, fighters purse, and of course promotion': 'Boxing Promotions', *Fight.com.au* (Web Page) <https://www.fight.com.au/find/boxing-promotions/>.

[117] *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [17].

[118] Ibid [17]

[119] Ibid [22].

[120] Ibid.

[121] Ibid [23].

[122] Ibid [17].

be caused in the course of the fight' and 'rules designed to secure that a boxer receives appropriate medical attention when injured in the course of a fight'.[123]

The third category required the adoption of specific rules designed to minimise harm to boxers, and was noted by Phillips MR as being 'of particular importance in the context of this action'.[124] The relevant rules included that '[t]he *promoter shall procure* that two doctors, who must be approved by the Area Medical Officer, attend at all promotions, one of whom must be seated at the ringside at all times during the contest'; that 'each boxer must … be examined after every contest and a report sent to the Board or Area Council concerned if necessary'; that '[a] doctor must be available to give immediate attention to any boxer should this be required'; and that '[t]he promoter shall procure that at all promotions a stretcher is available for use near the ring'.[125]

The first category included several rules relating directly to the care of boxers and from which, in conjunction with other safety protocols, a reasonable boxer could conclude that the BBBC was 'comprehensive' in regulating the medical care in the boxing profession. The relevant rules included that 'a licence may be suspended or withdrawn if … the licence holder is not medically fit to box'; that 'if a fight is stopped by the referee or a boxer is counted out, the boxer's licence is suspended for at least 28 days and until the boxer is certified fit to box by a doctor'; that 'any boxer selected to take part in a championship contest shall submit to the Board a satisfactory centralised tomography (CT) brain scan report not less than 28 days before the contest and a further scan report annually'.[126]

The BBBC, collectively and as individuals, did not undertake to provide medical assistance at matches but contractually assigned that responsibility to the fight promoter who was, in addition, required to provide to injured boxers the specific medical assistance the BBBC had mandated. In similarity with Australian contact sport, other than confirming the fitness of players for representative teams, an SGB does not generally procure medical services for players, but contractually assigns the obligation to district clubs.[127]

Phillips MR found that in the circumstances, it was reasonable that Watson relied on the BBBC's undertaking to care 'comprehensively' for the health and safety of its boxers. Had Watson, say, appointed his own medical team at ringside (a not uncommon event in championship bouts) the guarantees of the BBBC would not be operative. On this question, Phillips MR said:

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[123] Ibid [17].

[124] Ibid [20].

[125] Ibid. Emphasis added.

[126] Ibid [18].

[127] It can also be noted that the BBBC, through the recommendation of a Chief Medical Officer, was responsible for approving doctors to attend a fight.

> The Board had, or had available, medical expertise. The Board held itself out as treating the safety of boxers as of paramount importance. It carried out this function by making and imposing rules dealing with the safety of boxers, by approving medical officers and by giving detailed guidance as to the qualifications and equipment those officers should bring to the ringside. Boxer[s] … including Mr Watson, could reasonably rely upon the Board to have taken reasonable care in making provision for their safety … Thus the Board was a body with special knowledge giving advice to a defined class of persons in the knowledge that it would rely upon that advice in the defined situation of boxing contests. … [T]he judge was entitled to conclude that there was in this case reliance by Mr Watson on the exercise of skill and care by the Board in looking after his safety. This is a further factor which tends to establish the proximity necessary for a duty of care.[128]

To the extent that *Watson* provides a guide to the application of a 'salient features' approach to contact sport in Australia, the assurances of 'care' by Australian SGBs are — in contrast to *Watson* — more general; but in the totality of circumstances they are argued, nonetheless, to have induced athletes to reasonably rely on their SGBs to take reasonable steps to mitigate the risks and emerging risks of harm in the playing of their sport.

B The fact of 'salient features'

Phillips MR found that there was no category of case from which a duty of care could be established as 'a small increment'. Therefore his Lordship looked to specific factors in previous cases that had been found to create a duty of care and considered whether, 'on the facts of this case, they should also give rise to such a duty'.[129] In doing so, Phillips MR drew upon criteria identified by Hobhouse LJ in *Perrett v Collins*[130] indicative of 'the existence of a duty of care' and applied these to Watson's circumstances of harm.[131] These factors, several aligning with the 'salient features' extracted from High Court of Australia cases and promulgated by Allsop P in *Caltex Refineries (Qld) Pty Ltd v Stavar*[132] — which were largely absent in in *Agar v Hyde*[133] and *Green v Country Rugby Football League of New South Wales Pty Ltd*[134] — included the following:[135]

1. Mr Watson was one of a defined number of boxing members of the Board.

2. A primary stated object of the Board was to look after its boxing members' physical safety.

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[128] *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [85].

[129] Ibid [86].

[130] [1998] 2 Lloyd's Rep 255, 262.

[131] *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [87].

[132] (2009) 75 NSWLR 649, 676, [102]–[104].

[133] (2000) 201 CLR 552.

[134] [2008] NSWSC 26.

[135] *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [87].

3. The Board encouraged and supported its boxing members in the pursuit of an activity which involved inevitable physical injury and the need for medical precautions against the consequences of such injury.

4. The Board controlled every aspect of that activity.

5. In particular, the Board controlled the medical assistance that would be provided.

6. The Board had, or had access to, specialist expertise in relation to appropriate standards of medical care.

7. The Board's assumption of responsibility in relation to medical care probably relieved the promoter of such responsibility. If Mr Watson has no remedy against the Board, he has no remedy at all.

8. Boxing members of the Board, including Mr Watson, could reasonably rely upon the Board to look after their safety.

In summary, Phillips MR found that had the Board simply given advice 'to all involved in boxing' as to 'appropriate medical precautions', it is unlikely a duty would have arisen.[136] However, the Board 'went far beyond this'; it made 'provision in its rules for the medical precautions to be employed and made compliance with these rules mandatory'.[137] In this sense the Board 'arrogated' to itself the task of 'determining what medical facilities' are to be provided and required the 'boxer and the promoter to contract on terms under which the Board's rules will apply'.[138] In so doing,

> the Board reduces this aspect of the promoter's responsibility to the boxer to the contractual obligation to comply with the requirements of the Board's rules in relation to the provision of medical facilities and assistance. The Board assumes the responsibility of determining the nature of the medical facilities and assistance to be provided.[139]

His Lordship concluded that '[t]hese facts bring the Board into close proximity with each individual boxer who contracts with a promoter to fight under the Board's rules'.[140]

3.5.2 *Green v Country Rugby Football League*

In *Green v Country Rugby Football League of New South Wales Pty Ltd*[141] the controlling authority, the Country Rugby Football League (CRL), was argued to have negligently failed to take reasonable steps to introduce safety rules and to warn participants of the possibility of neck injury. The plaintiff, Shane Green, a 16-year-old schoolboy playing hooker in an open-

[136] Ibid [82].

[137] Ibid [82].

[138] Ibid [79].

[139] Ibid [79].

[140] Ibid [80].

[141] [2008] NSWSC 26.

age rugby league game, suffered tetraplegia following a scrum collapse in a second-grade match for the Laurieton Rugby League Club against The Old Bar Pirates.[142]

The relevance of *Green* is the court's recognition that the CRL 'owed the plaintiff a duty to take reasonable care'[143] or, as otherwise expressed, 'if a duty arose, it was a general duty of care'.[144] Ultimately, however, Walmsley AJ found that a failure to take steps to make scrummaging safer was not a breach of duty.[145]

A Why did the CRL owe a general duty of care?

The facts, or salient features, promoting Walmsley AJ to find that the CRL 'owed the plaintiff a duty to take reasonable care'[146] include the following:

> (a) CRL was ultimately responsible, within rugby league circles, for administering the sport in country New South Wales, although it did not organise games, or rosters, choose coaches or select teams;
>
> (b) the plaintiff's club and Group Three [competition] were constituents of CRL. Under their constitutions, whilst they had common objects, when it came, for example, to their amending their by-laws, CRL had the power to disallow the amendments [to depower the scrum];
>
> (c) CRL arranged insurance for all country players and insisted players be registered with their clubs, and, to ensure cover, that the registration forms be sent to it;
>
> (d) the plaintiff was a country rugby league player who played in a competition organised by CRL's constituent; …[147]

These salient features apply even more compellingly to a premier organisation with total control over the game, its rules and protocols, and material aspects of professional and private lives of its elite players — as do the SGBs of contact sport in Australia.

3.6 SGBs and athletes: A 'duty' relationship

The SGBs of major sport in Australia do not 'just organise' their elite competitions, nor are they the 'passive actors' described in *Green v Country Rugby Football League of New South*

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[142] Of note, the harm to Shane Green occurred in 1994, but the judgment of Walmsley AJ in *Green v Country Rugby Football League of New South Wales Pty Ltd* [2008] NSWSC 26 was not delivered until 2008. During that time, rugby authorities had in fact introduced rule changes and training protocols designed to minimise the risk of spinal injury, in part because of the injury to Green. The *Green* case discusses some of these measures.

[143] *Green v Country Rugby Football League of New South Wales Pty Ltd* [2008] NSWSC 26, [94].

[144] Ibid [85].

[145] While Walmsley AJ found that a general duty of care existed, he concluded that 'no breach is established' (at [130]) and added: 'I do not accept the submission. I am not persuaded reasonable care required [depowering]' (at [138]).

[146] *Green v Country Rugby Football League of New South Wales Pty Ltd* [2008] NSWSC 26, [94].

[147] Ibid [92]. Other factors were specific to *Green*: that the CRL organised coach accreditation courses, knew some were under 18 years of age, knew some front rowers were slight in physique, and knew some coaches had not taken coaching courses.

Wales Pty Ltd.[148] They are a controlling force in the professional and personal lives of the players who compete under their banner. Through 'control', other salient features listed from High Court authority by Allsop P in *Caltex Refineries (Qld) Pty Ltd v Stavar*[149] ensue, ranging from vulnerability to reliance, alongside several others. Legal commentary has also considered a 'salient features' approach relevant to the duty question and is discussed below. Greenhow suggests that 'the duty relationship between a governing body and professional players falls outside the established duty relationships and presents a novel case for consideration'.[150]

3.6.1 A matrix of 'salient' facts

Unlike one or two determinative salient features in a case such as *Laoulach v Ibrahim*[151] discussed above at **[3.3.1]**, the establishment of a duty of care in premier contact sport concerns a matrix of facts that underpin several salient features.

McHugh J spoke of a coactive relationship between several salient features in *Perre v Apand Pty Ltd*:

> In my view, reliance and assumption of responsibility are merely indicators of the plaintiff's vulnerability to harm from the defendant's conduct, and it is the concept of vulnerability rather than these evidentiary indicators which is the relevant criterion for determining whether a duty of care exists.
>
> Reliance may therefore be seen — for the purposes of duty of care — as an indicator of vulnerability: the plaintiff is specially vulnerable to the words and/or conduct of the defendant because he or she reasonably relied on the defendant. Reliance may also, of course, be relevant to causation. In terms of a duty of care, however, it is not reliance that is relevant, but its consequence, vulnerability.[152]

A question not addressed by Allsop P in *Caltex Refineries (Qld) Pty Ltd v Stavar*[153] is why 'control' would necessarily disclose a duty of care. Why should control not be utilised by the parent body solely for its own benefit? Most basically, 'control' permits the governing body of a sport to alter the rules of play to ameliorate risk of harm, to mandate protocols such as 'head injury assessment' and compulsory time out of play, and to issue warnings of material risks. Part of the reason also includes, as proposed here, an expectation or reliance in the minds of players that the organisation's power to control will be reasonably exercised with their health and welfare in mind. This proposition flows from the matrix of contractual and other

[148] [2008] NSWSC 26.

[149] (2009) 75 NSWLR 649, [103].

[150] Annette Greenhow, 'Mitigating the Concussion Crunch: League Governance, Passive Actors and Regulators of Risk' (2021) 14(1) *Australian and New Zealand Law Journal* 36, 59. See also Henrietta Gelber, 'Suing the AFL for Negligence: An Analysis of the Process and Prospects of a Potential Class Action Proceeding Based on Concussion-related Injuries Sustained in AFL Football' (2021) 14(1) *Australian and New Zealand Law Journal* 36, 120.

[151] [2011] NSWCA 402, [107].

[152] *Perre v Apand* (1998) 198 CLR 180, [125]–[126].

[153] (2009) 75 NSWLR 649, [103].

interactions between the governing organisation and its premier players, including the salient feature 'proximity or nearness in a physical, temporal or relational sense of the plaintiff to the defendant'.[154] Unlike the circumstances supporting the judgment in *Agar v Hyde*[155] the SGBs of Australian contact sports possess, as proposed, a 'relational proximity' to their premier-level athletes.

In exercising control, the lives of their athletes are both directly and collaterally affected. In *Watson* the undertaking moved from a set of rules to rules that created reliance — for example, a reliant boxer may proclaim to himself: 'I will not engage my own medicos to attend the fight, because you are supplying medicos properly trained for boxing matches.'

A *Agar* and *Watson*: A 'salient features' approach?

While the courts in both *Agar v Hyde*[156] and *Watson* found that no accepted duty category covered the cases before them, the determination of each court with respect to a duty of care was different. In *Agar*, the High Court of Australia found that the defendants — who were members of the IRFB, an organisation responsible for formulating rules of rugby — did not owe a duty of care to the plaintiffs, who were harmed in a scrum collapse. In *Watson*, the Court of Appeal found that a duty of care *was* owed in the circumstances by the BBBC to an injured boxer. Two Courts of Appeal, one of which is the final legal arbiter in Australia, decided differently as to the liability of two different SGBs.

As argued, however, while the judicial determinations in each case differ, the facts relevant to a 'salient features' approach are fundamentally similar. For example, the recognised salient feature of 'control'. In *Agar v Hyde*, this 'salient feature' was considered, but found absent. In *Watson*, 'control' was a determinative fact in recognising a duty. In *Agar v Hyde*, although the IRFB (and its members) were empowered to change the laws of rugby it could 'not compel the home Unions to adopt them'[157] — a fact indicative of an absence of 'control'. In *Watson*, in contrast, the BBBC controlled all aspects of the sport, including its rules, the registration of boxers and licensing of masters of ceremonies, and many more.[158] In other words, both the absence or presence of a salient feature goes to the question of 'duty'.

3.6.2 Control over the hazards of play

What is meant by 'control' as a 'salient feature'? Allsop P described it as 'the degree and nature of control able to be exercised by the defendant to avoid harm'[159] — in context, the capacity to

[154] *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649, [103].

[155] (2000) 201 CLR 552.

[156] Ibid.

[157] *Agar v Hyde* (2000) 201 CLR 552, [123] (Callinan J).

[158] *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [13].

[159] *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649, [103].

control the hazard of head trauma faced by a plaintiff athlete. Of note, the absence of meaningful control was a determining fact in *Agar v Hyde*. Although the IRFB proposed changes to the rules of rugby, the Australian Rugby Union maintained the power to accept or reject those changes as it wished; as such, there was no control by the IRFB of the domestic rules of play; nor over the implementation of the IRFB rules. In contrast, the governing bodies of contact sport in Australia exercise near total control over their sport, suggesting that ameliorating risk is merely a matter of that body deciding to do so.[160]

A Limitations on 'control'?

It is important to realise that the exercise of 'control', as it is directed to a sport's rules and protocols, is confined to 'reasonable steps' within the context of the contact sport under consideration. As argued in depth in Chapter 4 at **[4.5]**, civil liability legislation concerned with 'obvious risk' has firmly established that the obligation to take reasonable steps in amelioration is unlikely to extend to the removal or even mitigation of harm that is unavoidable in contact sport. Gleeson CJ in *Agar v Hyde* conveyed the point that '[t]he only way to avoid risk of injury is not to play'.[161] Those who are fully aware of the risks of play must be taken to have accepted that risk; risk is the very nature of contact sport.

However, a possible exception, Windholz points out, is the initiation of safety protocols that do not alter the nature of the sport but rather are collateral to it, such as '[r]ules that protect the head from unnecessary collision, heat policies that prevent play in extreme conditions, padding on goal posts, and pitch inspections to ensure the safety of the playing surface'.[162]

There is of course a legal difference between an elite athlete playing at premier level who accepts the known and obvious risks of a sport and, as argued in this thesis, the obligation of an SGB to be informed of emerging risks in the sport it controls and to warn athletes where those risks are material.

3.6.3 The base contractual means of 'control'

'Control' must include the power vested in an SGB to mandate adherence to its protocols.[163] Power to this degree reveals a capacity to 'control' the athletes who play and, given the reality of contact sport, the risks of harm those athletes face. The securing of control by the organisation, concomitantly generates reliance by athletes that the organisation to fill any void resulting from the athlete's loss of control — particularly in relation to those hazards that pose

[160] An exception may apply to Football Australia, which must adhere to certain protocols mandated by FIFA, the international controlling body. This does not, of course, interfere with the issuance of certain protocols and warnings of harm.

[161] *Agar v Hyde* (2000) 201 CLR 552, [18].

[162] Eric Windholz, 'Professional Sport, Work Health and Safety Law and Reluctant Regulators' (2015) *Bond University Sports Law ejournal* 7.

[163] For example, 'control' leading to 'reliance' was, as noted, lacking in *Agar v Hyde* (2000) 201 CLR 552.

risks to health and welfare. In *Perre v Apand Pty Ltd*, an economic loss case, McHugh pointed out a broader concern that

> the vulnerability of the plaintiff may often be a justifiable, but not sufficient, reason for imposing a duty of care in cases of negligence resulting in pure economic loss where the plaintiff could not have protected itself in contract. One of the assumptions of the law of contract, for example, is that the parties can bargain to protect their interests. A plaintiff who is vulnerable — for whatever reason — cannot do this in any meaningful way. In its quest for corrective justice, the law of negligence may be able to fill the gap which the law of contract has left out.[164]

3.6.4 Contractual terms and general control

The governing body of a premier-level contact sport in Australia is the dominant contracting party and proferens of the several contracts and contract annexures guiding the relationship between itself, the athletes who play in its premier competition and the clubs they play for. As the proferens, the SGB reserves to itself almost total control of the sport and those athletes and clubs. Control over the terms of engagement, along with control of the rules of play not only permits the SGB to adopt reasonable measures to ameliorate risks of harm but, given the violence of contact sport and the commercial returns associated with the nature of the sport, obliges it to monitor emerging risks to its players and, where material, to warn players of those risks. For purposes of context, it should again be noted that a salient features approach is not an attempt to identify reasonably foreseeability under the *Donoghue v Stevenson*[165] formulation, but to identify the presence of a fact that goes to establishing a duty of care.

A An ongoing history of control over the game and its players

As an exemplar of the degree of control exercised by the governing bodies of major contact sports in Australia, the contractual arrangements governing the relationship between the NRL (and its predecessors) and its premier players are considered below.

A review of past contractual arrangements reveals a long-held preference of the League to maintain control over all aspects of its sport. In the 1971 rugby league restraint of trade case *Buckley v Tutty*, indications of extensive control exercised by the SGB were recorded by the High Court:

> The General Committee [of the SGB] has power to deal with complaints of misconduct and to 'disqualify, suspend, fine or otherwise deal with any person, Club, League, Association or constituent body or any player or team'. …
>
> One of the principal functions of the League is to control, either directly (as in the case of district clubs) or indirectly (through The Country Rugby Football League of New South Wales), the bodies who are employers of Rugby League professionals.[166]

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[164] *Perre v Apand Pty Ltd* (1998) 198 CLR 180, [123].

[165] [1932] AC 562.

[166] *Buckley v Tutty* (1971) 125 CLR 353, 367, 373 (Barwick CJ, McTiernan, Windeyer, Owen and Gibbs JJ). The New South Wales Rugby League was the principal body controlling rugby league in Australia at the time.

The High Court noted further that 'the jurisdiction of the League is said to extend to all its constituent bodies and all members thereof', such that

> the League is declared to be the final court of appeal in the State in all matters of dispute, misconduct or otherwise except where the rules otherwise provide. Power to supervise and control the activities of district clubs is given to the League by a number of provisions of the rules.[167]

For many years the Australian Rugby League (a forerunner to the present Australian Rugby League Commission) as the dominant contracting party exerted *direct* control over the clubs and the professional and personal lives of premier players. As direct control is generally advantageous to the dominant party, why would the League adopt a less direct means of player control? The answer is found in a passage of the Federal Court's judgment in *News Ltd v Australian Rugby Football League*, commenting on the present contractual scheme's original antecedent:

> Until 1992, the players' contracts were tripartite arrangements, involving the League, the club and the player as parties. The decision to move to bilateral contracts was made deliberately, in consequence of legal advice that such contracts would not be subject to attack under the exclusionary provisions of the [*Trade Practices Act 1974* (Cth)].[168]

The court went on to state:

> Certain provisions imposed obligations on the player that are capable of being construed as being for the benefit of the League. For example, the player agreed to be bound by the memorandum and articles of the League and ARL …[169]

In fact the League did not, in moving to a bilateral contract between itself and the clubs, relinquish control over the players in its competition.

Similar terms binding a player to the governing organisation are found in the registration agreement between the New South Wales Rugby League (a forerunner to the NRL and the Australian Rugby League Commission). The High Court noted in *News Ltd v Australian Rugby Football League* that

> [t]he standard contract has a player's registration form printed on it. That form is addressed to the League and requires the signature of the player. Among other things, the player agrees to be bound by the League's memorandum, articles and rules and to make himself available for selection in representative teams.[170]

Consequently, a governing organisation, while not the employer of the premier players, preserves its enforcement of contractual terms by annexing the player contract to the player registration agreement, along with further rules and regulations.

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[167] *Buckley v Tutty* (1971) 125 CLR 353, 364.

[168] *News Ltd v Australian Rugby Football League* (1996) 139 ALR 193, 326.

[169] Ibid.

[170] Ibid 242.

B Contractual terms of control

The NRL Player Contract cedes foundational control over a player to the SGB, while nonetheless granting day-to-day authority to the player's club. For example, the NRL Player Contract includes a term requiring the player to 'participate in all activities reasonably required by … the NRL' and 'submit to the jurisdiction of, and comply with any decisions, determinations, directions or rulings made by the NRL Board [and] the Chief Executive Officer'.[171] Ordinarily the NRL as a 'non-contracting party, would be unable to enforce such rules; however, by incorporating the Player Contract into the player's registration agreement, along with several other policies and rules, the SGB establishes an enforceable and controlling arrangement between itself and the premier players.[172]

Consider the NRL Player Registration Application, the player's assent to which is necessary for admission to the NRL's premier competition, which begins with the following:

> I hereby apply to be registered by National Rugby League Limited (NRL) as a Player in the NRL Competition and/or the National Youth Competition, if eligible, with [in this example] the Wests Tigers Club.[173]

Clause 2 of the Registration Application provides:

> In signing this Application, and in return for the NRL agreeing to consider my application for registration as a Player in the NRL Competition and/or the National Youth Competition, if applicable, I agree:
>
> (a) To comply with, and be bound by, the NRL Rules including:
>
> (i) Schedule One — NRL Code of Conduct;
>
> (ii) Schedule Two — NRL Anti-Doping Rules;
>
> (iii) Schedule Three — NRL Anti-Vilification Code;
>
> (iv) Schedule Four — NRL Judiciary Code;
>
> (v) Schedule Five — NRL Appeals Committee Procedural Rules;
>
> (vi) Schedule Six — NRL Playing Contract and Remuneration Rules;
>
> (vii) Schedule Severn — NRL Player Welfare Rules;
>
> (viii) Schedule Eight — NRL Operations Manual;

[171] NRL Player Contract, Section 3.1(j), (l). Power to compel 'participation' is also that of the district clubs

[172] *Coulls v Bagot's Executor and Trustee Co Ltd* (1967) 119 CLR 460.

[173] NRL Player Registration Application, 2016.

(ix) Schedule Nine — NRL Testing Policy (Illicit & Hazardous Drugs); and

(x) Any guidelines to the NRL Rules,

as amended from time to time, and whether those amendments are effected before or after this date.

Notably, Clause 2 of the Registration Application is expressed in the form of 'offer and acceptance'. In return for the NRL registering the player, the player agrees to abide by the various rules incorporated into the contract. To emphasise the point, in *De Belin v Australian Rugby League Commission Limited*, Perry J recorded that 'a player must be registered under the NRL Rules and hold a Playing Contract with a club in order to participate in these competitions'.[174] Her Honour explained that

> the Club Licence Agreements and the Playing Contracts comprise the means by which the NRL, the clubs and the players are bound by the Rules.[175]

Broader still, the contractual matrix includes 'the [Australian Rugby League Commission], NRL, clubs, players, club officials, coaches and trainers [who] are bound by the same set of rules as amended from time to time'.[176]

Perry J stated further that, although

> registration does not create an employer/employee or principal/agent relationship between the NRL and a player … [this] does not, of course, mean that registration did not create a contractual relationship between Mr de Belin and the NRL.[177]

In fact, *De Belin* is a good example of the degree of control an SGB in Australia may exert over the sport and its athletes. The NRL Rules were amended in March 2019 by the inclusion of Rule 22A, which provided that a player will be automatically stood down from the competition where charged with a 'Serious Criminal Offence' as defined by the NRL 'as an offence punishable by a maximum penalty of 11 years' imprisonment or more'.[178] A player charged with a criminal offence carrying a lesser maximum penalty may be stood down at the absolute discretion of the CEO or Chief Operating Officer of the NRL. Jack de Belin was charged with a crime carrying more than 11 years imprisonment on 13 December 2018. Rightly or wrongly, Rule 22A was introduced unilaterally and retrospectively by the organisation to cover the charges against de Belin. In another example, Football Australia utilised its governing

[174] *De Belin v Australian Rugby League Commission Limited* [2019] FCA 688, [48].

[175] Ibid.

[176] Ibid [98].

[177] Ibid [63].

[178] Ibid [5].

rules to ban the Melbourne Victory club from selling tickets to home games following a 'pitch invasion' by supporters.[179]

The capacity to introduce rules that are averse to the interests of players is not only indicative of plenary control but also of a capacity to change rules and protocols that will benefit players.

3.6.5 Reliance and expectations of care for player health and welfare

Allsop P described 'the degree of reliance by the plaintiff upon the defendant' as a salient feature.[180] How to define or embody 'reliance' was not, however, mentioned by his Honour — just that '*degree*' of reliance is a determining measure. As a salient feature, 'reliance' is likely to depend on how the plaintiff comes to 'rely' on the SGB and whether, in the circumstances, they are reasonable in so relying. Similar to 'control', other salient features may act in combination to further a plaintiff athlete's claim of 'reliance'. These may include 'vulnerability', 'control', 'assumption of responsibility', 'proximity', 'personal connection', 'the degree of hazard' and 'the consequences', among several others.

To illustrate, a base of reliance may be formed through relational proximity or a personal connection between a premier-level athlete and his or her governing organisation. Reliance, as argued, is enhanced where the organisation expressly or impliedly indicates its concern for players' health and welfare; more so where the player is implicitly encouraged to throw off autonomy and place trust in the organisation and its judgment.

A combination of salient features was noted above in *Watson v British Boxing Board of Control; World Boxing Organisation Inc*.[181] Had the BBBC merely given general advice about medical precautions, a duty of care would likely not have arisen. As Phillips MR said, the BBBC, however, 'went far beyond this' — it 'arrogated' to itself the task of 'determining what medical facilities' were to be provided and required the 'boxer and the promoter to contract on terms under which the Board's rules [would] apply'.[182] The BBBC assumed the responsibility for determining the nature of the medical facilities and assistance to be provided, and in so doing placed itself '[in] close proximity with each individual boxer who contracts with a promoter to fight under the Board's rules'.[183]

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[179] 'Melbourne Victory Sanctions Imposed as Show Cause Process Continues', *Football Australia* (Web Page, 23 December 2022) <https://www.footballaustralia.com.au/news/melbourne-victory-sanctions-imposed-show-cause-process-continues>.

[180] *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649, [103].

[181] [2001] QB 1134.

[182] Ibid [79].

[183] Ibid [80].

The assurances of care by governing bodies of major sport in Australia are found in extensive lists of medical protocols mandated by contract to apply beneficially to the athletes who participate in their premier competitions. Nonetheless, a difference, perhaps significant, lies in the express and direct undertakings of the BBBC (itself) to care for the boxers' safety in contrast with those of, say, the NRL contract which mandates that players to seek medical advice from their club medico. In similarity, though, both organisations allocated the responsibility to subordinate actors with, it may be argued, an obligation to ensure both compliance and that medical protocols are relevant to the risks of the sport including emerging risks.

Irrespective, terms of player contracts, which are in standard form and are imposed on clubs and players by the SGB, deal extensively with health and welfare. Consider the following sections of the NRL Player Contract that highlight the obligations of the club, the player and the 'club medical officer'.[184] Again, these contractual mandates are enforceable by the NRL through the Player Registration Application.

Under Section 3.1 'Players' general obligations', the player agrees to

> (n) immediately report any illness, accident or injury of any nature to the Club Medical Officer and carry out any reasonable instruction given to him by the Club Medical Officer including the wearing or use of any protective equipment recommended by the Club Medical Officer.
>
> …
>
> (p) save in an emergency situation, not undertake any medical treatment for injuries sustained whilst training or playing the Game for the Club without the prior consent of the Club, such consent not to be unreasonably withheld.

Section 5.2 'Standing down due to health risk' provides:

> Where in the reasonable opinion of the Club Medical Officer, the Player would, by reason of some physical or medical condition, be exposed by playing the Game to a greater than usual risk to his health or to a greater than usual risk of injury, the Club may, at any time during the Employment Term, stand the Player down from playing with immediate effect.

Section 6.2 'Independent medical opinion' concerns a player who is thought by their club to be suffering a 'career ending injury':

> In any case where the Player wishes to obtain an independent medical opinion as to his bodily capacity to play the Game, and whether such an opinion is sought for the purposes of protecting rights under this clause or not, he shall be free to do so at his own cost.

Section 5.2 'Standing down due to health risk' is particularly telling with respect to the obligation to be informed and to warn. This contractual term naturally raises the expectation that events such as RHT, 'as a medical condition' exposing the athlete to 'a greater than usual risk of injury', will be the subject of warning.

[184] National Rugby League Playing Contract as in force 2020.

It is worth noting that, as a matter of law, the Club Medical Officer's duties are not limited to those expressed as contractual terms, but include the overarching obligation to 'exercise reasonable care and skill in the provision of professional advice and treatment'.[185] As noted by Lord Hewart:

> If [the physician] accepts the responsibility and undertakes the treatment and the patient submits to his direction and treatment accordingly, he owes a duty to the patient to use diligence, care, knowledge, skill and caution in administering the treatment. No contractual relation is necessary, nor is it necessary that the service be rendered for reward.[186]

Having mandated through contract that a club medical officer will watch over the athlete's medical needs, an expectation is raised in the consciousness of players that the governing organisation is concerned for their health and welfare and in consequence will ensure that the contractual mandate is enforced and that risks of harm, including emerging risks, specific to the sport will be monitored. At the very least, had the player *not* been instructed to attend the club doctor, or if the services of the club doctor were *not* mandatory, the athlete would as a matter of necessity consult a medico of his or her choosing. In effect the governing organisation has vouched for the efficacy of the system it has introduced and is obliged to take reasonable steps on an ongoing basis to monitor, through its own resources or those of its associated medicos, for known and emerging risks of harm.

3.7 The vulnerable athlete: Reliance and control

A judicial acceptance of the salient feature 'vulnerability' will turn in large part on the effective autonomy a plaintiff possesses to protect his or her interests over the sporting organisation's contractual or regulatory influences. For example, is an athlete subject to a wide range of contractual mandates? Does the content of these mandates lead to a mentality of reliance and consequential vulnerability? Is the athlete equipped to investigate emerging risks to health or initiate risk-reduction protocols? Is the athlete encouraged to seek independent advice from medicos or other professionals? Is the athlete's full autonomy restricted behaviourally or psychologically by an ethos that places the needs of the team above those to self?

As a salient feature, an athlete's 'vulnerability' to physical harm with respect to establishing a duty of care rests heavily upon the degree of control an SGB has over the hazards of play — or, more specifically, the rules, practices and protocols associated with play and, as noted, the capacity of the athlete to challenge that control. For example, as seen in *Green v Country Rugby Football League of New South Wales Pty Ltd*, the CRL's effective incapacity to unilaterally introduce rules to 'depower the scrum' was a determinant in Walmsley AJ's finding of no liability.[187] Vulnerability is also associated with the salient feature 'reliance'. McHugh J remarked that the concept of control 'is simply another way of saying that the plaintiff is

[185] *Wallace v Kam* (2013) 250 CLR 375, [8].

[186] *R v Bateman* (1925) 94 LJKB 791, 794.

[187] *Green v Country Rugby Football League of New South Wales Pty Ltd* [2008] NSWSC 26, [138].

vulnerable to the defendant's conduct because the defendant controls the situation'.[188] Or, expressed differently,

> reliance and assumption of responsibility are merely indicators of the plaintiff's vulnerability to harm from the defendant's conduct, and it is the concept of vulnerability rather than these evidentiary indicators which is the relevant criterion for determining whether a duty of care exists. …
>
> Reliance may therefore be seen — for the purposes of duty of care — as an indicator of vulnerability: the plaintiff is specially vulnerable to the words and/or conduct of the defendant because he or she reasonably relied on the defendant.[189]

In contextual terms, the SGB maintains control over all aspects of a violent contact sport. The athlete, given the realities of contact sport, relies on the SGB to reasonably exercise that control to ameliorate risks to his or her health. Reliance on the organisation's conduct as it applies to risks of harm is a source of athlete vulnerability. This section, therefore, considers 'reliance' and 'vulnerability' conjunctively.

In *Perre v Apand Pty Ltd*[190] the defendant supplied diseased potato seeds to the Sparnons, who were potato growers in South Australia, causing the crop to be infected with 'bacterial wilt'. Western Australian regulations prohibited the import of any potatoes grown within 20 km of a bacterial wilt outbreak. The appellants, which exported up to 79 per cent of their potato crops to Western Australia, were situated within the 20-km limit of the Sparnons' property, suffered loss and sued the supplier of the diseased seeds in negligence. The trial judge (and the Full Federal Court) recognised that although the loss suffered was foreseeable, an absence of 'proximity' excluded liability. On appeal to the High Court, however, a 'neighbour' relationship was accepted.

Gleeson CJ found that in supplying diseased seeds, the 'respondent controlled the activity on the Sparnons' land'.[191] Internal communications revealed that the respondent recognised a need to avoid damaging potato growers' crops on properties within a 20-km radius of any farm it serviced. On this fact, his Honour found 'actual foresight of the likelihood of harm, and knowledge of an ascertainable class of vulnerable persons'.[192] Gleeson CJ also stated that 'knowledge (actual, or that which a reasonable person would have) of an individual, or an ascertainable class of persons, who is or are reliant, and therefore vulnerable, is a significant factor in establishing a duty of care'.[193] McHugh J stated similarly that 'vulnerability to risk

[188] *Perre v Apand Pty Ltd* (1998) 198 CLR 180, [127].

[189] Ibid [125]–[126].

[190] (1998) 198 CLR 180.

[191] Ibid [14].

[192] Ibid [15].

[193] Ibid [10].

and the defendant's knowledge of the risk and its magnitude are, I think, relevant in determining whether a duty exists'.[194]

Gaudron J, although not using the word 'vulnerability', framed a general application describing a state of vulnerability:

> Where a person is in a position to control the exercise or enjoyment by another of a legal right, that position of control and, by corollary, *the other's dependence on the person with control* are, in my view, special factors or, which is the same thing, give rise to a special relationship of 'proximity' or 'neighbourhood' such that the law will impose liability upon the person with control if his or her negligent act or omission results in the loss or impairment of that right and is, thereby, productive of economic loss.[195]

Callinan J proposed several facts that go to establish 'a sufficient degree of proximity' as 'foreseeability, a special relationship, determinacy of a relatively small class, a large measure of control on the part of the respondent, and special circumstances justifying the compensation of the appellants for their losses'.[196] The specific facts cited by his Honour were a 'combination' of the respondent's 'leading role in the industry' and its involvement 'in all phases' of it, 'from the introduction of particular varieties of seed, and the encouragement of growers to sow that seed, to the acquisition of the produce of that seed … and the distribution of that commodity in the community'.[197] In summary,

> its role and position in the market were commanding ones. This role of itself tended very much to place the respondent in a special relationship to growers and handlers of potatoes.
>
> The respondent was in effective control of the particular operation which led ultimately to the imposition of the embargo upon the appellants' properties.[198]

With respect to 'vulnerability', Callinan J found it 'notorious' that 'commodity markets are fragile … and in particular are very vulnerable in modern times to contamination both prospective and actual'.[199] Being 'notorious', the risk of harm was known and, in similarity with the known risk of CTE in the playing of contact sport, a duty to take reasonable care emerged.

On the other hand, where an individual is autonomous and free to act as he or she wishes, a court is unlikely to recognise 'vulnerability'. In *CAL Pty Ltd v Motor Accidents Insurance Board*,[200] the plaintiff's husband, Shane Scott, who was drinking in the defendant's hotel,

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[194] Ibid [105]. The application, although general, was referring specifically to economic loss.

[195] Ibid [38]. Emphasis added.

[196] Ibid [406].

[197] Ibid [406], [407].

[198] Ibid [407]–[408].

[199] Ibid [414].

[200] (2009) 239 CLR 390.

agreed to hand the keys to his motor bike to the licensee and further agreed that his bike be locked in a storeroom. Later, after drinking for several hours, he was refused service and, in response, insisted his keys be returned. The licensee suggested calling Scott's wife to pick him up. Scott refused, and the licensee handed over the keys. Scott rode away from the hotel, lost control of his bike and was killed. His blood alcohol reading was 0.253. His wife sued the defendant claiming negligence in returning the bike to her drunk husband. The High Court found that there was no negligence, and spoke of a conflict between the autonomy of an individual and purported vulnerability:

> This conflict does *not* arise where for some supervening or overriding reason a person who is owed the putative duty is not autonomous, or fully autonomous — because, for example, some control must be exercised by the defendant over another person who either was vulnerable before the control was first exercised, or has become vulnerable by reason of the control having begun to be exercised.[201]

As argued, the control exercised by the governing bodies of sport over their players is reflected in the rules of play and the protocols concerned with post-injury treatment, including head injury assessments and compulsory stand down orders following head trauma. It is the SGB that possesses the resources to investigate emerging medical risks in a violent sport, along with a contractual entitlement to issue warnings of material risks to all players in its competition.

3.8 Reliance, vulnerability and the violent nature of the sport itself

The governing bodies of Australian contact sport, as discussed extensively above, control all aspects of the sports they administer. Within the contractual and administrative power of these bodies lies the final capacity to warn of harm, adjust the rules of play, and initiate ameliorating protocols.

Consider this proposition: Some premier-level contact sports are so violent that the entity exercising sole control over the relevant competition is duty-bound to take reasonable steps to ameliorate risks of harm and to monitor for emerging risks. As argued previously, 'reasonable steps' excludes an obligation to alter the rules that are the essence of the sport.

Possessing 'control' over risks of harm has been found to support several duty relationships, particularly where no other entity possesses the power of harm mitigation. Consider comments by the authors of *Balkin & Davis Law of Torts* on the broad-based obligations of a doctor to a patient:

[201] *CAL Pty Ltd v Motor Accidents Insurance Board* (2009) 239 CLR 390, [38]. Emphasis added.

> The basis of liability in these cases … appears to stem from the fact that the doctor as the repository of specialized information or knowledge, is, effectively, the only source upon which the individual can draw and on whom the individual must therefore rely.[202]

As a further example, statutory bodies, which are subject to the common law of negligence 'unless excluded',[203] usually exercise extensive control over their area of responsibility. In *Schiller v Mulgrave Shire Council*, a statutory body controlling and managing land came under a duty of care to users. Although based in the obligations of an occupier of land, Barwick CJ considered capacity to act as paramount, stating that 'the capacity to care for and control the reserve was in this case the source of duty'.[204]

3.8.1 The examples of *Watson* and *Green*

With respect to premier-level sport, the governing organisation, as the controlling entity, is alone entitled to respond to risks of harm and to ameliorate those risks. In *Green v Country Rugby Football League of New South Wales Pty Ltd*, for example, Warmsley AJ accepted the visitation of financial consequences on the CRL if it had, without the assent of the dominant organising authority (the New South Wales Rugby League), 'introduced a rule change unilaterally'[205] to make scrummaging safer. A single entity, the New South Wales Rugby League, was empowered to protect the well-being of players through rule adjustment. It chose not to, and no recourse was open to the CRL.

In *Watson v British Boxing Board of Control; World Boxing Organisation Inc*, Phillips MR drew a distinction between the 'general reliance' that the public places on services such as the fire brigade or police, and the reliance that a limited group of athletes closely associated with an organising authority places on that authority. There is 'no close proximity between the services and the general public' and there are also 'reasons of public policy for not imposing a duty of care on individuals regarding the performance of their functions'.[206] 'In contrast', said his Lordship,

> the injuries which are sustained by professional boxers are the foreseeable, indeed inevitable, consequence of an activity which the Board sponsors, encourages and controls. The conduct of the activity of professional boxing carries with it, for the small body of men that take part in it, the need for the provision of medical assistance to treat the injuries that they sustain and minimise their adverse consequences.[207]

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[202] D Rolph et al (eds), *Balkin & Davis Law of Torts* (LexisNexis Australia, 6th ed, 2021) 272 [7.22]; quoted with approval in *Green v Country Rugby Football League of New South Wales Pty Ltd* [2008] NSWSC 26, [193].

[203] *Crimmins v Stevedoring Industry Finance Committee* (1999) 200 CLR 1, [26] (Gaudron J).

[204] *Schiller v Mulgrave Shire Council* (1972) 129 CLR 116.

[205] *Green v Country Rugby Football League of New South Wales Pty Ltd* [2008] NSWSC 26, [131].

[206] *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [77].

[207] Ibid [78].

Had the BBBC not sought to provide medical assistance, 'the promoter would probably owe a common law duty to the boxer to make reasonable provision for the immediate treatment of his injuries'.[208]

In essence, the 'foreseeability' of 'inevitable' harm generated a duty of care. That the BBBC 'arrogated to itself' that obligation put the BBBC, rather than a different entity, 'in proximity' to 'each individual boxer'.[209] Had another entity stepped in, the BBBC had the power to overrule any of its initiatives.

Furthermore, the BBBC mandated the rules and protocols of boxing, the wearing of gloves, the size and weight of gloves, the length of rounds, the number of rounds, the Board appoints and monitors referees, dictates the time away from boxing following a knockout, among a myriad of other responsibilities. In short, most aspects of the sport of boxing and the professional life of the boxer are influenced by the BBBC's decisions. In the words of Phillips MR, the BBBC is in 'proximity' with the boxers under its charge.[210]

In similarity to Australian contact sports like rugby or AFL, harm to a professional boxer is inevitable and foreseeable. The BBBC promotes, through its rules and administration, the violence that leads to harm. The BBBC controls the sport. These facts contribute to establishing a duty relationship between the BBBC and the individual boxer, and are identical to those subsisting between the governing bodies of major sport in Australia and their premier-level athletes.

An overarching fact, which is common to Australian governing bodies of contact sport, is that the BBBC was 'not at arm's length' from the violence of the sport it administered; indeed, it was a financial beneficiary of the risks boxers faced.

3.8.2 Vulnerability and reliance on the sole decider of policy

Few greater relationships of reliance and concomitant vulnerability exist than where a single organisation alone can influence an individual's health and well-being. This, as argued, is also the basis of the relationship between the athletes who play premier contact sport in Australia and their governing bodies.

A belief that athletes in the heat of competition, or where the needs of the team dictate, will not exhibit vulnerability by embracing risks of harm is an unreality that must be considered by an SGB. In these circumstances, the obligation of the SGB to a plaintiff may, as explained by Dietrich, 'be framed as a "protective duty" such that the scope of [the defendant's] duty extends

[208] Ibid.

[209] Ibid [79]–[80].

[210] Ibid [75].

to protecting [the plaintiff] against the risk that [the plaintiff] might fail to take reasonable care of her own safety'.[211] Dietrich continued:

> In some circumstances, plaintiffs are not expected to take care of their own safety to the standard of a reasonable, objective person, because the scope of the defendant's duty extends to protecting the plaintiff precisely against that risk … [and] rests upon the plaintiffs' diminished capacity to take care for their own safety.[212]

Given the hierarchical structure of Australian premier sport, the SGB retains the ultimate power and the financial means to do such things as make policy, change rules initiate safety protocols and outlaw dangerous play. The athlete competing under an autocratic sporting structure is reliant on the governing organisation to introduce policies that mitigate a risk of harm where a subordinate organisation can only suggest policy change.

3.9 Foreseeability as a salient feature

The salient features of control, reliance and vulnerability were discussed above (at **[3.7]**). This section considers 'foreseeability' as a further feature relevant to the imputation of duty residing in an SGB, which is to say that it considers foreseeability as an element of negligence. For purposes of clarity, it should be noted that 'foreseeability', although listed as a salient feature by Allsop P in *Caltex Refineries (Qld) Pty Ltd v Stavar*,[213] does not serve the same purpose as the other features. Allsop P said: 'I have described "foreseeability" as a salient feature; it is perhaps better expressed that the use of salient features operates as a control measure on foreseeability employed at the level of abstraction earlier discussed … as the foundation for the imputation of duty of care'.[214]

As a salient features approach — that is, a multi-factorial analysis — is the approved methodology to determine in a novel case whether the relationship between the parties is such that a duty of care should be imposed on the defendant,[215] this section considers the application of the feature 'foreseeability' in identifying whether an SGB possesses an obligation to inform itself of the risk of CTE and to warn its premier-level athletes of that risk. In doing so, several examples of mesothelioma cases are used in illustration.

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[211] J Dietrich and I Field, 'The "Reasonable Tort Victim": Contributory Negligence, Standard of Care and the "Equivalence Theory"' (2017) 41(2) *Melbourne University Law Review* 602, 616.

[212] Ibid 624. Expedition and word length preclude deeper consideration of this argument.

[213] (2009) 75 NSWLR 649.

[214] Ibid [106].

[215] Ibid.

Two interrelated questions of foreseeability are pertinent: (1) Is CTE a foreseeable risk of playing the SGB's contact sport? (2) Is there a foreseeable risk that a plaintiff athlete will contract CTE if not warned by his or her SGB?[216]

Although it is an indispensable requirement, foreseeability of harm does not alone determine the establishment of a duty of care. Gleeson CJ remarked in *Tame v New South Wales* that 'reasonable foreseeability of the kind of injury that has been suffered by the person to whom the duty is owed is a necessary, but not sufficient condition of the existence of a legal duty of care'.[217] Allsop P said similarly in *Stavar* that, '[i]n a novel area, reasonable foreseeability of harm is inadequate alone to found a conclusion of duty', and commented further that the other salient features operate as a 'control measure on foreseeability'.[218] The test of foreseeability *at the duty stage* of inquiry is 'undemanding', such that a 'risk which is not far-fetched or fanciful is real and therefore foreseeable'.[219] That is, 'a risk of injury which is quite unlikely to occur ... may nevertheless be plainly foreseeable'.[220]

3.9.1 Analogous cases

The foreseeability of CTE as a 'real' or 'not fanciful' risk is based primarily on the availability of medical reports that were accessible to SGBs in Australia in the decades prior to 2010, along with knowledge gained through common experience and observation. The fact that information on the risk of CTE was not available until some time after Dr Bennet Omalu et al's 2005 revelations[221] offers for a defendant SGB a viable argument as to why it did not know and could not warn of the risk of long-term cognitive harm in the playing of its contact sport.

Arguments founded on an apparent absence of information on a risk of harm are, however, not uncommon to Australian courts in cases concerned with the contraction of disease following exposure to a risk-producing agent. Discussed below are several mesothelioma cases, where defendants argued that knowledge of the disease had not developed or been disseminated to the point where its risk was foreseeable. These cases offer a general insight into approaches to determining the foreseeability of CTE, which is argued as a re-emerging or incipient disease.

In the novel case of *Caltex Refineries (Qld) Pty Ltd v Stavar*,[222] Mrs Stavar's husband worked from 1964 until 1991 at the oil refinery of Ampol Pty Ltd, where he handled asbestos materials daily. Mrs Stavar came to suffer mesothelioma from inhaling asbestos dust when

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[216] Discussed in detail in Chapter 8: 'The Question of "Generic Capacity": Does the SGB's Contact Sport Cause CTE?'

[217] *Tame v New South Wales* (2002) 211 CLR 317, [12].

[218] *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649, [106].

[219] *Wyong Shire Council v Shirt* (1980) 146 CLR 40, 47–8 (Mason J).

[220] Ibid.

[221] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[222] *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649.

washing her husband's work clothes, while residing at home, and when riding in the family car. She claimed that Caltex Refineries (which acquired the business from Ampol) owed her a duty to take reasonable steps to prevent her inhaling asbestos brought home by her husband from his workplace.

In determining whether the risk of mesothelioma to the class of persons to which Mrs Stavar belonged was foreseeable, the court considered two matters in particular: (1) Was information on that risk available to the defendant? (2) Ought the defendant to have been aware of that information?

In respect to the question of foreseeability through attainable knowledge, a 'number of important journal articles'[223] were examined by O'Meally P at first instance.[224] The initial article appeared in the *British Journal of Industrial Medicine* in 1960, and was the first to confirm a link between asbestos and mesothelioma. Twelve further articles dating up to 1976 referred to the dangers of exposure outside the workplace, 'including exposure of family members of asbestos workers to asbestos in the domestic context being presumed to be the dust brought home in overalls'.[225] All but one of the listed journals were foreign publications, notably the *New England Journal of Medicine* and the *British Medical Journal*. The one Australian report, published in the *Medical Journal of Australia* in 1968, expressed concerns as to the possible dangers of 'minor exposure', the implication being that Ampol could have extrapolated from knowledge acquired in this article that family members who come into contact with minor quantities of asbestos on clothes worn home by a refinery worker may be exposed to a risk of mesothelioma. In addition, several later articles in Australian medical journals identified the causative link between asbestos and mesothelioma. O'Meally P at first instance found that all these papers were available in Australia, and 'demonstrate[d] a growing awareness of the dangers of exposure to asbestos with the effluxion of time'.[226]

Other indicators of foreseeable risk referred to by O'Meally P included expert evidence accepted as a finding of fact that: from the mid-1960s the medical community recognised the foreseeable risk of mesothelioma through exposure to small quantities of asbestos and that Ampol could have become aware of that risk had it made inquiries; and that from the mid-1980s a firm known as Bells required its employees working with asbestos to wear full protective clothing and that Ampol was aware of these precautions.[227] Moreover, a statutory

[223] Ibid [32].

[224] *Stavar v Caltex Refineries (NSW) Pty Ltd [2008] NSWDDT 22.*

[225] *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649 [32].

[226] Ibid [33].

[227] Ibid [48]–[51].

edict known as the 'Asbestos Rule', which came into effect in 1971, was found to have placed Ampol on notice of the relevant risks.[228]

In another mesothelioma case, *CSR Ltd v Young*, Giles AJA stated comprehensively that '[i]n many situations foreseeability is an influential, if not decisive, determinant of the existence of a relationship of proximity, and this is such a situation'.[229] In that case, people from the town of Wittenoom in Western Australia alleged that they suffered asbestos-related illnesses as a result of CSR's nearby blue asbestos mine. CSR claimed in its defence that knowledge of the risk of 'external' exposure to asbestos was nascent, such that it did not necessitate a duty to warn; and, in the alternative, that the state of knowledge extended only to the diseases of asbestosis and bronchogenic carcinoma.

In a formulaic manner, Giles AJA considered the availability of evidence of the risk of asbestos-related disease that suggested foreseeability. He found that CSR knew that it had used asbestos tailings around the town of Wittenoom, and ought to have known that asbestos created a risk to persons beyond the confines of the mine. His Honour also found that it was well known that asbestos fibres could cause asbestosis and bronchogenic carcinoma. He referred to: a 1930 report on the effects of asbestos dust in the lungs that informed of the risk; a 1955 article by a researcher named Doll that also informed of the risk; a 1952 publication of the Department of Health in which it was said that it 'may be doubted whether any dust, provided the exposure is sufficient, can be regarded as harmless', and included asbestos in its list of such dusts; and an article by the medical director of General Motors Holden, published in *Australian Factory*, which spoke of a permissible limit of asbestos as being 2.5 million parts per cubic foot, and in doing so alerted to a danger from that source in general

In addition to the several general sources of information available through the references given above, his Honour found that CSR 'knew' of the risk because in 1959 a Dr McNulty, a chest physician employed by the Public Health Department of Western Australia with a brief to monitor the health of miners, wrote to the manager of Australian Blue Asbestos Ltd (ABA) (CSR's subsidiary, which managed the mine) stating it was 'unfortunate that people who worked in the mine and in the mill, who were exposed to so much dust at work, should also have to face it at home and at recreation', and that he thought it was 'a bad thing'.[230] In addition, a 1960 article in the *British Medical Journal* reporting on the risk of mesothelioma was forwarded by the ABA manager to ABA's Sydney offices.

Giles AJA found that CSR also knew that blue asbestos was dangerous to workers; that asbestos fibre was deposited in the town at several public sites; that there was no known safe dosage; and that exposure in the town was a risk to its citizens' health. As such, 'in and from

[228] Ibid [52].

[229] *CSR Ltd v Young* (1998) 16 NSWCCR 56, BC 19 (Cohen AJA agreeing).

[230] Ibid BC 20.

1959 it was foreseeable to CSR and ABA … that there was a risk which was not far-fetched or fanciful'.[231]

In another asbestos case, *Bale v Seltsam Pty Ltd*,[232] the plaintiff's husband was employed by the defendant, Seltsam, at its asbestos works. The plaintiff contracted mesothelioma after being exposed to asbestos dust as she laundered her husband's work clothes and rode in his truck. She claimed that Seltsam owed her a duty to warn of the risk of harm associated with asbestos dust, having permitted 'her husband, who worked for the [defendant] from 1 June 1962 to 30 August 1965, to return home with asbestos dust on his person'.

Although Seltsam was 'indifferent to the effect on their workers of exposure to levels of asbestos dust well known for many years to be beyond the widely accepted safe level', the trial judge found that Seltsam did not owe the plaintiff a duty of care as there was insufficient foreseeability of the risk of mesothelioma to those in close contact with asbestos workers to establish that 'harm was likely'.[233] The Court of Appeal determined otherwise.

Knowledge came Seltsam's way through multiple sources — including medical literature, written reports, statements and expert evidence — revealing a risk of lung disease generally and mesothelioma in particular, that, according to the court, 'ought' to have been known:

- Papers were published in the 1930's which tended to show that even relatively short exposure to heavy doses of asbestos dust by workers could eventually cause asbestosis …;
- Papers seeking to make a link between lung cancer and asbestosis were published from about the mid-1930's, throughout the 1940's with the link finally confirmed in a seminal paper by R Doll in 1955.[234]

In 1956, the Victorian Government declared asbestos trades as dangerous under the Dangerous Trades Regulation; this included any factory process involving asbestos.

Fitzgerald P determined that

> [t]he [defendant] was engaged in commercial activities involving a material which was known to be extremely toxic. Its operation was one of the largest in Queensland. … It knew, or must be taken to have known, that there was scientific and medical uncertainty concerning the toxic effects of asbestos dust … Further, the [defendant] knew, or ought to have known, that the effects of exposure to asbestos dust on persons such as the appellant might not emerge for years and that any illness might be serious or even fatal.[235]

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[231] Ibid.

[232] [1996] QCA 288.

[233] Ibid 2, BC 3.

[234] Ibid BC 6.

[235] Ibid BC 15.

In addition, reference was made to several medical journals, mainly international, that carried information regarding the risks associated with asbestos inhalation: *British Medical Journal*, *The Lancet*, *Journal of the American Medical Association*, *Australian Medical Journal*, *British Journal of Industrial Medicine*, *Journal of Occupational Medicine* and *Archives of Environmental Health*.

According to the trial judge, 'a safety and fire officer at another large asbestos plant owned by a company during the relevant period … became aware through his reading in the late 1950's and the early 1960's of a possible link between asbestosis and cancer and between exposure to asbestos dust and mesothelioma'.[236] Another witness, an industrial hygienist, referred to several of the medical journals noted above and said that 'by the early 1960's, I would expect that a reasonably competent industrial hygienist employed by a company such as the [defendant] in these proceedings [would] have access to general medical publications'.[237]

The Court of Appeal found that Seltsam 'ought to have known' of the possible toxic effects of exposing domestic workers to asbestos dust by the end of 1962 — an opinion based on medical journals and statements presented as evidence.

3.9.2 Using the examples above: Fact and foreseeability

The mesothelioma cases cited above identify facts that are likely to be relevant in determining whether an SGB should have foreseen CTE as a risk in the playing of contact sport prior to *circa* 2010.

3.9.3 CTE, foreseeability and a 'known uncertainty'

A relevant aspect of the mesothelioma cases cited above with respect to an emerging disease — or one argued as newly emerged — is the recognition of foreseeability where a risk of harm is less than foreseeably certain and thereby less than certain. This principle operates in opposition to a defendant's argument that knowledge of CTE in the years prior to 2010 was less than fully formed; indeed, that the histological processes by which CTE develops were unknown before 2005, thereby precluding actual foreseeability. In other words, given that foreseeability at the duty level requires only that harm not be 'fanciful', a risk is foreseeable although its existence is uncertain and its cause merely suspected.[238]

In *Bale v Seltsam Pty Ltd*, Fitzgerald P found that among the 'unidentified and unknown' possible toxic effects of asbestos inhalation were those experienced outside the worksite, when asbestos dust was brought into the home by a returning worker. His Honour found:

> [B]ecause of the known toxicity of asbestos dust, the known uncertainty with respect to its effects, and the knowledge that injury or illness might not emerge for many years, that risk could not be

[236] Ibid BC 12.

[237] Ibid.

[238] Discussed at length in Chapter 4 is the obligation of a duty-holder be informed of emerging scientific knowledge and to warn of material risks — an obligation that rests on the foreseeability of harm.

> dismissed as 'remote', 'slight', 'far-fetched', or 'fanciful' or otherwise ignored so as to absolve the [defendant] of any duty of care towards the wives of its employees.[239]

Although exposure to asbestos dust presented known risks, it was evident that other unknown or uncertain harm associated with asbestos was also possible, and this was sufficient to establish foreseeability as not 'far-fetched'. Seltsam was not therefore entitled to 'assume' that low-level exposure though asbestos dust brought home on the clothes of a worker was safe. Fitzgerald P found that 'alarms bells' should have rung, and concluded that 'even if [Seltsam] considered risk of injury or illness improbable, I think that it became incumbent upon the respondent to respond by introducing the inexpensive and convenient systems which would have protected its workers' wives and warned those women to avoid exposure to asbestos dust even in small quantities'.[240]

In summary: 'Obviously, "foreseeability" of injury and hence "proximity" of relationship can be influenced by hazards known or suspected in connection with a product or activity.'[241] The risk of a serious lung disease from exposure to asbestos dust was sufficiently foreseeable, even if the risk of mesothelioma was not specifically known, for Fitzgerald P to find that 'the risk inherent in that uncertainty [was] sufficient to create a duty of care'.[242]

In *CSR Ltd v Young*, Giles AJA emphasised in italics a section of a 1960 article in the *British Medical Journal*, in effect indicating that the uncertainties by which mesothelioma was contracted required a defendant to exercise a level of foresight extending beyond the obvious: '[A]sbestos dust is most toxic *and the amount needed to cause asbestosis is not known*, so constant vigilance and new preventive methods are needed if this disease is to be abolished.'[243] Giles AJA also noted that the 'known uncertainty' associated with exposure to asbestos dust, along with knowledge that harm may not emerge for years, gave rise to a risk that 'could not be dismissed as remote, slight, far-fetched, or fanciful'.[244]

3.9.4 Foreseeability and access to knowledge of CTE

Foreseeability is a determination on the facts that supports the premise.[245] What facts support the proposition that a reasonable SGB ought to have foreseen the risk of CTE? Should 'alarm bells', to use the wording of Fitzgerald P, have rung when medical journals and professional

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[239] *Bale v Seltsam Pty Ltd* [1996] QCA 288, BC 17.

[240] Ibid.

[241] Ibid BC 21.

[242] Ibid BC 15.

[243] *CSR Ltd v Young* (1998) 16 NSWCCR 56, BC 20. Emphasis added by the court.

[244] Ibid BC 22.

[245] *Perre v Apand Pty Ltd* (1998) 198 CLR 180, 253.

medicos drew a possible connection between head trauma in the playing of contact sport and permanent cognitive disfunction in the pre-2010 era?

In the cases cited in the section above the courts formulated a judgment based on medical reports that revealed information on the risk of mesothelioma was available and therefore the risk was foreseeable prior to the plaintiff's exposure to asbestos dust — information that could have formed the basis of a warning. These journals were principally international, and were listed as *British Medical Journal, The Lancet, Journal of the American Medical Association, Australian Medical Journal, British Journal of Industrial Medicine, Journal of Occupational Medicine and Archives of Environmental Health. British Journal of Industrial Medicine*, *New England Journal of Medicine*, the *British Medical Journal* and the *Medical Journal of Australia*, *The Lancet*, *The Journal of the American Medical Association* and *The Australian Medical Journal*, *The British Journal of Industrial Medicine*, *The Journal of Occupational Medicine*, and *The Archives of Environmental Health*.

A similar approach logically applies to the question of whether an SGB ought to have foreseen the risk of CTE through the multitude of medical studies linking RHT in contact sport with CTE. Medical and other studies examining the risk of CTE in contact sport were discussed at length in Chapter 2 and highlighted throughout other chapters and appendices of this thesis. These studies were collated in a timeline beginning with the 1928 article in the *Journal of the American Medical Association* by Dr Harrison Martland,[246] which was confined to CTE in boxing, and concluded with the 2009 article of Dr Ann McKee et al,[247] listing several contact sports that posed a risk of CTE. From the 1970s, multiple medical journal reports are argued to provide foreseeable knowledge of the risk of CTE across a range of contact sports to Australian SGBs and, from the 1990s, notifications by medicos associated with AFL and rugby league of possible CTE-like harm associated with those contact sports.[248] It is unnecessary to list these reports again, it being sufficient to acknowledge that each publication provided a means by which SGBs in Australia could reasonably foresee a risk of CTE in playing contact sports that was not far-fetched and, at the very least, ought to have prompted further inquiry and the initiation of CTE-ameliorating protocols.[249]

Reliance on international publications prompts the question of why Australian medicos did not, and do not, rely solely on Australian publications. The answer, clearly, is the near impossibility of the limited number of Australian medical journals being able to cover the vast area of medical science and fulfil the centuries-based tradition of sharing discoveries, means of diagnosis and innovations.

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[246] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

[247] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

[248] See Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times'.

[249] See also Chapters 2, 4, 7 and 10.

3.9.5 Foreseeability and the boxing studies

What then of CTE caused in a contact sport such as AFL, which is suggested as foreseeable though references to studies of CTE in boxing? On its surface, the link is less than certain even though each sport shares a common cause of CTE, being RHT. Nonetheless, the value of the boxing studies lies predominantly in confirming that, from the 1970s onwards, RHT in the *non*-boxing sports carries the same risk of cognitive harm as RHT in boxing.[250] That is, alerted to the possibility of CTE caused in the playing of contact sports, a reasonable SGB would be motivated to review all emerging and historical reports to the degree necessary to respond to questions of foreseeability, either positively or negatively.

Irrespective, articles published in medical journals from the 1970s, along with statements made by Australia medicos associated with major contact sports in Australia during the 1990s, are, as argued, adequate to have informed a reasonable SGB of a major contact sport in Australia of a 'real' risk of CTE — a risk beyond the speculative.

3.9.6 Precise risk of harm need not be foreseeable, but …

A notification of a risk of harm can only be foreseeable where it describes the type of harm that the defendant's breach may cause to a plaintiff. Consequently, much depends on whether information supplied to an SGB through medical reports or by a qualified intermediary corresponds with a recognisable description of CTE. It is proposed here that CTE is not foreseeable through a mere description of the risk of frank concussion appearing in a medical journal, despite both frank concussion and CTE being forms of cognitive harm. This proposition is considered in greater detail in Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences' and Appendix 3: 'A Brief Consideration of the Scope of Liability'; but at this point it is sufficient to say that a symptomatic concussion, from which an athlete will generally recover within a few days or weeks, falls within an entirely different category of medical complaint to CTE, a disease which almost always results in a long period of debilitation followed by death. As argued in Chapter 9, CTE is most accurately characterised as a neurodegenerative form of dementia or tauopathy. In terms of symptoms, sequelae and prognosis — in short, harm — it is certainly not concussion or mere head trauma.[251]

As discussed in Chapter 4 and Appendix 2, Australian medical journals lagged behind international journals in reporting the risk of CTE. In addition, some medical commentators — for example, Dr Paul McCrory who was associated with the AFL and was Chair of the Concussion in Sport Group, denied at least initially a substantive connection between head trauma and CTE in Australian sports.[252] Does the argued lassitude of Australian medical

[250] See Chapter 2: 'The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments'.

[251] See Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences'.

[252] See the detailed discussion in Appendix 2: 'CTE and "Negativing Influences": Do They Determine Liability?'

journals in reporting on the risk of CTE diminish the foreseeability of CTE in playing contact sport? This question raises three points:[253] (1) Medical specialists associated with a specialist medical duty are likely negligent in sourcing emerging medical information from a single (local) source — and should also refer to a number of foreign specialist journals (a well-recognised practice). (2) Australian medicos were placed on notice of the risk of CTE in the 1990s by two publications: an article by Dr Nathan Gibbs indicating a possible risk of CTE in the playing of rugby league,[254] and a report of the National Health and Medical Research Council that offered some evidence of 'long term problems' caused by cumulative head trauma in contact sport.[255] (3) The standard of care employed in stating clinical opinions may be inadequate, negligent or irrational.[256]

Foreseeability at the duty stage requires only that a risk of harm to a plaintiff, or a class of persons to which the plaintiff belongs, is reasonably foreseen as 'real' or 'not unlikely'.[257] When considering the foreseeability of CTE across the decades prior to Dr Omalu et al's report in 2005,[258] multiple medical studies stated that RHT in the playing of contact sport is a clear cause of CTE. Studies linking non-boxing contact sports with CTE were published in the 1990s in Australia, and, prior to that, in multiple international medical journals from the 1970s. As explained in Chapters 5 and 8, while the risk of CTE in the non-pugilistic sports sport would seem less certain than in the sport of boxing, the commonality of repetitive blows to the head suggests at the very least that the 1970s reports reveal a real risk of foreseeable harm that a reasonable SGB would have inquired into. On these facts alone, it is arguably implausible that a governing body of a major contact sport in Australia would not have had access to sufficient information on the risk CTE-like harm to have foreseen a risk of 'known uncertainty'.

Even where doubt is said to exist between cause and effect, a tortfeasor is not absolved of liability on doubt alone. The extracts cited above reinforce the judicial view that the threshold of foreseeable knowledge for the purposes of negligence occurs where a 'known uncertainty' is evident, where the effects of a disease might not 'emerge for many years', or a 'known or suspected hazard' may harm a plaintiff. [259] This is a major countervailing point to a defendant's argument that knowledge of CTE, and therefore foreseeability of CTE, did not emerge until after Dr Omalu et al's report in 2005, or after *circa* 2010.

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[253] For further discussion, see Chapter 5 and Appendix 2.

[254] Nathan Gibbs, 'Common Rugby League Injuries' (1994) 18(6) *Sports Medicine* 438, 445.

[255] National Health and Medical Research Council (NHMRC), *Football Injuries of the Head and Neck*, (1994) 18, 19. See the discussion in Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times'.

[256] *Rogers v Whitaker* (1992) 175 CLR 479.

[257] *Caterson v Commissioner of Railways (NSW)* (1973) 128 CLR 99, 102.

[258] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[259] See *Bale v Seltsam Pty Ltd* [1996] QCA 288.

3.10 Questions of policy

When one considers the potential liability of SGBs in failing to warn their players of the risk of CTE, it has been argued that policy considerations could determine the outcome of a trial, irrespective of the accuracy and pertinence of the facts underpinning the other relevant elements of negligence. Some sport cases have referred to matters of policy as influencing the course of a judgment. For example, in *Singh v Lynch*[260] the Court of Appeal noted that the *Ipp Report*[261] of 2002 indicated that exemption from liability should not extend where members of schools and other institutions were required to participate in recreational activities, in contrast to 'self-regarding reasons' for such participation; that is, for personal enjoyment.[262] The court said that '[i]n policy terms one can understand why that might be', differentiating between a commercial provider of diving facilities to holiday-makers, which could reply on an exclusion from liability, and a facility provided to people in training or employment.[263] The court, however, dismissed the argument, stating that while policy reasons could lead to a view that 'voluntariness' was not an element to protect a provider from liability, that 'rationale for the protection need not be pursued: it is sufficient that the language adopted by the legislature did not reflect the language of the [Ipp] Report'.[264] In context, neither the common law history of cases such as *Agar v Hyde*,[265] and more particularly the exemptions provided under state civil liability legislation, work to exclude professional athletes nor their administrative organisation from liability in negligence.

Relevant to the discussion of CTE with respect to voluntariness is the date in time at which it is reasonable to expect a premier-level athlete to have become aware of the risk of CTE. This thesis proposes a date of *circa* 2010 at which the risk of CTE would be deemed by a court as obvious to an athlete plaintiff for the purpose of state civil liability legislation. Prior to this estimated date, an objective athlete would be deemed ignorant of the risk of CTE.

Policy impingements that have been argued to relieve an SGB from the consequences of negligence include those where liability would so adversely affect the functioning of the sport in question that the provision of its commercial and social functions — its 'social utility' — would be unsustainable, or where finding of liability would 'open the floodgates' or result in indeterminant liability (as was a concern in *Agar v Hyde*, discussed above). The question of social utility is discussed in Chapter 4 at **[4.3.3]**: 'The "calculus of negligence"'. It should be said, however, noting the wording of the statutory provisions regarding breach and reasonable

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260 [2020] NSWCA 152.

261 *Review of the Law of Negligence* (Final Report, September 2002).

262 *Singh v Lynch* [2020] NSWCA 152, [25], [28], [29].

263 Ibid [25].

264 Ibid [28].

265 (2000) 201 CLR 552.

precautions to be taken,[266] that the social utility — in terms of the rationality and justification for running a risk of harm — of an ambulance speeding to an emergency, as in *Logar v Ambulance Service of NSW Sydney Region*,[267] and a conscious decision to not inform players of a contact sport of a risk to their health and well-being due to apparent concerns as to possible financial implications, differ entirely in character.

3.10.1 Indeterminant liability

A 'floodgates' argument of concern involves a situation where 'if any individual claim were to be accepted, there would be no limit to future claims'.[268] Unlike the IFRB in *Agar v Hyde*, which would have been at least theoretically exposed to litigation by every injured rugby player in the world who was able to claim that a change of rules would have mitigated the risk of injury, each major contact sport in Australia is a confined and identifiable group.

Gleeson CJ stated:

> [T]he extent of the potential liability is confined only by the number of people who choose to play the sport anywhere in the world. … For practical purposes, the liability is indeterminate.
>
> In our opinion, when an appellant attended meetings of the Board, the law of negligence did not require him to conclude that thousands, perhaps hundreds of thousands, of rugby players were so closely and directly affected by his presence as a Board member that he ought to consider whether he should propose an amendment to the laws of the game to protect each player from injury. Unless it did, no duty of care to the respondents could arise.[269]

And tellingly, in contrast to an elite premier domestic competition is the court's consideration of a purported duty owed to players throughout the world:

> [T]he basic rule of the law of negligence is that it is 'incumbent on a claimant to establish breach of an independent duty to himself as a particular individual'. If the appellants owed a duty to these respondents, they must have owed a similar duty to the many thousands, perhaps hundreds of thousands, of persons who played rugby union throughout the world under the laws of the game which the IRFB had made. To hold that each of the individual appellants owed a duty of care to each person who played rugby under those laws strikes us as so unreal as to border on the absurd.[270]

However, the SGBs of major Australian sports do not have a duty relationship with 'perhaps hundreds of thousands' of players but, as argued, do owe a duty of care to the relatively few athletes who play in their premier competitions.

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[266] See *Civil Law (Wrongs) Act 2002* (ACT) s 43(2); *Civil Liability Act 2002* (NSW) s 5B(2); *Civil Liability Act 2003* (Qld) s 9(2); *Civil Liability Act 1936* (SA) s 32(2); *Civil Liability Act 2002* (Tas) s 11(2); *Wrongs Act 1958* (Vic) s 48(2); *Civil Liability Act 2002* (WA) s 5B(2).

[267] [2017] NSWCA 274.

[268] DL Fricke, 'Nervous Shock — The Opening of the Floodgates' (1981) 7(1) *University of Tasmania Law Review* 113, 113.

[269] *Agar v Hyde* (2000) 201 CLR 552 [19], [70].

[270] Ibid [67], quoting JG Fleming, *The Law of Torts* (LBC Information Services, 9th ed, 1998) 60.

3.11 Conclusion

A plaintiff athlete must prove in law that the defendant SGB owed a duty to the athletes who played in its premier competition to take reasonable steps to inform itself of the risk of CTE and warn those players of that risk. Establishing a duty of care requires proof of both 'foreseeability' of the risk of CTE or similar neurological diseases, and 'neighbourhood' as characterised through the relationship between a premier-level contact sport athlete and the defendant SGB.[271] The more difficulty limb to be proven is that of 'neighbourhood'

Some legal commentary has proposed that through the precedent of the High Court decision in the rugby case of *Agar v Hyde*,[272] no duty is owed in Australia by an SGB to those who play its contact sport. This chapter argues, to the contrary, that *Agar v Hyde* did not establish through precedent a recognised category of case. The foundation of this argument is twofold: first, that the rule-setting organisation in *Agar v Hyde*, the IRFB, was not an SGB in the Australian sense and, second, the facts (salient features) depicting the relationship between the plaintiff Agar and the IRFB were not those that characterise 'neighbourhood' in the sense necessary to impute a duty of care. For example, the IRFB had no power to control the decisions of the national rugby authorities to which it communicated or to mandate the adoption of safety rules. In contrast to *Agar v Hyde*, this chapter argued that the facts bearing upon the relationship between a major SGB controlling contact sport in Australia and its premier-level athletes characterise salient features recognised judicially as those through which a duty of care is imputed.

As no appeal court in Australia has found a duty of care to subsist between an SGB and its premier-level athletes, the matter is to be decided as a novel case through a salient features approach. A plaintiff athlete, therefore, must be able to show facts acceptable as salient features signifying a duty of care residing in the defendant SGB.

In each sport case examined in this chapter, the court's finding of a duty of care was based on either the presence or absence ('factors for and against')[273] of the salient features listed by Allsop P in *Caltex Refineries (Qld) Pty Ltd v Stavar*,[274] suggesting the relevance of those facts in the recognition of a duty of care residing in an SGB. Although none of the courts deciding these cases possessed any authority by which an accepted category of case was created, the judicial uniformity across several cases is informing and perhaps propitious.

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[271] Discussion of accessible knowledge of the risk of CTE is the subject of Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times'. The cut-off date of *circa* 2010 is the approximate date that the risk of CTE is argued in this thesis to have become obvious to premier-level contact sport athletes thereby excluding liability of the SGB under civil liability legislation. For further discussion, see Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences'.

[272] (2000) 201 CLR 552

[273] *Sullivan v Moody* (2001) 207 CLR 562, 579–80 [50].

[274] (2009) 75 NSWLR 649.

Although several salient features were noted in this chapter as pertinent, those of 'control' of the SGB over rule formulation, 'reliance' by the athlete on the SGB to exercise its control with reasonable regard to his or her health and welfare given the reality of contact sport, and the 'vulnerability' of the athlete should the SGB not take reasonable steps to safeguard the health and welfare of its players given the context of contact sport.

The case of *Watson v British Boxing Board of Control; World Boxing Organisation Inc*[275] was presented as an exemplar of a duty relationship identified through salient features. The BBBC (the SGB) exercised control over the sport, impliedly assumed an obligation to care for the health and well-being of the boxers under its administration by mandating regulations that medicos attend at ringside, medical examination and standdowns from competition after a knockout, the licensing of boxers and, expressly, through documents stating its concern for maintaining safety standards and that safety should take precedence over commercial interests. In very simple terms, the BBBC controlled the sport of boxing throughout Britain and possessed the power to adjust or introduce rules regulating safety, to conduct inquiries and make announcements regarding the welfare of boxers, the boxers under its authority relied on the BBBC to enforce the safety measures it provided and to proactively inquire into the welfare needs of its boxers, vulnerability arose from the boxers inability to control any rules of the sport, mandate safety regulations or inquire into the adequacy of welfare protocols. In *Agar v Hyde* the relationship between the athlete plaintiffs and the rule-making rugby organisation consisted of none of these features. The IRFB had insufficient control over the sport and its players, an inability to enforce any proposed rule changes, nor did the organisation expressly or impliedly assume responsibility for the health and well-being of players in the position of the plaintiff.

Although it was stated initially that harm in the form of CTE, or CTE-like harm 'ought to' be known to a reasonable SGB, facts specifically relevant to that limb were presented in this chapter. The means by which foreseeability of harm in the mesothelioma cases was presented as offering an insight into judging foreseeability with respect to CTE. In some cases, heard in the 2000s, information on the risk of exposure to asbestos dust being published in medical journals during the 1930s to 1960s was considered to provide a means of foreseeability that defendants ought to have been aware of and acted upon. Foreseeability of a risk that was classified as a 'known uncertainty' as to its toxic nature was considered sufficient establishing a duty of care. Nor, it was argued, did foreseeability require precise harm to be identified as foreseeable.

The duty of care owed by a governing body of a major contact sport to its premier players is perhaps best identified by a passage of Phillips MR in *Watson*:

> It seems to me that the authorities support a principle that, where A places himself in a relationship to B in which B's physical safety becomes dependent upon the acts or omissions of A, A's conduct can suffice to impose on A a duty to exercise reasonable care for B's safety. In such circumstances

[275] [2001] QB 1134.

> A's conduct can accurately be described as the assumption of responsibility for B, whether 'responsibility' is given its lay or legal meaning.[276]

The formulation is of course of wide application — wide enough to include the imposition of a duty in a governing organisation to exercise reasonable care for the safety of its premier-level athletes. Phillips MR also considered cases where liability followed the acceptance of responsibility for another, stating that these 'support the proposition that the act of undertaking to cater for the medical needs of a victim of illness or injury will generally carry with it the duty to exercise reasonable care in addressing those needs'.[277]

The recognition by a court of relevant salient features with respect to the relationship of the SGBs of the major contact sports in Australia and the athletes who play in the premier competitions they organise and administer will determine, as argued, that a duty is owed by those SGBs to take reasonable steps to safeguard the health and well-being of those athletes.

[276] Ibid [49].

[277] Ibid [57].

Chapter 4:
CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care

4.1 Introduction

The previous chapter, Chapter 3, examined the question of an SGB's legal obligation to care for the health and well-being of those who play in its premier-level competition. That chapter argued that the claimed duty must be established as a novel case determined through a salient features approach.

Chapter 4 considers the question of an SGB's breach of duty owed to its premier-level athletes in failing to meet the requisite standard of care necessary to inform itself of the risk of CTE and to warn its premier-level athletes of that risk. The chapter highlights that the SGB's duty to inform itself of the risk of CTE necessarily precedes the duty to warn of the risk of CTE. The chapter reviews case law concerned with meeting the standard of care in relation to a duty to be informed and to warn. With respect to a plaintiff athlete's case, three broad forms of obligation are argued as highly relevant to CTE caused through RHT in playing contact sport: (1) the obligation to be informed where knowledge is available; (2) the obligation to take proactive steps to secure relevant information on a risk of harm and act to ameliorate possible risks prior to their concrete affirmation, along with the obligation to search, discern and consider possible risks of harm; and (3) the obligation to structure a system by which extant and emerging knowledge of the risk of CTE is accessed.

The next chapter, Chapter 5, is material to this chapter in that it examines the factual question underpinning the legal proposition that sufficient accessible information on the risk CTE *was* available to SGBs in the decades leading up to 2010. This information is argued to form the basis to the obligation of an SGB to be informed and to warn its premier players of the risk of CTE.

This chapter is directed primarily to the question of whether an SGB of a contact sport in Australia breached its duty of care in failing to fulfil the standard of care by informing itself of the risk of CTE and, in turn, warning its premier players of that risk.

To fulfil the requisite standard of care, state civil liability legislation requires a duty-holder to take reasonable precautions against a risk of harm that is 'foreseeable' and 'not insignificant' in circumstances where a reasonable person would take precautions.[1] A failure to perform these obligations was described by Rolph as a 'failed step':

> The task of the plaintiff is to establish a prima facie case that, on the balance of probabilities the fault of the defendant caused or materially contributed to the injury; one measure being whether the defendant failed to take a step which more probably than not would have prevented or minimised the damage.[2]

The 'failed step', as argued in this thesis, is the failure of SGBs of contact sports in Australia to inform themselves of the risk of CTE and to warn their premier players of that risk

This chapter is concerned primarily with elucidating the content — the 'step' — that a reasonable SGB would take to eliminate or reduce the risk of CTE. In that respect, three obligations recognised through case law are argued as relevant in meeting the requisite standard of care:

- the base obligation to be informed of the scientific knowledge of the day, and how this differs from the obligation to warn;
- the proactive obligation to warn where scientific knowledge is not fully developed;
- the obligations to inquire, discern, search and consider.

This chapter argues that in failing to fulfil these obligations, SGBs in Australia fell below a reasonable standard necessary to care for the health and well-being of their premier-level athletes.[3]

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1 *Civil Law (Wrongs) Act 2002* (ACT) s 43(1); *Civil Liability Act 2002* (NSW) s 5B (1); *Civil Liability Act 2003* (Qld) s 9(1); *Civil Liability Act 1936* (SA) s 32(1); *Civil Liability Act 2002* (Tas) s 11(1); *Wrongs Act 1958* (Vic) s 48(1); *Civil Liability Act 2002* (WA) s 5B(1). The common law applies in the Northern Territory.

2 David Rolph et al, *Balkin & Davis Law of Torts* (LexisNexis Australia, 6th ed, 2021) [9.2].

3 It is worth noting that the claims set out in this thesis are to be prosecuted and defended in the Jarad Rooke players class action against the Australian Football League (AFL) *Jarad Maxwell Rooke v Australian Football League*, Supreme Court of Victoria, Case: S ECI 2023 00969, Date 6 December 2023.

4.1.1 This chapter proceeds as follows

Section 4.2: The link between the SGB and medicos

This section reviews the relationship between SGBs and their medical associates who advise on matters of player health and fitness. The sole authority to introduce protocols for monitoring risks to player health and to direct its associated sports physicians to inquire into those risks is that of the SGB. It is through this association that the duty to be informed and to warn of the risk of CTE is argued to primarily arise.

The section also proposes that the 'medical model', or the medical standard, is the most apt for determining the level and content of care that an SGB owes to a premier-level athlete. This standard, perhaps adjusted by degrees for the absence of clinical skills, is based primarily on a necessity in contact sport for a proactive monitoring of threats to the health of the SGB's athletes. In addition, the section considers the SGB's obligations with respect to monitoring medicos, issuing directives to medicos, and warning its players of extant and emerging risks to their health and well-being.

Section 4.3: The law: Breach of duty

This section explores the legal principles of negligence relevant to a claim based on an SGB's failure to inform itself of the risk of CTE and to warn its premier players of that risk.

The section considers the statutory pre-conditions of 'foreseeability' and 'not insignificant' with respect to whether the SGB should have taken reasonable steps to inform itself of the risk of harm in the playing of its contact sport, and with respect to a failure to warn its players of the risk of CTE.

In addition, the section considers the 'calculus of negligence', asking the statutory question: Would a reasonable SGB have taken precautions against the risk of CTE? Reviewed are: the probability of harm; the likely seriousness of harm; and, with respect to whether a reasonable SGB would have warned of the risk of CTE, the probability of CTE occurring in the absence of warnings, the seriousness of CTE, the burden of taking precautions to ameliorate the risk of CTE through warnings, and the social utility of the activity.

Section 4.4: The content of the standard of care

Having considered the legislative requirements to establish that CTE presented a risk of harm for which an SGB is required to take precautions, this section examines the standard of care necessary to address the risk of harm through the monitoring of potential harm and the issuance of warnings.

The section is divided into several sub-sections discussing matters pertinent to an SGB's obligation to be informed of the risk of CTE. In sub-section 4.4.1, three key cases that determine the relevant standard of care applicable to a governing body of a premier-level contact sport in Australia form the basis of examination.

Sub-section 4.4.2, on the obligation to be informed, considers the law applicable to the obligation of a duty-holder to take reasonable steps to inform themselves of risks of harm to those to whom a duty is owed. In essence, an SGB will be negligent where information on the risk of CTE 'was or ought reasonably to have been possessed' and reasonable steps had not been established to ensure its possession. The 'fact' of available information on the risk of CTE is the subject of the following chapter — Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times'.

Sub-section 4.4.3 is concerned with medical developments, judgment and proactive obligations, and examines these with respect to a duty-holder SGB's obligation to be informed of emerging risks of harm, risks that are less than certain, risks that are less than scientifically ascertainable, or situations where the risk is harm is potentially serious but may not have risen above the level of 'might be a risk'. A duty-holder must take proactive steps to put itself in a position where it can acquire knowledge; the point being that the consequences of a delay in fulfilling its duty or know and to warn is to expose the SGB's athlete to a greater risk of contracting CTE.

Sub-section 4.4.4, on the obligation to inquire and discern, examines the obligation of a duty-holder to put in place a system by which it can be informed of extant and emerging risks of harm and, in essence, to 'stay up to date' with advancing knowledge. The duty-holder cannot merely wait passively for developing knowledge to arrive, but must establish a system to ensure information that ought to be known becomes known.

Section 4.5: Rule changes and the standard of care: Safety measures — voluntary or duty?

This section examines the question of whether an SGB is obliged to alter the rules of play to make the sport safer. The section argues that there is no such responsibility, although noting that exceptions may apply where rules do not change the basic nature of the sport but are rather collateral to it.

The section also considers whether there is any precedent value flowing from rule changes made by an SGB to improve safety in the sport.

Section 4.6: Conclusion

The chapter concludes that the risk of contracting CTE through playing contact sport at a premier level is both foreseeable and not insignificant. A reasonable SGB would be aware that while the probability of contacting CTE was low, the risk was nonetheless not remote. Potentially serious harm in the form of mental and physical debilitation, and ultimately death, must also form part of the SGB's calculation. As such, the standard of care requires the SGB to establish a proactive medical-level response to informing itself of the risk and warning its players of that risk.

4.2 The link between the SGB and medicos

Because the administrative members of SGBs are unlikely to be educated in the science of medicine, it is essential that they put in place protocols by which they are informed, or become informed, of material risks (both extant and emerging) to the health and well-being of their premier players.

Medical professionals have advised major SGBs in Australia on matters likely to affect the health of the premier players of their sport for several decades.[4] For example, in 1945, in a rugby league match between Newtown and St George at Henson Park in Sydney, St George prop Bill McRitchie had part of his ear bitten off by legendary 'tough', Frank 'Bumper' Farrell. So damaged was McRitchie's ear that an attending doctor at the nearby Marrickville Hospital thought stitching to be futile. 'Next morning he was examined by the *League's medical officer*, Dr HC Finn, who admitted him to Lewisham Hospital.'[5]

In their capacity as advisers to SGBs and their players, medical professionals are expected to be informed of injuries and maladies that affect athletes, including clinical developments that may influence the decision of athletes to play or to continue to play a given sport.[6] Discussed below is the expectation that such experts — as a matter of course and as directed by a dutiful SGB — are required to advise the SGB of extant and emerging risk of harm to its premier players.

Sport and exercise medicine is a specialised practice under the auspices of the Australasian College of Sport and Exercise Physicians, formed in 1985 as the Australian College of Sports Physicians.[7] Recognised by the Council of Presidents of Medical Colleges, 'Fellows' must hold a medical degree, have completed 'foundational training' in sport and exercise medicine, passed an entrance exam and an interview, and gained clinical experience in sports medicine over a minimum of four years' full-time training. The range of specialised skills within a sport setting and the consequential obligation of the medico to the SGB and athlete are reflected in the representations of the Australian College of Sport and Exercise Physicians: 'All Sport and Exercise Physicians have experience looking after premier-level athletes in team environments and apply these principles to the management of all patients.'[8] The College provides additional

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4 See 'History of SMA', *Sports Medicine Australia* (Web Page) <https://sma.org.au/about-sma/history-of-sma/>; Nathan Gibbs, 'Common Rugby League Injuries' (1994) 18(6) *Sports Medicine* 438; AFL, 'Responsible Approach to Concussion in the AFL', Information Paper, May 2012, 1: '[Medical research on concussion] has been ongoing since 1985.' See multiple references in Chapter 2 at **[2.4]**: 'The importance of the timeline of revelations of CTE'.

5 Larry Writer, *Bumper: The Life and Times of Frank 'Bumper' Farrell* (Hachette, Sydney, 2011) 151. Emphasis added.

6 *Wallace v Kam* (2013) 250 CLR 375.

7 'History', *Australasian College of Sport and Exercise Physicians* (Web Page) <https://www.acsep.org.au/page/about/the-college/history>.

8 'Medical Practitioner Referral Guide', *Australasian College of Sport and Exercise Physicians* (Web Page, June 2020) <http://www.acsep.org.au/content/Media/Medical%20Practitioner%20Referral%20Guide%202018.pdf>.

resources to allied health professions, including physiotherapists, dietitians, psychologists and accredited exercise physiologists.

Several independent, though often affiliated, bodies support the professional practice of sports medicine in Australia. The organisation Sports Medicine Australia was 'formed in 1963 following the amalgamation of the Australian Sports Medicine Association and the Australian Federation of Sports Medicine',[9] with membership originally restricted to medical doctors. In time, membership widened to include a range of medical professionals, including physiotherapists, psychologists, exercise physiologists, sports scientists, academics and chiropractors. Sports Medicine Australia states that it is 'widely acknowledged internationally as a leading multidisciplinary sports medicine body'.[10]

Another organisation, Sports Doctors Australia, was launched in 1999:

> [A] number of doctors undertaking postgraduate training in sports medicine felt there was a need for an association representing non-sports physician doctors practicing in sports medicine. It was envisaged to form a group within Sports Medicine Australia as well as being an independent on-going professional society.[11]

Among medical professionals, a system to monitor and implement advances in medical science is a required legal duty,[12] hence the importance of resources such as the official journal of Sports Medicine Australia, *Journal of Science and Medicine in Sport*, which was first published in 1964 under the name *Australian Journal of Sports Medicine*.[13] Other accessible and well-regarded resources include the *British Journal of Sports Medicine*, launched in January 1964, the *International Journal of Sports Medicine*, first published in 1980, and the *Exercise and Sports Science Review*, published by the American College of Sports Medicine from 1973, among many other international sports medicine journals and many specialist medical journals relevant to sport.

Once appointed by an SGB, the medico's obligation is to be informed of medical developments material to the health of athletes, to inform their affiliated SGB and, where relevant, to warn the athletes under their care of risks of harm associated with play and training.

4.2.1 Applying the 'medical model' as the standard of care

While several legal formulae are available to guide the obligation to warn of possible harm, the most practical methodology by which to judge the failure or otherwise of an SGB to inform

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9 'History of SMA', *Sports Medicine Australia* (Web Page) <https://sma.org.au/about-sma/history-of-sma/>.

10 'About', *Sports Medicine Australia* (Web Page) <https://sma.org.au/about-sma/>.

11 'About SDrA', *Sports Doctors Australia* (Web Document) <https://www.sportsdoctors.com.au/about>.

12 *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2, [60].

13 'History of SMA', *Sports Medicine Australia* (Web Page) <https://sma.org.au/about-sma/history-of-sma/>.

itself and warn its athletes of the risk of CTE is that applicable to medical practitioners — what may be referred to as the 'medical model'. There is nothing remarkable about this proposal as a matter of logic. The general approach to any legal obligation to warn of a potential risk broadly mirrors the 'medical model': investigation, deliberation or assessment, and mitigation. Such an approach itself reflects a duty of care and breach of that duty — a foreseeable risk that a reasonable person would take precautions to avoid.[14] For example, in *Alameddine v Glenworth Valley Horse Riding*,[15] a company conducting quad-bike treks through the Australian bush investigated the risk of harm, deliberated on the possibility of harm and issued a warning: 'Please be advised that quad biking is an inherently dangerous activity. You are required at all times to ride at a speed which is within your ability and that is suitable for the ground conditions you may experience.'[16]

In assigning a medical standard of obligation to an SGB, it must also be noted that unlike, say, a duty to warn of hidden rock ledges at a diving site, which remains constant over time such that a single posting of a warning sign will suffice, the revealed risks of harm associated with sport, especially contact sport, are liable to change with time. As medical science progresses new methods of detecting risks of harm emerge and new risks to health and well-being are detected. Aspects of a sport may change to elevate the risk of harm, recent examples of which include the increased size and speed of contact sport athletes, brought about by focus on body building exercise and nutrition, which serve to magnify the force of exerted between an attacker and defender. The sole means and authority by which to direct the monitoring of developments in the risk profile of a sport at premier level resides with the SGB.

Most pointedly, the risk encountered by an athlete is, in fact, a 'health risk'. As noted above, the accepted benchmark for evaluating the standard of care in providing health-related services is the 'medical model'. In the sport case of *Watson v British Boxing Board of Control; World Boxing Organisation Inc*, for example, Phillips MR said that the 'most obvious category of case of a duty of care to administer medical treatment to restrict the consequences of injury or illness … is that of the duty owed by a doctor or a hospital authority to a patient'.[17] As discussed in the previous chapter, a duty of care was formed when the British Boxing Board of Control held itself out as 'as treating the safety of boxers as of paramount importance' and as possessing 'special knowledge to a defined class of persons'.[18] His Lordship then referenced two non-medical cases as supporting authority on the obligations of a non-medically trained defendant charged with the delivery of medical-related care: *Hedley Byrne v Heller*,[19] which concerned

[14] See state civil liability legislation; for example, s 5B(1) of the *Civil Liability Act 2000* (NSW).

[15] [2015] NSWCA 219.

[16] Ibid [6]. The company was nonetheless held liable, as other information was considered to contradict the warning.

[17] *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [49].

[18] Ibid [85].

[19] [1964] AC 465.

financial misrepresentation, and *Capital and Counties plc v Hampshire County Council*,[20] which concerned breach of duty by a fire brigade. That is, Phillips MR applied the medical standard to a case that did not concern the obligations of a medico. Relevant statements include:

> As a general rule a sufficient relationship of proximity will exist when someone possessed of special skill undertakes to apply that skill for the assistance of another person who relies upon such skill and there is direct and substantial reliance by the plaintiff on the defendant's skill.[21]

In summing up, Phillips MR stated:

> It seems to me that the authorities support a principle that, where A places himself in a relationship to B in which B's physical safety becomes dependent upon the acts or omissions of A, A's conduct can suffice to impose on A a duty to exercise reasonable care for B's safety. In such circumstances A's conduct can accurately be described as the assumption of responsibility for B, whether 'responsibility' is given its lay or legal meaning. [22]

Special skill may be discerned from an SGB's knowledge of the health risks of playing contact sport, its appointment of a dedicated physician to the sport, recognising through its contractual mandate that players attend medical examinations or standing down following incidents like concussive events, among others.

In *Cassidy v Ministry of Health*, Denning LJ said:

> I take it to be clear law, as well as good sense, that, where a person is himself under a duty to use care, he cannot get rid of his responsibility by delegating the performance of it to someone else, no matter whether the delegation be to a servant under a contract of service or to an independent contractor under a contract for services.
>
> In my opinion authorities who run a hospital, be they local authorities, government boards, or any other corporation, are in law under the selfsame duty as the humblest doctor. Whenever they accept a patient for treatment, they must use reasonable care and skill to cure him of his ailment.[23]

Given that the SGB is under a duty to take reasonable steps to care for the health and well-being of its premier-level athletes, can the 'reasonable standard' of care be anything less than the 'medical standard' — which, among other obligations discussed below, includes a proactive approach to monitoring for threats to the players' health by ensuring that its medical associates report extant and emerging risks? It is only through notification of the material risk of CTE that a contact sport athlete can respond to that threat, and it is the SGB as the controlling body that possesses the means to establish a system by which to monitor for emerging risks of harm, to mandate steps to be taken in mitigation of risks such as the issuance of warnings regarding risks.

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[20] [1997] QB 1004, 1034–5.

[21] *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [85].

[22] Ibid [47].

[23] [1951] 2 KB 343, 360. See also *Hedley Byrne & Co Ltd v Heller & Partners Ltd* [1964] AC 465.

The alternative 'standard' of care is one that does not oblige an SGB to act until it is informed through happenstance or comes across news (perhaps in the media) of a risk of harm.

A sport administrator or board member does not usually possess the clinical skills of a physician. Nonetheless, the conditions of play, the forms of body-on-body contact and the observed experience of obvious serious injury are quite clear to reasonably observant sport officials. Equally, a sport official does not need to offer a histological explanation for CTE; it is sufficient that he or she be aware of the general process of occurrence of harm and the damaging effects of that harm. At its most simple, a blow to the head followed by well-known symptoms of concussion, which may be one of many. It is the SGB that possesses the power to instruct its associated medicos to investigate and advise on reports of emerging risk of harm to its players.

4.2.2 The SGB's obligations

As information is necessarily sought from the SGB's associated medicos there is perhaps a temptation to believe that liability for failing to warn of the risk of CTE is that of the advising 'team doctor'. However, in appointing such a doctor, granting access to the playing and training facilities, and directing players to him or her for medical care, the SGB in effect 'vouches' for the competency of the doctor. In this sense, the SGB is not an actor independent of the team doctor.

A sport medico's access to athletes, 'as athletes' rather than private patients, occurs only under licence or with the contractual assent of the SGB. The medico's attendance at games and training, a physical presence on the side-line, entry to the field of play and to change rooms in granted only with the permission of the organisation. Each consultation is an opportunity to question the athlete on his or her history of head trauma and to advise of possible cognitive disfunction.

As a condition of selection to representative and premier-level teams, an athlete's contract requires, almost universally, medical examination by an assigned physician, along with ongoing consultation. For example, the National Rugby League (NRL) contract, which is mandated for use by all premier-level clubs, stipulates that players 'immediately report any illness, accident or injury of any nature to the Club Medical Officer and carry out any reasonable instruction given to him by the Club Medical Officer'.[24] Ian Kennedy J said at trial in *Watson v British Boxing Board of Control; World Boxing Organisation Inc* of the Board's duty of care to an injured boxer under its authority:

> The Board professes … its lively interest in his safety. I do not believe there is any difference in principle between giving advice about safety and laying down rules to provide for safety. … To my

[24] NRL Player Contract, Section 3.1: 'Player's general obligations'.

> mind it is difficult in such a situation to profess a concern for safety and to deny a duty such as I have described.[25]

To draw a parallel, it can equally be said that in mandating consultation with a team physician, the SGB professes a 'lively interest' in the safety of the athletes under its mandate.[26] Although treatment of an athlete-patient falls to the medico, in *Watson* the Board was liable in failing to introduce a system by which the medico would attend promptly and efficiently to the boxer's needs. There being an arguable 'distinction between instructions as to duties and instructions as to how to perform duties',[27] the mere appointment of a medical officer is not sufficient to offset the organisation's obligation to be conscious of developments likely to affect the well-being of its athletes and to warn its athletes of material concerns.

The rationale of an SGB mandating through contract an athlete's attendance upon a team doctor is only explicable in terms of a belief that the sport carries a risk of physical harm. It should be noted as a matter of practical reality that a warning of risk by a team medico will inevitably be conveyed to other medicos throughout the sport, and indeed to other sports. This is particularly so where the only way to protect club players from risks of harm is to convey concern to the SGB, which alone possesses the power to alter the rules of the sport and the protocols under which it is conducted.

As a further practical matter, without the SGB mandating consultation with the team doctor and without the club appointing a team doctor for this purpose, an athlete, of necessity, is forced to seek advice from a personal physician unrelated to the organisation. And while the right of independent consultation is not denied outright, the likely response of an athlete — particularly a young one — would be to adhere to the organisation's direction and accept without question the convenience of attending an on-site team doctor. As noted by Ian Kennedy J at trial with regard to boxing in *Watson*: 'There is always a risk, and the pool from which professional boxers tend to be recruited is unlikely to be one with an innate or well-informed concern about safety.'[28] On appeal, Phillips MR stated of the provision of medical expertise that the boxer 'would not be in a position to know whether the provisions that the Board required to be put in place represented all that it was reasonable to provide for his safety'.[29]

The logical outcome of a mandate to consult the team doctor is the removal of the player from the free market for medical services, effectively diminishing the athlete's autonomy of choice. In these circumstances, as argued, an obligation falls upon the SGB to ensure that the provision of medical services is to a reasonable standard and to supervise the well-being of the

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[25] Quoted with approval by Phillips MR in the Court of Appeal: *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [6].

[26] Discussed extensively in Chapter 3: 'A Duty Relationship between an SGB and the Athletes Who Play Contact Sport'.

[27] *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134, [114]. Phillips MR thought the defendant's argument as to such a distinction elusive, given the facts of this case.

[28] Quoted with approval by Phillips MR in the Court of Appeal: ibid [6].

[29] Ibid [85].

athlete with respect to those services within the context of the risks associated with contact sport. Some similarity in application is found in the case of *Barrett v Ministry of Defence*,[30] where a naval petty officer placed a drunken and comatose sailor on a stretcher and left him unsupervised in a cabin. The sailor vomited while unconscious and died of asphyxiation. The Ministry was vicariously liable in negligence, the Court of Appeal holding that 'when the defendant assumed responsibility for [the sailor], it accepts that the measures taken fell short of the standard reasonably to be expected. It did not summon medical assistance and its supervision of him was inadequate.'[31] Correspondingly, athletes who reasonably perceive they have been 'taken under the wing' of the SGB, and to some degree isolated from alternative assistance, have a reasonable expectation that those caring for their health will be competent and monitored for ongoing competency including awareness of extant and emerging risks of harm.

At the most basic, in delegating is duty of care, the SGB is to ensure that its medical professionals tend to the health needs of its athletes, to make inquiry as to whether an athlete is fit for play, and to ask whether medical developments such as new treatments or threats to the health of athletes have emerged and been appraised.[32] With respect to CTE, when — through observation, conversation, or medical or media reports — the organisation becomes aware of cognitive disfunction caused by head trauma in sport, it is duty-bound to make inquiry of its medical professionals, to direct those professionals to make further inquiry where information is contradictory or insufficient in forming a reasonable judgment, and to critically consider any advice given.[33]

On this point, Hunt AJA said in *South Eastern Sydney Area Health Service v King*,[34] as regards delegation in 'a system based on collaborative teamwork', that the principal had to 'do more … than merely rely in a purely reactive manner' to information 'that may randomly have been received'; rather: 'If he could not do it himself, he had to take reasonable care to ensure that someone else actively searched for material which was relevant to the task he was to undertake.'[35]

4.2.3 Distinct duties to be informed and to warn

As discussed at length throughout this chapter, a professional SGB must, as a singular entity or through its associated medical staff, investigate and understand the risks of harm in playing

[30] [1995] 1 WLR 1217.

[31] Ibid 1217.

[32] *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2; *Wallace v Kam* (2013) 250 CLR 375.

[33] *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2; *Wallace v Kam* (2013) 250 CLR 375.

[34] [2006] NSWCA 2, [60] (Mason P and McColl AJA agreeing).

[35] Ibid.

its sport and warn its players of any 'material risk' of which it is aware.[36] A material risk is one 'to which a reasonable person … would be likely to attach significance', or one 'to which [the duty-holder] knows or ought reasonably to know [an affected person] would be likely to attach significance'.[37] Most reasonable athletes will consider the risk of cognitive damage and the behavioural manifestations associated with CTE to be significant; whether those athletes cease playing in response to information regarding such risk is a different question.

The obligation to inform oneself and to know is distinct from the obligation to warn.[38] Only upon receiving information and weighing its value can a duty-holder determine whether a warning of a material risk should be given. There is no obligation to warn of a non-material risk.

4.3 The law: Breach of duty

The legislative provisions applicable to a breach of duty due to a failure to meet the requisite standard of care, along with relevant case law, are considered in this section.

4.3.1 The statutory provisions

Section 5B(1) of the *Civil Liability Act 2002* (NSW)[39] provides:

> (1) A person is not negligent 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.

Section 5B(2) provides:[40]

> (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,

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36 *Wallace v Kam* (2013) 250 CLR 375, [8] (a medical case).

37 Ibid.

38 *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2.

39 See also *Civil Law (Wrongs) Act 2002* (ACT) s 43(1); *Civil Liability Act 2003* (Qld) s 9(1); *Civil Liability Act 1936* (SA) s 32(1); *Civil Liability Act 2002* (Tas) s 11(1); *Wrongs Act 1958* (Vic) s 48(1); *Civil Liability Act 2002* (WA) s 5B(1) (where the focus is on being 'at fault' rather than 'negligent'). The common law applies in the Northern Territory.

40 See also *Civil Law (Wrongs) Act 2002* (ACT) s 43(2); *Civil Liability Act 2003* (Qld) s 9(2); *Civil Liability Act 1936* (SA) s 32(2); *Civil Liability Act 2002* (Tas) s 11(2); *Wrongs Act 1958* (Vic) s 48(2); *Civil Liability Act 2002* (WA) s 5B(2). The common law applies in the Northern Territory.

(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.

Section 5B(2) is the so-called 'calculus of negligence' and is a statutory adjustment of the words of Mason J in *Wyong Shire Council v Shirt*:

> The perception of the reasonable man's response calls for a consideration of the magnitude of the risk and the degree of the probability of its occurrence, along with the expense, difficulty and inconvenience of taking alleviating action and any other conflicting responsibilities which the defendant may have.[41]

The calculus of negligence is, therefore, a process for determining through factual evidence if a legal duty of care has been breached.

A finding of a breach of duty requires that 'the three pre-conditions set out in s 5B (1) are satisfied'[42] and, in turn, satisfaction of the elements of s 5B(2). To illustrate, if a risk is extremely dangerous but not foreseeable at the time of the purported breach the defendant cannot be negligence.

All states, other than Victoria, interpret the terms 'significant' and 'not insignificant' through the common law. The term 'not insignificant' has been interpreted by Hodgson JA in *Drinkwater v Howarth* as follows:

> Assuming that s 5B [of the New South Wales Act] applies, … there is no possibility of a different result of applying a test that the risk in question be not insignificant, from applying the test as formulated in [*Wyong Shire Council v*] *Shirt*, namely that the risk be not far-fetched or fanciful.[43]

In the later case of *Shaw v Thomas*, Macfarlan JA held:

> In *Wyong Shire Council v Shirt*, Mason J referred to a risk 'which is not far-fetched or fanciful' as 'being real and therefore foreseeable' … The requirement in s 5B(1)(b) [of the New South Wales Act] that the risk be 'not insignificant' imposes a more demanding standard but in my view not by very much.[44]

In contrast, in Victoria the *Wrongs Act 1958* (Vic) at s 48(3) gives content to 'insignificant risk' stating:

(3) For the purposes of subsection (1)(b) —

(a) ***insignificant risks*** include, but are not limited to, risks that are far-fetched or fanciful; and

---

41 *Wyong Shire Council v Shirt* (1980) 146 CLR 40, 48.

42 *Mungis (No 2) Pty Ltd v Still* [2011] NSWCA 261, [51] (Sackville AJA).

43 [2006] NSWCA 222, [25].

44 [2010] NSWCA 169, [44] (Beazley and Tobias JJA agreeing).

(b) risks that are ***not insignificant*** are all risks other than insignificant risks and include, but are not limited to, significant risks.

Although headed 'duty of care' the provisions of s 5B(1) and (2), as the High Court observed, 'are evidently directed to questions of breach of duty'.[45] In essence, what would a reasonable person do in the circumstances of a risk of harm by way of the listed considerations?

The application of the provision involves weighing paras (a) and (b) against paras (c) and (d).[46] It is also well recognised that s 5B(2) 'operates against the backdrop of the common law of negligence and incorporates concepts derived from the common law'.[47] A plaintiff must adduce evidence of each factor to prove that a reasonable person would have taken precautions to ameliorate the risk.[48]

Discussion of s 5B as it relates to an SGB's breach of duty follows this progression:

- First, s 5B(1) is considered regarding the foundational prerequisites that CTE was a foreseeable risk of playing contact sport that an SGB knew or ought to have known and presented a not insignificant risk of harm to an SGB's premier players.
- Second, s 5B (2), the 'calculus of negligence', is reviewed with respect to the elements a court is to consider in deciding whether a reasonable SGB would have taken precautions in respect to the risk of CTE, a risk that was 'foreseeable' and 'not insignificant'.
- Third, the actions necessary for an SGB to fulfil its obligation to be informed of emerging and extant risks of harm and to warn its premier players of the risk of CTE are considered.

4.3.2 The pre-required conditions: 'Foreseeability' and 'not insignificant'

Section 5B(1) sets out two substantive elements to be proven before proceeding to consideration of the 'calculus of negligence' at s 5B(2). One, was the risk 'foreseeable' or, expressed alternatively, 'ought' the defendant have 'known' of the risk? Two, was the risk 'not insignificant', referring to the existence of a risk that is something 'not very much more' than 'far-fetched or fanciful'?[49] That is, the standard to determine whether a risk is foreseeable is set quite low as regards the operation of s 5B(1).[50]

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45 *Adeels Palace Pty Ltd v Moubarak* (2009) 239 CLR 420, [13].

46 *Roads and Traffic Authority of NSW v Dederer* (2007) 234 CLR 330.

47 *Stephens v Giovenco* [2011] NSWCA 53, [28].

48 *Adeels Palace Pty Ltd v Moubarak* (2009) 239 CLR 420.

49 *Shaw v Thomas* [2010] NSWCA 169, [144].

50 See *Review of the Law of Negligence* (Final Report, September 2002) [7.15] ('*Ipp Report*'); *Drinkwater v Howarth* [2006] NSWCA 22.

Two closely linked questions are to be consider. One, concerned with the foreseeability and not insignificant risk of harm befalling a premier-level athlete due to the SGB's failure to inform itself of the risk of CTE, or sport-caused injuries in general. Two, concerned with the foreseeability and not insignificant risk of harm befalling a premier-level athlete due to the SGB's failure to warn its premier-level athletes of the risk of CTE.

The 'risk' in question, as discussed extensively in Chapter 9, is the risk of CTE, CTE-like harm, a tauopathy or a neurodegenerative disease, or indeed a risk that is expressed by its symptoms and sequelae.[51]

A 'Ought' the SGB have informed itself of the risk of CTE?

On the basis that a defendant SGB is argued to have possessed a duty to take reasonable steps to safeguard the health and well-being of its premier players, 'ought' the SGB have known — to have foreseen — that playing contact sport exposed those players to the risk of CTE-like harm through RHT? The standard is whether a reasonable SGB in its position should have been aware of the risk. This is an inquiry of fact. Several chapters of this thesis have referred to the stock of medical articles available to have provided a dutiful SGB through its medical associates with the means to be informed of the risk of CTE. Most basically, had an SGB instituted reasonable protocols to monitor for extant and emerging risks, the harm associated with CTE would have been known and certainly 'ought to have known'.[52] As discussed above, medicos appointed to monitor and report to an SGB are held to a higher standard of care and are expected to 'take reasonable care to keep informed' of medical developments.

(i) Sources of knowledge available to an SGB or as informed through its medical associates

Dr Martland's article in the *Journal of the American Medical Association*[53] is identified as a foundational in the understanding of CTE. Martland's work established a cause-and-effect relationship between RHT and long-term cognitive disfunction. Medical journals have frequently published warnings about CTE-like harm associated with boxing. Researchers such as Critchley (1957)[54] and Mawdsley and Ferguson (1963)[55] described symptoms and personality deficiencies in ex-boxers that were consistent with CTE, and emphasised RHT as a causative factor. In the 1970s–1990s, knowledge of CTE expanded into other contact sports,

[51] See Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences'.

[52] See the discussion in *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2.

[53] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

[54] M Critchley, 'Medical Aspects of Boxing, Particularly from a Neurological Standpoint' (1957) 1(5015) *British Medical Journal* 357.

[55] C Mawdsley and FR Ferguson, 'Neurological Disease in Boxers' (19 October 1963) *The Lancet* 795.

such as rugby, soccer and American football. Tysvaer (1992)[56] investigated the effects of head trauma in soccer. *The Lancet* published a letter to the editor in 1976 raising concerns about the effects of head trauma in rugby and soccer, suggesting that players who have suffered more than two head injuries should stop playing.[57] Australian publishers such as Dr Nathan Gibbs (1994)[58] and the National Health Medical Research Council (1994),[59] and more recent studies by Omalu et al (2005)[60] and McKee et al (2009),[61] are well covered and need not be reviewed further.

Given the available resources stretching back decades into the twentieth century and the association between SGBs and their appointed medicos, the question of whether a reasonable SGB ought to have known of the risk of CTE in the playing of contact sport can be answered in the positive.

B 'Ought' the SGB to have known of a risk of playing if not warned?

On a more general level, 'ought' the SGB have known that a player who was not warned of the risk of CTE would be more likely to elect to play in its competition and, in so doing, risk contracting CTE? On the basis that a 'real risk is one that would occur to the mind of a reasonable man in the position'[62] of the defendant, it can be said correctly that it is reasonably foreseeable to the SGB that if it did not warn the premier player of the risk of CTE or neurological harm through RHT in playing its sport, the player would be exposed to neurological damage that he or she may have elected to avoid if warned. The question at this stage is not whether the player would have acted on the warning, but that a risk of harm followed from not giving the warning — a warning that 'a reasonable person in the respondent's situation would have taken to guard against the foreseeable risk of injury which existed'.[63]

(i) The obligation to be informed generally

Because the risk of CTE had been known for decades prior to 2010, a plaintiff athlete may merely point to the fact that the SGB ought to have known of the risk. However, the obligation

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[56] AT Tysvaer, 'Head and Neck Injuries in Soccer: Impact of Minor Trauma' (1992) 14(3) *Sports Medicine* 200.

[57] See ID Adams, 'Brain Damage in Sport' (13 March 1976) *The Lancet* 585.

[58] Nathan Gibbs, 'Common Rugby League Injuries' (1994) 18(6) *Sports Medicine* 438.

[59] National Health Medical Research Council, *Football Injuries of the Head and Neck* (Report, 1994).

[60] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[61] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

[62] *Overseas Tankship (UK) Ltd v The Miller Steamship Co Pty (Wagon Mound (No2))* [1967] 1 AC 617, 643.

[63] *Nagle v Rottnest Island Authority* (1993) 112 ALR 393, 398.

is broader and extends to establishing a system that permits an SGB to reasonably monitor for all extant and emerging risks to its premier players. In fact, had the risk of CTE not emerged until, say, 2000, it would be reasonable to have expected the SGB to have a system in place to monitor for that risk. In short, the failure to monitor for CTE-like harm is a failure to have initiated a system of monitoring for risks to player health and well-being in general.

C Was the risk of CTE 'not insignificant'?

According to the *Ipp Report* in 2002, the 'phrase "not insignificant" is intended to indicate a risk that is of a higher probability than is indicated by the phrase "not far-fetched" or "fanciful"'.[64] Little is likely to turn on this altered phrase given that judgments have taken the position that the difference between the two is not likely to be material.[65] The test of 'not insignificant' has been described by the New South Wales Court of Appeal as 'not particularly high'.[66] Or similarly: 'The increase in the necessary degree of probability is not quantifiable and it might be so minor as to make no difference at all.'[67]

It is important to note, especially where a risk of harm is low, that each element is to be considered in combination, the danger having been noted 'of treating the various elements as divisible and the importance of treating the concept of the reasonable person's response to a risk as part of a single overall test of reasonableness in the concept of the tort'.[68] This was reiterated by the Court of Appeal in *South Sydney Rugby Leagues Club v Gazis*: 'The significance of risk will depend upon a variety of factors, including obviousness, likelihood of occurrence and seriousness of consequences.'[69] In essence, was the risk that materialised reasonably foreseeable from the defendant's actions or inactions?

Is, then, the risk of a plaintiff athlete contracting CTE due to an SGB's failure to inform itself of the risk of CTE and, in respect to the major question, due to the SGB's failure to warn the plaintiff athlete of the risk of CTE, 'not insignificant'?

The first question takes two forms. One, in the playing of a violent contact sport, is it reasonable to appreciate a risk of a serious 'sport type' of harm made known through medical journal publications or announced to an SGB through its medical associates? An example would be blood-borne infection through the communal use by players of blood-soaked sponges when wiped down by medical attendants on the field of play. As such, there is a 'not insignificant risk' that an SGB's failure to establish a means to be notified of harm caused in playing contact sport will be unable to warn its premier players of emerging risks, including

64 *Review of the Law of Negligence* (Final Report, September 2002) [7.15].

65 *Drinkwater v Howarth* [2006] NSWCA 22, [16] (Basten JA; Tobias and Hodgson JJA agreeing).

66 *JFIT Holdings Pty Ltd v Powell* [2021] NSWCA 137, [39]; *Sibraa v Brown* [2012] NSWCA 328, [49].

67 *Meandarra Aerial Spraying Pty Ltd v GEJ Geldard Pty Ltd* [2013] 1 Qd R 319, [26].

68 *Drinkwater v Howarth* [2006] NSWCA 22, [22].

69 *South Sydney Rugby Leagues Club v Gazis* [2016] NSWCA 8 [89].

neurological diseases associated with RHT. Two, on the basis that CTE-like harm has been the subject of medical journal reporting since at least Dr Martland's study published in the *Journal of the American Medical Association* in 1928, does a failure to institute a protocol of medical inquiry indicate a 'not insignificant risk' that the SGB would consequently fail to inform itself of CTE as a type of neurological disease associated with RHT while participating in its premier-level competition?

The second question concerns whether there was a 'not insignificant risk' that a premier player who was not warned of the risk of CTE would play in a contact sport that he or she would have otherwise avoided, and in so doing was exposed to neurological harm through RHT.

It can rightly be said that both risks are 'not insignificant', there being at this point in the legislation not consideration of the probability of the harm.

4.3.3 The 'calculus of negligence': Would a reasonable SGB have taken precautions against the risk of CTE?

Whether a reasonable person would have taken precautions against a risk of harm requires consideration of what is referred to as the 'calculus of negligence'. The phrase originated with the judgment of Mason J in *Wyong v Shirt*,[70] and the common law rules are now incorporated in s 5B(2) of the *Civil Liability Act 2002* (NSW) and its equivalents.[71]

As reviewed in the previous section, two types or breaches may be referred to: the failure to be informed of extant and emerging risks of harm in the playing of contact sport and the failure of the SGB to warn its premier players of the risk of CTE-like harm. For purposes of expedience, this section will concern only the failure to warn (with the failure to be informed being subsumed within that breach).

The negligence claimed is the failure of an SGB to take reasonable steps to warn its premier players of the risk of CTE. This failure caused the plaintiff athlete to participate in the SGB's sport, where RHT manifested in actual harm in the form of CTE.

The application of the provisions of s 5B(2) as they apply to the obligation of an SGB to be informed and to warn of the risk of CTE is briefly considered in this section. The adequacy of the defendant's response to a risk of harm is a question of fact.[72] Many of the 'facts' that determine whether a reasonable SGB would have 'taken precautions' were discussed in previous chapters, describing — among other things — the process of contraction through RHT, the seriousness of the risk, the means of prevention and amelioration through timely

[70] *Wyong v Shirt* (1980) 146 CLR 40, 47–8.

[71] See **[4.3.1]** above.

[72] *Wyong v Shirt* (1980) 146 CLR 40, 47–8.

warnings, the availability of knowledge of the risk of CTE, the disabling course of the disease, its symptoms and sequelae displayed as extreme forms of mental and physical disfunction, and a prognosis that leads finally to death.

It should also be noted that the applicable facts do not operate in isolation one from the other but must be 'balanced out [so] that the tribunal of fact can confidently assert what is the standard response to be ascribed to the reasonable man placed in the defendant's position'.[73]

The first requirement in assessing the probability of contracting CTE, is the identification of the 'true source of the risk' of harm. In the context of this thesis, an SGB in failing to warn of the risk of CTE exposed its premier players to the risk of a neurological disease which materialised.

4.3.4 Accessing information on the risk of CTE

On the simplest of levels, an SGB possesses a duty to warn its premier players of the risk of CTE. The standard of care required to fulfil this duty necessitates that the SGB take reasonable steps to inform itself of the risk of CTE and, upon assessing the materiality of that risk, to warn its players.

An assumption can reasonably be made that medical practitioners know or ought to know of the probability of harm in the form of CTE caused by playing contact sport. As argued in Chapter 4, an SGB also possesses a duty to warn its premier players of the risk of CTE. But no assumption of knowledge can necessarily be made.

4.3.5 The probability that the harm would occur if care was not taken

The concept of 'probability of the risk of harm' refers to the probability of that of harm actually materialising. In contrast 'foreseeability of harm' under s 5B(1), may concern foreseeability of harm that may in fact be 'quite unlikely to occur'.[74] As noted by the High Court, 'the measure of care increases in proportion with the danger involved'.[75]

A very low risk, or no probability of that risk eventuating, will likely indicate that the defendant was not negligent in taking the precautions proposed as necessary by the plaintiff. That is, why take precautions where there is no, or low, risk of harm. In the spinal injury case of *Dederer*, the probability of harm was, at least demonstrably, so low that liability did not attach. Gummow J the High Court found:

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[73] Ibid.

[74] Ibid 47.

[75] *Swinton v China Mutual Navigation Co Ltd* (1951) CLR 553.

> The frequency of jumping and diving was only startling if one ignored the fact that no-one was injured until Mr Dederer's unfortunate accident. Far from being a risk with a high probability of occurrence, the probability was in truth very low …[76]
>
> Despite the frequency of jumping and diving from the bridge, no-one was injured until Mr Dederer's unfortunate dive.[77]

Callinan J referred to 'a risk that had not been realised for 40 years'.[78]

While the probability of CTE being contracted by a premier player is low, the risk is far from non-existent. Medical publications for decades had cited case studies of CTE-like harm experienced by players of contact sports involving RHT. Brief reference can be made to Chapter 2, where several authorities indicated their involvement with CTE sufferers or those thought to be suffering the symptoms of CTE. Dr Rowena Mobbs indicated that she had treated 180 patients suspected of suffering traumatic encephalopathy syndrome (TES), with '52 confirmed with probable TES on cognitive testing'.[79] Suter et al revealed the statistics on CTE in Australia through the donation of 21 brains to the Australian Sports Brain Bank. All 21 had participated in sports with risks of RHT. Further, '[a]ll but one donor exhibited some form of neurodegeneration, and 13 presented two or more neurodegenerative pathologies. The most frequent neuropathology was CTE: 12 donors had pathognomonic CTE lesions.'[80]

The Boston University CTE Centre reported that 345 former NFL players out of 376 subjects had been diagnosed with CTE at autopsy.[81] Although it appears that the percentage of players of American football who contract CTE markedly exceed those who play the common Australian contact sports, the commonality of RHT reinforces the obligation of SGB's to warn of the 'real risk' of CTE.

With respect to the obligation of an SGB to warn of the risk of CTE, and to have done so on a continuing basis, is the well-recognised nexus between RHT in playing contact sport and the later onset of neurological disfunction in the form of CTE. Let us return to a study by Dr McKee et al. McKee et al found that in a series of 85 subjects with a mean age of 59.5 years — all with a history of RHT in playing American football or ice hockey, boxing, wrestling, military service or head-banging behaviours — 68 were diagnosed at autopsy as afflicted with

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[76] *Roads and Traffic Authority of NSW v Dederer* (2007) 234 CLR 330, [61].

[77] Ibid [71].

[78] Ibid [728].

[79] Rowena Mobbs, Submission No 1 to Senate, Community Affairs References Committee, Parliament of Australia *Concussions and Repeated Head Trauma in Contact Sports* (September 2023) 3. Because, like most neurodegenerative diseases, CTE can only be diagnosed definitively at autopsy, 'traumatic encephalopathy syndrome' (TES) is the term used to describe the *in vivo* clinical manifestation of CTE.

[80] CM Suter et al, 'Chronic Traumatic Encephalopathy in Australia: The First Three Years of the Australian Sports Brain Bank' (2022) 216(10) *Medical Journal of Australia* 530, 530.

[81] 'Researchers Find CTE in 345 of 376 Former NFL Players Studied', *Chobanian & Avedisian School of Medicine* (Web Page) <https://www.bumc.bu.edu/camed/2023/02/06/researchers-find-cte-in-345-of-376-former-nfl-players-studied/>.

CTE.[82] McKee et al had earlier implicated 'a wide variety of contact sports, including football, boxing, wrestling, rugby, hockey, lacrosse, soccer, and skiing', as well as 'karate, horse riding, and parachuting'.[83] These are not the figures that would justify an SGB in failing to perceive the risk of CTE.

Importantly, where the risk of materialisation is not probable, as with CTE, 'probability' is considered in conjunction with 'the likely seriousness of the harm'.[84]

4.3.6 The likely seriousness of the harm

The more serious the gravity of harm, the greater the level of precaution and vigilance required of a duty-holder. Noting again, that 'seriousness' is considered alongside 'probability'.

A risk of serious harm must weigh heavily in the plaintiff's favour. For example, in *Basha v Vocational Capacity Centre Pty Ltd*,[85] a physiotherapist took inadequate care in preventing injury to a plaintiff who had undergone recent shoulder surgery. The New South Wales Court of Appeal said that a reasonable response must have had 'regard to the magnitude of the consequences if the perceived risk came home', such that the conduct that was engaged in 'was an inadequate response'.[86] Similarly, in the well-known case of *Paris v Stepney Borough Council*,[87] the defendant was required to take additional measures — providing safety glasses — to a one-eyed mechanic, given the more serious consequences to this particular plaintiff of injury to his good eye.

In *GEO Group Australia Pty Ltd v O'Connor*,[88] the Parklea Correctional Centre received information that the respondent (an inmate) was to be attacked by other prisoners. Although the information was not certain, its credibility was supported by its source (a trusted inmate, who relayed the threat to a nursing unit manager) and its level of detail (the information described the target, the location and the timing of the attack, and the reason for the attack, being the discovery of the true nature of the respondent's offence). The New South Wales Court of Appeal utilised the 'calculus of negligence' to determine that the consequences of the risk eventuating were foreseeably serious injury or even death, and that the non-identification of the aggressors had heightened the risk.[89] As such,

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82 Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43.

83 Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 709.

84 *Paris v Stepney Borough Council* [1951] AC 367.

85 [2009] NSWCA 409.

86 Ibid [71].

87 [1951] AC 367.

88 [2019] NSWCA 323.

89 Ibid [109].

> in those circumstances, reasonable care — consistent with the GEO's own policies — required that, for his own protection [the plaintiff] be segregated from the threat.[90]

Reviewed in several chapters of this thesis are the serious debilitating symptoms and sequelae of CTE leading ultimately to death. For a reasonable SGB informed of these consequences of CTE a recognition of the seriousness of the disease is inescapable. The general application is not overly different to the reasoning employed by the Court of Appeal in *O'Connor* above with respect to the risk of CTE: information provided through medical reports is credible, the source (being medical researchers) is reliable, and the source of CTE is known, as is the threat to the players should the risk eventuate — including ongoing and increasingly severe physical and cognitive disfunction.

4.3.7 The burden of taking precautions to avoid the risk of harm

The probability of harm and the seriousness of the risk of harm must be weighed against the burden of taking precautions. Notably:

> [A] reference to 'precautions' is a reference to conduct which might minimise or remove the risk of harm which has been identified as not insignificant.[91]

In *Bunnings Group Ltd v Giudice*,[92] the New South Wales Court of Appeal said that each available precaution must be considered individually, as each may vary in the burden of its provision. In that case, the difference between the burden of installing a 'watch your step' sign and the burden of realigning two concrete surfaces provided alternate approaches to the risk of tripping.[93]

Commonly, 'the burden' refers to the cost of implementing the precaution, but as s 5(2)(c) does not stipulate, consideration 'may extend to factors such as time or distance or communication … [or it] may be [that a precaution is] difficult to implement'.[94]

Again, it is for the plaintiff athlete to show that a reasonable SGB in the defendant's position would have taken those precautions.[95] What is reasonable will often depend on the cost of implementing the ameliorating measures as against the seriousness of the risk of harm. For example, in *Endeavour Energy v Precision Helicopters Pty Ltd*[96] a helicopter struck a redundant catenary wire strung across a small valley by Telstra Corporation. The pilot managed

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90 Ibid [110].

91 *Endeavour Energy v Precision Helicopters Pty Ltd* [2015] NSWCA 169, [33].

92 [2018] NSWCA 144.

93 Ibid [34].

94 *New South Wales v Mikhael* [2012] NSWCA 338, [82].

95 *Bunnings Group Ltd v Giudice* [2018] NSWCA 144, [34].

96 [2015] NSWCA 169.

to land in a nearby paddock, but the helicopter rolled and the main rotor struck a passenger on the head causing catastrophic injuries although he survived. The New South Wales Court of Appeal took the view that the likelihood of the risk materialising was 'very low' though 'not insignificant', but tellingly added that 'the consequences if it did materialise were likely to be catastrophic for any individual involved, in circumstances where only a small number of individuals would likely be involved'.[97] Relevantly, with respect to 'burden', the court said that the burden of taking precautions and the social utility of the activity creating the risk 'are factors' that

> cannot operate against a finding of negligence in the present case. Removal of the wire would have been cheap: indeed, the appropriate 'precaution' would have been not to re-erect the wire in the first place. There was no demonstrated social utility in maintaining the wire across the valley.[98]

The burden of an SGB informing itself of the risk of CTE is unlikely to exceed the burden of a physician's ongoing general obligation to 'exercise reasonable care and skill'.[99] In short, the professional research required of all sport medicos 'to be informed', to 'proactively consider emerging threats' and to 'inquire and discern' will naturally include the monitoring of emerging threats to athlete health.[100] And, as importantly, while an SGB is required to remunerate the medico for services, the costs of doing so are relatively modest, particularly when weighed against the potential harm of CTE. As said by McHugh JA,

> it is generally a powerful indication of negligence that the cost of precaution is small compared with the consequences of a breach even where the risk of occurrence is small.[101]

The burden of warning its players of the risk of CTE obviously involves little cost or effort.

4.3.8 The social utility of the activity that creates the risk of harm

A court is to consider the social utility of the SGB's activity in deciding whether a reasonable SGB would have taken precautions to ameliorate the risk of CTE. 'Social utility' refers to 'the activity that creates the risk of harm'.[102] The question is whether a reasonable SGB should have warned its premier players that RHT in playing its contact sport exposed them to the risk of CTE and ultimately death? The relevant activity in respect to the obligation of an SGB is warning of the risk of CTE.

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97 Ibid [32].

98 Ibid [33].

99 *Wallace v Kam* (2013) 250 CLR 375, [8].

100 See **[4.4.1]**: 'Three principal cases establishing the standard of care', which discusses the following: *Roe v Minister of Health* [1954] 2 QB 66; *H v Royal Alexandra Hospital* [1990] 1 Med LR 297; *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2.

101 *Western Suburbs Hospital v Currie* (1987) 9 NSWLR 511, 524.

102 *Doherty v New South Wales* [2010] NSWSC 450.

Importantly, social utility is not an excuse for failing to take reasonable precautions. As Tobias JA (Beazley JA agreeing) stated in *Wilson v Nilepac Pty Ltd*,

> there is nothing in the *Ipp Report* which recommended s 5B or in the text of the legislation which suggested that the standard of reasonable care required the taking of fewer precautions against an acknowledged risk of harm simply because the activity which created that risk had some social utility.[103]

Most cases where social utility has been accepted in excusing argued negligence concern an act or failure to act that furthers a higher community purpose. In *E v Australian Red Cross*,[104] for example, the plaintiff contracted AIDS from HIV infected blood in 1984, prior to when a test to detect the disease had been developed. The plaintiff claimed that had the Red Cross tested all blood for hepatitis antibodies this would probably have resulted in the transfused blood being discarded. However, Sheppard J stated:

> Blood was needed to meet the community's continuing need for it; and it was important that the community did not become alarmed by thought that the blood supply was extremely short or that AIDS was spreading much faster than it really was. … in all the circumstances … the Society was not shown to have acted unreasonably in not withholding untested blood.[105]

An ambulance with lights and sirens displayed on its way to an unconscious patient with breathing difficulties crashed into a car at an intersection in *Logar v Ambulance Service of NSW*. Emmett J held:

> The primary judge had to take into account the social utility of the response to the emergency. … Having regard to the medical emergency [the driver's] actions in proceeding slowly through the intersection against the red light …justified her action in proceeding.[106]

A concern that an SGB may conceivably suffer commercially should it warn or report that CTE had emerged as a risk of its contact sport, does not fall within the recognised application of the social utility element, which is commonly directed towards accepting a risk in order to achieve a higher social object. For example, in *Watt v Hertfordshire County Council*[107] a fireman was injured when he was struck by an unsecured jack in a speeding fire engine on its way to rescue a woman trapped under a vehicle. The jack was required for the rescue, but the fire engine that was specially fitted to carry the jack was unavailable. As a result, the jack was put on a fire engine that had no means of securing it. The court found no negligence, stating as follows:

> It is well settled that in measuring due care you must balance the risk against the measures necessary to eliminate the risk. To that proposition there ought to be added this: you must balance the risk against the end to be achieved. If this accident had occurred in a commercial enterprise without any

[103] [2011] NSWCA 63, [130], referring to *Review of the Law of Negligence* (Final Report, September 2002) ('*Ipp Report*').

[104] (1991) 31 FCR 299.

[105] Ibid, 86 (Lockhart and Pincus JJ agreeing).

[106] *Logar v Ambulance Service of New South Wales* [2017] NSWCA 274, [29].

[107] [1954] 1 WLR 835.

> emergency there could be no doubt that the servant would succeed. *But the commercial end to make profit is very different from the human end to save life or limb*. The saving of life or limb justifies taking considerable risk.[108]

In *Doherty v New South Wales*,[109] a case in which a police forensic investigator claimed his employer had caused him post-traumatic stress disorder through exposing him to 'horrific' accident and crime scenes without the provision of psychiatric treatment, Price J considered the social utility in determining 'what a reasonable person would have done in response to that risk'.[110] Here, however, the social utility required the provision of care. His Honour found that the employer had breached its duty:

> I consider it to be of fundamental importance to the criminal justice system that these important activities [that is, crime scene investigation, detection of the offence and appearing as an expert witness] are carried out by properly qualified objective police officers who are free from psychological injury. Crime scene investigation was the activity which created the risk of harm.[111]

Where there was the need to ensure that forensic investigation was not hampered, the court found it remained necessary to ensure the well-being of those exposed to risks to their health, including their mental health.

The concern of an SGB should it announce CTE as a risk of playing its sport is widely believed to be the fear of losing junior players and a curtailment in sponsor revenue were the sport be perceived as 'too violent and dangerous'. In these circumstances the motive in failing to warn of the risk of CTE is not altruistic but rather commercial.

In summary, the risk of CTE was, or ought to have been foreseeable to the SGBs of contact sport in Australia for several decades prior to *circa* 2010. The significance of contracting CTE is extreme, putting a sufferer through ever-increasingly debilitating and distressing physical, psychological and behavioural symptoms. Taking account of and balancing out the probability of contracting CTE, the seriousness of the symptoms and sequelae, the minor burden of issuing warnings and the absence of true social cost, there can be little doubt that the major SGBs in Australia breached their duty to care for the well-being of their premier-level athletes in the pre-2010 era.

4.3.9 The burden of taking precaution against similar risks

At this point it is necessary to briefly note that a court when assessing the burden of taking precautions against a risk of harm it is required to include within the assessment not only those

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[108] Ibid 838. Emphasis added.

[109] [2010] NSWSC 450.

[110] Ibid [167].

[111] *Doherty v New South Wales* [2010] NSWSC 450 [167], [201].

burdens associated with the risk of harm that materialised, but any burden associated with precautions to avoid similar other burdens.

Section 5B(2)(a) of the *Civil Liability Act 2002* (NSW)[112] provides:

> [T]he burden of taking precautions to avoid a risk of harm includes the burden of taking precautions to avoid similar risks of harm for which the person may be responsible …

Where the claim against an SGB is a failure to warn of the risk of CTE, an additional burden may include the burden of taking steps to inform itself of other risks to the health and well-being of its premier players and warning where those risks are material.

The discussion above regarding the burden of taking precautions to avoid the risk of harm is pertinent to this additional burden. In short measure, the burden of taking precautions to inform itself of extant and emerging risks of harm and to warn its premier players of the risk of CTE will likely include the burden of informing itself of other risks to the health and well-being of its premier players. On the basis that its medical associates are the most usual conduit for information on matters of health, the burden cannot be said to have increased given that the obligation of the medico remains constant; that is, to provide comprehensive advice on all matters affecting the health and well-being of the players of the parent SGB.[113]

4.4 The content of the standard of care

Although s 5B(1) of the *Civil Liability Act 2002* (NSW) (and its equivalents) provides that a finding of negligence necessitates a 'foreseeable risk' that is 'not insignificant' and for which a 'reasonable person in the [defendant's] position would have taken … precautions', it says nothing about what actual precautions should have been taken by a reasonable person. Basten JA remarked:

> The content of the duty [to take reasonable steps to avoid foreseeable risks of harm] … must be defined by reference to the steps which a reasonable person would take to eliminate or reduce the risk.[114]

In *Benic v New South Wales*,[115] a police officer received threats to his life that led to post-traumatic stress disorder, associated anxiety and depression spectrum conditions. Garling J spoke non-exhaustively of how a defendant may acquire knowledge of a risk of harm, and where the absence of knowledge reveals a falling short of the standard of care:

> [T]he plaintiff must satisfy the Court that the defendant, at the date of the alleged negligence, knew of the alleged risk of harm, or else, by reference to other facts, matters and circumstances ought to

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[112] See also *Civil Law (Wrongs) Act 2002* (ACT) s 44(a), (b); *Civil Liability Act 2002* (NSW) s 5C(2)(a), (b); *Civil Liability Act 2003* (Qld) s 10(a), (b); *Civil Liability Act 2002* (Tas) ss 11(3), 12(a); *Wrongs Act 1958* (Vic) s 49(a), (b).

[113] *Rogers v Whitaker* (1992) 175 CLR 479.

[114] *Caltex Refineries (Qld) Pty Ltd v Stavar* (2009) 75 NSWLR 649, [157].

[115] [2010] NSWSC 1039.

> have known it. Those other matters will vary from case to case but may include such things as the *common knowledge* and *experience of others* in the similar position of the defendant, *public notoriety* of a particular risk of harm, publications and *academic knowledge* which might be expected to be read by people in the defendant's position and the *obviousness or the likelihood of the event* happening when using common sense.[116]

This chapter considers in large part the content of the duty associated with 'publications' and 'academic knowledge which might be expected to be read' by an SGB through its medical associates. Note, most importantly, that the standard of care is based on what a reasonable person would do in the circumstances. Would a reasonable person warn of a risk of harm that carried life threatening consequences even where that risk had yet to be fully confirmed?

The presence of publications that ought to have been read but were not, indicates a falling short of the expected standard owed by an SGB in fulfilling its duty to care for the health and well-being of its premier players. Other relevant matters listed in the passage above include 'common knowledge', the knowledge of others in a similar position to the defendant, and public notoriety.

In the medical case of *Rogers v Whitaker*, the High Court remarked that the 'comprehensive duty' of a medical practitioner includes the 'provision of information'.[117] This statement of obligation does not, however, reveal what actions are sufficient in discharging the obligation, the court observing that '[i]t is of course necessary to give content to the duty in the given case'.[118] As argued below, the applicable standard of care owed by an SGB to care for the health and well-being of those participating in its premier competition, is that of the 'medical model', an argument based primarily on the fact that in a dangerous activity without the proactive searching out of risks to health, a plaintiff athlete is unprotected should newly discovered threats of disease or injury emerge.

4.4.1 Three principal cases establishing the standard of care

Three principal cases are considered to describe the content of an SGB's obligation to take the reasonable steps to 'be informed' of the risk of CTE (and from which the risk of CTE could have been the subject of an SGB's warning to its premier-level athletes):

- *Roe v Minister of Health* established the principle that conduct 'is only judged in the light of knowledge which then was or ought reasonably to have been possessed'.[119]
- *H v Royal Alexandra Hospital* established the principle that foreseeability of a risk of harm may exist prior to identification of the full nature of the risk. In that case, the risk

[116] Ibid [92]. Emphasis added.

[117] (1992) 175 CLR 479, [14].

[118] Ibid [5].

[119] [1954] 2 QB 66, 92.

that 'AIDS might be a viral disease transmissible in blood products … cast upon [the defendant hospital] a duty to give an appropriate warning of that risk'.[120]

- *South Eastern Sydney Area Health Service v King* established the principle that there is a positive obligation on the part of a medical practitioner to seek out knowledge relevant to the area of practice — to 'take reasonable care to inform himself of "the most current and considered guidelines available in the world"'[121] — and, as expressed in expert evidence, 'to find the relevant literature, to read it, to understand it and to discuss it'.[122]

In summary, CTE was recognised nearly 100 years ago in the *Journal of the American Medical Association* and in hundreds of scientific journal articles since. As discussed in the next chapter, these articles were readily accessible to Australian SGBs through their associated medical practitioners.[123] Had the SGBs taken the reasonable precaution of initiating a system of monitoring for emerging risks to the health and well-being of their athletes, the threat of CTE-type harm would almost certainly have been realised and have formed the basis for the issuance of warnings that would have permitted athletes, of their own volition, to take steps to ameliorate the risk of CTE.

4.4.2 The standard of care: The obligation to be informed

Only where information regarding a risk of harm is available is a duty-holder obliged to access it and to know it. A risk of harm is foreseeable (or ought to be foreseeable) where it is available, and failure to know it — and to act upon it if material — is a breach of duty. This section considers the law on when information regarding a risk of harm is sufficiently available that a duty-holder is obliged to know of it. The factual question of when knowledge of the risk of CTE is sufficiently available that it 'ought to have been known' by an SGB is considered in the next chapter.

In *Roe v Minister of Health*,[124] two patients (Mr Roe and Mr Woolley) were paralysed when their spines were injected by their anaesthetist with nupercaine contaminated with the acid phenol. Floating in the sterilising agent phenol, the glass ampoules of nupercaine collided with each other, causing invisible cracks through which the phenol passed. The harm to the patients occurred in England in 1947. The attending anaesthetist, Dr Graham, was not aware of the risk of contamination. That risk was, however, known in some medical circles in England and internationally. Evidence that 'some hospitals' placed coloured dye in the phenol solution to detect percolation into the ampoule was given.[125] Had Dr Graham known of this information,

[120] [1990] 1 Med LR 297, 299.

[121] [2006] NSWCA 2, [60], quoting Newman AJ at trial.

[122] Ibid [68].

[123] See Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times'.

[124] [1954] 2 QB 66.

[125] Ibid 77–8.

and acted on it, his patients would not have been paralysed. The Court of Appeal, unpersuaded that information not known by Dr Graham could constitute negligence, rationalised that the evidence did not support negligence. The court observed that while mention had been made in 'American publications to colouring … the only paper traced on "immersion" in this country made no reference to deep tinting as an ingredient of the process'.[126]

The most important factual difference between *Roe* and the other principal cases, discussed below, is that the defendant Dr Graham did not know, and most tellingly *could not know*, of the specific risk of harm that befell his patients. The decision in *Roe* was based primarily on the factual circumstances of the day characterised in the main by ponderous communication methods, and perhaps the personal lassitude of holders of knowledge that denied the conscientious inquirer even a hint of danger.

'There is no duty of care owed to a person,' said Lord Denning in *Roe*, 'when you could not reasonably foresee that he might be injured by your conduct.'[127] Once, however, a risk ought reasonably be known, a duty arises: 'Nowadays it would be negligence not to realize the danger, but it was not then.'[128] To revisit Lord Morris's comment on the same point: 'care has to be exercised to ensure that conduct in 1947 is only judged in the light of knowledge which then *was or ought reasonably to have been possessed*.'[129] Somervell LJ found that 'Dr Graham had never heard of deep tinting as a precaution'.[130] The duty in these circumstances, given the general unawareness among anaesthetists as a class at the time of the danger of unperceived fissures, extended only to visually checking for cracks in the ampoules. In the judgment of Somervell LJ, his Lordship quoted the trial judge:

> In 1947 the general run of competent anaesthetists would not appreciate this risk [of imperceptible cracks]. Dr Graham certainly did not appreciate this as a risk. I accordingly find that by the standard of knowledge to be imputed to competent anaesthetists in 1947, Dr Graham was not negligent in failing to appreciate this risk and *a fortiori* the theatre staff were not negligent.[131]

At its most basic, Dr Graham's ignorance absolved him from a charge of professional negligence, prompting the question: when is knowledge sufficiently available that a reasonable medical practitioner should 'know' and 'ought reasonably have been possessed' of such knowledge?

In England 'medical ignorance' of contaminated ampules of nupercaine ended with the 1951 publication of *Lumbar Puncture and Spinal Anaesthesia* by the eminent anaesthetist

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[126] Ibid.

[127] Ibid 94.

[128] Ibid 96.

[129] Ibid 92. Emphasis added.

[130] Ibid 78.

[131] Ibid 77.

Prof Macintosh, who cautioned: 'Never place ampoules of local anaesthetic solution in alcohol or spirit. This common practice is probably responsible for some of the cases of permanent paralysis reported after spinal analgesia.'[132] No reference to a book or medical journal prior to that date carrying such critical information is mentioned in the *Roe* judgment. Electronic communications or online journals, or discussions by online conference or private text, were of course non-existent. Prof Macintosh's publication formed an endpoint in time beyond which an ignorant physician in England could, *ceteris paribus*, avoid liability.[133]

Lord Denning emphasised foreseeability as the focal point of an obligation to warn: Dr Graham 'did not know that there could be un-detectable cracks, but it was not negligent for him not to know it at that time. We must not look at the 1947 accident with 1954 spectacles.'[134]

Dr Graham's ignorance extended to two medical facts that could have prevented 'the disaster which has befallen these two unfortunate men':[135] (1) that phenol can pass through microscopic cracks in glass ampoules; and (2) that some medical institutions dyed the phenol solution so as to reveal transportation into the nupercaine. These risks were, though, known to at least some medical professionals in England:

> There was evidence that in some hospitals where the immersion system was used the disinfecting liquid, whether a phenol solution or surgical spirit, was stained a deep tint with methylene blue or some other dye. … This would make it easier, of course, to detect percolation. It was a method used by Ciba Laboratories [which manufactured and supplied the nupercaine] and was known to analytical chemists.[136]

Dr Graham did not foresee the risk of ampoule percolation. Why? The answer is because he did not know of the risk. The question to ponder, however, is not whether the harm was unforeseeable, but *why* was it unforeseen by Dr Graham? The corollary of this is: 'ought Dr Graham have known?'

Knowledge of the precautionary measure of deep tinting existed, but it was scattered, it was not reported in medical writings, and it was seemingly confined to local or parochial discussion. It could not, thereby, be accessed by the broader English medical fraternity. The precautionary information had not been published; nor was it the subject of wide professional discussion. The

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[132] Ibid 86.

[133] Perhaps interesting to note is an editorial from a 1975 *British Journal of Anaesthesia* titled 'Spinal Anaesthesia': 'Since the [*Roe* case], reported in 1954, in which two patients developed painful and permanent paraplegia following spinal anaesthesia, the use of the technique in the United Kingdom has been confined to a few enthusiasts. … Ten months ago, the manufacturers of cinchocaine (Nupercaine) stopped supplying the drug because very fine cracks had been found in the glass of some of their ampoules': Editorial, 'Spinal Anaesthesia' (1975) 47(4) *British Journal of Anaesthesia* 421, 421.

[134] *Roe v Minister of Health* [1954] 2 QB 6684.

[135] Ibid 81.

[136] Ibid 77 (Somervell LJ).

only way for 'outsiders' to be informed, it would seem, was by direct contact with those who 'knew' — in essence, by working at the same hospital or through personal relationships.

The question remains: given that some medicos knew of percolation, should Dr Graham have sought information from the informed? There must be something to prompt an inquiry; an indication of a development worth knowing. Dr Graham was aware, as were all physicians, that acid injected into a spinal cord causes catastrophic harm. To combat the risk, he checked the nupercaine ampoules for visible cracks. He had no reason to suspect 'invisible cracks' in the ampoules short of some prompt — which did not come. Nor was medical science on the subject at a moment of rapid change where important developments could be expected — which would, if realised, put a physician on inquiry.

The common law descriptor of knowledge that '*ought* reasonably to have been possessed'[137] and the statutory application of 'the person *knew or ought to have known*'[138] are imperatives. A defendant who does not know, or ought reasonably to have known but does not, is, *ceteris paribus*, in breach.

For a reasonable duty-holder a glance at information is inadequate to fulfil the obligation to know facts, understand research or grapple with studies relevant to decision-making. Accordingly, the medico and diligent SGB must contemplate and understand the information revealed in order to warn athletes who are possibly at risk. This requirement is augmented, as discussed below in the case of *South Eastern Sydney Area Health Service v King*,[139] by a proactive obligation to seek out relevant information and to exercise judgment.

4.4.3 Standard of care: Medical developments, judgment and proactive obligations

In *H v Royal Alexandra Hospital*,[140] according to medical opinion accepted by the court, the plaintiff was infected with HIV through infected blood on either 20 March 1982 or 25 September 1983 — an 18-month span. Badgery-Parker J found that 'it is abundantly clear that there was no evidence at all that AIDS was transmissible in blood before July 16, 1982. Before March 1982, information about the disease was very limited.'[141] But, by September 1983, the later time of the plaintiff's possible infection, knowledge was sufficient to require preventative measures: 'Certainly … persons in the relevant fields ought to have been well

[137] Ibid 92. Emphasis added.

[138] For example, s 5B(1)(a) of the *Civil Liability Act 2000* (NSW). Emphasis added.

[139] [2006] NSWCA 2.

[140] [1990] 1 Med LR 297.

[141] Ibid 307.

aware of the possibility that AIDS was blood borne and giving active consideration to the adoption of appropriate precautions.'[142]

Importance lies in the actions of medicos in the period between 'no evidence', and being 'well aware of the possibility' of risk and the precautions to be taken. To Badgery-Parker J knowledge short of absolute proof was sufficient to necessitate action by responsible physicians and other duty-holders — an action guided by common sense and prudence, given the potential costs to those to whom the duty was owed:

> [T]he fact is that however the risk be quantified in the light of knowledge available in January 1983 [that is, when US medicos began taking precautions], and whether they were convinced or not that AIDS was blood borne, physicians treating haemophilia and blood scientists and persons and organizations involved in the collection of plasma and the manufacture of blood products in the United States *began to perceive the need to take such precautions* as could be devised in case it should turn out that AIDS was a blood-borne disease, and within a *comparatively short time thereafter, it was reasonable that similar persons in Australia*, and hence the defendants, *should have adopted similar attitudes and precautions*.[143]

In March 1982, 'the existence of the [specific] virus HIV was unknown'.[144] At that point in time the causal virus had not been identified, but 'there was a foreseeable risk that recipients of blood products would become infected'.[145] Even by September 1983, 'the existence of the virus was strongly suspected but its identification was far from clear'.[146] No direct test for it was available, and yet reasonable scientists would have had a 'strong apprehension'.[147]

With respect to the issuance of warnings, the judgment of Badgery-Parker J concerns two stages: (1) when it was clear that some unknown agent in blood products was causing a disease; and (2) when the disease was discovered to be a virus. The giving of a warning was to be aligned to the state of knowledge. His Honour said:

> It is clearly not the case that prior to March 1982 there was any obligation on the … defendants to give warning of the risk that blood products distributed by them might carry, specifically, the agent responsible for the syndrome recently reported as occurring in American homosexuals. It is equally clear that from soon after the beginning of 1983 the … defendants were aware of the risk then becoming known that AIDS *might be* a viral disease transmissible in blood products and in particular, in factor VIII preparations. That knowledge, with their inability to do much by way of screening of donors or otherwise to eliminate the risk, cast upon them a duty to give an appropriate warning of that risk …[148]

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[142] Ibid.

[143] Ibid. Emphasis added.

[144] Ibid 311.

[145] Ibid 310

[146] Ibid 311.

[147] Ibid.

[148] Ibid 316. Emphasis added.

From early 1983 the obligation was to warn that blood might be contaminated with an 'agent' that causes an identified disease syndrome. This agent was unknown, but the malady had become known. The obligation to warn occurred at the point of 'might be' — not certainty. That is, despite information on the cause and aetiology available to physicians being less than absolute, the duty to warn was nonetheless imposed.

And what of the obligation to 'be informed', and of clinical judgment assembled from known but diverse facts? The first case of AIDS in Australia was reported by publication in April 1983. On this, Badgery-Parker J commented:

> I have no difficulty in concluding that reasonably informed physicians, scientists and blood transfusion services in this country ought to have been well aware by at latest April 1983 that there was a real risk that among the unknown and unidentified sources of infection which blood and blood products had the capacity to carry must be numbered whatever agent was responsible for the production of AIDS.[149]

The obligation to take reasonable steps to gain knowledge of a developing disease was enlivened despite the absence of absolute proof of cause. It was sufficient to know that a disease 'might' be carried in blood and, later, that it 'might' be caused by a virus — knowledge gained from reflection and deliberation.

In September 1983, 'there was still a lot to learn about this new syndrome', and yet Badgery-Parker J found that what had 'been mere suspicions would necessarily have been elevated in the minds of responsible Australian physicians and blood scientists into a strong apprehension'.[150] What caused the suspicions that drove 'responsible' physicians to strongly apprehend the risk of blood-borne infections? These physicians came to a clinical judgment by stitching together pieces of relevant, though imprecise, information: their 'knowledge of the demonstrated long incubation period', the 'undoubted fact that the incidence among haemophiliacs was greater than in the general population', and their knowledge that a medical researcher had 'produced evidence which, if it did not demonstrate the fact, strongly suggested that AIDS was due to a virus'.[151]

4.4.4 Standard of care: The obligation to inquire and discern

In *South Eastern Sydney Area Health Service v King*,[152] the plaintiff, Ms King, received treatment at Prince of Wales Children's Hospital in 1989, at age 13, for a cancerous tumour surrounding the nerve root at C6-C7 of her spinal cord. The treatment, which two years earlier had been reported in a medical journal as causing neurological damage in another patient, left Ms King quadriplegic. At age 29 she sued the Health Service (which operated the hospital) and

[149] Ibid 311.

[150] Ibid.

[151] Ibid.

[152] [2006] NSWCA 2.

the paediatric oncology specialist who had treated her, Prof O'Gorman-Hughes, for negligent performance and failing to warn her parents fully of the dangers of paraplegia or quadriplegia in the proposed treatment plan. An associate of Prof O'Gorman-Hughes, Dr White, was also sued in negligence for failing to inform himself of vital clinical information and, in turn, failing to inform the medical team treating Ms King.

The treatment devised was a chemotherapy and radiological therapy known as the 'IRS-II protocol', which included the administration of three chemicals — methotrexate, cytosine arabinoside ('Ara-C') and hydrocortisone, together known as 'triple intrathecal therapy' ('TIT'). The IRS-II protocol was founded on a study, carried out between 1978 and 1984 by a body called the Intergroup Rhabdomyosarcoma[153] Study Group ('IRS Group'), which was based mainly in the United States. Prof O'Gorman-Hughes followed the IRS-II protocol in determining the doses, number and frequency of the TIT chemicals to be administered. In 1984 the IRS-II protocol was augmented by the IRS-III protocol. Importantly, in 1985 a Dutch group warned that TIT could cause paraplegia because the systemic chemotherapy increased the effect of radiation. This was not known by Prof O'Gorman-Hughes at the time he treated Ms King, four years later.

To again emphasise the relevant dates, the new protocol, IRS-III, had been published by the IRS group in 1984. In 1987, two years before Ms King was treated, an 'important amendment' to IRS-III, of 'vital importance in the treatment of the respondent',[154] revealed that one patient under the IRS-II protocol and six patients under the IRS-III protocol had suffered 'neurological complications' including 'lower extremity weakness and bladder/bowel dysfunction'.[155] While the IRS Group did not know what factor or factors had caused the complications, it recommended adjustments to the protocols as follows: 'it is felt that the doses of [chemicals in TIT] … should be limited to 4 doses in 20 weeks', and in addition that the radiation should be reduced. Prof O'Gorman-Hughes did not, however, know of the 'important amendment'.[156] Consequently, Ms King's 'treatment differed substantially from that advised by the 1987 amendment to IRS-III, and these differences formed the substance of the respondent's case'.[157]

Prof O'Gorman-Hughes 'had no direct association with the IRS Group'.[158] However, another specialist paediatric oncologist at the Prince of Wales Hospital, Dr White, was a '"corresponding" member of the Children's Cancer Group in the United States, which was

[153] Rhabdomyosarcoma is a highly malignant tumour affecting children, which occurs frequently in the head and the neck.

[154] Labelled as such by Hunt AJA in *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2, [16].

[155] Ibid.

[156] Ibid.

[157] Ibid [17].

[158] Ibid [19].

itself a member of the IRS Group'.[159] Dr White received information on the amendment in what were called the 'Orlando minutes'. Dr White knew Prof O'Gorman-Hughes based his treatment of Ms King 'at least in part on what was known about IRS-II, and he agreed that the current studies being undertaken by the IRS Group were regarded as being studies of importance'.[160] Dr White did not, however, circulate the information 'within the [oncology department] team' or pass it to Prof O'Gorman-Hughes as he 'normally would have' because it was a large document and 'he had not got around to reading it'.[161] Moreover, he implied that his obligations were not proactive: 'had he been asked by Professor O'Gorman-Hughes whether he had any documentation in relation to the IRS-II protocol, he would have searched through his documents and would have produced this document.'[162]

Further, a reference to the amendment was published before Ms King's treatment by a member of the IRS Group Committee in a new medical journal, *Cancer in Children*, dated December 1987, offering another avenue of knowledge to Prof O'Gorman-Hughes. The reference stated that the amendment was prompted by an awareness of brain stem disfunction in a small number of patients, and proposed the reduction of TIT and the avoidance of 'simultaneous TIT and radiotherapy. It identified in detail the reduced TIT as being administered in weeks 0, 6, 12 and 20 and then stopped, and the changes in the dosages for Ara-C already referred to'.[163]

The Court of Appeal found that *had* Prof O'Gorman-Hughes been made aware of the Orlando minutes, he 'would have adopted the advice given by the amendment, and the respondent would not have been rendered quadriplegic by the treatment administered to her'.[164]

The Court of Appeal, through Hunt AJA,[165] found Dr White possessed a duty of care 'in relation to the dissemination of the Orlando minutes to the members of the oncology department team' and was negligent in not passing on information he had received.[166]

A The issue of 'non-peer reviewed' information

Importantly, the 1987 amendment to the IRS-III protocol warning of a risk of neurological complications was 'part of an ongoing clinical trial, [and] had not been analysed, peer-reviewed or published'; accordingly, it was argued, the amendment 'was still experimental' and as such

[159] Ibid [19].

[160] Ibid [20].

[161] Ibid [23].

[162] Ibid.

[163] Ibid [26].

[164] Ibid [47].

[165] The judgment was that of Hunt AJA, with Mason P and McColl JA agreeing entirely.

[166] *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2, [40].

'not binding on Prof O'Gorman-Hughes'.[167] Nor, as argued, was 'it necessarily demonstrative of the best standards of practice'.[168] Hunt AJA found these challenges 'misdirected':

> The relevant issue in the case was whether — having already adopted an earlier version of the IRS protocol at a time when it too had not been analysed or peer-reviewed, and advice having now been given to reduce the dosages and the frequency of the treatments it had recommended — Professor O'Gorman-Hughes would have followed the advice given in the amendment.[169]

Witness evidence, accepted by the court, was that 'had Professor O'Gorman-Hughes been aware of the 1987 amendment to the protocol on which he had relied, he would have acted unreasonably if he had *not* followed the recommendations made by the amendment'.[170]

B Keeping informed — novelty and foreseeable complications

The treatment given by Prof O'Gorman-Hughes to Ms King was 'radical, experimental and controversial',[171] and concerned her central nervous system. These factors of novelty and potential serious harm carried foreseeable risk, necessitating 'a duty to take reasonable care to keep informed before embarking on the treatment of the nature prescribed in this case … whether or not it was reasonably foreseeable that it could cause paraplegia or quadriplegia'.[172] Quite clearly the adoption of a new treatment demands active investigation sufficient to reveal possible adverse effects. As noted by the court: 'The whole nature of this particular treatment … indicated the reasonable foreseeability of some form of neurological complication.'[173]

C Practical limits

While there is a duty on the part of a medical practitioner to 'keep abreast of literature and publications relevant to the treatment he or she is prescribing',[174] such a duty is not, however, absolute. It is in keeping with the duty to take reasonable care to be informed, but '[m]uch will turn on the nature of the treatment being prescribed and on the nature of any risk which could reasonably be involved in that treatment'.[175]

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[167] Ibid [45].

[168] Ibid.

[169] Ibid.

[170] Ibid [46]. Original emphasis.

[171] Ibid [59].

[172] Ibid.

[173] Ibid.

[174] Ibid [58].

[175] Ibid.

The Court of Appeal, in agreement with the trial judge, found in 'the particular circumstances of the case' that the duty of the professor was to 'take reasonable care to inform himself of "the most current and considered guidelines available in the world"'.[176]

D The standard for delegation

Dr White, it will be recalled, had in his possession the amendment to the IRS-III protocol, but had not found time to read it, although he knew that Prof O'Gorman-Hughes was utilising the protocol in treatment.

While it is acceptable that Prof O'Gorman-Hughes delegate responsibility for conducting a literature search and making inquiries of the IRS Group, fulfilling his duty required him to ensure that 'in the particular circumstances of this case …, he was placed in as good a position by a system based on collaborative teamwork as he would have been had he performed the duty himself'.[177] In specific terms, Prof O'Gorman-Hughes

> had to do more in this case than merely rely in a purely reactive manner to whatever information Dr White may randomly have received from his overseas contacts. If he could not do it himself, he had to take reasonable care to ensure that someone else actively searched for material which was relevant to the task he was to undertake.[178]

E A proactive 'personal' duty to keep informed

A part of the general duty of a physician to exercise reasonable skill and care, is 'a duty to take reasonable care to keep informed'.[179] Unlike Dr Graham, the defendant in *Roe v Minister of Health*,[180] who had no practical means of accessing information or even knowing it existed, Prof O'Gorman-Hughes possessed several avenues of inquiry. One was an article by Dr Frederick B Ruymann, a member of the IRS Group Committee and Director of the Division of Haematology/Oncology in the Children's Hospital in the Ohio State University School of Medicine, which referenced the amendment to the IRS-III protocol in the journal *Cancer in Children and Adolescents* — a relatively 'new' journal — in December 1987. Ms King was treated between March and August 1989. The title of Ruymann's article was 'Rhabdomyosarcoma in children and adolescents: A review' (rhabdomyosarcoma being the disease from which Ms King suffered). The article stated that 'brain stem dysfunction in a small number of patients had prompted the amendment, which reduced TIT and avoided simultaneous TIT and radiotherapy'.[181] The review also detailed the different weeks when the reduced TIT should be administered to avoid harm.

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[176] Ibid [60].

[177] *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2, [60].

[178] Ibid.

[179] Ibid [59]. Although the passage referred to the specific treatment of Ms King, the obligation is general in application.

[180] [1954] 2 QB 66.

[181] *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2, [26].

Dr Ruymann's review was available in both the University of Sydney and the University of New South Wales libraries. In fact, Prof O'Gorman-Hughes became aware in late 1989 of the IRS Group recommendation that 'central nervous system [treatment] including TIT be changed because of known toxic consequences … when he noted a reference to that fact in Dr Ruymann's paper … in the medical school library at the University of New South Wales'.[182] Prof O'Gorman-Hughes could also have accessed the review through a publication called *Index Medicus*, 'which enabled medical practitioners to search for specific articles of interest', and an 'internet search tool known as "Medline"'. The Court of Appeal accordingly found Prof O'Gorman-Hughes negligent in failing to seek out information pertinent to his treatment of Ms King.

The Court of Appeal found that Dr White also had a proactive duty to act. Dr White possessed information on the amendment to the IRS-III protocol through his association with the IRS Group, but did not find time to read it or to share it with Prof O'Gorman-Hughes. The mere possession of knowledge of preventative treatments served no purpose and, through his passivity Dr White was a causative factor in the harm befalling Ms King. Agreeing with the trial judge, Hunt AJA found that

> Dr White was negligent in failing either to make himself aware of the information in his possession relating to the amendment to the IRS protocol on which Professor O'Gorman-Hughes relied or to establish a system for the proper dissemination of such information throughout the oncology department at the hospital.[183]

Although Dr White was negligent, Prof O'Gorman-Hughes was not absolved of his personal duty to stay informed. While delegation is entirely acceptable, relying on the provision of knowledge by third parties through an unreliable system affords no relief.

F A positive obligation to search and consider?

The obligation to be informed and to know is not passive; those who are subject to this obligation must actively search for information relevant to their task, rather than passively assume that it will arrive through the courtesy of others, and must then consider its use within their sphere of obligation. The Court of Appeal stressed that the duty to access relevant information takes precedence over doubts regarding the apparent 'newness' of a journal or its apparent 'impact'.

Accordingly, the argument that the journal *Cancer in Children and Adolescents* had 'an impact value of virtually zero' and, as such, 'was not one Professor O'Gorman-Hughes would have been expected to have seen',[184] and 'did not … appear in the first 10 or 20 journals read by oncologists,'[185] was quickly disposed of by Hunt AJA, who said: 'That evidence did not

[182] Ibid [61].

[183] Ibid [50].

[184] *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2, [62].

[185] Ibid [64].

lessen the weight to be given to [plaintiff witness] Professor Stevens' evidence as to the accessibility of the paper when searches were made'.[186]

A witness called by the defendants, Dr Kellie, told the court that the new journal 'was part of a well-regarded series, and that it would be an important source to access in relation to the treatment of a patient such as [Ms King]'.[187] Nonetheless, he admitted that he would not expect to find 'new information in a review', prompting the court's derisory comment:

> The circumstances in which Professor O'Gorman-Hughes was about to treat [Ms King] did not permit him to place such limited reliance on Dr Ruymann's paper merely because it was a review. Even Dr Kellie described it as an excellent review; he merely preferred to go back to the original research identified in the 167 references or citations in the endnotes.[188]

Whether the paper was 'an excellent review' or not is beside the point; rather, the obligation is to seek out relevant material and then evaluate its clinical worth. Duty requires, in the words of Prof Bleyer (a witness called by Ms King) given in evidence, that

> Prof O'Gorman-Hughes should have taken steps to find the relevant literature, to read it, to understand it and to discuss it; a simple literature search would have revealed the Ruymann paper, which [Prof Bleyer] described as a review of the experience of the IRS Group, and which should have been looked at before embarking on any consideration of TIT.[189]

G The obligation to inquire where potential harm is discerned

The obligation to be informed does not end at the reading of single journal or study. All relevant information is not necessarily found in one journal or paper. More importantly, the obligation extends to following a chain of information and reasoning — one item pointing to another of possible relevance. In Ms King's case, for example:

> If Professor O'Gorman-Hughes had read Dr Ruymann's paper, he would have become acutely aware of the advice given in the amendment to the IRS-III protocol in 1987 … If what the paper disclosed of the reasoning for that advice did not satisfy him to change his own protocol, I am satisfied that he, like Dr Kellie, would have gone to the source of the information, the 1987 amendment to IRS III.[190]

Dr Ruymann's paper reported only one patient as suffering brain stem disfunction due to excessive dosage of Ara-C by way of TIT and the interactive effects of chemotherapy and radiation, but this is not relevant to the duty to search and consider. The 'information … was nevertheless of importance' and Hunt AJA did not consider that 'the discovery that only one patient had suffered from brain stem dysfunction would have dissuaded Professor O'Gorman-

[186] Ibid.

[187] Ibid [65].

[188] Ibid [66].

[189] Ibid [67].

[190] Ibid [69].

Hughes from pursuing the reason for the reduction advised by the amendment'.[191] In fact, being 'up to date' necessitated a 'literature search'.

H Breach by failure to take steps to be informed

A crucial aspect of Ms King's case, 'the failure to search for and find Dr Ruymann's paper',[192] was borne out on two fronts. First, according to Hunt AJA, '[i]n my opinion, Professor O'Gorman-Hughes would have been obliged to read this paper if it had been discovered before treating the respondent'.[193] As it was,

> by proceeding with the treatment of [Ms King] without ensuring that he had been placed in as good a position as if he had conducted a literature search and any necessary follow-up inquiries himself, he failed in his duty to [Ms King] in the particular circumstances of this case to be in possession of all the necessary up-to-date information in relation to that treatment.[194]

Second, the system in place for sharing information, specifically that residing with Dr White, 'was insufficient for the treatment prescribed'.[195]

In consequence, had Prof O'Gorman-Hughes fulfilled his 'duty to be informed' and consequently 'to know',

> he would have modified the regime he had prescribed for [Ms King]. As the excessive dosages of Ara-C by way of TIT and their frequency associated with the radiotherapy contributed to [Ms King's] myelopathy and thus her quadriplegia, causation is established ...[196]

4.5 Rule changes and the standard of care: Safety measures — voluntary or duty?

Sport has not uncommonly encountered threats to the health and well-being of its athletes. Often the response, albeit somewhat haphazard at times, has been to investigate a risk and, where it is affirmed, to introduce mitigation procedures including the issuance of warnings — a pattern, as noted above, resembling the 'medical model'. The obligation to initiate mitigation protocols raises the question of whether, in fulfilling that duty, an SGB is in breach should it not change the rules to make its contact sport safer. The response to the question is not a contentious and, as such, discussion below is truncated and expeditious.

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[191] Ibid [70].

[192] Ibid.

[193] Ibid [68].

[194] Ibid [71].

[195] Ibid.

[196] Ibid [72]. Ms King was awarded $7 million by previous arrangements between the defendants.

4.5.1 Is there a legal obligation to change the rules of play to make a sport safer?

Several reasons can be cited as to why the response to this question is, with some inevitability, in the negative. Although, in contrast, there is an arguable obligation to adjust rules that are collateral to the nature of the sport and do not work to change its basic nature. Some reasons why such an obligation has not been imposed on an SGB by an appeals court are given below in brief form.

A The *Ipp Report*: Object and recommendations

In 2002 all Australian states and territories introduced civil liability legislation[197] based on recommendations of the *Ipp Report*.[198] The report states: 'The Panel's starting point is that personal injury law comprises a set of rules and principles of personal responsibility.'[199] It then declares as an object that '[t]he Panel sees its task as being to recommend changes that impose a reasonable burden of responsibility on individuals to take care of others and to take care of themselves'.[200] Under the heading 'Recreational activities and services generally', the report states its conclusion: that there is wide public support for

> the idea that people who voluntarily participate in certain recreational activities can reasonably be expected … to take personal responsibility for, and to bear risks of, the activity that would, in the circumstances, be obvious to the reasonable person in the participant's position.[201]

B Precedent

Rather than an SGB being held liable for not adjusting its rules of play to provide for greater safety, in contradistinction courts have found against such a positive obligation. In *Agar v Hyde*,[202] before the High Court, two plaintiffs injured in a rugby scrum collapse claimed that the rules should have been changed to make scrummaging safer. Callinan J commented: 'Each participant in the match was adult and must be taken to have consented to the application of physical force in accordance with the laws of the game.'[203] As Gleeson CJ remarked:

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[197] *Civil Law (Wrongs) Act 2002* (ACT); *Civil Liability Act 2002* (NSW); *Personal Injuries (Liabilities and Damages) Act* 2003 (NT); *Civil Liability Act 2003* (Qld); *Civil Liability Act 1936* (SA); *Civil Liability Act 2002* (Tas); *Wrongs Act 1958* (Vic); *Civil Liability Act 2002* (WA).

[198] *Review of the Law of Negligence* (Final Report, September 2002).

[199] Ibid [1.24].

[200] Ibid.

[201] Ibid [4.13].

[202] (2000) 201 CLR 552.

[203] Ibid [88].

> It cannot be the case that all avoidable risks have to be eliminated. The only way to avoid risk of injury is not to play.[204]

C Obvious risk and assumption of risk

State civil liability legislation[205] provides that an SGB is not liable for harm that results from risks that are obvious when participating in a recreational activity.[206] Moreover, a participant is assumed to have volunteered for a risk of harm that is 'obvious'.[207] Clearly, the risks that arise through known activities of a sport, or that fall within and are permitted under the recognised rules of a sport, are 'obvious' risks. On this point, Callinan J remarked in *Agar v Hyde*:

> When adults voluntarily participate in sport they may be assumed to know the rules and to have an appreciation of the risks of the game.'[208]

Moreover, consent to implied rules is determined objectively and '[does] not depend upon the views of individual players'.[209] The defence of 'obvious risk' and its several applications within civil liability legislation is discussed extensively in Chapter 9 and need not be considered further in this chapter.[210]

D Judicial recognition of athlete and spectator acceptance

The High Court has recognised that risk generated through the physical nature of contact sport is not only accepted by players and spectators, but often sought after. In commenting on the argument that the 'law-makers' of the sport (that is, SGBs) should be liable because 'they could have made the activity that the participant chose to undertake less dangerous', Callinan J in *Agar v Hyde* commented: 'The absurdity of this proposition is highlighted by the fact that, in many activities, the danger is part of the activity's attraction. The participant may therefore not have chosen to engage in the activity at all if it was less dangerous.'[211] Gleeson CJ spoke on the issue at length, and said similarly:

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[204] Ibid [18].

[205] *Civil Law (Wrongs) Act 2002* (ACT); *Civil Liability Act 2002* (NSW); *Personal Injuries (Liabilities and Damages) Act 2003* (NT); *Civil Liability Act 2003* (Qld); *Civil Liability Act 1936* (SA); *Civil Liability Act 2002* (Tas); *Wrongs Act 1958* (Vic); *Civil Liability Act 2002* (WA).

[206] See the full discussion in Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences'.

[207] *Civil Liability Act 2002* (NSW) s 5G; *Civil Liability Act 2003* (Qld) s 14; *Civil Liability Act 1936* (SA) s 37; *Civil Liability Act 2002* (Tas) s 16; *Wrongs Act 1958* (Vic) s 54; *Civil Liability Act 2002* (WA) s 5N.

[208] (2000) 201 CLR 552, [127].

[209] *R v Barnes* [2005] 2 All ER 113, [15]. See also *R v Carr* (New South Wales Court of Criminal Appeal, Lee CJ, Allen and Badgery-Parker JJ, 17 October 1990); *Re Jewell* [1987] 1 VAR 370, 372.

[210] See Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences'.

[211] (2000) 201 CLR 552, [89].

> People who pursue recreational activities regarded as sports often do so in hazardous circumstances; the element of danger may add to the enjoyment of the activity. Accepting risk, sometimes to a high degree, is part of many sports.[212]

In addition, his Honour spoke of competitors seeking to 'test the limits' of their capabilities, which may result in physical harm, and added, somewhat definitively: 'Sporting activities of a kind that sometimes result in physical injury are not only permitted; they are encouraged.'[213]

Burns also points out that players must accept a compromise between safety and playing a sport they enjoy: '[T]he majority of the risks of the sporting activity [are] part of the trade-off for the preservation of the perceived essential characteristics of sport.'[214]

E Autonomy and parliamentary interference

In a general sense, sport has been afforded a high degree of autonomy from government and legal interference. Sports commentator Donnelly highlighted that sports take place 'in a sort of separate autonomous sphere, detached from normal rules and regulations in society'.[215] This concept of freedom of decision-making was further characterised in Donnelly's statement that '[a]utonomy refers to "the ability of a sports body, without undue external influence, to establish, amend and interpret sporting rules …; this autonomy is commonly justified as an important tool through which the values inherent to sport can be safeguarded from political, legal, and in the modern era, commercial influences'.[216]

F Who is to decide what level of risk is acceptable?

Should it have the authority, by what measure is a court differentiate between the rules of a sport that create an unnecessary risk of harm from those that do not? Courts do not ordinarily possess the objective skill or expertise to decide whether a certain rule in a certain sport is 'too dangerous' or 'unnecessarily dangerous' or to differentiate between the opinions of experts, the views of which are not likely to extend beyond the personal. As said by Gleeson CJ in *Agar v Hyde*:

> There is no objective standard by reference to which it is possible to decide that a given level of risk involved in rugby is acceptable, but that beyond that level, it is "unnecessary".[217]

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[212] Ibid [15].

[213] Ibid.

[214] Kylie Burns, 'It's Just Not Cricket: The High Court, Sport and Legislative Facts' (2002) 10 *Torts Law Journal* 234, 234–5.

[215] Peter Donnelly, 'Autonomy, Governance and Safe Sport' in J Stevens (ed), *Safe Sport: Critical Issues and Practices* (Encampus Ontario, 2022), quoting H Bruyninckx, 'Obsession with Rules vs Mistrust in Being Ruled' (Conference Paper, Play the Game Conference, 2011). See also JL Chappelet, *Autonomy of Sport in Europe* (Council of Europe Publishing, 2010).

[216] Ibid.

[217] Ibid [18].

The degree of subjectivity, continued his Honour, 'weighs against a conclusion that there is a legal duty which, in its practical application, depends upon such an assessment'.[218] Gaudron J said similarly that courts are not trained to differentiate between the possibly subtle differences between an existing rule and a proposed change to that rule: 'The laws of a game like rugby football differ from norms of conduct enforced by the courts. … What is "unduly" rough play in a body contact sport? What is "dangerous" play? All these and many other judgments must be made by the officials.'[219]

A concern of no small matter is the risk that a court mandating rule changes to a contact sport may invite opprobrium upon itself where the adjustment had no effect upon safety or, more tellingly, increased the risk of harm. For example, a study by Stokes found that the intuitively sensible rule change of mandate tackling in rugby below the armpits of an attacker, actually increased the occurrence of concussion by exposing the tackler to the pumping thighs of a hard running attacker.[220] A further example is the intuitive belief that wearing a helmet in contact sport will reduce the risk of head and neck trauma, when in fact the sense of invulnerability instilled in players increases the risk of injury.[221]

G Consistency of mandated adjustments across all dangerous sports

If there were, in fact, a legal obligation to change the rules of contact sports to improve safety, consistency would arguably require that all sports carrying a high risk of harm and death be subject to parliamentary of judicial amendment to their rules and protocols. In addition to being an unwelcome intrusion into people's lives and perhaps invite governmental election defeat, such an action would eliminate what Gleeson CJ described as 'the element of danger' that 'may add to the enjoyment of the activity'.[222] Consider the many popular sports and pastimes that would be affected by the initiation of such legal mandates as illustrated by the statistics below.

The government organisation Sports Injury Australia[223] provided figures for sports with the highest number of injuries resulting in a hospital stay in 2022. Cycling accounted for the highest number of injuries (8,638, or 15% of the total 56,070 injuries). Other sports included:

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[218] Ibid.

[219] Ibid [80].

[220] Keith Stokes et al, 'Does Reducing the Height of the Tackle through Law Change in Elite Men's Rugby Union (The Championship, England) Reduce the Incidence of Concussion? A Controlled Study in 126 Games' (2021) 55 *British Journal of Sports Medicine* 220.

[221] JC Maroon, PB Steele and R Berlin, 'Football Head and Neck Injuries: An Update' (1980) 27 *Clinical Neurosurgery* 414.

[222] *Agar v Hyde* (2000) 201 CLR 552, [15].

[223] 'Sports Injury in Australia', *Australian Institute of Health and Welfare* (Web Page, 17 July 2024) <https://www.aihw.gov.au/reports/sports-injury/sports-injury-in-australia/contents/about .

- AFL: 4,317;
- roller sports (such as roller skating): 4,143;
- wheeled motor sports (such as car racing and motorbike racing): 3,827;
- soccer: 3,744;
- rugby: 3,386;
- combat sports: 1,777.

Outdoor team sports combined (comprising rugby, AFL and soccer) accounted for 26% of sports injuries leading to hospitalisation in 2022.

Between 2000 and 2019, 1,192 deaths resulting from sport participation were reported in Australia, with the highest number occurring in wheeled motor sports (321 deaths, or 27% of the total 1,192 deaths).[224] Deaths in playing other sports included:

- wheeled non-motor sports (such as cycling and skateboarding): 193;
- individual water sports: 157;
- aero sports (such as parachuting and hang gliding): 116;
- equestrian: 77; and
- ice and snow sports: 25.

Team ball sports (including rugby league and AFL) accounted for 25 deaths, or 2.1%.

Not included within the statistics given above are the socially and legally acceptable so-called 'adventure sports' and other recreational pastimes. According to *Worldmetrics*,[225] BASE jumping has a death rate of 1 in 60 participants and big wave surfing has a death rate of 1 in 140 participants. Mountaineering results in 1.6 deaths per 1,000 climbs, and bull riding has a catastrophic injury rate of 9.8 per 1,000 rides.

H Legal tolerance of violent sport

Militating against liability in negligence is a centuries old high legal tolerance to violence and the risk of harm in sport and recreational pursuits, particularly where the plaintiff is taken to have consented to the risk of harm. For example, in *Hilton v Wallace*[226] the defendant, in

[224] L Fortington, A McIntosh and C Finch (2021) 'Injury Deaths in Australian Sport and Recreation: Identifying and Assessing Priorities for Prevention' 16(4) *PLoS One* e0250199.

[225] A Eser, *Worldmetrics Report 2024 — Most Dangerous Sports Ranked: Shocking Fatality and Injury Rates Revealed* (Report, 2024).

[226] [1989] Aust Torts Reports 68,448.

attempting to lift the plaintiff off his teammate in a rugby league game, inserted his finger to the plaintiff's eye socket, crushing the eyeball and causing loss of sight. The plaintiff was not in possession of the ball at the time. Although wrestling with a player not in possession of the ball is not within the express rules of the game, Kelly SPJ found that the act 'frequently [occurred] without any penalty being incurred' and could, provided the action was not done with undue force, be regarded as being within the 'spirit and intendment' of the game'; and found the defendant not liable.[227] There was no call by the court to ban such actions. In *Pallante Stadiums Pty Ltd (No 1)*,[228] the plaintiff boxer lost sight in one eye as a result of injuries received during a bout. He claimed that the sport involved the deliberate infliction of grievous bodily harm, not unreasonably citing as evidence that the object of boxing was:

(1) to render the opponent insensible for at least 10 seconds;

(2) to inflict such bodily harm on the opponent as to disable him from continuing the fight;

(3) to subdue the other by violent blows;

(4) to inflict serious bodily harm on the opponent;

(5) to inflict actual bodily harm not of a trifling and transient nature and going beyond mere physical discomfort.[229]

This very true assessment did not prevent McInerney J from finding for the defendants, stating that the intentional infliction of grievous bodily harm, which is illegal under statute and common law,[230] was nonetheless acceptable in the sport of boxing: 'To be an assault the infliction of force to the person of another must be done: (1) in a hostile or angry manner, and (2) without the consent of that other person.'[231]

Boxing, mixed martial arts[232] and other combat sports, despite being known to cause serious injury and death, are legal for those above the age of 18 as long as certain criteria are fulfilled. For example, in New South Wales, under the Boxing and Wrestling Control Act 1986, the following is required: completion of a 'certificate of fitness for boxing' by a medical practitioner, registration as a boxer by the Boxing Authority of New South Wales, and submission to medical examination after a contest.[233]

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227 Ibid 68,450–1.

228 [1976] VR 331.

229 Ibid 338.

230 See *R v Brown* [1994] 1 AC 212, 231; *Attorney-General's Reference (No 6 of 1980)* [1981] All ER 1057.

231 *Pallante v Stadiums Pty Ltd (No 1)* [1976] VR 331, 389.

232 Sometimes referred to as the Ultimate Fighting Championship (UFC).

233 *Boxing and Wrestling Control Act 1986* (NSW) ss 8, 56.

(i) But SGBs have been successfully sued in negligence?

Judicial determinations possess a plethora of examples of SGBs being successfully sued by athletes following a negligent operational decision. Almost universally these cases do not involve the express and known foundational rules of the sport. To give one example, in *Simmons v Rockdale Council*[234] a bike-rider struck a boom gate that had been installed at the entrance of a carpark on a route that was commonly used by cyclists. The plaintiff had his leg amputated below the knee. Hall J found that although the plaintiff was contributorily negligent in not adjusting his speed to the circumstances, the defendant council — having installed the gate — was 'under a duty to ensure that the system it established continued to provide, on a daily basis, for the safe passage, inter alia, of cyclists who, it was known were frequent users of the cycle route'.[235]

4.5.2 The (collateral) rules that SGBs have volunteered to adjust

As noted above, SGBs have not uncommonly responded to threats to the safety of their athletes by adjusting the rules of play — specifically, the 'collateral' or 'associated' rules that do not alter the basic nature of the sport. This does not mean, as discussed, that an SGB is obliged by law to change the rules of a sport to make it safer. The mitigatory actions taken by SGBs discussed in this section were undertaken voluntary and without legal compulsion. No finding by a court compelled the introduction of these safety measures. While the motivation of the SGBs is undoubtedly a concern for player welfare, it must be equally recognised that commercial interests — including spectator and television audiences, advertising, endorsement and sponsorship, and the willingness of parents to permit their junior children to participate in an apparently safer contact sport — were salient motivators.

Most importantly, the changes adopted did not alter the base conditions of play: rugby scrums were still set; tackling and body contact continued to cause bloodletting in football; bowlers in cricket still strained their backs sending down head-high bouncers at batters; and, after a short lay-off following loss of consciousness, boxers continued their punishing regimen in the ring. Whenever contact sport is played, that is, where athletic opponents make heavy contact or collide one with the other there is a risk of physical harm. This is an unavoidable truism, a point affirmed by Professor of Applied Policy Sciences, Mike Weed:

> [B]lows to the head, neck, or body, be they accidental or purposive, are to some extent a risk for virtually every athlete in virtually every sport (including non-contact sports such as alpine skiing).[236]

The illustrations below indicate that SGBs are not usually unaware of the risks posed in playing their contact sport; but they also indicate that while SGBs are prepared to introduce

[234] [2013] NSWSC 1431.

[235] Ibid [609].

[236] Mike Weed, 'Informing Evidence-based Policy for Sport-related Concussion: Are the Consensus Statements of the Concussion in Sport Group Fit for this Purpose?' (2024) 18(3–4) *Sport, Ethics and Philosophy* 433, 436.

safety protocols, they are not prepared to alter the rules of play that define the basic nature of the sport.

In these examples, the threat of harm was addressed by an adjustment to the collateral or associated rules of play (which did not remove the risk of harm), accompanied by a warning of the risk — either expressly, or as would reasonably be implied through the athletes' knowledge of the object to be achieved by changing the rules.

Following several catastrophic spinal injuries to front row forwards in the 1980 and 90s, the rugby codes in Australia responded by investigating the nature and cause of the harm and introducing modified scrum protocols to minimise the risk of such harm.[237] *Green v Country Rugby Football League of NSW Inc*[238] concerned a scrum collapse that resulted in a 16-year-old boy's tetraplegia. The plaintiff was described as having a 'slight build and had a long, thin, neck — sometimes called a *"swan neck"*',[239] a build that was unsuitable for safe front row play. Spinal trauma specialist Dr Yeo, who gave evidence in the case, said: 'In 1994 I and others involved in contact sport had emphasised to the community including the Rugby League authorities, the importance of adequate preparation for contact sport and appropriate choice of players for certain positions on the field.'[240] Warmsley AJ, in recording his judgment in *Green*, said of another spinal injury case, *Watson v Haines*,[241] that 'Mr Corcoran, a former [Australian Rugby League] director of coaching, told me that after the *Watson* case became known in rugby league circles, player safety was given greater attention'.[242] His Honour continued: 'Mr Corcoran worked a great deal with Dr Yeo on issues of spinal safety, and was the co-author of a publication initially sponsored by the Rothmans Foundation, called "National Coaching Scheme, Coaching Rugby League: Level 1 Coaching Certificate Course"'. This publication included a section on selection of players for the front row and scrummaging techniques, for example: 'It is important that [front rowers'] builds should include short, strong necks and strong shoulders.'[243]

In *Hyde v Agar; Worsley v Agar*[244] two rugby players were rendered quadriplegic in separate scrum incidents. In the Court of Appeal, the appellant players placed reliance on a

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[237] See *Hyde v Agar; Worsley v Agar* (1998) 45 NSWLR 487, 500 (Court of Appeal); *Agar v Hyde* (2000) 201 CLR 552; *Green v Country Rugby Football League of NSW Inc* [2008] NSWSC 26; *McCracken v Melbourne Storm Rugby League Football Club* [2005] NSWSC 107; *Maroubra Rugby League Football Club Inc v Malo* [2007] NSWCA 39.

[238] [2008] NSWSC 26.

[239] Ibid [38].

[240] Ibid [40].

[241] (1987) Aust Torts Reports 80-094.

[242] *Green v Country Rugby Football League of NSW Inc* [2008] NSWSC 26, [119].

[243] Ibid.

[244] (1998) 45 NSWLR 487 (Court of Appeal).

1987 article in the *Medical Journal of Australia*, which reviewed spinal-cord injuries to 107 rugby players over several years. The article argued that 'collision at scrum engagement caused the majority of such injuries' and, tellingly, that such harm was 'largely preventable by appropriate rule changes'.[245] The court noted that '[t]he paper led to changes in the rules in 1988 (introducing the "crouch, touch, pause" requirements), unfortunately too late for the appellants'.[246] In keeping with developing knowledge, the pre-scrum routine was modified several years later to 'crouch, bind, set', to emphasise a more deliberate packing of the front rows. Coaching and refereeing practices were adjusted commensurately. For example, to gain accreditation referees must now be 'smart rugby compliant'.[247] The Rugby Australia *Refereeing in Practice* manual presents a 'scrum checklist', and informs that 'scrum engagement is managed in sequence by the referee to ensure that it occurs safely'.[248] A component of the manual requires a referee to respond to a player's 'Mayday' call by 'blowing the whistle loudly'.[249]

When blood-borne diseases became a concern in the 1980s, contact sport responded by introducing 'once only' use of water sponges to mop the bloodied faces of cut players. Before long, players were required to leave the field if cut, to return only when a tamponade had quelled the flow of blood. In Australia, 'spear tackles', which were seen to increase the risk of neck injury, were banned in the rugby codes. Other dangerous tackles such as the 'chicken wing' (which was reported to risk a shoulder dislocation, as the arm was bent backwards from the shoulder) and the 'cannonball' (where a tackler's shoulder was directed to the knee joint of an opponent) were outlawed in rugby league. The AFL introduced match suspensions for 'shoulder bumps' to the head, and rugby outlawed contact above the armpits when tackling.

In cricket, as the risk of stress fractures to the lower back in fast bowlers became apparent, Dr A Warren advised in 2018 that 'practitioners should consider changing training or match bowling workloads to reduce their risk of injury'.[250] The SGB, Cricket Australia, warned: 'New data shows that players who sustain a lumbar bone stress injury coming through the [Junior Pathway] are 2.5 times more likely to have a lumbar bone stress injury (typically a stress

[245] Ibid 492. Spinal injury in scrummaging occurs in two ways. One: both scrums engage. The scrum collapses, resulting in a front rower's head striking the ground and forcing his head onto his chest. Further weight from the scrum forces the head to turn under the chest, causing a leveraging effect to the neck, dislocating the cervical vertebrae. Two: one front row crouches to pack, but the opposing front row does not. The packed scrum begins to move forward. The crouched front rowers are forced forward onto the stomachs or chests of the opponent front rowers. Further weight from the players behind them forces the head of a front rower onto his chest in a twisting motion, from which a subluxation of the cervical vertebra results.

[246] Ibid.

[247] Rugby Australia, *Referee Accreditation Policy*.

[248] Rugby Australia, *Refereeing in Practice 2020*, 41.

[249] Ibid 45.

[250] A Warren et al, 'High Acute: Chronic Workloads Are Associated with Injury in England & Wales Cricket Development Programme Fast Bowlers' (2018) 21 *Journal of Science and Medicine in Sport* 40, 44.

fracture)'.[251] In response to this and other developing medical advice, cricket authorities in the late 2010s restricted the number of overs a junior fast bowler was permitted to bowl in a match and across a week.

In each case, the sport's response to a threat of harm was to investigate, consider and, where deemed necessary, introduce mitigating protocols including the issuance of warnings — a similar process to the 'medical model'. This same response is argued in this chapter as warranted with respect to the threat of RHT to athlete health.

Decades before Omalu et al's revelations in 2005, it was not uncommon for medical journals to suggest that SGBs change the rules of play or their treatment protocols to better protect athletes from head trauma: see, for example, Lindsay's 1980 review of head trauma in sport,[252] which is an indictment of the SGBs of sport on two levels. First, Lindsay pointed to several studies — available as far back as the 1950s — that warned of a risk of long-term cognitive harm in playing contact sport.[253] Second, as a stand-alone contemporary study, Lindsay's review warned of cognitive harm at a time when many of the athletes now suffering CTE first entered premier competition. Consider some of Lindsay's references. He stated that '[t]he effects of repeated minor [head] injury are cumulative',[254] and suggested that a similar 'traumatic encephalopathy' to that experienced by boxers 'may occur in other sports, including Association football, rugby football, and wrestling'.[255] Lindsay also recorded Thorndike's 1952 recommendation 'that any player who has sustained three or more injuries with loss of consciousness or post-traumatic amnesia should be banned from further contact sports',[256] and quoted Murphey and Simmons as recommending that 'if head injury was severe enough to produce coma, or if a player has had a craniotomy, then further contact sports should be discouraged'.[257]

Lindsay noted that '[b]oxers must wait four weeks after loss of consciousness before fighting again' and opined that '[s]imilar rules should probably be applied to all sports', adding that 'if the post-concussional syndrome is present — namely persistent headaches, postural dizziness, irritability, and a failure of concentration — the player should not return to the sport

[251] Cricket Australia, *Cricket Australia Junior Bowling Guidelines 2019/20*, 4, <https://www.community.cricket.com.au/coach/news/bowling-guidelines/2019-11-14>.

[252] KW Lindsay et al, 'Serious Head Injury in Sport' (1980) 281 *British Medical Journal* 789.

[253] Notably, indicating the relatively early availability of information on cognitive harm in the playing of contact sport, Lindsay's study referenced several articles from British and American publications, including, of particular relevance to specialist 'sport medicos', the 1968 *Journal of Sports Medicine and Physical Fitness*.

[254] Ibid 790 (referencing D Gronwall and P Wrightson, 'Cumulative Effect of Concussion' (1975) 2(7943) *The Lancet* 995).

[255] Ibid (referencing Editorial, 'Brain Damage in Sport' (1976) 1(7956) *The Lancet* 401).

[256] Ibid (referencing A Thorndike, 'Serious Recurrent Injuries of Athletes: Contraindications to Further Competitive Participation' (1952) 247(15) *New England Journal of Medicine* 554).

[257] Ibid 791 (referencing F Murphey and JCH Simmons, 'Initial Management of Athletic Injuries to the Head and Neck' (1959) 98(3) *American Journal of Surgery* 379).

until all symptoms have resolved'.[258] This '1980s' approach to treatment is now standard in most contact sports in Australia and undoubtedly would have enabled players of the 1980s to contemplate the risk of cognitive harm had their organisation informed itself and its athletes. Lindsay concluded with a warning to athletes and the administrators alike: '[B]oth trainers and players may be reluctant to follow such policies, but if further damage should occur before recovery is complete then cumulative effects may be more serious.'[259]

Thus, it is clear that long-term cognitive harm in the playing of contact sport has been the subject of risk warnings for decades. The SGBs of contact sports 'made aware' in general terms, or informed by medical staff, must analyse that risk, consider its relevance to the sport they govern and, where material, warn their players of potential harm. At the very minimum, credible reports of risk are a prompt to investigate the foreseeability of harm.

As noted previously, sports officials are not commonly medically trained and are unlikely to have ready access to medical journals. However, where over time a link between head trauma in contact sport and CTE-like harm becomes 'notorious' or 'obvious', organising officials calling on their personal knowledge of their athletes and the conditions of play in their sport should, as a matter of law, reasonably foresee the risks of cognitive disfunction with little or no medical assistance, or at the very least direct their associated medicos to inquire and report.[260] To illustrate, given the abundant information flowing from the United States linking American football to CTE, officials of the Australian version of the game (often referred to as 'gridiron') could reasonably be expected to warn local players of the risk of long-term cognitive damage caused by, say, the repetitive clashing of helmets.

A A possible exception

Windholz implies that there is a case for mandating safety protocols do not alter the nature of the sport but rather are collateral to it, such as '[r]ules that protect the head from unnecessary collision, heat policies that prevent play in extreme conditions, padding on goal posts, and pitch inspections to ensure the safety of the playing surface'.[261]

Windholz also draws a line between what is reasonable as regards safety measures given the nature of contact and collision sports, stating: 'Eliminating all risks associated with the sporting contest (actual and simulated), while possible, is unlikely to be reasonable if it involves eliminating from the sport the physicality, excitement and contested essence which attracts participants and spectators, and on which it relies for its popularity and success.'[262]

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[258] Ibid 790.

[259] Ibid.

[260] *Bolton v Stone* [1951] AC 850.

[261] Eric Windholz, 'Professional Sport, Work Health and Safety Law and Reluctant Regulators' (2015) *Bond University Sports Law ejournal* 1, 7.

[262] Ibid.

Also noteworthy is the penalisation of deliberate or perhaps even reckless contacts to the head area of opponents. Weed points out that 'the primary concern of a policy purpose to reduce or eliminate the identified cause of SRC should not be isolated or accidental SRC, but rather the purposive direct blows to the head, neck, or body, which are a regular result of the constitutive rules of the sport'.[263]

4.5.3 Conclusion to this section

Whether, at some point — for example, under statutory direction or by developing precedent — SGBs will be required to adjust the rules of play to make a sport safer remains a possibility, though remote at best. A possible exception, noted above, is a legal direction to adjust the collateral protocols of a sport that do not alter the nature of the game. As observation, experience and medical reports reveal, it is not possible to eliminate head trauma from a contact sport given its very nature. While certain rule changes may reduce the quantity of head experienced, the risk nonetheless remains.

4.6 Conclusion

This chapter examined the law and facts regarding the obligation of an SGB of a contact sport to take precautions against a risk of harm in the form of CTE. A breach of the standard of care applicable to that duty may be assessed as positive on the basis of the application of law to fact established in this chapter.

In short measure, the contraction of CTE through the playing of contact sport is a foreseeable risk of harm. The medical, psychiatric and behavioural manifestations of CTE are 'not insignificant'; rather, they are physically and mentally debilitating and lead ultimately to death. Records of CTE in medical literature date back to the 1920s, indicating a disease that is not uncommon among those who engage in contact sport. The burden of gathering information on the risk of CTE and issuing a warning to premier players requires little effort or expense, and the failure to warn is unjustifiable on grounds of a higher moral purpose. With the development of digital communications, Martland's paper was accessible electronically, as were many other articles that advanced the understanding of long-term cognitive damage in contact and collision sports. Much was published in the pre-digital era. Much has also been published in the digital era. Although early publications referenced the sport of boxing, from the 1970s on CTE was liked to a range of other contact sports.

The breach of duty by an SGB in not taking reasonable steps to inform itself of the risk of CTE is a failure to apply the obligations propounded in the three principal cases discussed in this chapter. The SGB failed to inform itself that RHT may cause CTE, a known risk in boxing since Dr Martland's 1928 article in the *Journal of the American Medical Association*, and in other contact sports common to Australia from the 1970s on. Based on access to material

[263] Mike Weed, 'Informing Evidence-based Policy for Sport-related Concussion: Are the Consensus Statements of the Concussion in Sport Group Fit for this Purpose?' (2024) 18(3–4) *Sport, Ethics and Philosophy* 433, 439–40.

information, there is a strong argument that knowledge of CTE 'was or ought reasonably to have been possessed'[264] by an SGB.

The SGB failed to take reasonable steps by which to acquire knowledge of emerging risks — inclusive of CTE. These steps required the SGB to adopt a protocol by which scientific and medical journals conveying information on extant and emerging risks of harm were acquired, read and knowledge passed to it, to 'take reasonable care to inform itself.'[265] Where it is not able to acquire that knowledge and assess its materiality, the SGB must take reasonable care to ensure that someone else actively searched for information, assessed it, and transferred it to the SGB.[266] Had this been done effectively there can be little doubt that the SGB would have acquired knowledge of the risk of CTE in playing contact sport and from which it could have initiated warnings or other protocols in amelioration.

Where the SGB administers a contact sport — a violent undertaking — there is an arguable positive obligation to proactively seek information on emerging risks of harm to the players of its sport and where a possible threat is revealed to ensure that the chain of information is followed to a point of resolution. For example, where Dr Nathan Gibbs in 1994 reports in *Sports Medicine* [267] a concern that that *dementia pugilistica* could be caused by playing rugby league, or where the *British Medical Journal* in 1980 reports that 'repeated minor injury are cumulative' and that 'permanent damage (traumatic encephalopathy) is a risk in sports, 'including Association football, rugby football, and wrestling',[268] or where the *Clinical Journal of Sports Medicine* in 2001,[269] warned of the possible occurrence of CTE across several contact sports including 'soccer, football, ice hockey, and the martial arts', a literature search would reveal a history of CTE-like harm associated with contact sport going back decades and serve to substantiate the necessity of warning of this risk to players.[270]

An SGB breached the standard of care by failing to warn its premier players of the risk of CTE at the point where it was reasonable to consider it a 'real risk', a 'foreseeable risk', or given the seriousness of its consequences when, in paraphrasing Badgery-Parker J, a reasonable SGB should have 'began to perceive the need to take such precautions … *in case* it should turn

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[264] *Roe v Minister of Health* [1954] 2 QB 66, 92.

[265] *South Eastern Sydney Area Health Service v King,* [2006] NSWCA 2, [60].

[266] Ibid [59].

[267] Nathan Gibbs, 'Common Rugby League Injuries' (1994) 18(6) *Sports Medicine* 438, 438.

[268] KW Lindsay et al, 'Serious Head Injury in Sport' (1980) 281 *British Medical Journal* 789, 790.

[269] MH Rabadi and BD Jordan, 'The Cumulative Effect of Concussion in Sports' (2001) 11 *Clinical Journal of Sports Medicine* 194, 194.

[270] Consider also National Health Medical Research Council, *Football Injuries of the Head and Neck* (Report, 1994) iii; ALRC, *Essentially Yours: The Protection of Human Genetic Information in Australia* (Report 96, 2003). Discussion of the degree of accessible information available to SGBs is the subject of the next chapter.

out'[271] to be a physically and mentally destructive disease caused by RHT in playing the SGB's contact sport. In *H v Royal Alexandra Hospital* what were initially 'mere suspicions' of infection through blood contamination became in time 'a strong apprehension' as to what might be the cause.[272] At that point, well short of absolute proof, the duty to warn was triggered. Importantly, as 'there was still a lot to learn about this new syndrome',[273] the 'strong apprehension' that impelled the issuance of a warning was based on the reasonable vigilance of duty-holders, revealed facts and informed judgment. As noted several times, the legal obligation to take steps in response to a risk of harm does not require scientific certainty of that risk.

An SGB that had taken reasonable steps to inform itself of the risk of CTE would have been equipped to abide by its duty to warn its premier players of that risk. As argued, Australian SGBs delayed in fulfilling these obligations for a period of years and most likely decades, exposed its players to a risk of CTE; players that could have been avoided CTE had they decided to heed the warning and take mitigatory measures.

The overriding consideration, to be proven by a plaintiff athlete is expressed by Lord Morris in *Roe*:

> [C]are has to be exercised to ensure that conduct in 1947 is only judged in the light of knowledge which then was or ought reasonably to have been possessed. In this connexion the then-existing state of medical literature must be had in mind.[274]

Evidence of a link between head trauma in the playing of contact sport and the later onset of CTE-like symptoms dates to Martland's 1928 article. This study concerned boxers. Over succeeding decades, CTE-like harm in other contact and collision sports was increasingly reported. The relevant inquiry with respect to Lord Morris's dictum, then, concerns the availability and accessibility of these studies and whether a reasonable SGB — through observation, media reportage or as informed by its medical advisers — should reasonably have recognised a material risk of long-term cognitive damage in the playing of its sport. This is the topic of the next chapter — Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times'.

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[271] *H v Royal Alexandra Hospital* [1990] 1 Med LR 297, 307 and 311.

[272] Ibid 311.

[273] Ibid.

[274] *Roe v Minister of Health* [1954] 2 QB 66, 92.

Chapter 5:
Accessible Knowledge of CTE in Australia at Relevant Times

5.1 Introduction

The previous chapter, Chapter 4, argued that an SGB that failed to inform itself of the risk of CTE and to warn of that risk in the era prior to CTE becoming an obvious risk to premier-level players, breached its duty to take reasonable steps to care for the health and well-being of its premier players. Much of Chapter 4 concerned a discussion of the law applicable to a duty-holder's obligation to inform itself of a risk of harm and to warn those to whom it owed a duty of care.

Chapter 5 considers in brief form whether information on the risk of CTE was, as a factual matter, reasonably available to SGBs in Australia prior to *circa* 2010.[1]

The next chapter, Chapter 6, concerns the methodology of proving on the balance of probabilities that a plaintiff athlete is suffering from actual medical harm in the form of CTE.

The landmark 'rediscovery' of CTE by Dr Bennet Omalu et al in 2005[2] is, perhaps surprisingly, of limited value for athletes intending to sue their SGBs for failing to warn them of the risk of cognitive harm in playing contact sport. Almost all such athletes are currently more than 40 years old — and many are in their 50s, 60s and 70s — and played premier sport prior to the publication of Omalu et al's research. Where an athlete's suit is founded on a failure to warn, which is almost invariably the case with CTE, proof that a defendant '*ought* reasonably to have been possessed'[3] of knowledge of a risk of harm at the time of the breach is essential. Knowledge coming to light only in 2010 cannot qualify as knowledge that a major SGB 'ought reasonably' to have possessed prior to 2010. As such, the factual question concerns whether information on the risk of CTE in the playing of contact sport was reasonably available to Australian SGBs in the period prior to *circa* 2010, the roughly estimated year by which it is likely that players of contact sport in Australia at the premier level would be deemed by a court

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1 This is a somewhat truncated chapter that was reduced for reasons of word limitations. A full account can be found at: David Thorpe, 'Knowledge of Chronic Traumatic Encephalopathy and Concussion Cannot Support a Negligence Suit against major Sporting Organisations by Athletes. Or Can It?' (2022) 29(4) *Journal of Law and Medicine* 1052.

2 Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

3 *Roe v Minister of Health* [1954] 2 QB 66, 92. Emphasis added.

of law to have recognised CTE as an 'obvious risk'. 'Obvious risk', for purposes of statute and common law, is a question of fact.

Looking back to the 'three landmark medical studies' noted in Chapter 2,[4] the time that elapsed between Martland's article in 1928 and Omalu et al's in 2005 seems to indicate that there was little study into a link between the playing of contact sport and the risk of long-term cognitive harm carried out over a period of more than 70 years. In fact, the contrary is true. Literally hundreds of journal articles published across the world revealed just such a risk. These studies implicated boxing in the main, but progressively — particularly from the 1970s onwards — associated a range of contact sports common to Australia with the onset of CTE-like harm. As argued in this chapter, these studies were available for access by any reasonable SGB through its medical associates.

This chapter proposes that, between the publication in 1928 of Martland's 'Punch Drunk' article in the *Journal of the American Medical Association*[5] and the ground-breaking 2005 revelations of 'Chronic Traumatic Encephalopathy in a National Football League Player' by Omalu et al,[6] sufficient information *was* available from which the governing bodies of major contact sports could assess the risk of cognitive disfunction in their players. This information was either not accessed, or it *was* accessed and — for reasons uncertain, but suggestive of insouciance, wilful blindness, and fear of financial implications and parental backlash — was withheld from players. In each case, the SGB's obligation to safeguard the health and well-being of their athletes was, as argued by the author, not fulfilled.

As almost all litigation by athletes suffering symptoms of CTE is framed around, or includes, a duty to warn, reports published years or decades ago will necessarily form the basis of a case against the major SGBs.[7] The pertinent question in law is this: given that knowledge of CTE-like harm extends back to the 1920s, should the reasonable SGB prior to 2010 — through observation, acquired knowledge, direct research or being informed by its medical

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4 See **[2.4.1]**, regarding Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103; Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128; and Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

5 Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

6 Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

7 See, eg, *In Re National Football League Players' Concussion Injury Litigation*, No 2:12-md-02323-AB, MDL No 2323, Plaintiffs' Amended Master Administrative Long-Form Complaint, [8]; District Court of the Eastern District of Pennsylvania. In that case, the plaintiffs argued that the National Football League was 'aware' or 'should have been aware' of a 'growing body of scientific evidence and its compelling conclusions that professional football players who sustain repetitive [mild traumatic brain injury] during their careers are at greater risk for chronic neuro-cognitive illness and disabilities both during their football careers and later in life', and 'ignored, minimized, disputed, and actively suppressed broader awareness of the link between sub concussive and concussive injuries in football and the chronic neuro-cognitive damage, illnesses, and decline suffered by former players, including the Plaintiffs'.

personnel — have taken steps to protect its players from the risk of long-term cognitive harm caused by RHT?

Consideration, though, must also be given to the degree to which knowledge of CTE associated with one sport is transferable to another sport. For example, while RHT in contact sport leading to long-term cognitive disfunction was first reported medically in the 1920s, the focus of early reports was usually on the sport of boxing, and not contact sports such as rugby, Australian football, soccer and hockey.

Consider for purposes of orientation the case as put by the legal representative of ex-England international rugby player and CTE litigant Steve Thompson: '[T]he governing bodies have failed in their duty of care, having not acted upon the known risks of head injury.'[8] The writ in the Max Rooke class action[9] claimed similarly that the Australian Football League (AFL) 'knew or ought to have known' of the 'potential long-term consequences of concussion', but failed to inform players of that risk.[10] Axiomatically, though, an SGB cannot know of a risk of harm unless a source of such knowledge exists.

As discussed at length in Chapter 4, liability in negligence for failure to warn does not attach unless information regarding the relevant risk of harm was available to the defendant and 'ought reasonably to have been possessed'.[11] However, liability in negligence cannot be avoided by a claim of ignorance where knowledge 'ought reasonably to have been possessed'.[12] As a matter of logic and law, therefore, any duty-holder alerted to the possible existence of information that impacts on his or her duty of care 'ought reasonably' to inquire as to its relevance.[13] This formulation would make little sense if deliberate avoidance of inquiry or wilful blindness to risk absolved a duty-holder from liability. Such failures suggest a fear of inquiry and a quest for 'plausible deniability'.

5.1.1 This chapter proceeds as follows

Section 5.2: The fundamental legal question: Information indicating foreseeability

This section reviews the law relevant to the foreseeability of information regarding a risk of CTE-like harm in the playing of contact sport. The section briefly notes that an inference may

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8 Michael Alwyn and Andy Bull, 'Rugby World Cup Winner Steve Thompson Reveals He Has Dementia and Joins Landmark Legal Case', *The Guardian* (London, 8 December 2020).

9 Discussed in Chapter 2 at **[2.4.2]**: 'Two other influential periods'.

10 *Jarad Maxwell Rooke v Australian Football League*, Supreme Court of Victoria, Case: S ECI 2023 00969, Writ, cll 10 and 15.

11 *Roe v Minister of Health* [1954] 2 QB 66, 92.

12 Ibid.

13 *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2.

be drawn from facts that are singularly inadequate provided that collectively they combine to substantiate a fact on the balance of probabilities.

Section 5.3: Martland's enduring warning of 'traumatic encephalitis'

This brief section highlights that Martland's description of the symptoms of 'punch drunk syndrome' remains largely accurate as against modern descriptions of CTE to provide, among other facts, an indication to an SGB that RHT in playing contact sport should be investigated.

Section 5.4: Extrapolating from the boxing studies

This section examines the contention that although boxing is a different and outwardly more violent sport when compared to common Australian contact sports, such as AFL or rugby, the symptoms of CTE displayed in both boxing and Australian contact sports are the same and the mechanism of harm is identical — that of RHT.

Section 5.5: Non-sport concussive trauma as a source of information on CTE

CTE-like harm is a cellular reaction in the brain to RHT and has been recorded as far back as the 1800s. This section suggests that to know of the cause of CTE in general is to know of the cause of sport-related CTE. This fact ought to have prompted SGBs to warn their players of the risk of cognitive disfunction in playing contact sport.

Section 5.6: 'Pre-Omalu' reports of CTE in sports *other* than boxing

This section stresses that knowledge of CTE had been revealed in medical journals in sports other than boxing from at least the 1970s. It argues that a reasonably dutiful SGB 'ought' to have accessed this information and protected its players by warning them of the risks of CTE.

Section 5.7: The Australian studies relevant to knowledge of CTE prior to 2010

This section highlights several Australian studies in the 1990s and early 2000s that touched on the risk of CTE. While the point is made that, in combination, these reports could be argued to have informed of the risk of CTE, the expression of that risk was often ambiguous or merely indicated a need for further study and inquiry. As such, any legal redress sought by Australian athletes suffering symptoms of CTE pre-*circa* 2010 will inevitably have to rely on the research and information supplied through international medical journals with, at best, collateral support through the Australian publications.

Section 5.8: Extrapolating from American football

The common contact sports played in Australia most resemble American football, albeit with obvious differences. As such, the fact that players of the National Football League (NFL) in the United States had developed CTE — a fact widely reported in US medical journals — is proposed as a further source of information from which SGBs in Australia ought to have discerned a similar risk in their own contact sports.

Section 5.9: Conclusion

This section proposes that the SGBs of contact sports in Australia had sufficient access to information regarding the risk of CTE such that they were duty-bound to warn those who played in their premier competition prior to 2010 of the risk.

Appendix 2: CTE and 'Negativing Influences': Do They Determine Liability?

Appendix 2 supplements this chapter by reviewing an argument made by some commentators that certain journal articles so downplayed the risk of CTE from RHT in playing contact sports that Australian SGBs were justified in believing the risk was not foreseeable, thereby excusing them from failing to warn their players of that risk. It also examines claims that some journal articles published in Australia were sufficiently informative of the risk of CTE that liability should attach to Australian SGBs.

5.2 The fundamental legal question: Available information indicating foreseeability

Was information on the risk of CTE published in medical journals as far back as the 1920s sufficient to establish in law that Australian SGBs ought to have had knowledge of that risk prior to 2010? In other words, was the risk of CTE foreseeable?

Foreseeability of harm is only possible where information regarding that harm is available to a duty holder. The relevant statutory provision is s 5B(1)(a) of the *Civil Liability Act 2002* (NSW), which is similar to that of other states:[14]

> (1) A person is not negligent 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.

The statutory approach is reflected in the formulation of Mason J in *Wyong Shire Council v Shirt*:

> In deciding whether there has been a breach of the duty of care the tribunal of fact must first ask itself whether a reasonable man in the defendant's position would have foreseen that his conduct involved a risk of injury to the plaintiff or to a class of persons including the plaintiff. If the answer be in the

[14] *Civil Law (Wrongs) Act 2002* (ACT) s 43(1); *Civil Liability Act 2003* (Qld) s 9(1); *Civil Liability Act 1936* (SA) s 32(1); *Civil Liability Act 2002* (Tas) s 11(1); *Wrongs Act 1958* (Vic) s 48(1); *Civil Liability Act 2002* (WA) s 5B(1). The Western Australian Act uses the term 'fault' rather than 'negligence'. The common law applies in the Northern Territory. A full analysis of the operation of s 5B may be found in Chapters 3 and 4.

affirmative, it is then for the tribunal of fact to determine what a reasonable man would do by way of response to the risk.[15]

5.2.1 Drawing an inference

In this chapter, what may at times be seen as disparate or insignificant facts akin to 'feeble rays of light' are nonetheless, in the words of Lord Cairns, capable when 'all converging and brought to bear upon the same point, and, when united, [of] producing a body of illumination which will clear away the darkness which you are endeavouring to dispel'.[16] In other words, in drawing an inference of whether information on the risk of CTE was reasonably available to Australian SGBs from facts and circumstantial evidence separated across decades and involving a range of sports perhaps of varying materiality, 'we have to consider the weight which is to be given to the united force of all the circumstances put together'.[17]

That reasonable availability of information on the risk of CTE was reasonably available to an Australian SGB *circa* 2010 is a question of fact. As said by the High Court in *Holloway v McFeeters*: 'Inferences from actual facts that are proved are just as much part of the evidence as those facts themselves.'[18] Of course, the standard of proof is on the balance of probabilities:

> [Y]ou need only circumstances raising a more probable inference in favour of what is alleged … By more probable is meant no more than that upon a balance of probabilities such an inference might reasonably be considered to have some greater degree of likelihood.[19]

Consequently, in assessing the risk of CTE-like harm in boxing, one's judgment is not isolated from other sports and circumstances but considers a fact such as RHT as a cause of CTE, perhaps weighted against the greater frequency of deliberate blows to the head, when comparing the materiality of boxing to non-pugilistic contact sports.

5.3 Martland's enduring warning of 'traumatic encephalitis'

A measure of the relevance of the 'pre-Omalu' publications as a source of foreseeability of CTE, including the boxing studies, is the extent and degree to which symptoms and indicators of CTE (often referred to as *dementia pugilistica* or punch drunk syndrome) align with those displayed around the time of Omalu et al and McKee et al's articles.

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15 *Wyong Shire Council v Shirt* (1980) 146 CLR 40, 47.

16 *Belhaven and Stenton Peerage* (1875) 1 App Cas 278, 279.

17 Ibid. Drawing an inference in law and fact with respect to CTE is discussed extensively in Chapter 6 at **[6.5]** and **[6.6]**.

18 *Holloway v McFeeters* (1956) 94 CLR 470, 480.

19 Ibid 481 (Dixon CJ), quoting the unanimous judgment of Dixon, Williams, Webb, Fullagar and Kitto JJ in *Bradshaw v McEwans Pty Ltd* (High Court of Australia, 27 April 1951). Inference and fact are discussed extensively in Chapter 6: 'Proof *In Vivo* of Actual Harm in the Form of CTE: The Question of "Individual Agency"'.

Although the medical science of 1928, the date of Martland's article in the *Journal of the American Medical Association*, lacked the tools necessary to reveal the cellular leakage of tau discovered in 2005 by Omalu et al, the 'cause and effect' in Martland's 1928 revelations of 'punch drunk syndrome' are essentially those reported by Omalu et al as the 'cause and effect' of CTE. Martland found that repetitive blows to the head in boxing led to 'traumatic encephalopathy' in his patients. Omalu et al found that repetitive blows to the head in American football could cause 'chronic traumatic encephalopathy'. In addition to sharing a similar nomenclature, each medico presents a common theme: that repetitive blows to the head can lead to long-term cognitive disfunction.

The revelatory medical findings of Martland's research, together with ample non-medical 'proofs', are — arguably and perhaps conclusively — sufficient to have alerted the reasonable SGB and associated medical practitioners to the risk of 'punch drunk syndrome' (CTE) in all contact sports, and prompt the fulfilment of the legal duty to 'take steps' to find, to read and to understand.[20] First, in terms of broad medical evidence, Martland states:

> [T]he occurrence of the symptoms in almost 50 per cent of fighters who develop this condition in mild or severe form, if they keep at the game long enough, seems to be good evidence that some special brain injury due to their occupation exists.[21]

Second, Martland speaks of non-medical 'proofs' as follows:

> We are placed in the position of accepting a series of objective symptoms described to us by laymen. There is undoubted proof that for years fighters, fight promoters and the sporting world have recognized and talked about this condition.[22]

Of similar impact is Martland's illuminating dismissal of claims that the disease of cognitive deficiency known as 'punch drunk syndrome' in boxing was imaginary or exaggerated. 'One sports-writer', reported Martland, 'recently stated that punch drunk was greatly exaggerated and that he had consulted eminent neurologists who had assured him that such a condition did not exist.'[23] Whether the journalist had in fact consulted 'eminent neurologists' cannot be known, but Martland's retort, along with his medical assessment, points to a risk that places succeeding generations of duty-holders on inquiry:

> I have found that the opinion of shrewd laymen, many of whom are making a living by observing the physical fitness, actions and characteristics of the professional fighter, is perhaps more substantial than the opinion of medical experts.
>
> A fight promoter whose ability to judge the physical condition of fighters is unquestionable has given me the names of twenty-three fighters whom he considers punch drunk. Many of these men are scattered over the country and I have been unable to ascertain their exact condition at the present

[20] *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2, [67].

[21] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103, 1103.

[22] Ibid 1106.

[23] Ibid.

> time, especially those who are in asylums. I have examined five of these men and they present the clinical pictures as described.[24]

On one argument, Martland's warning of the harmful effects of repeated head trauma in boxing is enough to have alerted all contact sporting organisations to the risk of long-term cognitive harm. According to this argument, from 1928, the reasonable sporting organisation could access information of CTE-like harm sufficient to warrant further inquiry and to issue warnings to its athletes. What is important is not the terms used by Martland and Omalu et al to describe a common medical malady, but the mechanism by which the malady occurs — RHT in sport. The name is different, but the cause is common and known.

5.4 Extrapolating from the boxing studies

The boxing studies are integral to the history of CTE in sport and are mentioned in many, if not most, of the recent reports of CTE in contact sports. The extent to which medical journal articles concerned with boxing are relevant to discerning CTE in the non-boxing sports has been questioned — a reasonable question.[25]

The studies of boxing and CTE are relevant to the discussion of CTE in the non-pugilistic contact sports for several reasons. Foremost is the common link of RHT in both types of sports, such that the possibility of CTE in, say, Australian football, is recognised through CTE in boxing. The symptoms of *dementia pugilistica* and CTE are identical, as is the progression of the disease — a point that belies later claims that CTE is not a real disease.

To consider just one boxing study of the hundreds available — in 1984 Casson, to counter the argument that 'chronic encephalopathy' 'occurs only in an occasional poorly skilled fighter' with 'minimal education, lack of modern medical controls, and abuse of alcohol and other drugs',[26] studied 18 current and retired boxers (five amateur and 13 professional) who had no 'history of neurological, psychiatric, or medical illness or substance abuse'.[27] The subjects ranged in age from 18 to 60 years, with a mean age of 36 years:

> Three are college graduates, ten others are high school graduates, and the remaining five all had attended junior high school. The mean educational level was grade 12. All the former fighters are currently employed full time in civil service, education, or private industry.[28]

Total bouts boxed were 240, with an average of 83 per person. Professional bouts stood at 128, with a mean of 29. The length of career ranged from three months to 22 years. Casson found

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[24] Ibid.

[25] P McCrory, 'When to Retire after Concussion?' (2002) 5(3) *Journal of Science and Medicine in Sport* 169. Note that McCrory's article was retracted, but not for reasons relevant to this statement. For full discussion, see Appendix 2 at **[2.4]**.

[26] IR Casson, 'Brain Damage in Modern Boxers' (1984) 251(20) *Journal of the American Medical Association* 2663, 2663.

[27] Ibid.

[28] Ibid 2663.

> unequivocal evidence of organic cerebral dysfunction in 87% (13/15) of this group of ex-boxers and active professional fighters. The other two professional and three active amateur boxers had suggestive evidence of subtle brain injury …[29]

He concluded that

> the correlation between impairment index and number of professional fights is more clinically significant than that for age. This suggests that the longer one's professional boxing career, the greater is the likelihood of brain damage.[30]

Aligning with other studies, Casson found that '[a] cumulative effect of multiple sub-concussive blows to the head is a likely etiology',[31] to finish with an ominous warning:

> Existing medical controls and safety measures did not prevent chronic brain damage in this group of boxers. There is no reason to believe that future generations of boxers will be any more fortunate.[32]

The boxing studies are part of the provenance of CTE; they provide a historic legitimacy to later claims of a disease caused by RHT in sport. When Dr Omalu et al's research received notoriety in 2005, previous knowledge of *dementia pugilistica* gained through the boxing studies gave plausibility to the proposal that 'it had spread' to the contact sport of American football. With this background knowledge, tentative acceptance or, at least, an open mind to the prospect of CTE in the non-pugilistic contact sports should, arguably, have followed, leading to a 'strong apprehension' of a foreseeable risk of harm.[33]

5.5 Non-sport concussive trauma as a source of information on CTE

Cognitive disfunction caused by concussive head trauma 'has been known for thousands of years, its effects described in legend, and in historical and medical writings'.[34] For 'almost a century', notes Casper, 'single or repetitive HI [head injury] have been known to result in degenerative processes'.[35] Concussive trauma in sport is a subset of concussive trauma in general — to know the cause of concussion in general is to know of the cause of concussion in sport. In this sense, a medical report of concussive trauma in a non-sporting context is relevant to concussive trauma in sport, and serves equally to warn the conscientious medico or sports organisation of the relevant dangers of contact sport. In the form of a simple syllogism: Can

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29 Ibid 2666.

30 Ibid.

31 Ibid 2667.

32 Ibid.

33 *H v Royal Alexandra Hospital* [1990] 1 Med LR 297, 311.

34 Louise Gordy, 'Concussion of the Brain' (1955) *American Bar Association, 1955 Proceedings, Section of Insurance Law* 437, 452.

35 ST Casper, 'Concussion: A History of Science and Medicine, 1870–2005' (2018) 58 *Headache* 795, 808.

head trauma or repeated concussions cause permanent cognitive damage? Does head trauma or repeated concussions occur in contact sports? Is your sport a contact sport?

For more than 150 years prior to Omalu et al's report in 2005, medical journals warned of threats to the well-being of those who suffer concussive blows to the head, whether incurred in sport or otherwise. There is, however, little to be gained in citing large numbers of studies published since the 1850s to illustrate that which is not in contention:[36] that blows to the head cause cognitive trauma, which, if repetitive or severe, leads to long-term and permanent cognitive damage. Casper referred to this extensive historical account over time as 'a strong genealogical relationship across the generations'.[37]

Gronwall and Wrightson's 1975 article 'Cumulative Effect of Concussion'[38] did not concern sport, but it is clearly relevant to head injury in sport. Patients who suffered multiple concussions were 'tested individually within 48 hours of injury with four trials of a paced auditory serial addition task, shown to estimate the rate of information processing'.[39] On the issue of whether 'a number of mild concussions' would lead to permanent cognitive decline, Gronwall and Wrightson drew a clear connection:

> We have no data to show this, but there is evidence of the cumulation of repeated injury in Roberts' study of brain damage in boxers[40]. … Roberts' evidence does suggest that in the general area of concussion-like syndromes the effect of repeated damage is cumulative.[41]

The authors proposed that long-term damage was a result of multiple concussions:

> If, as seems likely from pathological studies, the mechanism of concussion of all grades of severity includes neuronal damage, the most probable explanation of the cumulation of the effects of concussion is that each event destroys neurons, diminishing the reserve available and making the loss evident under the stress of further injury.[42]

As said by Casper (in 2018), the foundational causes and cerebral changes associated with CTE were catalogued 60 years earlier than the publications of Omalu et al and McKee et al:

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[36] For example, the word 'concussion' searched at *The Lancet* results in 1,968 articles between 1900 and 2018, including letters and commentary.

[37] ST Casper, 'Concussion: A History of Science and Medicine, 1870–2005' (2018) 58 *Headache* 795, 796.

[38] D Gronwall and P Wrightson, 'Cumulative Effect of Concussion' (22 November 1975) *The Lancet* 995.

[39] Ibid 995.

[40] AH Roberts, *Brain Damage in Boxers* (Pitman, London, 1969).

[41] D Gronwall and P Wrightson, 'Cumulative Effect of Concussion' (22 November 1975) *The Lancet* 995, 997. Footnote added.

[42] Ibid.

> By the 1950s, sub-concussive blows, second-impact syndrome, tangle pathology, and concerns about cumulative effects of concussion contributed observations that are now key to current understanding of concussion. The science was secure.[43]

5.6 'Pre-Omalu' reports of CTE in sports *other* than boxing

This section considers medical research prior to 2005 concerned with concussion and CTE-like harm in sports *other* than boxing. The question of interest is this: should the debilitating effects of head trauma in the sports that were studied put all sporting organisations and associated medical staff 'on inquiry'? In other words: 'ought' the reasonable SGB have 'joined the dots' and foreseen the possibility of long term and progressive cognitive harm in the playing of its sport?

Some journal articles referred to below draw a direct parallel between cognitive disfunction in boxing and cognitive disfunction in a different sport. Other articles consider cognitive disfunction in one sport and implicate a similar sport.

It should be stressed that the sheer number of medical articles — circulated and cross-referenced in both specialised sports medicine journals and other professional medical journals — articulating a connection between cognitive debilitation and head trauma in sport make it is difficult to believe that a reasonable physician associated with sport, if not physicians in general, would be ignorant of the risk of CTE. Contemplation, where it did exist, was seemingly dismissed however, giving way to an unsure inertia that was content to await the revelations of a trailblazer.

Several studies concerned with the revelations of CTE-like harm in non-boxing sports in the 'pre-Omalu' era are considered below.

An emergency department medico in 1976 informed *The Lancet* that there appear to be 'reasonable grounds' to advise patients to 'stop collision sports' if they had suffered 'two episodes of head injury' that resulted in loss of consciousness or amnesia'.[44]

Lindsay, writing in the *British Medical Journal* in 1980, began with a historical statistic:

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[43] ST Casper, 'Concussion: A History of Science and Medicine, 1870–2005' (2018) 58 *Headache* 795, 808. Nonetheless, and not without irony, the sheer degree of punishment some boxers suffer is so severe that to extrapolate from boxing studies to other contact sports is difficult, if not arguably unreasonable. For further discussion, see David Thorpe, 'Knowledge of Chronic Traumatic Encephalopathy and Concussion Cannot Support a Negligence Suit against major Sporting Organisations by Athletes. Or Can It?' (2022) 29(4) *Journal of Law and Medicine* 1052.

[44] ID Adams, 'Brain Damage in Sport' (13 March 1976) *The Lancet* 585, 585.

> Of 1900 head injuries serious enough to be admitted to the neurosurgical unit in Glasgow over a five-year period, 52 (2–7%) were due to 'sport.' Golf, horse-riding, and Association football were the sports most commonly linked with serious head injury.[45]

Then, under the heading 'Prevention of cumulative damage', Lindsay drew a parallel not only with cognitive harm in boxing, but also with other contact sports:

> Injuries producing even a few minutes post-traumatic amnesia may cause some microscopic structural brain damage, and impaired psychological function for two to three weeks. The effects of repeated minor injury are cumulative; the occurrence of permanent damage (traumatic encephalopathy) in boxing and in jockeys has led to the introduction of statutory medical cover in these sports.
>
> More recently, it has been suggested that a similar encephalopathy may occur in other sports, including Association football, rugby football, and wrestling.[46]

After proposing that all sports should, in similitude with boxing, mandate four weeks' ineligibility after being knocked out, Lindsay warned that while it 'is understandable that both trainers and players may be reluctant to follow such policies … if further damage should occur before recovery is complete then cumulative effects may be more serious',[47] and then proposed that 'governing bodies of the individual sports should draw up rules to ensure that sports trainers and supervisors are aware of the correct management of head injuries'.[48]

In 1992, neurologist Robert Cantu published the article 'Cerebral Concussion in Sport',[49] which included the comment: '[B]rain injuries are one of the most common catastrophic athletic injuries and a leading cause of athletic death.'[50]

Daly, in the journal *Sports Medicine* in 1990,[51] examined the causes and effects of head injury in ice hockey. He noted that high skating speeds and quick changes of direction in an environment of 'rigid boards, goalposts, and opposing players eventually will result in high-impact collisions regardless of the player's skill. Such collisions result in blunt injuries to the extremities, viscera, and head.'[52]

Neurologist Wilberger wrote in 1993 that minor head trauma 'may result in temporary disturbances and also long term persistent clinical symptoms and neurological, cognitive and psychological sequelae', stating prophetically that 'with the possibility of many head injuries

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45 KW Lindsay et al, 'Serious Head Injury in Sport' (1980) 281 *British Medical Journal* 789, 789.

46 Ibid 790.

47 Ibid.

48 Ibid 791.

49 RC Cantu, 'Cerebral Concussion in Sport: Management and Prevention' (1992) 14(1) *Sports Medicine* 64.

50 Ibid 65.

51 PJ Daly, 'Ice Hockey Injuries: A Review' (1990) 10(3) *Sports Medicine* 122.

52 Ibid 123.

potentially occurring over an athlete's entire career, questions have been raised over the possible cumulative effects of these injuries'.[53]

In 1999 Erlanger introduced an article in *The Clinical Neuropsychologist* with the words: 'Neuropsychologists are increasingly examining and treating athletes sustaining head injuries ranging from the frequent uncomplicated mild concussion to the less frequent cerebrovascular compromise, oedema, and *Dementia Pugilistica*.'[54] The article listed the sports of 'football, rugby, hockey, and lacrosse — in which contact to the head is incidental — to non-contact sports not typically associated with head injury such as soccer, baseball, and basketball'.[55]

In the *Clinical Journal of Sports Medicine* in 2001,[56] four years prior to Omalu et al, neurologists Rabadi and Jordan once more informed the medical community that 'punch drunk syndrome' or 'chromic traumatic encephalopathy' was the result of 'cumulative, long-term neurologic consequences of repetitive concussive and sub-concussive blows to the brain',[57]

In the journal *Neurosurgery*, Bailes and Cantu reported in 2001 that concussion may be 'more common and more serious than previously thought' and that 'long-term consequences of repetitive blows to the head are now considered to be detrimental to the future well-being of the athlete'.[58]

5.7 The Australian studies relevant to knowledge of CTE prior to 2010

This section examines whether three Australian studies published in the 1990s and early 2000s provided information on the risk of CTE that Australian SGBs ought to have *reasonably possessed* regarding the dangers posed by playing contact sports. It also considers whether the information contained within those publications was of sufficient materiality to prompt further inquiries as a matter of duty.

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[53] JE Wilberger, 'Minor Head Injuries in American Football: Prevention of Long-Term Sequelae' (1993) 15(5) *Sports Medicine* 338, 338–9.

[54] DM Erlanger, 'Neuropsychology of Sports-related Head Injury: Dementia Pugilistica to Post Concussion Syndrome' (1999) 13(2) *The Clinical Neuropsychologist* 193, 193.

[55] Ibid.

[56] MH Rabadi and BD Jordan, 'The Cumulative Effect of Concussion in Sports' (2001) 11 *Clinical Journal of Sports Medicine* 194.

[57] Ibid 194.

[58] JE Bailes and RC Cantu, 'Head Injury in Athletes' (2001) 48(1) *Neurosurgery* 26, 26.

5.7.1 National Health and Medical Research Council (1994)

In 1994 the National Health and Medical Research Council (NHMRC) convened a panel of medical experts 'to inquire into head and neck injuries resulting from participation in the sport of football (all codes) and to suggest ways of minimising injuries'.[59] Under the heading 'Long-term problems' the following statement was made regarding head trauma:

> There is some evidence that there may be some long-term effects, particularly in terms of attentional processes and information processing rate, from a single head injury, even if there has not been actual loss of consciousness. The issue is still debateable.

Although indefinite, this statement could reasonably be considered to have provided sufficient information on the risk of CTE-like harm to SGBs (as perhaps informed by their medical associates) to have prompted a dutiful SGB to initiate inquiries with the view to confirming or disaffirming the possibility of harm and, where a material risk was affirmed, to have taken reasonable steps to ameliorate the risk.

A Doubts expressed

The panel referred to a study by Tysvaer[60] of Scandinavian soccer players which showed that 'cognitive deficits were noted. … Approximately 40 per cent of the control group were found to be neuropsychologically impaired!'[61] This 'cognitive deficit' was explained as follows: 'significant methodological problems flaw the studies — notably, the lack of pre-injury data, statistical bias and inadequate control subjects'.[62]

Of interest was the apparent indifference of the panel to the conclusion of the authors 'that the deficits noted in the former soccer players were explained by repetitive trauma such as heading the ball.'[63] The panel then appeared to excuse the study, stating: 'To date there has been no replication of these findings by other groups. The question of whether repeated headings of the ball causes long-term effects has not been answered.'[64]

The final comment indicates an awareness of a possible risk of long-term damage, but one that has been dismissed rather than leading to further investigation of a possible cause of serious

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[59] NHMRC, *Football Injuries of the Head and Neck* (Report, 1994) iii. This report was noted by A Greenhow in 'Mitigating the Concussion Crunch: League Governance, Passive Actors and Regulators of Risk' (2021) 14(1) *Australian and New Zealand Sports Law* 36.

[60] AT Tysvaer, OV Storli and NI Bachen, 'Soccer Injuries to the Brain: A Neurologic and Electroencephalographic Study of Former Players' (1989) 80(2) *Acta Neurologica Scandinavica* 151; AT Tysvaer and EA Lochen, 'Soccer Injuries to the Brain: A Neuropsychologic Study of Former Soccer Players' (1991) 19(1) *American Journal of Sports Medicine* 1119.

[61] NHMRC, *Football Injuries of the Head and Neck* (Report, 1994) 19. Exclamation mark in the original.

[62] Ibid.

[63] Ibid.

[64] Ibid.

harm to athletes. It is clear, however, that the NHMRC report placed the major sporting organisations on notice that repetitive heading of a soccer ball may lead to cognitive disfunction, and to the extrapolated argument that cognitive harm may be caused through repeated head trauma from any source.

5.7.2 Nathan Gibbs (1994)

Dr Nathan Gibbs — ex-premier and international representative rugby league player — published an article in *Sports Medicine*, that raised concern about *dementia pugilistica* due to repeated head trauma in contact sports 'such as rugby league'.[65]

In a passage worth recording in full, Gibbs placed the issue of 'punch drunk syndrome' before the medical fraternity and administrators of sports, albeit somewhat tenuously:

> The belief that there may be cumulative effects from repeated concussions in rugby league with resultant chronic brain damage is based on the evidence from boxing. However, head injury in boxing is much more common than in rugby league, as the aim of the former sport is to render your opponent unconscious and helpless through successive blows to the head. Numerous studies have shown the development of dementia pugilistica, or chronic boxers' encephalopathy, as a result of repeated head trauma from boxing. *Further research in contact sports such as rugby league is required to see if chronic brain changes can also occur*.[66]

Although *Sports Medicine* is not an Australian publication, Gibbs's reputation as an Australian sports medico is likely to have attracted the interest of local SGBs and their medical associates, which incidentally highlights the importance of international medical journals in informing of emerging risks of potential harm. However, Gibbs's qualification that further research was needed[67] may be viewed by a court as impeding an affirmative belief of a foreseeable risk of CTE. Having said that, the possibility of harm had been raised, and the standard of care[68] as described in *H v Royal Alexandra Hospital*[69] requires a warning to be issued where there 'might' be a real risk of serious harm.

5.7.3 Australian Law Reform Commission (2003)

In 2003 the Australian Law Reform Commission (ALRC) released a report concerned with certain aspects of the human genome,[70] which included a chapter titled 'Sport', a section of

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[65] Nathan Gibbs, 'Common Rugby League Injuries' (1994) 18(6) *Sports Medicine* 438, 438, discussed in more detail in Chapter 4: 'CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care'.

[66] Ibid 445. Emphasis added.

[67] Ibid.

[68] Discussed fully in Chapter 4: 'CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care'.

[69] [1990] 1 Med LR 297.

[70] ALRC, *Essentially Yours: The Protection of Human Genetic Information in Australia* (Report 96, 2003).

which considered 'Screening for predisposition to injury'.[71] The section included a quote from Jordan, who suggested screening athletes for genetic susceptibility to harm for the purpose of providing physicians with the opportunity to advise on potential risks, and to furnish those at some apparent risk with information that may guide them take up less risky sports or to use specialised safety equipment to mitigate risk.[72]

With respect to CTE, the ALRC report is at best uncertain. As a single reference it is unlikely to reveal foreseeability of harm to the level of a 'real risk'. This is particularly so given the report's comment that the use of genetic information to determine an individual's predisposition to sports injuries was 'still largely experimental'.[73] However, in combination with other publications, the report could, at least arguably, be expected to prompt a reasonable SGB to inquire into the potential risk of CTE in contact sport. Greenhow has suggested that from 2003 the ALRC report was 'worthy of further examination' for the purpose of initiating 'policy formation in addressing genetic predisposition to brain injury in sport'.[74]

The ALRC report, published in 2003, does little, however, to assist the litigatory prospects of athletes who endured RHT prior to that date — in effect, the majority of Australian athletes who now claim to be suffering the symptoms of CTE.

5.7.4 The combined significance of the Australian studies: Are they sufficient on their own?

Gibbs's article[75] is damaging to the proposition that an SGB did not, and could not, know that playing contact sport presented a risk of CTE, possible or otherwise. The standard to determine whether a warning should be issued, as noted several times, does not require scientific certainty or indeed certainty. This, coupled with the obligation to continually monitor for emerging risks of potential harm, adds to the difficulty of an SGB mounting a viable defence.

The NHMR[76] and ALRC[77] reports suggest a possibility of permanent or progressive cognitive deterioration related to CTE. Again, however, the language is non-committal. And yet, the combination of information from both sources, especially in conjunction with Gibbs's

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71 Ibid vol 2 chapter 38 'Sport', 964.

72 Ibid, referring to BD Jordan, 'Genetic Susceptibility to Brain Injury in Sports: A Role for Genetic Testing in Athletes?' (1998) 26(2) *The Physician and Sportsmedicine* 25.

73 Ibid.

74 A Greenhow in 'Mitigating the Concussion Crunch: League Governance, Passive Actors and Regulators of Risk' (2021) 14(1) *Australian and New Zealand Sports Law* 36, 53. Greenhow did not, however, offer any detailed reasoning as to what form the examination should take of the policy that could apply.

75 Nathan Gibbs, 'Common Rugby League Injuries' (1994) 18(6) *Sports Medicine* 438.

76 NHMRC, *Football Injuries of the Head and Neck* (Report, 1994).

77 ALRC, *Essentially Yours: The Protection of Human Genetic Information in Australia* (Report 96, 2003).

article, could arguably be sufficient to have compelled the issuance of warnings and other ameliorating protocols by SGBs.

Against this is the possibility that little attention was paid to these Australian studies during the pre-2010 era. As evidence of this possibility, consider a 2006 article by Hoskins in the Australian-based *Journal of Science and Medicine in Sport*.[78] Although noting that concussion is the second most prominent injury in rugby league, and that '[c]oncerns have been raised regarding the number of reported concussions in rugby league', the article makes no mention of the long-term consequences of RHT in the form of CTE. Furthermore, although it was printed a year after Dr Omalu et al's 2005 article, it makes no reference to the content of that study. Despite including a note of concern that those experiencing multiple brain injuries could suffer catastrophic or fatal consequences 'due to a variety of mechanisms including second impact syndrome',[79] Hoskins did not make the leap to the probability of CTE in Australian contact sport.

5.8 Extrapolating from American football

American football, a 'global sport' for decades in the form of the NFL, has the closest resemblance to the contact sports of rugby league, rugby, AFL and, to a lesser degree, soccer and hockey. For a diligent sporting organisation or medico associated with athletes, the similarities are arguably sufficient to raise a reasonable concern that the long-term cognitive harm evident in American football is visited upon players of other contact sports. With training regimens, fitness programs, injury types and a commercial orientation common to other professional contact sports, it would seem unlikely that knowledge of CTE-like harm in NFL would have escaped the notice of administrators, coaches, and medicos in non-NFL sports in Australia.

Although the NFL players' concussion litigation[80] was conducted several years after the publication of Omalu et al's article 'Chronic Traumatic Encephalopathy in a National Football League Player',[81] the litigation itself is of limited utility to athletes harmed prior to 2005. Nonetheless, several studies, inquiries and media reports dating back decades were listed within the NFL players' complaint[82] and are material to a claim by Australian athletes that information on CTE-like harm was available and ought to have been investigated by their SGBs. In fact, these studies are of such gravity that any reasonable Australian SGB, irrespective of code, ought to have been 'on notice' and duty-bound to ameliorate potential harm. For

78 W Hoskins et al, 'Injury in Rugby League' (2006) 9 *Journal of Science and Medicine in Sport* 46.

79 Ibid 51.

80 *In Re National Football League Players' Concussion Injury Litigation*, No 2:12-md-02323-AB, MDL No 2323.

81 Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

82 *In Re National Football League Players' Concussion Injury Litigation*, No 2:12-md-02323-AB, MDL No 2323, Plaintiffs' Amended Master Administrative Long-Form Complaint; District Court of the Eastern District of Pennsylvania.

example, Mike Webster — 'the first documented case' of CTE in American football, as reported by Omalu et al in 2005[83] — filed a request with the NFL in 1999 for 'complete disability benefits', supplying extensive medical reports that he 'suffered from "traumatic or punch drunk encephalopathy [brain disease]" sustained from playing football that left [him] totally and permanently disabled as of 1991'.[84] The NFL's six-member Disability Board[85] denied the request, despite the NFL's own physician determining that 'Webster was mentally "completely and totally disabled as of the date of his retirement and was certainly disabled when he stopped playing football sometime in 1990"'.[86]

A 1978 report in the *Journal of the American Medical Association* found that more deaths occurred in American football than in all other competitive US sports combined.[87] Should Australian SGBs and their associated medicos have discerned, from knowledge of fatalities in American football, a risk of long-term cognitive harm in all sports where head contact is common? Two opposing possibilities are proposed: (1) similar to boxing, fatalities in other contact sports are rare, such that no automatic parallel springs to mind; or (2) evidence of death by head trauma in American football should have alerted the prudent medico to the possibility of cognitive harm falling short of death in contact sports with similar playing conditions to American football. Of course, that harm might result is a prompt to a reasonable governing body to initiate inquiries.

5.9 Conclusion

A defendant can only be liable in negligence for failing to warn of a risk of harm that 'ought reasonably to have been possessed'.[88] It must therefore be shown that information regarding the risk of CTE-like harm was known, or ought to have been known by a reasonable SGB.

Two questions are relevant to this thesis. Was information on the risk of CTE reasonably accessible to a defendant SGB in the years prior to CTE being declared an obvious risk of playing contact sport, estimated here to be around 2010? And was there information

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[83] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128, 129.

[84] *In Re National Football League Players' Concussion Injury Litigation*, No 2:12-md-02323-AB, MDL No 2323, Plaintiffs' Amended Master Administrative Long-Form Complaint, [132]; District Court of the Eastern District of Pennsylvania.

[85] The Disability Board, which is made up of three NFL player union representatives and three NFL representatives, determines whether a player is entitled to a pension under the NFL Player Retirement Plan, following the onset of a disability.

[86] *In Re National Football League Players' Concussion Injury Litigation*, No 2:12-md-02323-AB, MDL No 2323, Plaintiffs' Amended Master Administrative Long-Form Complaint, [133]; District Court of the Eastern District of Pennsylvania.

[87] WR Barclay, 'Equestrian Sports' (1978) 240(17) *Journal of the American Medical Association* 1892.

[88] *Roe v Minister of Health* [1954] 2 QB 66, 92.

downplaying the risk of CTE to such an extent that an SGB would be excused in law from not warning of CTE because it was not a foreseeable risk of playing contact sport.

This chapter presented factual evidence of scientific studies extending over several decades prior to 2010 linking CTE-like harm with RHT in the playing of contact sport. From these studies it was argued that an inference can be drawn that the SGBs of major contact sport in Australia 'ought to' have known of the risk of CTE. From accessing this information, the thesis argues that the SGBs were duty-bound to warn their premier players of that risk. The bulk of information on the risk of CTE is that flowing to an SGB is through its associated medical practitioners and, as argued in this thesis, requires a dutiful SGB to establish a system by which that flow of information is readily conveyed.

With respect to the question of accessible knowledge, several key points are prominent.

Dr Harrison Martland's assessment of CTE (*dementia pugilistica*), its cause, symptoms and sequelae[89] remain remarkably accurate and are argued along with other studies to have added to the body revealing the risk of CTE in playing contact sport in Australia.

The dozens of boxing studies throughout the twentieth century and into the twenty-first century are argued to have provided a further link between RHT and CTE although, as noted, the extreme violence of the pugilistic sports may more readily be seen as no more than a 'feeble ray of light', to use the words of Lord Cairns, that combine with other evidence to establish a factual inference.[90]

The medical cause of CTE-like harm is repetitive concussive head trauma. To know of the risk of CTE as a medical condition is to know of the risk of CTE in playing contact sport, and as argued provided a further means by which information of the risk of CTE was available to SGBs in Australia.

From the 1970s onwards, scientific articles informed of the risk of CTE in the playing of several contact sports common to Australia including football, rugby, hockey, baseball, and basketball. In these studies the degree of extrapolation from one sport to another was minor or non-existent.

Almost all studies during the twentieth century on CTE-like harm were published in medical journals originating in the United States. Without these international sources it would be difficult to successfully claim in law that an SGB ought to have known and warned of CTE. Nonetheless, Australian studies from the 1990s and early 2000s, such as the NHMRC report[91] and Dr Nathan Gibbs's article,[92] raised concerns about long-term cognitive damage from head

[89] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103

[90] *Belhaven and Stenton Peerage* (1875) 1 App Cas 278, 279.

[91] NHMRC, *Football Injuries of the Head and Neck* (Report, 1994).

[92] Nathan Gibbs, 'Common Rugby League Injuries' (1994) 18(6) *Sports Medicine* 438.

injuries in sports. In 2003 the ALRC released a report concerned with CTE and the human genome.[93] Although these Australian reports were often non-committal as to a link between in playing contact sport and CTE, notification was sufficient to have prompted a reasonable SGB to at least initiate, through its medical associates, further inquiries into the risk posed to its players by RHT. In conjunction with the international studies, the obligation to inquire and warn is argued as sustainable in law.

Similarities between American football and other contact sports suggest that knowledge of CTE-like harm in the NFL should have raised concerns in Australian sports. Although reasonably argued as different from each other, the commonality of RHT in American football and the Australian contact sports is sufficient to have alerted a reasonable Australian SGB to the risk of CTE.

[93] ALRC, *Essentially Yours: The Protection of Human Genetic Information in Australia* (Report 96, 2003).

Chapter 6:
Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of 'Individual Agency'

6.1 Introduction

The previous chapter, Chapter 5, concerned whether information on the risk of CTE was available and accessible to Australian SGBs in the period before CTE could be deemed an obvious risk of contact sport. Chapter 5 is supplemented by Appendix 2, which considered commentary downplaying the risk of CTE in contact sport and the extent to which, if any, 'CTE denialism' acted to excuse an SGB from failing to informing itself of the risk of CTE and to warn its premier players of that risk.

Chapter 6 concerns the challenges a living plaintiff athlete faces in proving on the balance of probabilities that he or she is, in fact, suffering from CTE. A principal challenge faced by living athletes is that CTE is only definitively diagnosable at autopsy. As such, a living athlete must rely on indirect evidence which is inferred through symptoms, markers, behavioural manifestations and other indicators and is therefore less certain. This chapter evaluates *in vivo* methodologies and considers whether these, either singularly or in combination, are sufficient to establish actual harm in the form of CTE on the balance or probabilities.

The next chapter, Chapter 7, involves the question of factual causation due to the SGB's failure to warn its premier players of the risk of CTE in playing contact sport.

To establish negligence by a failure to warn of a material risk of harm a plaintiff must prove that the breach of duty manifested into actual physical harm, or 'damage'.[1] Proof is provided through factual evidence which, should direct evidence not be available, is determined through inference from facts or circumstantial evidence.[2] On the balance of probabilities, 'the burden lies on the plaintiff to prove injury or loss for which he seeks damages'.[3]

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1 See Chapter 4: 'CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care'.

2 *Luxton v Vines* (1952) 85 CLR 352.

3 *Todorovic v Waller* (1981) 150 CLR 402, 412 (Gibbs and Wilson JJ). The terms 'actual harm', 'actual physical harm' and 'damage' are all generally used to refer to physical harm.

Although at law the factual cause of the plaintiff athlete's harm is a failure to warn of the risk of CTE that induced the athlete to participate in the SGB's contact sport, the plaintiff bears the evidentiary burden of proving that he or she is, as a factual matter, suffering from symptoms of CTE rather than a different neurodegenerative disease. The connection in law between the failure to warn and physical harm is not the 'impairment of the … right to choose' or the 'exposure … to an undisclosed risk', but rather 'the occurrence and consequences of physical injury'.[4] A claim of failure to warn is unproductive without proof that the breach of duty resulted in physical harm to the plaintiff — an outcome often referred to as 'individual agency'.[5]

In many cases — indeed, in most failure to warn actions — the question of sustained damage is clear and flows naturally from the failure to warn (breach of duty) and, thereby, is not the subject of extensive judicial discussion. For example, in *Rogers v Whitaker*, damage to the plaintiff's eye was a consequence of the operation performed by her surgeon, to which she would not have consented had she been properly warned.[6] However, in cases where the latency between the stimuli causing harm and the harm itself is extreme, the question is less clear and will require proof that the defendant is in fact suffering from harm claimed to have been caused by the defendant's breach.[7]

Proof that an individual suffers from CTE is elementally different to proving that the sport the individual played is a cause of CTE.[8] As Stapleton opined, even if a medium is capable of causing a particular type of harm ('generic capacity'),[9] the plaintiff must still establish 'that it did so in this individual case (which we might call "individual agency")'.[10] In context, even though a plaintiff was exposed to RHT while participating in the SGB's contact sport, he or she must nonetheless prove that the symptoms displayed are those of CTE rather than a different cognitive impairment.

In addition, given the latency with which CTE develops after exposure to RHT, years may pass before the build-up of (p)tau causes 'measurable' symptoms marking the beginning of compensable damage. Handley JA explained in *Scarcella v Lettice* that

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4 *Wallace v Kam* (2013) 250 CLR 375, [9].

5 See Jane Stapleton, 'Reflections on Common Sense Causation in Australia' in S Degeling and J Edelman (eds), *Torts in Commercial Law* (Thompson Reuters Australia, 2011) 343–4.

6 *Rogers v Whitaker* (1992) 175 CLR 479.

7 See Jane Stapleton, 'Reflections on Common Sense Causation in Australia' in S Degeling and J Edelman (eds), *Torts in Commercial Law* (Thompson Reuters Australia, 2011) 343–4.

8 The use of the word 'sport' as a cause of CTE in this thesis refers, within context, to head trauma in playing a contact sport.

9 Generic capacity is considered in Chapter 8: 'The Question of "Generic Capacity": Does the SGB's Contact Sport Cause CTE?'

10 Jane Stapleton, 'Factual Causation' (2010) 38(3) *Federal Law Review* 467, 478–9.

> [a] cause of action is not complete until the plaintiff first suffers actual loss or damage. Damage which is prospective or contingent does not qualify as actual damage for this purpose. In order for the plaintiff's cause of action to be complete the plaintiff's actual damage must be 'measurable', or … the damage must be 'beyond what can be regarded as negligible'.[11]

This chapter proposes that methods of diagnosis or detection are sufficiently developed to allow a living plaintiff athlete to establish on the balance of probabilities that he or she is suffering actual harm in the form of CTE. Two broad forms of diagnostic methodologies are examined for this purpose: (1) a methodology known as 'traumatic encephalopathy syndrome' (TES), which is based on the application of multiple criteria to reach a clinical diagnosis assessment;[12] and (2) a collection of methods that emphasise medical imaging and biofluid markers along with clinical presentations of CTE as preferred means of diagnosis.[13]

Whichever methodology is applied, either singularly or in combination, evidence that an individual is, or is not, afflicted with CTE *in vivo* is provided through expert medical opinion, which, as discussed below,[14] is likely to involve expert disagreement that will ultimately be the subject of judicial resolution and explanation.[15]

This chapter is only collaterally concerned with proof that a *deceased* athlete was afflicted with CTE — proof that has the advantage of certainty upon histological examination of brain cells at autopsy to thereby avoid legal debate.

6.1.1 This chapter proceeds as follows

Section 6.2: Of law and fact: Two broad problems of proof

A living plaintiff athlete is faced with two broad problems in proving to the legal standard that symptoms suffered are those of CTE: Because CTE is only definitively diagnosable at autopsy, the athlete must rely on factual and circumstantial evidence to infer that the symptoms displayed are those of CTE.[16] And, because the initial physical changes in the brain associated with CTE are not usually perceived of the disease may not be discernible or measurable — a base requirement to establish harm recognised in law and to secure compensation.

Section 6.3: At what stage is CTE compensable at law?

CTE is a disease that progresses in stages, beginning with the release of tau from axons into the brain and leading to increasingly debilitating sequelae. This section considers the critical

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[11] (2000) 51 NSWLR 302, 306, referring to the judgment of Lord Reid in the personal injuries case *Cartledge v E Jopling & Sons Ltd* [1963] 1 All ER 341.

[12] See **[6.7]**.

[13] See **[6.8]**.

[14] See **[6.5]–[6.6]**.

[15] See, eg, *Smith v Southampton University Hospital NHS Trust* [2007] EWCA 387.

[16] *Holloway v McFeeters* (1956) 94 CLR 470.

question of when the symptoms and indicators of CTE in a living plaintiff progress to a point where damage, as recognised in law, is measurable and thereby compensable.

Section 6.4: Diagnosis of CTE post-mortem and *in vivo*

This section examines the fundamental distinction that while CTE is definitively diagnosable through histological examination at autopsy at death, a living athlete must rely on an inference of CTE through symptoms, markers and other indicators that collectively provide proof of affliction on the balance of probabilities.

Section 6.5: The standard of proof and inference of CTE *in vivo*

This section emphasises that a plaintiff athlete must prove that the symptoms he or she experiences are those of CTE. In essence, the question is whether a living plaintiff athlete is suffering symptoms of CTE or a different neurodegenerative malady. Proof, it is also emphasised, is on the balance of probabilities, and is not founded on certainty or to an absolute scientific standard. It is the task of the court to determine 'legal causation'. This crucial distinction runs through previous chapters of this thesis and is a key to countering proposals in commentary that downgrade or deny a causal connection between RHT in playing contact sport and CTE.

Section 6.6: CTE as a unique disease: Fact and inference

An athlete whose malady is not distinguishable as CTE may be excluded from recovery of damages. For a living athlete — who is obviously unable to rely on direct, definitive autopsy evidence — proof of CTE is a matter of inference drawn from symptoms, studies and other indicators, either independently or combined. This section refamiliarises briefly with studies documenting CTE as a distinct neuropathology despite sharing several clinical features with other neurodegenerative disorders.

The section also introduces the diagnostic methodologies that are discussed further below. These methodologies, as discussed, have been divided into two broad types by Mavroudis et al.[17]

Section 6.7: Traumatic encephalopathy syndrome (TES) and inference of harm in the form of CTE

Traumatic encephalopathy syndrome (TES) utilises various recognised indicator and medical methods of CTE to make an *in vivo* clinical diagnosis of CTE. This section explores TES in detail to enable an assessment of its usefulness in inferring to the legal standard of proof — perhaps in combination with other methodologies — whether a plaintiff athlete is afflicted with CTE.

[17] I Mavroudis et al, 'A Review of the Most Recent Clinical and Neuropathological Criteria for Chronic Traumatic Encephalopathy' (2023) 11(12) *Healthcare* 1689.

Section 6.8: Other methodologies in diagnosing CTE *in vivo*

While TES has been utilised as a diagnostic methodology, other credible methods have also been proposed in medical literature. Three separate studies are reviewed in this section. On the basis that an inference may be formed through 'strands in a cable' rather than 'links in a chain',[18] each study has potential to advance an athlete's claim of suffering the symptoms of CTE.

Section 6.9: Two notable core indicia of CTE

To emphasise the unique nature of CTE as a neurodegenerative disease, two indicators that are confined largely to contact sport athletes who have contracted CTE are considered and highlighted in this section. These are an 'athlete history of repetitive head trauma experienced in playing contact sport' and 'behavioural symptoms and indicators'. These indicators are common in cases of CTE across time — first seen in Dr Martland's 1928 study,[19] noted by Dr Omalu et al in 2005,[20] and again referred to in the coroner's report into the death of AFL player Danny Frawley 2019[21] — and thereby testify to the long provenance of CTE and further illustrate the usefulness of referring to the historical record.

Section 6.10: The Bradford Hill criteria

This brief section highlights that the Bradford Hill methodology — referred to in Chapter 8 as a 'generic capacity' test in establishing CTE as a disease caused by head trauma in playing contact sport in general — benefits an individual plaintiff athlete whose playing experience aligns with the criteria used by Bradford Hill to establish a causal relationship.

Section 6.11: Application: Multiple *in vivo* indicators to infer CTE

This section applies several *in vivo* tests for CTE in proxy to the case of AFL player Danny Frawley, who was confirmed at autopsy as afflicted with CTE. The facts and circumstances of Frawley's playing life and the symptoms he is recorded as suffering are used to gauge the accuracy of the *in vivo* tests against a diagnosed CTE sufferer. Although relatively few indicators of CTE are publicly available in Frawley's case, the argument is made that the application nonetheless reveals a high level of reliability in discerning CTE on the balance of probabilities.

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[18] *Shepherd v The Queen* (1990) 170 CLR 573, 579, referring to *Wigmore on Evidence* (3rd ed, 1981) vol 9, 412–44.

[19] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103, 1103.

[20] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[21] In the Coroners Court of Victoria at Melbourne, Finding into Death, Daniel Frawley, Court Reference: COR 2019 4895, 23 February 2021, [93(d)] per Coroner Paresa Spanos <www.coronerscourt.vic.gov.au/sites/default/files/2021-02/Finding%20D%20Frawley.pdf>.

Section 6.12: Conclusion

This section proposes that TES and other means of reaching a clinical diagnosis of CTE, used either singularly or in combination, are sufficient to prove on the balance of probabilities that an individual plaintiff athlete is suffering from CTE. It is noted that the degree of effectiveness will depend on the availability of adequate records of an athlete's playing history and the symptoms suffered.

6.2 Of law and fact: Two broad problems of proof

In law, whether a person suffers personal injury or damage to his or her body through the negligence of another is a question of fact.[22] CTE is a type of personal injury or damage. A living plaintiff athlete who claims to be suffering from CTE confronts two broad problems in proving in law that his or her symptoms are caused by CTE: (1) because CTE is only definitively diagnosable at autopsy, the athlete must rely on factual and circumstantial evidence to infer that the symptoms displayed are those of CTE;[23] and (2) because the physical changes associated with CTE usually begin imperceptibly, the early manifestations of the disease may not be discernible or measurable — a base requirement to establish harm recognised in law and to secure compensation.

The chapter is not concerned with causation of harm through an SGB's failure to warn, which relates to whether, if warned, the plaintiff would have taken ameliorating steps to avoid harm in the form of CTE, such as retirement.[24] Rather, this chapter is about the means of proving whether an individual plaintiff athlete is afflicted with CTE.

6.2.1 Doubts expressed as to CTE as a 'real' disease

A living plaintiff athlete must be able to prove to the satisfaction of the court that the symptoms suffered are those of CTE. Doubts as to whether CTE is a 'real' disease, or whether, if 'real', it is caused in the playing of contact sport, may jeopardise the athlete's capacity to prove affliction to the requisite standard of probability. The impact, or supposed impact, of contrary views expressed, in particular, by the CISG Consensus Statements and neurologist Dr McCrory are discussed extensively in Appendix 2, and may be referenced in relation to this chapter.[25] This short sub-section notes that questions regarding possible alternative causes of symptoms said to be CTE have been suggested since Dr Omalu et al's 2005 publication;[26] and that these

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22 *Cartledge v E Jopling & Sons Ltd* [1963] 1 All ER 341.

23 *Holloway v McFeeters* (1956) 94 CLR 470.

24 This is discussed in Chapter 7: 'Factual Causation of Harm: Failure to Warn and the Athlete's Response Had a Warning Been Given'. See also *Wallace v Kam* (2013) 250 CLR 375.

25 Appendix 2: 'CTE and "Negativing Influences": Do They Determine Liability?'

26 Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

questions run counter to the athlete plaintiff's claim that he or she is suffering from CTE and must be considered as part of the matrix of facts in a determination of 'individual agency'.

As far back as Dr Bennet Omalu et al's first reports of CTE in a National Football League (NFL) footballer in the journal *Neurosurgery* in 2005 and 2006,[27] uncertainty had been expressed as to whether the symptoms reported as CTE were in fact those of diseases other than CTE. Various doctors gave their views in the 'comments' sections beneath Omalu et al's articles. For example, a Dr Guskiewicz, although recognising the importance of the information, pointed out that 'the case study by itself cannot confirm a causal link between professional football and chronic neurodegenerative diseases such as CTE'.[28] Dr Valadka said that 'the putative association between suspected repetitive mild traumatic brain injury in professional football players and histological findings consistent with chronic neurodegenerative processes is, at the very least, questionable'.[29] Dr Bailes expressed the view that

> [i]t is uncertain whether or not these two case reports, unprecedented for such athletes, represent random findings or if there is a posttraumatic state which is akin to, but dissimilar enough not to satisfy all the classic neuropathological findings of, dementia pugilistica.[30]

Dr Park said of the human subject of Omalu's 2006 autopsy that '[t]he contribution of anticoagulant rodenticide and ethylene glycol ingestion to his demise is unclear', given that a year before his retirement from football 'he attempted suicide by the ingestion of rat poison and cold medications'.[31] Dr Smith thought the report was 'in keeping with traumatic encephalopathy', but noted that 'it is not possible to exclude a coincidental association of a psychiatric disorder in a professional NFL player'.[32]

That several cognitive diseases share some common symptoms may confound a diagnosis of CTE. For example, a sufferer of Alzheimer's disease may display symptoms of parkinsonism, which may also manifest in the latter stages of CTE. Becker noted that '[c]oncussion and other traumatic brain injuries (TBIs) and Alzheimer's disease (AD) share many overlapping pathologies and possible clinical links'.[33] In a 2019 media report Dr Martin Raftery, World Rugby's Chief Medical Officer, questioned the validity of a CTE diagnosis in

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27 Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128; Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player: Part Two' (2006) 59(5) *Neurosurgery* 1086.

28 Kevin Guskiewicz, 'Comments' (2005) 57(1) *Neurosurgery* 128, 134.

29 Alex Valadka, ibid 133.

30 Julian Bailes, 'Comments' (2006) 59(5) *Neurosurgery* 1086, 1093.

31 Min Park, ibid 1093.

32 Colin Smith, ibid 1093.

33 Robert Becker, 'Does Traumatic Brain Injury Hold the Key to the Alzheimer's Disease Puzzle?' (2018) 14 *Alzheimer's & Dementia* 431, 431.

two National Rugby League (NRL) players based on several medical reports that found 'the current pathological findings of CTE mimic other neurodegenerative diseases, as well as ageing, meaning the diagnosis [of CTE] is doubtful'.[34] The media report referred to a 2015 article in *Neurosurgery* that stated:

> There are many confounding variables … that can account for brain tau deposition, including genetic mutations, drugs, normal aging, environmental factors, post-mortem brain processing, and toxins … It is likely that many of the cases with neuropathological findings represent the normal ageing process ...[35]

These briefly mentioned comments and reports reveal the challenge that living athletes face in proving that the symptoms they suffer are manifestations of CTE. The athlete must nonetheless prove actual harm in the form of CTE.[36] This chapter argues that while a histological examination of brain tissue at autopsy is the 'best' form of diagnosis, medical and scientific techniques have advanced over recent years — indeed, recent months — to the point where proof of CTE *in vivo* may be inferred at law on the balance of probabilities. Unlike the absoluteness expected of scientific proof, an inference to the legal standard 'does not require certainty'; just that it is 'more probable'.[37]

6.3 At what stage is CTE compensable at law?

Proof that a plaintiff suffers measurable harm (damage) has been described as 'the gist of an action in negligence'.[38] In the progression to CTE a critical threshold is reached when the body's repair mechanisms are no longer 'able to keep pace' with the accumulation of head trauma and tau begins to be released from the axon walls of neurones.[39] This initial release is clearly a change in a sufferer's body — a minute contributor in the progression to serious disease — yet it occurs without detectable symptoms. Then 'slowly over decades … widespread regions of the neocortex, medial temporal lobe, diencephalon, basal ganglia, brainstem and spinal cord' are affected.[40] Cognitive disfunction emerges, increasingly severe symptoms follow, and then, ultimately, death.

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[34] Roy Masters, 'World Rugby's Medical Chief Doubts Breakthrough CTE Findings', *Sydney Morning Herald* (Sydney, 30 June 2019.

[35] Gavin Davis et al, 'Neurodegeneration and Sport' (2015) 76(6) *Neurosurgery* 655, 655.

[36] See, eg, *Civil Liability Act 2002* (NSW) s 11, which states that '"injury" means personal injury and includes the following — … (b) impairment of a person's physical or mental condition, (c) disease'.

[37] *Tabet v Gett* (2010) 240 CLR 537, [145].

[38] Ibid [23] (Gummow ACJ).

[39] ML Lipton et al, 'Soccer Heading is Associated with White Matter Microstructural and Cognitive Abnormalities' (2013) 268(3) *Neuroradiology* 850, 854.

[40] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43, 60.

The gradual movement towards symptoms and disability raises the issue of whether harm in the form of CTE is to be recognised by a court at time of the release of damaging tau into the brain of a sufferer. Or should it be recognised at some other point, such as with the onset of symptoms? What of symptoms that are subtle or barely discernible, or an athlete's sense that, having been concussed multiple times, 'I must have contracted CTE'? More generally what is the 'personal harm' for which damages are payable?

In the legal assessment of harm in the form of CTE, several cases involving industrial lung disease offer guidance. In *Rothwell v Chemical & Insulating Co*[41] the claimant, who had been exposed to asbestos at work, showed pleural plaques around his lungs when x-rayed. Such plaques do not 'in themselves threaten or lead to other asbestos-induced conditions and were not a necessary pre-condition to such conditions but they evidenced exposure to asbestos'.[42] And although causing 'fibrous thickening of the pleural membrane', except in 'very exceptional circumstances, they cause no symptoms'.[43] With no evident disease, the claimant argued psychiatric injury in the form of anxiety after being notified of his x-ray results. On appeal before the House of Lords, all judgments affirmed that detectable harm is essential to sustaining an action in negligence.

Lord Hoffman said:

> Proof of damage is an essential element in a claim in negligence and in my opinion the symptomless plaques are not compensatable damage. … The risk of the future disease is not actionable and neither is a psychiatric illness caused by contemplation of that risk.
>
> … [A] claim in tort based on negligence is incomplete without proof of damage. Damage in this sense is an abstract concept of being worse off, physically or economically, so that compensation is an appropriate remedy. It does not mean simply a physical change, which is consistent … with being neutral, having no perceptible effect upon one's health or capability.[44]

Lord Hope said that the physical effects of the pleural plaques 'cannot, in any normal sense of the word, be described as harmful' and that 'the use of … descriptions [such as 'injury' or 'disease'] does not address the question of law, which is whether a physical change of this kind is actionable. There must be real damage, as distinct from damage which is purely minimal.'[45] And in the words of Lord Scott: 'Pleural plaques are characteristic of asbestos exposure but are in themselves asymptomatic. … They cause neither impairment of lung function nor disablement.'[46]

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41 [2006] 4 All ER 1161.

42 Ibid 1161.

43 *Johnston v NEI International Combustion Ltd; Rothwell v Chemical & Insulating Co Ltd* [2007] UKHL 39, [1].

44 Ibid [2], [7].

45 Ibid [38]–[39].

46 Ibid [63].

It is clear that a 'trivial injury' — a *de minimis* physical change to an individual's body — is not 'damage' for the purposes of law. [47] But where, as a question of fact, is the cut-off point between *de minimis* change and compensable harm — or, as expressed by Lord Hoffman: 'How much worse off must one be?'[48]

In *Cartledge v E Jopling & Sons Ltd*[49] the plaintiffs, affected by silicosis, had not displayed symptoms (such as shortness of breath) within the statutory limitation period. Nonetheless, Lord Pearce, in considering the factual question, described a concrete, identifiable symptom as evidencing harm that exceeds the *de minimis*:

> It is a question of fact in each case whether a man has suffered material damage by any physical changes in his body. Evidence that those changes are not felt by him and may never be felt tells in favour of the damage coming within the principle of *de minimis non curat lex*. On the other hand, evidence that in unusual exertion or at the onset of disease he may suffer from his hidden impairment tells in favour of the damage being substantial.[50]

The initial leakage of tau from the axons of brain cells does not produce symptoms of CTE. Moreover, according to McKee et al's four pathological stages of CTE,[51] early symptoms such as headaches, loss of attention or concentration are vague, or may be symptoms of other diseases. Consequently, not until symptoms, or a cluster of symptoms, can be defined as *probably* those of CTE, can harm in the form of CTE be established as a matter of law.

6.4 Diagnosis of CTE post-mortem and *in vivo*

Because CTE is a progressive histological reaction to RHT over many years, most afflicted athletes do not suffer symptoms until their early to late middle age. Symptoms of CTE 'begin focally' and spread 'slowly over decades to involve widespread regions of the neocortex, medial temporal lobe, diencephalon, basal ganglia, brainstem and spinal cord'.[52] The 'clinical course of CTE is slow, progressing at a rate of 11–14 years between pathological stages'.[53] As time passes, a sufferer develops increasingly severe and debilitating symptoms, including aggression, impulsivity, apathy, headaches, cognitive impairment and suicidality, ultimately resulting in death.[54]

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47 Ibid [8].

48 Ibid [8].

49 [1963] 1 All ER 341.

50 Ibid 349.

51 Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43. See Chapter 2: 'The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments'.

52 Ibid.

53 Ibid 61.

54 Ibid 43.

Proof that a plaintiff athlete suffers, or suffered, CTE is provided through two diagnostic methodologies: (1) histological examination of brain tissue post-mortem, which, as noted, provides definitive proof of CTE; or (2) inferential diagnosis through the fact of *in vivo* symptoms, markers, and other indicators, which, as argued, affords proof on the balance of probabilities that an individual is affected by CTE.

6.5 The standard of proof and inference of CTE *in vivo*

A living athlete will, with some certainty, face the burden of proving that the symptoms with which he or she suffers are those of CTE rather than those of other forms of cognitive disease, particularly Alzheimer's disease. An SGB may propose that because CTE is diagnosed *definitively* only at autopsy, any other means of assessment is less than the best available and cannot provide accurate proof. For the living athlete suffering with CTE and seeking compensation, these propositions are of course untenable.

There is a difference, though, between definitive proof and proof at law — a difference advantageous to the plaintiff athlete.

Definitive proof is a scientific standard expressed only where proof is near to absolute: where there is little or no doubt at all. Where substantiation of a scientific fact or process is less than absolute, terms such as 'near certainty', 'probably' and 'theoretically' are usually applied. Physicist Jack Fraser said that the scientific standard of proof 'implies that there is no room for error — that you can be 100% sure that what you have written down on the piece of paper is 100% representative of what you are talking about'.[55]

Whatever its possible advantages, attempts to achieve complete certainty set an unrealistic standard that is unhelpful in promulgating a satisfactory real-world outcome. In any case, the scientific standard is not the legal standard. The legal standard — or *proof at law* — in claims of negligence is not perfection, beyond reasonable doubt, or even comfortable satisfaction, but rather, 'on the balance of probabilities'. Kiefel J remarked in *Tabet v Gett*:

> All that is necessary is that … the more probable inference appearing from the evidence is that a defendant's negligence caused the injury or harm. 'More probable' means no more than that, upon a balance of probabilities, such an inference might reasonably be considered to have some greater degree of likelihood; it does not require certainty. … The purpose of proof at law, unlike science or philosophy, is to apportion legal responsibility.[56]

In the mesothelioma case of *Amaca v Ellis*, the High Court explained:

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[55] Jack Fraser, 'There's No Such Thing as Proof in the Scientific World — There's Only Evidence', *forbes.com* (online, 14 December 2017) <https://www.forbes.com/sites/quora/2017/12/14/theres-no-such-thing-as-proof-in-the-scientific-world-theres-only-evidence>.

[56] (2010) 240 CLR 537, [111].

> The court's response to uncertainty arising from the absence of knowledge must be different from that of the medical practitioner or scientist. The courts cannot respond to a claim that is made by saying that, because science and medicine are not now able to say what caused [the victim's] cancer, the claim is neither allowed nor rejected. …
>
> The central question is whether it had been shown to me more probable than not that exposure to asbestos was a cause of [the victim's] death.[57]

The High Court stated further that

> despite this uncertainty, the courts must, and do, 'reduce to legal certainty [a question] to which no other conclusive answer can be given'. The courts do that by asking whether it is more probable than not that X was a cause of Y.[58]

Dixon J in *Bank of NSW v Commonwealth* remarked similarly:

> There are few, if any, questions of fact that courts cannot undertake to inquire into. In fact it may be said that under the maxim *res iudicata pro veritate accipitur* [a thing adjudicated is received as the truth] courts have an advantage over other seekers after truth. For by their judgment they can reduce to legal certainty questions to which no other conclusive answer can be given.[59]

Legal scholar Eggleston said of the interplay between science and law that '[s]cience has more leisure but also has to accept that its conclusions are true only "as far as they have been tested"'.[60] Adeney opined similarly: 'Though a situation may fall far short of satisfying a scientist that causation is present it may nevertheless satisfy a judge. From that moment on, causation will exist for legal purposes.'[61]

Ultimately the question for the court is not about statistical probabilities but, as said by Spigelman CJ, '[t]he predominant position in Australian case law is that a balance of probabilities test requires a court to reach a level of actual persuasion. This process does not involve a mechanical application of probabilities.'[62]

Proof that a *living* athlete is afflicted with CTE is necessarily established through inference rather than direct and irrefutable scientific evidence. While CTE and other cognitive maladies may cause some common symptoms, the totality of symptoms, biomarkers and scans, along with an extensive history of playing contact sport, are, as argued, sufficient to infer harm in the form of CTE on the balance of probabilities.

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57 (2010) 240 CLR 111, [6].

58 Ibid [70], quoting *Bank of NSW v Commonwealth* (1948) 76 CLR 1, 340.

59 (1848) 76 CLR 1, 340.

60 Richard Eggleston, *Evidence, Proof and Probability* (Weidenfeld and Nicholson, 2nd ed 1983); quoted in E Magnusson, 'Statistical Proof of Causation' in Danuta Mendelson and Ian Freckelton (eds), *Causation in Law and Medicine* (Taylor & Francis Group, 2002) 411.

61 E Adeney, 'The Challenge of Medical Uncertainty: Factual Causation in Anglo-Australian Toxic Tort Litigation' (1993) 19 *Monash University Law Review* 23, 25.

62 *Seltsam Pty Ltd v McGuinness* [2000] NSWCA 29, [136].

6.5.1 Drawing an inference

In vivo proof of actual harm in the form of CTE is not 'direct evidence' capable of establishing 'a clear connection between an event and an outcome'; rather, the provision of proof in such circumstances is 'arrived at via circumstantial evidence and by expert evidence framed in terms of possibility and probability'.[63] Of this form of proof the High Court's comments in *Luxton v Vines* are material:

> [W]here direct proof is not available, it is enough if the circumstances appearing in evidence give rise to a reasonable and definite inference; they must do more than give rise to conflicting inferences of equal degrees of probability so that the choice between them is more a matter of conjecture.[64]

In like vein, Gibbs J in *TNT Management Pty Ltd v Brooks* commented of the standard of proof in negligence that 'you need only circumstances raising a more probable inference in favour of what is alleged'.[65]

The High Court in *Holloway v McFeeters* explained the validity of a test of probable inference to establish proof on the balance of probabilities from facts:

> Inferences from actual facts that are proved are just as much part of the evidence as those facts themselves. In a civil cause 'you need only circumstances raising a more probable inference in favour of what is alleged … they must do more than give rise to conflicting inferences of equal degree of probability so that the choice between them is mere matter of conjecture … All that is necessary is that according to the course of common experience the more probable inference from the circumstances that sufficiently appear by evidence or admission, left unexplained, should be that the injury arose from the defendant's negligence. By more probable is meant no more than that upon a balance of probabilities such an inference might reasonably be considered to have some greater degree of likelihood'.[66]

To illustrate the application in general terms, *Holloway v McFeeters* concerned an apparent 'hit and run' collision that killed the plaintiff's pedestrian husband and for which there were no witnesses and no direct evidence. By considering facts such as the length of skid marks outside the proper lane, weather conditions, street lighting, the position of the body of the deceased and his injuries, and the location of car debris, the High Court majority was able to infer that the defendant drove at excessive speed and failed to keep a proper lookout. Dixon CJ framed the question as one that 'depends upon the sufficiency of circumstantial evidence to discharge the burden of proving that the death of the deceased was occasioned by negligence on the part of the driver of the unidentified car'.[67] His Honour went on to note that

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[63] I Freckelton, 'Epilogue: Dilemmas in Proof of Causation' in Danuta Mendelson and Ian Freckelton (eds), *Causation in Law and Medicine* (Taylor & Francis Group, 2002) 433.

[64] (1952) 85 CLR 352, 358.

[65] *TNT Management Pty Ltd v Brooks* (1979) 53 ALJR 267, 269

[66] *Holloway v McFeeters* (1956) 94 CLR 470, [8] per Dixon CJ, quoting the unanimous judgment of Dixon, Williams, Webb, Fullagar and Kitto JJ in *Bradshaw v McEwans Pty Ltd* (High Court of Australia, 27 April 1951).

[67] (1956) 94 CLR 470, [6].

> [i]n the present case the true cause of the accident is in truth unknown. The state of facts reached by inferences is itself compatible with a number of hypotheses, some of them implying fault on one side, some on the other, some on both sides. Hypotheses of this kind are not inferences. What is required is a basis for some positive inference involving negligence on the part of the driver as a cause of the deceased's death. The inference may be made only as the most probable deduction from the established facts, but it must at least be a deduction which may reasonably be drawn from them.[68]

Below, it is argued that specific and unique symptoms and various medical tests permit a finding of CTE *in vivo* that is both 'reasonable' and not 'speculation'.[69] Worth noting is the correctness of a court in taking a retrospective view of relevant facts in its determination, about which the New South Wales Court of Appeal in *Zabic v Alcan Gove* stated that '[h]indsight is frequently employed when one is endeavouring to ascertain the cause or causes of an injury or damage which does not become manifest until some later time'.[70]

6.5.2 Inference through multiple facts

In *Chamberlain v The Queen*[71] Gibbs CJ and Mason J referred to the standard by which to determine a sustainable inference: '[I]n a civil case the circumstances must raise a more probable inference in favour of what is alleged, and in a criminal case the circumstances must exclude any reasonable hypothesis consistent with innocence.' As seen in *Holloway v McFeeters*, [72] an inference is likely to be drawn from several disparate sources. On facts coalescing into the foundation of a judgment, Lord Cairns in *Belhaven and Stenton Perlerage* stated that

> in dealing with circumstantial evidence, we have to consider the weight which is to be given to the united force of all the circumstances put together. You may have a ray of light so feeble that by itself it will do little to elucidate a dark corner. But, on the other hand, you will have a number of rays, each of them insufficient, but all converging and brought to bear on the same point, and when united, producing a body of illumination which will clear away the darkness which you are endeavouring to dispel.[73]

Legal commentor Freckelton remarked that a proper inference is 'based upon primary facts … and it is the only inference which is reasonably open upon the whole body of primary facts', stating further that 'jurors are entitled to draw an inference of guilt from a combination of facts, none of which viewed alone would support that inference'.[74] A 'body of primary facts' that 'converge' 'or accumulate' is a method of proving actual harm in the form of CTE and, as

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68 Ibid [10].

69 Again, note the living athlete's 'contingency' position of absolute proof upon autopsy examination.

70 (2015) 292 FLR 413, [20].

71 (1984) 153 CLR 521, 536.

72 (1956) 94 CLR 470.

73 (1875) 1 App Cas 278, 279.

74 I Freckelton, 'Epilogue: Dilemmas in Proof of Causation' in Danuta Mendelson and Ian Freckelton (eds), *Causation in Law and Medicine* (Taylor & Francis Group, 2002) 437. Freckelton was referring directly to the criminal standard.

Deane J said in *Chamberlain v The Queen*, '[t]he jury [is] entitled to pay regard to all of them'.[75] As said by Spigelman CJ, '[c]ausation, like any other fact can be established by a process of inference which combines primary facts like "strands in a cable"'.[76]

Below, multiple factual indicators of long-term cognitive damage in athletes who played contact sport, when drawn together, are argued to sustain an inference that the living plaintiff is probably suffering from CTE.

6.6 CTE as a unique disease: Fact and inference

Athletes claiming the failure of their SGBs to warn of the risks of cognitive harm in playing contact sport must prove that CTE is not a disease common to the general population or due to causes other than sport-related head trauma — that CTE is not, for example, a form of Alzheimer's disease or merely a product of advancing age. In short, they must establish that CTE is a disease characterised by specific and identifiable symptoms and indicators. An athlete whose malady is not distinguishable as CTE may be excluded from recovery of damages given that a connection to on-field head trauma is not probable.

As previous chapters and sections of this chapter affirm, CTE is well documented and medically acknowledged as a distinct disease. To refamiliarise, consider three studies in brief form. Neurologist McKee et al, in 2009, found that although CTE 'shares many features of other neurodegenerative disorders, including Alzheimer disease (AD), progressive supranuclear palsy (PSP), postencephalitic parkinsonism, and the amyotrophic lateral sclerosis/Parkinson-dementia complex of Guam (ALS/PDC)', it is 'a neuropathologically distinct progressive tauopathy with a clear environmental etiology'.[77] In a study reported by Bieniek, eight neuropathologists, 'blinded to all clinical and demographic information', individually examined microscopic slides of 25 cases of various forms of tauopathies.[78] Each 'successfully identified CTE pathology [from] amongst 29 cases of various tauopathies and determined that CTE was a distinct disease'.[79] Alosco et al also reported on the clinical differences between the two diseases: 'Although CTE and AD have similar (3R/4seltsamR) tau

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[75] (1984) 153 CLR 521, 627.

[76] *Seltsam Pty Ltd v McGuinness* [2000] NSWCA 29, [91], referring to *Wigmore on Evidence* (3rd ed, 1981) vol 9, 412–44; referred to in *Shepherd v The Queen* (1990) 170 CLR 573 at 579.

[77] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 710.

[78] KF Bieniek, 'The Second NINDS/NIBIB Consensus Meeting to Define Neuropathological Criteria for the Diagnosis of Chronic Traumatic Encephalopathy' (2021) 80(3) *Journal of Neuropathology & Experimental Neurology* 210, 211. The other tau pathologies examined included: Alzheimer's disease (AD), progressive supranuclear palsy (PSP), corticobasal degeneration (CBD), Guamanian parkinsonism dementia complex (GPDC), argyrophilic grain disease (AGD) and primary age-related tauopathy (PART). CTE, as with other tauopathies, concerns the leakage or dislodging of the tau protein from the wall of the axon (neurone cell) which forms clumps leading to a breakdown in transmissions between neurons.

[79] Ibid.

isoforms, there are differences at the molecular level, and … in CTE the ratio of 3R:4R differs across disease stage and between neuronal and glial tau.'[80]

Nonetheless, as with many 'newly discovered' diseases, much is to be learned, and McKee et al's 2015 comment on the 'unknowns' of CTE is still relevant: '[T]he mechanisms underlying the spread of tau and … accumulation … in the brain remain to be defined.'[81] It should be stressed, though, that it is not the disease (CTE) or its physical cause (RHT) that is uncertain, but rather 'the morphologies of tau filaments in CTE and the mechanisms by which brain trauma can lead to their formation [that] are unknown'.[82]

Also instructive is a recognition that diagnoses of *other* tau-related brain disorders are definitive only at autopsy, and that a 'non-definitive' *in vivo* diagnosis of such disorders nonetheless merits extensive medical treatment. Revisiting a statement of Pierre et al: '*Like most* neurodegenerative diseases, CTE can only be diagnosed by post-mortem neuropathologic examination of the brain tissue.'[83] Alosco et al writes: 'Consistent with the clinical diagnosis of *other* neurodegenerative diseases, an accurate diagnosis of CTE during life will require validated biomarkers of the underlying pathophysiology of the disease.'[84]

In addition, a diagnosis of disease is often that of clinical judgment rather than a biological test or medical imaging. When Dr Harrison Martland in 1928 observed boxers with 'a staggering, propulsive gait with the facial characteristics of the parkinsonian syndrome, or a backward swaying of the body, tremors, vertigo and deafness',[85] he had no doubt they suffered from 'punch drunk syndrome', which he described as 'a very definite brain injury due to single or repeated blows on the head or jaw'.[86]

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[80] ML Alosco et al, 'Developing Methods to Detect and Diagnose Chronic Traumatic Encephalopathy during Life: Rationale, Design, and Methodology for the DIAGNOSE CTE Research Project' (2021) 13(1) *Alzheimer's Research & Therapy* 136, 139.

[81] Ann McKee et al, 'The Neuropathology of Chronic Traumatic Encephalopathy' (2015) 25 *Brain Pathology* 350, 361.

[82] Benjamin Falcon, 'Novel Tau Filament Fold in Chronic Traumatic Encephalopathy Encloses Hydrophobic Molecules' (2019) 568 *Nature* 420, 420.

[83] K Pierre et al, 'Chronic Traumatic Encephalopathy: Update on Current Clinical Diagnosis and Management' (2021) 9 *Biomedicines* 415, 417. Emphasis added.

[84] ML Alosco et al, 'Developing Methods to Detect and Diagnose Chronic Traumatic Encephalopathy during Life: Rationale, Design, and Methodology for the DIAGNOSE CTE Research Project' (2021) 13(1) *Alzheimer's Research & Therapy* 136, 138. Emphasis added.

[85] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103, 1103.

[86] Ibid. And see **[6.6.1]** below with regard to the comments of Dr Rowena Mobbs.

6.6.1 Diagnosis and inference of CTE *in vivo*: Proof of affliction through multiple indicators of harm

To begin with a revealing quote by Dr Rowena Mobbs, neurologist and CTE specialist, in a submission to the Senate concussion inquiry:

> I note that doctors have long held the ability to diagnose 'dementia pugilistica' or CTE in boxers purely based on clinical suspicion according to their training, and that CTE is commonly described in neurology textbooks. The claim that CTE cannot be diagnosed in life is a later phenomenon through the lens of modern neuroimaging available for other dementias. Just as we diagnose Alzheimer's disease clinically over time, I am confident in assessing patients as having suspected CTE.[87]

Three matters are salient: CTE has long been diagnosed in boxers through the specialist training of the examining medico; modern neuroimaging has supplanted to some degree the confidence that may rightly be assigned clinical judgment; the author of the submission, presumably through her training and experience, is confident in diagnosing CTE. These observations are somewhat of a benchmark against which to assess the worth of comments downgrading or denying the reality of CTE based on a shortfall or in exactitude in scientific measurement.

CTE presents with a range of clinical symptoms, biomarkers and aberrant behavioural manifestations that may be, and in fact have been, utilised in the diagnosis of CTE *in vivo*. Neuroscientists have conducted medical research using these manifestations to develop methodologies to diagnose or detect CTE. These methodologies, either independently or in conjunction, are argued as a means by which a living athlete may prove on the balance of probabilities that he or she is suffering from harm in the form of CTE.

CTE diagnostic methodologies, according to Mavroudis et al, are divided into two broad types: (1) that known as traumatic encephalopathy syndrome (TES) methodology, which formulates a diagnosis based on the clinical manifestations of CTE; and (2) those methodologies that utilise other means of diagnosis (or in conjunction with TES) through neuroimaging (magnetic resonance imaging (MRI), positron emission tomography (PET) and magnetoencephalography (MEG)) and fluid biomarkers (including increased plasma and exosomal tau levels, and changes in amyloid β40 and β42).[88]

Medical commentators refer to the diagnostic methodologies variously as: the TES methodology, the *in vivo* symptoms of CTE, CTE/TES manifestations, or similar. This chapter will use the terms interchangeably. It should be noted that no organisation possesses the authority to determine that one methodology is necessarily superior. Having said that, as explained below, it is quite evident that TES has been, and continues to be, used for medical

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[87] Rowena Mobbs, Submission No 1 to Senate, Community Affairs References Committee, Parliament of Australia *Concussions and Repeated Head Trauma in Contact Sports* (September 2023) 3.

[88] I Mavroudis et al, 'A Review of the Most Recent Clinical and Neuropathological Criteria for Chronic Traumatic Encephalopathy' (2023) 11(12) *Healthcare* 1689, 2.

diagnostic purposes, a point favouring the acceptance of this method to infer on the balance of probabilities that a plaintiff athlete's symptoms are those of CTE.

In determining harm *in vivo*, consideration of *all* relevant markers and symptoms is arguably more valid than reflecting on merely the singular. Given that the researchers discussed in the following sections are examining for symptoms of the same disease and referring to common studies on CTE, some overlap in the proposed criteria and methodologies can be expected.

6.7 Traumatic encephalopathy syndrome (TES) and inference of harm in the form of CTE

An athlete's traumatic encephalopathy syndrome (TES) profile, as argued in this chapter, provides a means of determining actual harm in the form of CTE *in vivo* on the balance of probabilities and, to a degree, has become accepted in that capacity within some medical circles and used as a basis for treatment. TES is 'the clinical disorder associated with neuropathologically diagnosed [CTE]'[89] and, as such, functions to describe the *in vivo* diagnosis of CTE. That is, because a definitive diagnosis of CTE is only possible at autopsy, TES is a clinical diagnosis based on symptoms and other indicators, not dissimilar in process to the diagnosis of influenza through symptoms although a definitive diagnosis eliminating other possible causes would require pathological assessment. As Mavroudis et al explained, '[c]urrently, the diagnosis of CTE can only be assumed postmortem. Thus, the clinical manifestations associated with CTE are referred to as traumatic encephalopathy syndrome (TES), for which diagnostic multiple sets of criteria can be used.'[90]

In the initial iteration of TES in 2014, Montenigro reviewed more than 200 medical articles in 'world literature' on the clinical manifestations of CTE, the stated purpose being 'to address many of the limitations of the previously published criteria'.[91] The indicators of CTE were distilled to 'research diagnostic criteria' and, where denoting CTE *in vivo*, these are collectively termed the '2014 TES criteria'. Although 'not meant to be used for clinical diagnosis', Montenigro envisaged that the 2014 TES criteria could 'be employed' in 'studies of the underlying causes, risk factors, differential diagnosis, prevention, and treatment of CTE and related disorders'.[92]

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[89] DI Katz et al, 'National Institute of Neurological Disorders and Stroke Consensus Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2021) 96 *Neurology* 848.

[90] I Mavroudis et al, 'A Review of the Most Recent Clinical and Neuropathological Criteria for Chronic Traumatic Encephalopathy' (2023) 11(12) *Healthcare* 1689, 2.

[91] PH Montenigro et al, 'Clinical Subtypes of Chronic Traumatic Encephalopathy: Literature Review and Proposed Research Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2014) 6(5) *Alzheimer's Research & Therapy* 68, 75.

[92] Ibid 69. The term 'traumatic encephalopathy syndrome' in the title was selected 'to describe the clinical presentation of CTE as well as other possible long-term consequences of repetitive head impacts (for example, chronic or progressive axonopathy without tauopathy) but is not meant to include the acute or post-acute manifestations of a single concussion,

The 2014 TES criteria were revisited by Katz and the NINDS expert consensus panel in 2021[93] and used as a 'starting point' and 'organising structure' for the development of the 'NINDS Consensus Diagnostic Criteria for TES' (the 'NINDS TES criteria'). The NINDS TES criteria are, as would be expected, largely the same as the 2014 TES criteria, although with the stated object of improving on the earlier study 'by maintaining adequate sensitivity and increasing the specificity for underlying CTE pathology … and [developing] a criteria structure amenable to future updates'.[94] A main change in the NINDS TES criteria was the exclusion of biomarkers, as these were thought 'not sufficiently mature' by the panel at the time of the study. In contrast, other studies (discussed below) include biomarkers as a criterion for diagnosis.

Katz's adoption and alteration of the 2014 TES criteria do not diminish their previous function as a recognised diagnostic tool; in fact, several studies noted that they fulfilled that purpose.[95]

The NINDS expert consensus panel comprised 20 clinician-scientists across the disciplines of neurology, neuropsychology, neurosurgery, psychiatry, physical medicine and rehabilitation, whose areas of clinical and research expertise encompassed TBI, sports concussion and neurodegenerative disorders. The panel members were given 40 articles on CTE published between 1957 and 2019, which were chosen according to defined inclusion and exclusion criteria. The articles were discussed, summaries of comments from each member were provided anonymously to each other member with a view to forming a consensus on the diagnostic components of TES. In what was described as a 'modified Delphi procedure', members voted on the criteria to be included or excluded, with a threshold of ≥ 80% agreement required for acceptance and with voting limited to four rounds.[96]

post-concussion syndrome, or moderate to severe TBI' (at 76). The word 'syndrome' describes 'the cluster of clinical features that make up this condition [CTE]' (at 76).

[93] DI Katz et al, 'National Institute of Neurological Disorders and Stroke Consensus Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2021) 96 *Neurology* 848. NINDS is the National Institute on Neurological Disorders and Stroke, part of the US National Institutes of Health.

[94] Ibid 850.

[95] See, eg, R Grashow et al, 'Premortem Chronic Traumatic Encephalopathy Diagnoses in Professional Football' (2020) 88 *Annals of Neurology* 106; R Cantu and A Budson, 'Management of Chronic Traumatic Encephalopathy' (2019) 19(10) *Expert Review of Neurotherapeutics* 1015; PS Kim and RG Beran, 'Legal Medicine Implications of a Multidisciplinary Approach to Managing Traumatic Encephalopathy Syndrome in Australia' (2023) 14 *Frontiers in Neurology* 1179319.

[96] DI Katz et al, 'National Institute of Neurological Disorders and Stroke Consensus Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2021) 96 *Neurology* 848, 850. The study was conducted by 'a panel of 20 expert clinician-scientists in neurology, neuropsychology, psychiatry, neurosurgery, and physical medicine and rehabilitation, from 11 academic institutions.'

6.7.1 The NINDS TES criteria

The NINDS TES criteria consist of 'primary criteria' and 'supportive criteria'.

A The primary criteria

The primary criteria are divided into four parts.[97]

(i) Substantial exposure to RHT

This is a 'required criterion'. Exposure is revealed by participation in 'high exposure contact or collision sports',[98] including (but not limited to) boxing, American football, ice hockey, soccer, rugby, professional wrestling, mixed martial arts, and some other sports such as motocross and bull riding. RHT can be with or without clinical symptoms or signs of concussion.

Exposure also involves a time element, which was listed as a minimum of five years for American football of two or more at high school level. Thresholds for other contact sports had not been established, but 'should be a substantial number of years (eg ≥ 5 y) at a level of play involving routine [RHTs]'.[99]

(ii) Cognitive impairment and/or neurobehavioral dysregulation and/or progressive course

This criterion is sub-divided into three parts.

1. *With respect to cognitive impairment*: This is a 'required criterion' and must include a report by self or a clinician. There must be a significant decline from baseline functioning, together with deficits in episodic memory and/or executive functioning (additional domains may be impaired in addition to these), substantiated by impaired performance on formal neuropsychological testing (if available), 'as defined by performance at a level of at least 1.5 SDs below appropriate norms'.[100]

2. *With respect to neurobehavioral dysregulation*: This must include a report by self or a clinician. The neurobehavioral dysregulation must represent a significant change from baseline functioning, with symptoms or observed behaviours that include: poor regulation or control of emotions and/or behaviour, including (but not limited to) explosiveness, impulsivity, rage, violent outbursts, having a short fuse (exceeding what might be described as periodic episodes of minor irritability); or emotional lability (often reported as mood swings).

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97 The criteria are considered in greater detail in the case study of AFL player Danny Frawley at **[6.11.2]** below.

98 DI Katz et al, 'National Institute of Neurological Disorders and Stroke Consensus Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2021) 96 *Neurology* 848, 853.

99 Ibid 851. Military service involving RHTs, such as blast exposure and head banging syndrome, were also noted.

100 Ibid 852.

3. *With respect to progressive course*: There must be evidence of progressive worsening of these clinical features over a period of at least one year in the absence of continued exposure to RHTs or TBIs.

(iii) Not fully accounted for by other disorders

This criterion requires that the pattern of the cognitive deficits is not fully accounted for by other pre-existing, established, or acquired nondegenerative nervous system, medical, or psychiatric disorders and conditions. However, a comorbid diagnosis of another neurodegenerative disease does not exclude a TES diagnosis, but TES may be excluded if, based on clinical judgment, the clinical features and any available biomarkers are fully accounted for by another neurodegenerative disorder. In addition, a comorbid diagnosis of substance use disorder, PTSD, mood or anxiety disorders, or a combination of these can be present, and do not exclude a TES diagnosis, unless they are determined to account for all core clinical features.

(iv) Level of functional dependence/dementia

This criterion requires that the level of functional dependence should be based on the impact of cognitive impairment and/or neurobehavioral dysregulation, and not on physical limitations or medical illness; and further that it should represent a change from baseline functioning. Information is obtained from self-report, an informant and/or clinical records.

Functionality is divided into five levels.[101]

1. *Independent*: Performed at the usual level at household tasks, or social and family roles, and fully independent at activities of basic daily living (ADLs).

2. *Subtle/mild functional limitation*: 'Slightly reduced performance in job, household responsibilities, or family, social and community roles; slight problems in hobbies and intellectual interests … and in some instrumental ADLs [such as paying bills, preparing meals and medication management] ... [F]ully independent in basic ADLs'; for example, (personal hygiene and grooming, toilet hygiene, cleaning oneself, dressing, self-feeding, and functional mobility, such as walking.

3. *Mild dementia*: 'Definite impairment of instrumental ADLs; may be engaged in some home, family, social, and community activities; more difficult activities abandoned; needs cues for some basic ADLs.'

4. *Moderate dementia*: 'Not independent but can be taken to some functions outside the home; only simple chores preserved; very restricted interests; needs assistance with basic ADLs.'

[101] Ibid 864.

5. *Severe dementia*: 'Cannot participate in functions outside the home; no significant function in home; impaired basic ADLs; not independent with self-care; frequently incontinent.'

B The supportive criteria

The supportive criteria are divided into three components. These have insufficient predictive value to qualify as core clinical features but are frequently present in individuals with underlying CTE pathology and, as such, are used in determining the provisional levels of certainty for CTE.

(i) Delayed onset

This refers to a period of stable functioning after RHT exposure ends (for example, at the end of a playing career) before the onset and progression of symptoms, to suggest a history consistent with a degenerative disorder rather than problems associated with TBI or other pre-existing conditions.

(ii) Motor signs

Motor signs include:

- *Parkinsonism*: Symptoms of parkinsonism must suggest a history consistent with a degenerative disorder rather than problems associated with TBI or other pre-existing conditions.
- *Other motor signs*: These include dysarthria, ataxia and imbalance. They should not be more consistent with the clinical features of other neurologic conditions or primary orthopaedic problems.
- *Motor neuron disease*: Symptoms include weakness, dysphagia, other lower motor neuron signs.

(iii) Psychiatric features

The supportive psychiatric features refer to those that have not been accounted for by neurobehavioral dysregulation described in the core clinical features. They include:[102]

- *Anxiety*: 'pervasive worries, excessive fears, agitation, or obsessive or compulsive behaviour (or both); a formal diagnosis of anxiety disorder would meet this criterion but is not necessary.'
- *Apathy*: 'loss of interest in usual activities and loss of motivation or drive.'

[102] Ibid 856.

- *Depression*: 'feeling overly sad, dysphoric, or hopeless, with or without a history of suicidal thoughts or attempts; a formal diagnosis of major depressive disorder or persistent depressive disorder would meet this criterion but is not necessary.'
- *Paranoia*: 'delusional beliefs of suspicion, persecution, or unwarranted jealousy; a formal diagnosis of a psychotic disorder would meet this criterion but is not necessary.'

6.7.2 Provisional levels of certainty for CTE pathology

The primary and supportive criteria of the NINDS TES criteria were utilised to establish four levels of certainly with respect to the diagnosis of CTE pathology.

A Suggestive of CTE

This level of certainty is established where the NINDS TES criteria are met, but the additional supportive criteria for 'possible CTE' are not met.

B Possible CTE

This level meets the NINDS TES criteria of substantial exposure to RHT and cognitive impairment. In addition, a minimum of two supportive criteria are required: delayed onset, motor signs, one or more psychiatric features, neurobehavioral dysregulation, and subtle/mild functional limitation or worse.

C Probable CTE

This level meets the NINDS TES criteria of substantial exposure to RHT and cognitive impairment. Also required are three of the following five criteria: delayed onset, motor signs, one or more psychiatric features, neurobehavioral dysregulation, and a severity of functional dependence that equals mild dementia or worse.

D Definite CTE with TES

This level requires that the NINDS TES criteria be met and confirmed by postmortem neuropathologic diagnosis.

6.7.3 A TES case study example

The NINDS methodology does not include a case study example to offer a useful assessment of how the criteria would operate in practice to deliver a diagnosis of CTE to the legal standard. However, the Montenigro study of 2014[103] utilised most of the same criteria as NINDs and is useful in providing such an example. This example is reproduced below as a guide to the application of the TES. It should be emphasised, however, that there is no one model for determining the contraction of CTE *in vivo*. Other studies, some of which are considered below, use different criteria to NINDS. In this sense, the Montenigro study remains relevant.

[103] PH Montenigro et al, 'Clinical Subtypes of Chronic Traumatic Encephalopathy: Literature Review and Proposed Research Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2014) 6(5) *Alzheimer's Research & Therapy* 68.

The following example of 'probable CTE' was offered in Montenigro's paper:

> Case C: A 59-year-old man presented to his primary care physician with complaints of progressive memory and concentration problems. Prior to going to college, the patient entered the Army, where he boxed competitively for 4 years. He did not experience any combat. He was an avid rugby player in college and continued playing in formal competitive clubs until the age of 54, when he stopped because of a cervical disk injury. He received an MBA and had been a successful business consultant. He was divorced at the age of 45 and lived alone. He reported one concussion at the age of 30, when he briefly lost consciousness during a rugby game, although he stated he got his 'bell rung' countless times in boxing and rugby. He reported to his primary care physician that he had been having difficulty remembering details of conversations and meetings at work and that this was beginning to interfere with his productivity. His medical history was significant for the cervical disk injury and for migraine headaches for many years. He was referred to a local academic medical center memory clinic, where a formal neuropsychological evaluation demonstrated moderately impaired performance on a word list recall task, compared with age and education norms, as well as severely impaired fine motor dexterity. All other areas were intact, although his performance on a measure of psychomotor speed and response set maintenance was slightly below expected levels given his history. A neurological examination revealed mild bilateral resting tremor and mild upper extremity rigidity. An MRI scan was read as normal, and all laboratory findings were within normal limits. As part of a clinical research study, he was given two PETs: one with a new tau radiotracer and another with an amyloid tracer. Results indicated no meaningful amyloid uptake, although his tau scan was abnormal with scattered increased tracer uptake in the dorsolateral frontal cortex and the medial temporal lobes. Diagnosis: TES-COGv, with motor features, progressive course; probable CTE.[104]

6.7.4 TES and the question of *in vivo* diagnosis of CTE

While NINDS and Montenigro both stated that the TES criteria are not meant to be used for a clinical diagnosis or as evidence of an underlying disease, they were, and are, being utilised for just such a purpose, as revealed in some publications listed below. In addition, Montenigro noted nonetheless that the criteria are useful in 'differential diagnosis'; for example, between CTE and Alzheimer's disease.[105] Moreover, proof of the cause of actual harm need not, as discussed above, be definitive.

Katz points out that some medicos have used the criteria for the clinical diagnosis of CTE: '[A] recent survey study of former National Football League players found that 108 (of 3,913) respondents reported having been given a diagnosis of CTE by a clinician.'[106]

Katz appears to accept the reality of the NINDS diagnostic use, stating that the consensus criteria for TES 'should be used cautiously in clinical and medicolegal settings, avoiding

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[104] Ibid 80.

[105] Ibid.

[106] DI Katz et al, 'National Institute of Neurological Disorders and Stroke Consensus Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2021) 96 *Neurology* 848, 860. The survey study referred to is R Grashow et al, 'Premortem Chronic Traumatic Encephalopathy Diagnoses in Professional Football' (2020) 88 *Annals of Neurology* 106.

equivalence with a diagnosis of CTE, and using appropriate care when communicating a diagnosis of TES'.[107]

Australian authors Kim and Beran say that despite expressions of caution in using the NINDS TES criteria as a diagnostic tool, the 'authority of the NINDS and the simplicity of the criteria have prompted physicians to ignore the caution and either to adopt the criteria or use them as a guideline to diagnose TES'.[108]

6.7.5 An Australian application of the NINDS TES criteria

In an early, though limited, legal recognition of the NINDS TES criteria, Richmond Australian Football League (AFL) player Ty Zantuck in 2022 sought an extension of time to bring an action beyond the statutory limitation period, claiming that his club and two of its medical staff 'breached [their] duty of care to manage concussions', and as a result he 'has a brain injury'.[109] The judgment quoted a report of neurologist Dr Rowena Mobbs, who stated that 'Mr Zantuck presented with a complex neurological picture that was characterised by decline in cognition, mood and behaviour … [He] fulfils the criteria for suggestive TES and CTE.'[110] The judgment then quoted a report of neuropsychologist Assoc Prof Jennifer Batchelor, who opined that

> Mr Zantuck meets the criteria for probable CTE. Further, … he meets each of the primary diagnostic criteria for TES … [T]he time during which Mr Zantuck played for [Richmond Football Club] would qualify as a substantive risk factor for TES.[111]

On the basis of this and other evidence, Ierodiaconou AsJ granted the extension of time.

Zantuck's condition, assuming the fact of CTE, will likely deteriorate over time as the disease progresses, in which case a TES-based diagnosis will move through the probability scales to that of a more certain categorisation.

6.7.6 TES diagnosis and treatment as a fact in inferring contraction of CTE

Dr Rowena Mobbs, neurologist and CTE specialist, told the Senate Inquiry into *Concussions and Repeated Head Trauma in Contact Sports* that she utilises TES as a diagnostic method:

> I have experience in managing a cohort of 180 patients with suspected traumatic encephalopathy syndrome (TES), 52 confirmed with probable TES on cognitive testing. TES is the in-life syndrome

[107] DI Katz et al, 'National Institute of Neurological Disorders and Stroke Consensus Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2021) 96 *Neurology* 848, 860.

[108] PS Kim and RG Beran, 'Legal Medicine Implications of a Multidisciplinary Approach to Managing Traumatic Encephalopathy Syndrome in Australia' (2023) 14 *Frontiers in Neurology* 1179319, 2.

[109] *Zantuck v Richmond Football Club* [2022] VSC 405, [27].

[110] Ibid [164].

[111] Ibid [165].

> of CTE. This is arguably Australia's largest clinical cohort, as I am unaware of any other medical research program with double figures, let alone triple figures, of patients with CTE symptoms.[112]

Mobbs stated — perhaps suggestively of confidence in the TES methodology — that '[f]or clarity, I will refer to "CTE" rather than "probable TES" in describing my patient's clinical diagnosis'.[113]

With reference to an article by Cantu and Budson, Kim and Beran (both medicos) state that '[t]he survey [in Cantu and Budson] demonstrated that physicians were diagnosing CTE, during life, even though it cannot be officially diagnosed antemortem'.[114] Kim and Beran argue that even though TES is not officially recognised, its use to diagnose CTE falls within the scope of a physician's 'comprehensive duty of care',[115] and refer to the judgment of Brereton J in *Paul v Cooke*:

> [I]n a failure to diagnose case, breach of the duty results in a condition not being discovered and treated, so that the condition persists and potentially deteriorates, continuing to cause harm that could otherwise be avoided or becoming more difficult to treat in the future …
>
> [The duty's] purpose is … to enable the appropriate treatments to be identified.[116]

As is clear from Kim and Beran's statement, AFL player Zantuck's application for extension of time, and other examples, some medicos are using TES as a diagnostic tool, on the basis of which symptoms of CTE are treated. The willingness of medical professionals to frame treatment around a diagnostic methodology suggests a level of satisfaction with that methodology and represents a further fact going to establishing an overall inference of CTE on the balance of probabilities. Moreover, Katz suggested that because 'probable CTE' is 'weighted toward high specificity but low sensitivity', it would be 'appropriate for targeted therapeutic trials'.[117]

While the argument of Kim and Barron regarding an obligation to utilise TES as a diagnostic tool is debatable as a legal obligation, the same cannot be said for failing to warn of the risk of CTE on the basis that its clinical diagnostic use is likely to be considered, at a

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[112] Rowena Mobbs, Submission No 1 to Senate, Community Affairs References Committee, Parliament of Australia *Concussions and Repeated Head Trauma in Contact Sports* (September 2023) 3.

[113] Ibid.

[114] PS Kim and RG Beran, 'Legal Medicine Implications of a Multidisciplinary Approach to Managing Traumatic Encephalopathy Syndrome in Australia' (2023) 14 *Frontiers in Neurology* 1179319, 2, referring to R Cantu and A Budson, 'Management of Chronic Traumatic Encephalopathy' (2019) 19(10) *Expert Review of Neurotherapeutics* 1015.

[115] *Rogers v Whitaker* (1992) 175 CLR 479, 483.

[116] PS Kim and RG Beran, 'Legal Medicine Implications of a Multidisciplinary Approach to Managing Traumatic Encephalopathy Syndrome in Australia' (2023) 14 *Frontiers in Neurology* 1179319, 2, quoting *Paul v Cooke* (2012) NSWSC 840.

[117] DI Katz et al, 'National Institute of Neurological Disorders and Stroke Consensus Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2021) 96 *Neurology* 848, 859.

minimum, indicative of a real risk of harm.[118] As Chapter 4 discussed, the obligation to warn requires the defendant to take a proactive approach to a potentially serious risk of harm *prior* to confirmation of that risk. For example, in *H v Royal Alexandra Hospital*[119] Badgery-Parker J said the fulfilment of the defendant hospital's duty required it to warn recipients of blood products of the emerging risk of HIV before the risk had been substantiated.[120]

6.8 Other methodologies in diagnosing CTE *in vivo*

While the TES criteria of NINDS, and previously those of Montenigro, have gained recent currency and would seem on balance to offer a recognised means of diagnosing the contraction of CTE *in vivo*, there is no overseeing authority that determines one methodology is superior to any other. As such, several different methods offered by researchers in diagnosing CTE in a living patient are relevant. These methods, or the indicia collectively, provide a further means of establishing a probable factual inference of harm.[121]

Given that the NINDS TES methodology is based on the clinical manifestations of CTE, it would be possible to refer to any methodology that uses these sequelae as a TES methodology. However, given that other methodologies utilise some criteria that the NINDS TES methodology does not, this chapter maintains the distinction to avoid confusion.

6.8.1 Pierre et al: The spectrum of indicators and inference of harm in the form of CTE

Researchers Pierre et al reviewed 'neurobehavioral commonalities, neuropathology, cognitive and functional symptomatology, imaging findings, and biomarkers' to 'illuminate our perspective on *in-vivo* diagnosis and facilitate improved characterization of the spectrum of CTE'.[122] Again, the indicators are strongly indicative of CTE, rather than definitive.

The criteria were referred to as 'innovations in diagnosis' and were classified broadly under three headings: 'clinical diagnostic criteria', 'imaging' and 'fluid biomarkers'.[123] The last two do not feature in the NINDS TES criteria.

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[118] See *H v Royal Alexandra Hospital* [1990] 1 Med LR 297.

[119] Ibid.

[120] See Chapter 4: 'CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care'.

[121] See, eg, *Zantuck v Richmond Football Club* [2022] VSC 405.

[122] K Pierre et al, 'Chronic Traumatic Encephalopathy: Update on Current Clinical Diagnosis and Management' (2021) 9 *Biomedicines* 415, 416. The study was largely an account of ongoing research, and mentioned briefly that of Montenigro.

[123] Ibid 418–19.

The research revealed positive *in vivo* indications of CTE: '[P]reliminary research supports the use of plasma t-tau as a marker of injury severity following repetitive head trauma' and 'exosomal tau' was able to 'reliably differentiate former NFL players from healthy controls and that levels correlate with cognitive and psychomotor decline'.[124] Further, 'sTREM2 correlated with total tau levels, and strengthened the relationship with tau and head trauma when included in regression models'.[125] Some criteria, while not absolute in themselves, were described as 'a tool for assisting in the diagnosis of CTE'.[126]

6.8.2 Clinicopathological evaluation and inference of harm in the form of CTE

Mez et al [127] compared the symptoms that characterise the different stages of CTE — as described in McKee et al's 2013 article[128] — with the degree of post-mortem tau deposition in a sufferer's brain. For example, the histological findings of a stage III CTE individual would be checked against the external symptoms of CTE associated with a stage III sufferer, such as frequent depression and impulsivity.

According to Mez et al, '[t]he strengths of this study are that this is the largest CTE case series ever described to our knowledge … and that all participants were exposed to a relatively similar type of repetitive head trauma while playing the same sport'.[129] The results confirm that the more an athlete engages in contact sport, the greater the risk of sustaining actual harm in the form of CTE.

Where, as in Mez et al's research, the degree of internal damage accurately correlates to the external or clinical symptoms, and the cause of the internal damage is the playing of American football, logic suggests that the playing of American football correlates to the external or clinical symptoms. More broadly, *ceteris paribus*, the symptoms and behavioural manifestations an athlete displays are caused by RHT in the playing of contact sports, including those played in Australia. While a correlation alone is not definitive proof of harm, where it is consistent across similar cohorts and across time, it is highly inferential. In short, where an

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[124] Ibid 418.

[125] Ibid.

[126] Ibid 419.

[127] J Mez et al, 'Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football' (2017) 318(4) *Journal of the American Medical Association* 360. Mez's study was referred to by Greenhow in a sub-section titled 'CTE according to the UNITE brain bank — Accumulation of cases': see A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024) 110. Although not stated, Mez's study presumably is one of several relevant to providing proof of CTE as a generic cause of RHT in the playing of contact sport.

[128] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43. See Chapter 2: 'The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments'.

[129] J Mez et al, 'Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football' (2017) 318(4) *Journal of the American Medical Association* 360, 367.

athlete's *in vivo* symptoms correspond with the known indicators of CTE, one may reasonably argue that the athlete is suffering probable harm in the form of CTE.

6.8.3 Turk: Cerebrospinal fluid as biomarker and inference of harm in the form of CTE

Turk's 2022 study tested cerebrospinal fluid (CSF) post-mortem for markers of CTE, noting that 'biomarkers with the ability to detect and distinguish CTE from AD [Alzheimer's disease] would be of great value if they show a high degree of correlation with CTE neuropathology [*in vivo*]'.[130] The brains of 192 subjects were divided into four 'pathological groups' based on AD and CTE criteria. One group possessed CTE neuropathology, one group possessed AD neuropathology, one group possessed both CTE and AD neuropathology, and a control group possessed neither. Importantly in terms of comparison, the groups were also diverse in the degree of CTE and AD. CSF was collected at autopsy and examined for p-tau (phosphorylated tau), and Aβ (beta-amyloid).[131] The study revealed that 'CSF $A\beta_{1\text{-}42}$ was found to be decreased in both low stage CTE compared to the no CTE/no AD (control) group as well as in high stage CTE compared to [intermediate/high] AD'.[132]

6.9 Two notable core indicia of CTE

Emphasised below, given their largely unique association with CTE over other cognitive maladies, are two features noted in almost all studies concerned with the *in vivo* diagnosis of CTE: an athlete's history of RHT and the aberrant behavioural manifestations commonly associated with CTE. Again, these facts, along with other facts, are argued to form a the basis leading to an inference on the balance of probabilities that a plaintiff athlete is afflicted with CTE.

6.9.1 Athlete history of RHT: CTE

Lesman-Segev noted that '[c]urrent evidence suggests that exposure to [RHT] is necessary for the evolution of CTE, though the "threshold" of cumulative [RHT] exposure that puts a person at risk for CTE is unknown'.[133] Knowledge of CTE-like harm caused by repetitive trauma through engagement in contact sport dates back at least as far as Martland's *Journal of the*

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[130] KW Turk, 'A Comparison between Tau and Amyloid-B Cerebrospinal Fluid Biomarkers in Chronic Traumatic Encephalopathy and Alzheimer Disease' (2022) 14(28) *Alzheimer's Research & Therapy* 1, 2.

[131] 'Aβ is a peptide of 39–43 amino acids produced by β-secretase-1 cleavage of Amyloid Precursor Protein. Beta amyloid is normally secreted from cells and degraded. In Alzheimer Disease patients, it is secreted and aggregated into insoluble plaques that may be neurotoxic': 'Beta Amyloid — An Overview', *ScienceDirect* (Web Page, May 2020) <https://www.sciencedirect.com/topics/neuroscience/beta-amyloid >. Turk tested for $A\beta_{1\text{-}42}$ and for p-tau_{231}.

[132] KW Turk, 'A Comparison between Tau and Amyloid-B Cerebrospinal Fluid Biomarkers in Chronic Traumatic Encephalopathy and Alzheimer Disease' (2022) 14(28) *Alzheimer's Research & Therapy* 1, 8.

[133] OH Lesman-Segev et al, 'Chronic Traumatic Encephalopathy: A Comparison with Alzheimer's Disease and Frontotemporal Dementia' (2020) 40 *Seminars in Neurology* 394, 397.

American Medical Association article in 1928.[134] Just as exposure to a carrier of measles is foundational to the diagnosis of that disease, extensive participation in contact sports is a logical element to be considered in the diagnosis of CTE. For example, where two patients present with similar cognitive symptoms, differential diagnosis between CTE and, say, Alzheimer's disease is aided by one patient's long participation in contact sport.

6.9.2 Behavioural symptoms and indicators

A well-accepted indicator of CTE is the aberrant behavioural manifestations generated as a sufferer's brain progressively deteriorates. Most studies concerned with the *in vivo* diagnosis of CTE emphasise the consistency of this criterion across time and across individuals at any point in time. Irrespective of the period in which CTE is discussed and the tools available for diagnosis, the one constant linking all eras over time — from Martland in 1928[135] to McKee et al in 2009,[136] onto the present day — is the effect of CTE on behaviour. This section briefly highlights this important criterion and its utility in discerning CTE in a living athlete. The aberrant alterations in a CTE sufferer's behaviour are considered in the case study of AFL player Danny Frawley.[137]

According to Lesman-Segev's study, '86% of patients presenting with behavioral changes later developed cognitive symptoms'.[138] In fact, the most common motivation for relatives in seeking medical advice on behalf of an athlete displaying symptoms thought to be CTE and, later, to agree to autopsy examination, are the aberrant behavioural manifestations associated with CTE.

When Dr Bennet Omalu decided to dissect NFL player Mike Webster's brain, he believed Webster was probably suffering from a cognitive malady. As Laskas wrote, Omalu asked himself: 'How did this big athletic man, only fifty years old, end up crazy in the head?'[139]

Laskas also recorded Webster's altered states of behaviour:

> In possession of an assault rifle, a semiautomatic pistol and a .357 Magnum revolver Webster could not remember where he bought the weapons. When his teeth started falling out Webster tried to stick

[134] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

[135] Ibid.

[136] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

[137] See **[6.11.2]**.

[138] OH Lesman-Segev et al, 'Chronic Traumatic Encephalopathy: A Comparison with Alzheimer's Disease and Frontotemporal Dementia' (2020) 40 *Seminars in Neurology* 394, 399.

[139] JM Laskas, *Concussion* (Random House, 2015) 98.

> them back with Super Glue. To write letters he wrapped his shaky hands in duct tape, through which he inserted his pen. Unable to sleep he bought a Taser and 'zapped' himself into unconsciousness.[140]

Martland in his 1928 article noted that 'a marked mental deterioration may set in necessitating commitment to an asylum'.[141] Pierre' et al s 2021 paper said that the 'spectrum of symptoms' includes 'changes in behavior, cognition and motor symptoms like impulsivity, paranoia, rage behaviors, headaches, memory deficits, impaired attention, dysphagia, dysarthria, and coordination problems'.[142]

Omalu et al's second CTE article — 'Chronic Traumatic Encephalopathy in a National Football League Player: Part Two' in 2006 — lists several indicative behaviours exhibited by the subject player (Terry Long) prior to his death:

> He became progressively incapable of mentally handling very complex rational thoughts in matters of daily living and business. He became increasingly impulsive and paranoid. His erratic behaviour continued to worsen; he exhibited disinhibition, began having financial problems, and could not sustain his businesses. … His behaviour became increasingly characterized by constant thoughts of suicide, and he was admitted for psychiatric treatment three times.[143]

Gavett found that '[i]n some individuals, the early manifestations of CTE affect behavior; … [including] being more irritable, angry, or apathetic or … having a shorter fuse. Increased suicidality seems to be a particularly salient symptom of CTE.'[144]

The behavioural traits of Australian CTE sufferers, or those believed to be suffering with CTE, have been described similarly by friends and relatives. To illustrate, in February 2020, ex-AFL player Graham 'Polly' Farmer, who died in 2019 aged 84, was found at autopsy to have stage III.5 CTE. At the age of 64 he had been diagnosed 'with Alzheimer's, with elements of depression or mood swings, explosivity, loss of attention and concentration, short-term memory loss and headaches'. Upon autopsy, these symptoms were attributed by researchers at the Australian Sports Brain Bank as 'the first case of CTE in an Australian Rules footballer'.[145] In February 2021, the Coroners Court of Victoria found that ex-AFL player Danny Frawley,

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[140] Paraphrased from ibid 94–7.

[141] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103, 1103.

[142] K Pierre et al, 'Chronic Traumatic Encephalopathy: Update on Current Clinical Diagnosis and Management' (2021) 9 *Biomedicines* 415, 415. See the discussion above at **[6.8.1]**.

[143] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player: Part Two' (2006) 59(5) *Neurosurgery* 1086, 1087.

[144] BE Gavett et al, 'Chronic Traumatic Encephalopathy: A Potential Late Effect of Sport-related Concussive and Sub-concussive Head Trauma' (2011) 30 *Clinical Journal of Sports Medicine* 179, 180.

[145] 'Graham "Polly" Farmer Is the First Australian Rules Player Diagnosed with Chronic Traumatic Encephalopathy', *ABC News* (online, 27 February 2020) <https://www.abc.net.au/news/2020-02-27/australian-rules-great-graham-polly-farmer-diagnosed-with-cte/12005508>.

aged 56, had 'intentionally taken his own life' when he crashed his car at 132 km/h at Millbrook, Victoria, in 2019.[146]

These official finding of death associated with CTE are supported by statements published in the media of players who report they are suffering from symptoms of CTE. A media publication unsupported by medical evidence is of limited probative value, though in the absence of official documents it is nonetheless informative. Former rugby league player Shaun Valentine says 'dizzy spells and memory loss … have plagued his life after a series of head knocks in 2000'.[147] Ex-AFL player Greg Williams 'is suffering memory loss. His moods have become erratic.'[148] Koby Stevens, former AFL player, 'has largely been housebound for months while struggling with symptoms of concussion', and on 'advice from doctors' decided to retire from the game.[149] A 2019 news article titled 'The Concussion Class Action that Could Punch a Hole in the AFL' reported as follows:

> [R]oguish Geelong and Essendon flag ruckman John Barnes, 49, [is] now dealing with random, horrifying seizures. … Hawthorn's champion rover John Platten, 55, [is] struggling to cope with acute memory loss. … [F]ormer Melbourne and North Melbourne high-flyer Shaun Smith, 49, [is] beset by painful mood swings, anxiety and depression. … [Barnes] retired in 2001 … [after an estimated] dozen serious concussions in his career. [His wife, Rowena Barnes, said that she] recently awoke in bed as a seizure took hold, hearing him choke through convulsions. When he recovered, he remembered nothing. 'He just looked around the bedroom and asked, "Who are these people?"' Her voice breaks now. 'They were his sons!'[150]

From the several histories cited above, along with consistency through time, the behavioural manifestations of CTE are one of the key indicators of CTE *in vivo*. The medico or SGB that becomes aware of a player's disfunctional behaviour, particularly when associated with declining cognitive faculties has, through the studies of the past, a means to form a preliminary diagnosis of CTE and initiate further research and inquiries.

6.10 The Bradford Hill criteria

Sir Austin Bradford Hill, an English epidemiologist and statistician, proposed to the Royal Society of Medicine in 1964 a set of criteria to detect 'relationships between sickness, injury

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[146] In the Coroners Court of Victoria at Melbourne, Finding into Death, Daniel Frawley, Court Reference: COR 2019 4895, 23 February 2021, [93(d)] per Coroner Paresa Spanos <www.coronerscourt.vic.gov.au/sites/default/files/2021-02/Finding%20D%20Frawley.pdf>.

[147] C Barrett, 'Lawyer Keen to Act for NRL Players on NFL-style Concussion Lawsuits', *Sydney Morning Herald* (Sydney, 10 November 2014).

[148] P Sheehan, 'Concussion a Concern from Elite to Schools', *Sydney Morning Herald* (Sydney, 28 February 2013).

[149] C Walsh, 'St Kilda's Koby Stevens Succumbs to Concussion Symptoms', *The Australian* (Sydney, 24 July 2018).

[150] K Marshall, 'The Concussion Class Action that Could Punch a Hole in the AFL', *The Age* (Melbourne, 16 March 2019).

and conditions of work'[151] — in effect, to move from a believed association between an environmental feature and a disease, to affirming that the environmental feature was a *cause* of the disease. The 'environmental feature' in the context of CTE is RHT.

The nine criteria proposed by Bradford Hill as a 'framework to assess causal inferences in epidemiological research'[152] are: strength of association, consistency, specificity, temporality, biological gradient, plausibility, coherence, experiment and analogy.

The Bradford Hill methodology is discussed in detail in Chapter 8 as a recognised epidemiology means to establish contact sport as a cause of CTE. Nonetheless, a causative relationship between head trauma and CTE established through the Bradford Hill criteria is logically helpful to an individual athlete whose personal experience aligns with that of the criteria that establish a general causal relationship. For example, where 'strength of association' based on repetitive concussion is high, this advantages an athlete who has experienced repetitive concussion in claiming that he or she is suffering symptoms of CTE.

6.11 Application: Multiple *in vivo* indicators to infer CTE

Once an athlete exhibits cognitive disfunction, the question necessarily turns to whether these *in vivo* symptoms are sufficient to draw an inference of actual harm in the form of CTE, rather than some other disease.

Multiple means to discern CTE *in vivo* were presented above, ranging from the TES criteria, diagnostic imaging, the use of fluid biomarkers, the degree of tau deposition in spinal fluid, and the aberrant behavioural manifestations of CTE.

All diagnostic criteria that indicate that an individual athlete is either afflicted by CTE or is not, are arguably relevant. Mavroudis et al state that '[t]he diagnosis of TES/CTE requires a multidisciplinary approach', and list a range of categories to be included: 'neurodegenerative disorders, systemic illnesses, and/or psychiatric conditions that can account for the symptoms; and also complex investigations of patient history, psychiatric assessment, and blood and cerebrospinal fluid biomarker evaluation'.[153] These are included in discussion below.

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[151] Austin Bradford Hill, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965) 295, 295. The Bradford Hill criteria are discussed extensively in Chapter 8: 'The Question of "Generic Capacity": Does the SGB's Contact Sport Cause CTE?'

[152] Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1, 3.

[153] I Mavroudis et al, 'A Review of the Most Recent Clinical and Neuropathological Criteria for Chronic Traumatic Encephalopathy' (2023) 11(12) *Healthcare* 1689, 11.

6.11.1 Application: Points of consideration

The *in vivo* indicators and symptoms of CTE recited above, either as single indicia or when joined to form a methodology can be distilled into a series of facts leading to an inference as to whether an athlete is, for purposes of the legal standard of proof 'probably' suffering from CTE — in a sense, comparing the 'textbook' indicators of CTE with the symptoms and playing history of an athlete. This is not dissimilar to diagnosing influenza by reference to the symptoms of influenza. An inference drawn from facts must be sufficient to prove that the living athlete is 'probably' suffering symptoms of CTE.[154]

Where the 'tests' or 'matters for considerations' listed are positive for CTE, a causal explanation of actual harm *in vivo* is, as argued, reasonable. Some variation among individual complainants can be expected. Further, it is not expected that all tests will be positive for CTE; rather, one looks to a prevalence or preponderance of positive indicators that together raise an inference.

In discerning through factual inference that an athlete is afflicted with CTE actual harm in the form of CTE the following, non-exhaustive, tests are proposed:

1. consideration of whether an athlete is suffering symptoms indicative of CTE (obviously, actual harm in the form of CTE cannot be proven without *in vivo* symptoms);

2. consideration of the athlete's playing history and career. Years of play, time within junior and senior competitions, the playing of other contact sports, and any employment where head trauma is likely — for example, as an artillery officer — are pertinent;

3. utilisation of core criteria (for example, a history of head impacts and recorded concussions) and the 'core clinical features' (for example, impairment of executive function) used to initiate a TES profile;[155]

4. utilisation of 'supportive features' of a TES profile (for example, loss of impulse control and delusional beliefs);[156]

5. utilisation of the tools used to support 'diagnostic criteria' as proposed by Pierre et al and Mez et al, such as symptoms being present for at least 12 months, fMRI and PET scans, and fluid markers;[157]

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[154] *Bennett v Minister for Community Welfare* (1992) 176 CLR 408, 412–13. A guide to the reception of evidence in general was given by Gummow J in *Rosenberg v Percival* (2001) 205 CLR 445, [163], who said that the overriding concern is for judicial decision-makers 'to rest their conclusions, as far as possible, on objective considerations, contemporaneous facts and logical inferences, rather than a self-claimed capacity to tell truth from falsehood by visual or aural impression'.

[155] See **[6.7]**.

[156] Ibid.

[157] See **[6.8.1]–[6.8.2]**.

6. testing of proposed symptoms against the Bradford Hill criteria;[158]

7. consideration of the full range of behavioural manifestations (noting that these have been recorded as manifestations of CTE-like harm for more than a century), ranging from irritability and parkinsonism-like movement to personality changes.

While each singular methodology proposed in the various studies[159] may be sufficient to establish CTE in a living plaintiff, when applied in conjunction with each other and alongside the behavioural manifestations of CTE and the playing history of plaintiff athlete, the establishment of CTE as the affecting malady is, as argued, probable in law.

6.11.2 Analytical illustration: Inference of harm, the Danny Frawley case in proxy

Many of the tests listed above are multifaceted, but no study appears to have combined all tests to diagnose, or prove, actual harm in the form of CTE in a purported sufferer. In part, this is explicable because of the relatively recent development of most tests and the need to wait for histological confirmation at autopsy. Bearing in mind that 'where direct proof is not available, it is enough if the circumstances appearing in evidence give rise to a reasonable and definite inference',[160] and, as noted by Gibbs J, 'you need only circumstances raising a more probable inference in favour of what is alleged',[161] the case of ex-AFL player Danny Frawley is considered in proxy to illustrate the application of the diagnostic methodologies with respect to an athlete's factual claim that the *in vivo* symptoms and aberrant behaviour he or she suffers are caused by CTE.

The Coroners Court of Victoria found that 56-year-old Danny Frawley had suicided by deliberately crashing his car into a tree at an estimated speed of 132 km/h in an 80 km/h speed zone. At autopsy, the Australian Sports Brain Bank found 'demonstrated features of chronic traumatic encephalopathy (CTE), Stage I with no other evidence of other neurodegenerative disease'.[162] A second opinion by the Boston University Center for the Study of Traumatic Encephalopathy found stage II CTE.[163]

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158 See **[6.10]**.

159 See **[6.6]**.

160 *Luxton v Vines* (1952) 85 CLR 352, 358.

161 *TNT Management Pty Ltd v Brooks* (1979) 53 ALJR 267, 269

162 In the Coroners Court of Victoria at Melbourne, Finding into Death, Daniel Frawley, Court Reference: COR 2019 4895, 23 February 2021, [18] per Coroner Paresa Spanos, <www.coronerscourt.vic.gov.au/sites/default/files/2021-02/Finding%20D%20Frawley.pdf>.

163 The court noted (ibid): 'Stage I and II pathology are often grouped together as "low stage" or "mild" CTE which has been frequently associated with mood and behavioural symptoms and slightly less frequently with cognitive and memory impairment. Accordingly, the clinical interpretation in A/Prof Buckland's [of the Australian Sports Brain Bank] original report remains unchanged.'

Although the autopsy revealed that Frawley was suffering from CTE, our interest lies in whether the diagnostic techniques that are available today would have confirmed prior to death that his symptoms were those of CTE.

The inquiry begins with the onset of symptoms and markers. 'Mrs [Anita] Frawley first noticed symptoms of mental illness once her husband finished up [as senior coach of] the Richmond football club' in 2004.[164] It was suggested that Frawley's removal as coach may have been associated with 'low feelings of self-worth'.[165] Mrs Frawley said that during this time he suffered 'wildly oscillating moods that were only seen by his family'.[166]

The next step is to consider Frawley's playing history, number of games, level of play and incidents of recorded head trauma, including time off after head trauma and severity of head trauma. Frawley played 240 games in his professional career between 1984 and 1995 — a substantial period in premier contact sport — to which is added junior participation and practice sessions (including those at full contact) throughout his playing life. As noted by the Coroner,

> Mr Frawley played football between the ages of 16 and 34. He experienced 20 concussions, including loss of consciousness, and was admitted to The Epworth Hospital emergency department five or six times. During these incidents, he experienced vomiting and was unable to lift his head. Despite this, Mr Frawley always returned to the playing field the following week.[167]

Other indicators of CTE followed: 'Mrs Frawley observed her husband's behaviour became more erratic and he would often disappear, purportedly to the gym, but in reality his whereabouts were largely unknown.'[168] Frawley consulted a psychiatrist, Dr Brendan Hayman, in June 2014, presenting with

> a significant constellation of depressive symptoms including significant insomnia, depressive ruminations, relative anhedonia, self-reproachment, amotivation, difficulty functioning and episodic passive suicidality. He had also engaged in poor decision-making and conduct that caused conflict in his marriage.[169]

Dr Hayman prescribed several medications. Frawley was described as 'extremely needy' by his physician and family members.[170] In 2014 he was 'admitted to The Albert Road Clinic with agitated depression, insomnia and mood-congruent depressive thinking', from which he

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[164] Ibid [21].

[165] Ibid.

[166] Ibid.

[167] Ibid [59].

[168] Ibid [21].

[169] Ibid [23].

[170] Ibid [24].

slowly improved.[171] Nonetheless, Mrs Frawley felt that 'it took her husband at least a year to recover after he commenced treatment with Dr Hayman, but he was never the same'.[172]

In June 2019,

> Mr Frawley's decision making was still erratic and his personality had changed. He became even more self-absorbed and had gained about 25 kilograms. He avoided his family's Christmas in July celebrations, preferring to spend the time with a country AFL team that he had been coaching.[173]

The aberrant behaviour Frawley engaged in, is similar to with that displayed by almost all athletes who, at autopsy, were later confirmed as afflicted with CTE.

Although CTE was confirmed at Frawley's autopsy, the purpose of an *in vivo* diagnosis is to establish that a plaintiff suffers CTE prior to death so as to further the athlete's negligence claim during the living years. As such, possible confounding facts must be considered:

> Mr Frawley began his football career in his formative years and likely experienced head trauma before his personality was well established. This coupled with the inability to diagnose CTE before death, confounds evaluation of the contribution of CTE to personality, behaviours, any cognitive deficits, or lability of mood.[174]

The point, seemingly, is that Frawley may have had a different personality had he not experienced head trauma as a youngster, in effect making it difficult to discern which aspects of his personality were 'intrinsic' and which 'might be attributable to CTE'.[175] In any case, changes brought on by head trauma as a youth nonetheless serve to establish a possible cause of CTE. The aberrant behaviour of Frawley in the later years of his life was extremely different to that of the outgoing 'knockabout guy' he was known to be earlier.[176] Further, although 'Mr Frawley was diagnosed with major depression in 2014, he may well have experienced depression for several years prior'.[177] Depression, again, is a common symptom of CTE in its earlier stages.

A Provisional certainty for CTE pathology

Should one review Frawley's experience against the primary and supportive criteria of the NINDS TES methodology, would it reveal that the ex-player was 'probably' suffering from CTE? Four levels of certainty were proposed in diagnosing TES across various strengths of indicators. Here only 'probable' is considered. The selection of the probable category does not

[171] Ibid [26].

[172] Ibid [24].

[173] Ibid [33].

[174] Ibid 'Comments' [1].

[175] Ibid [60].

[176] Ibid.

[177] Ibid [61].

mean that the 'possible' category is necessarily excluded for purposes of law, as the scale of indicators is directed to a form of scientific standard which, to those conducting the study, is sufficient to warrant diagnosis with TES associated with 'treatment of neuropathologically diagnosed chronic traumatic encephalopathy'.

As discussed above, what is referred to as 'Probable CTE' requires two of three criteria listed here to be met: He played high-level contact sport for a period 12 years, including prior to entry into the premier competition. He had a recorded history of exposure to high levels of RHT. He had deficits in executive function as demonstrated perhaps by his removal as coach as the Richmond Club, recorded poor decision making and difficulty functioning. These three, rather than two, can be said to have been confirmed in the case of Frawley.

In addition, three of the following five must be shown in Frawley's case.

- *Delayed onset*: Frawley did not display symptoms of CTE until well after both his retirement in 1995 as a player and, apparently, when dismissed as premier coach. There was clearly, as required, a period of stable functioning prior to onset.
- *Motor signs*: Motor signs are not directly recorded.
- *Psychiatric features*: Frawley suffered from a range of symptoms including wildly oscillating moods. He became erratic and suffered significant insomnia and anhedonia.
- *Neurobehavioural dysregulation*: Frawley experienced agitated depression, which may have included poor regulation and control, anger outbursts, passive suicidality and perhaps suicide.
- *Severity of functional dependence indicative of mild dementia or worse*: Frawley avoided his family at Christmas in July, preferring time in the country with his team. This conduct caused conflict in his marriage and reduction in his social and family interests.

Even from the very limited reporting of Frawley's symptoms, behavioural traits and other indicators under the TES methodology the player would, if examined prior to death, have been found to have suffered from CTE. Again, there was no intent to seek out many of the TES criteria given the positive autopsy findings, so a preliminary finding, while positive, is incomplete.

At the time Frawley first experienced symptoms, clinical, fluid biomarker and imaging tests for CTE had not been developed. Had they been available, and had they been used in the above proxy, the following symptoms and indicators could have revealed the cause of his debilitation: examination for gait disturbance, features of parkinsonism and neuroinflammation markers; MRI, fMRI and PET imaging to detect changes in haemoglobin oxygenation; and measurements of total tau in body fluids, and p-tau$_{231}$ and A$\beta_{1\text{-}42}$ in cerebrospinal fluid. As they were not available, an inference of affliction with CTE has to be drawn from the symptoms and indicators listed above.

When facts such as these are considered, it is well to keep in mind comments of Lord Cairns noted above. Several feeble rays of light 'all converging' may 'produce a body of illumination' that clears 'away the darkness which you are endeavouring to dispel'.[178]

6.12 Conclusion

A plaintiff claiming to be suffering from actual harm must prove this on the balance of probabilities.

'Actual harm' is a question of fact. Whether a plaintiff is suffering from CTE must be proven as a fact. Proof of affliction with CTE is only definitive upon the histological examination of a claimed sufferer at autopsy. A living athlete is therefore unable to prove affliction with CTE through direct evidence.

Where direct evidence of actual harm is not available, the plaintiff athlete must prove *in vivo* affliction through facts and circumstances that provide a reasonable and definite inference of harm in the form of CTE on the balance of probabilities. The scientific standard requires proof that is near certain or absolute. The legal standard, with the object of apportioning legal responsibility, requires no more than 'it is more probable' that the plaintiff has the disease in question.

Ascertaining a 'probable inference' of harm in the form of CTE begins with recognition that the plaintiff athlete suffers from a cognitive malady for which there must be a medical explanation. An assessment of other facts follows, including the athlete's playing history, years of participation, level of play, number of games, position played, age at first game, number of concussions or blows to the head, and how often live practice was attended — all matters scientifically established as increasing the risk of CTE.[179] In reaching an *in vivo* diagnosis, medical technologies and techniques are employed, including scans, biomarkers and symptomatic diagnosis. Aberrant behavioural traits associated with CTE are reviewed. Finally, other medical conditions are eliminated as improbable. These indicators are used variously in the *in vivo* methodologies to diagnose or detect CTE described above.

At this point, the known history of CTE going back to Dr Martland's 1928 article,[180] along with the symptoms and behavioural traits of those who were confirmed at autopsy to have been suffering from CTE, can be compared to those of the plaintiff. The assessment of these facts will almost certainly require affirmation through expert evidence.

Each of the studies described in this chapter is useful in determining whether an athlete's symptoms are those of CTE, the NINDS methodology describing 'traumatic encephalopathy syndrome' (TES) is, given its comprehensive criteria and at least apparent acceptance by some

[178] *Belhaven and Stenton Perlerage* (1875) 1 App Ca 278, 279.

[179] See Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times'; Chapter 8 'The Question of "Generic Capacity": Does the SGB's Contact Sport Cause CTE?'

[180] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

medicos, especially useful in revealing probable CTE/TES. The TES methodology was considered appropriate by the Victoria Supreme Court, which granted an extension beyond the statutory limitation period for ex-AFL player Ty Zantuck's 'CTE claim' against the Richmond AFL club in 2022.[181] Other studies noted above also refer to the use of the TES criteria for diagnostic purposes.

In determining harm *in vivo*, consideration of *all* relevant markers and symptoms is argued as more valid than reflecting on merely the singular. Over time, as seen in the NINDS report on TES[182] (which built upon the original study of Montenigro),[183] the diagnostic criteria utilised to diagnose CTE *in vivo* may change, be emphasised or be de-emphasised over time as more is learned about the disease. Given that the researchers of these methods of diagnosis are examining for symptoms of the same disease and referring to common studies on CTE some overlap in proposed methodologies can be expected.

Of foundational relevance is a recognition that an athlete has presented with cognitive symptoms for which there must be a medical explanation. On this, Bradford Hill made a point of wide relevance on detecting the cause of a disease, asking: 'is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?'[184]

The application of the combined diagnostic methods discussed above was analytically tested in proxy against the symptoms and other indicators of CTE displayed by ex-AFL player Danny Frawley who, following his suicide, was affirmed by the Boston University Center for the Study of CTE to be afflicted with stage II CTE. Frawley displayed many of the symptoms and indicators of CTE, including a premier-level playing career of 12 years along with junior participation, multiple concussions and loss of consciousness, feelings of low self-worth, wild mood swings, disappearing from family contact, insomnia, depression, amotivation and suicidality. Each symptom and each indicator is a fact leading singularly and in combination to, as proposed, a probable inference of CTE prior to death.

Ultimately a determination of CTE by inference is a matter of judgment. As French CJ noted in *Amaca v Booth*, '[b]ecause the inferential process involves assessing multiple unranked factors, some of which may be more or less appropriate with regard to a specific causal assessment, judgment is required'.[185]

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[181] *Zantuck v Richmond Football Club* [2022] VSC 405.

[182] DI Katz et al, 'National Institute of Neurological Disorders and Stroke Consensus Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2021) 96 *Neurology* 848.

[183] PH Montenigro et al, 'Clinical Subtypes of Chronic Traumatic Encephalopathy: Literature Review and Proposed Research Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2014) 6(5) *Alzheimer's Research & Therapy* 68.

[184] Austin Bradford Hill, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965) 295, 299.

[185] *Amaca Pty Ltd v Booth* (2011) 246 CLR 36, [44], referring to the *American Restatement of the Law (3d) of Torts*.

This chapter proposes that a living athlete is able to prove on the balance of probabilities that he or she is afflicted with CTE through the two broad methodologies discussed above: that of TES and the other methods that utilise means that TES does not.

Where sufficient, the symptoms, history, medical markers and other indicators of CTE distil down to what Freckelton described as 'primary facts' from which, as proposed here, 'the only inference which is reasonably open' is that the plaintiff either is, or is not, suffering from CTE;[186] or, as expressed by Lord Cairns in *Belhaven and Stenton Perlerage*, facts that together serve to 'illuminate' or to 'dispel' the 'darkness',[187] and in so doing reach, through inference, a conclusion as to whether or not the plaintiff is suffering from CTE.

[186] I Freckelton, 'Epilogue: Dilemmas in Proof of Causation' in Danuta Mendelson and Ian Freckelton (eds), *Causation in Law and Medicine* (Taylor & Francis Group, 2002) 437.

[187] (1875) 1 App Cas 278, 279.

Chapter 7:
Factual Causation of Harm: Failure to Warn and the Athlete's Response Had a Warning Been Given

7.1 Introduction

The previous chapter, Chapter 6, concerned the challenges a living plaintiff athlete faces in proving on the balance of probabilities that he or she is, in fact, suffering with CTE.

This chapter, Chapter 7, concerns the question of factual causation of harm through an SGB's failure to inform itself of the risk of CTE and to warn its premier players of that risk, along with the essential element of proving had he or she been warned the athlete would have taken ameliorating action such as not participating in the SGB's competition.

The next chapter, Chapter 8, considers the question of whether the sport a defendant SGB administers, and which the plaintiff athlete participates, has the generic capacity to cause CTE through RHT.

An athlete claiming to have contracted CTE must prove that 'but for' the SGB's failure to warn, he or she would not have contracted CTE. As a matter of law, the failure to warn is the 'factual cause' because it 'causes' the plaintiff athlete to engage in an activity that he or she would have avoided if warned. For example, the player would not have played in the SGB's competition if warned. Or expressed differently, 'but for' the SGB's failure to warn, the player would have retired from contact sport, or at least would have not played without taking ameliorating steps to mitigate the risk of CTE.

On the other hand, a warning that would have been ignored cannot support a claim that the SGB caused the athlete's harm, as the athlete would have pursued the same risky course of action in any case.[1] The response of an athlete to a warning is judged subjectively — that is, how would *this athlete* have responded if warned? — but the athlete's testimony as to what he

[1] *Wallace v Kam* (2013) 250 CLR 375, [18]–[20].

or she would have done a*fter* suffering harm is not admissible unless it is against the athlete's interest. This chapter considers these issues.[2]

7.1.1 This chapter proceeds as follows

Section 7.2: The law and the athlete's response to warnings

This section outlines the law relevant to negligence arising from a failure to warn. The legal question is that of *factual causation* under state civil liability legislation — specifically, whether the failure to warn was a *necessary condition* of the harm (CTE) suffered by the plaintiff athlete. The section highlights the evidentiary challenges faced by the plaintiff: because a plaintiff's own statement about how he or she would have responded to a warning is generally inadmissible (unless made against the athlete's interest), the athlete must instead prove, on a hypothetical basis, that he or she would have taken ameliorative action — such as retiring from contact sport — had an adequate warning been provided by the SGB.

Section 7.3: The materiality of CTE as a subject of warning

This section reinforces the legal requirement that an SGB under a duty of care must warn of any risk of harm that a plaintiff would consider 'material'. The section notes that the degree of risk need only be minor, provided it would be material to the plaintiff.

Section 7.4: Context: Head trauma yet to be experienced

This section argues that, both practically and legally, a professional premier-level athlete cannot be expected to retire from a contact sport on the mere warning of a risk of CTE without at least having experienced several symptomatic concussions. This proposition is supported by scientific evidence indicating that CTE is almost always caused by exposure to multiple symptomatic concussions and many asymptomatic episodes of head trauma. In other words, as CTE is a 'dose response' disease, there can be no realistic expectation that an athlete's response to the question 'would you have retired from contact sport if warned of the risk of CTE?' would be to retire, unless he or she has already experienced some concussive level of head trauma.

Section 7.5: Means of inferring an athlete's response

This section examines the methodologies available to a court in determining how a player would have hypothetically responded if warned by an SGB of the risk of CTE. While the assessment must focus on the particular plaintiff, the plaintiff's own statement of what he or she would have done is generally inadmissible — unless it is against the plaintiff's interest. In making this determination, the court relies on objective factors, including what a reasonable person would have done in similar circumstances, the plaintiff's overall credibility, and

[2] This thesis proposes that, in relation to causation, the element of factual causation poses the greatest challenge for the plaintiff athlete. As such, due to word limitations, this chapter focuses primarily on that issue. The question of scope of liability is addressed separately in Appendix 3: 'A Brief Consideration of the Scope of Liability'.

extraneous considerations such as deeply held personal beliefs or domestic pressures, including financial obligations.

Section 7.6: Further indicators from which an inference may be formed

This section considers whether a player's reluctance to report on-field concussions, or their willingness to continue participating in contact sport despite experiencing multiple symptomatic concussions — particularly where medical warnings were made — may indicate a preparedness to accept the risk of CTE. This question, however, is largely theoretical in relation to athletes who played prior to 2010, as the risk of CTE was not, as argued, reasonably apparent to athletes during that period. However, the argument can be made on an objective basis that retirement of post-2010 athletes following multiple concussive episodes counters any suggestion that pre-2010 players were willing to accept the risk of long-term harm had they been properly warned of the potential adverse effects on their health and well-being.

Section 7.7: 'Running the risk' or not?

This section poses the question of how, in the latter half of the twentieth century, an ethos that was instilled in players of contact sport by coaches and administrators — that they should be prepared to 'take one for the team' (meaning that they should be prepared to bear physical pain and risk long-term injury to serve the needs of the team) — should be considered in calculating an athlete's hypothetical response to a warning had it been given.

Section 7.8: Conclusion

Where an SGB is duty-bound to warn of a risk of CTE, the failure to do so must be shown by an athlete plaintiff to have been the 'factual cause' of the player continuing to engage in the SGB's competition despite enduring head trauma. This section includes discussion of the elements to be proven by a plaintiff athlete including: proving a subjective response (such as retirement) from objective measures had a warning been given by the SGB; the argued impracticality of expecting a premier-level athlete to retire, short of several symptomatic concussion;; the role of credibility in making a judgment; and how criteria such as financial need, family responsibility and pressure imposed on an athlete by coaches and administrators should be considered.

Appendix 3: A brief consideration of the scope of liability

Although scope of liability was noted above to lie outside the application of this chapter, it is, for purposes of completeness, considered briefly in Appendix 3.

7.2 The law and the athlete's response to warnings

Because an athlete's claim is argued in this thesis to be based on a 'negligent failure to warn', the question of factual causation is determined under state civil liability legislation, the relevant provision of which focuses on the plaintiff athlete's hypothetical response had a warning been

given by the SGB. Section 5D ('General principles') of the *Civil Liability Act 2002* (NSW)[3] provides:

(1) A determination that negligence caused particular harm comprises the following elements:

(a) that the negligence was a necessary condition of the occurrence of the harm (*factual causation*), and

(b) that it is appropriate for the scope of the negligent person's liability to extend to the harm so caused (*scope of liability*).

Section 5D(3) of the *Civil Liability Act 2002* (NSW)[4]provides:

(3) If it is relevant to the determination of factual causation to determine what the person who suffered harm would have done if the negligent person had not been negligent —

(a) the matter is to be determined subjectively in the light of all relevant circumstances, subject to paragraph (b), and

(b) and any statement made by the person after suffering the harm about what he or she would have done is inadmissible except to the extent (if any) that the statement is against his or her interest.

In application of the provision, the High Court of Australia noted in the medical case of *Wallace v Kam* that '[b]ecause [the] determination of factual causation necessarily turns on a determination of what the patient would have chosen to do if the medical practitioner had warned of all material risks, the determination of factual causation is governed by s 5D(3)'.[5] In another High Court case, *Chappel v Hart*, Gaudron J explained that

> a defendant is not causally liable, and therefore legally responsible, for wrongful acts or omissions if those acts or omissions would not have caused the plaintiff to alter his or her course of action. The inquiry as to what the plaintiff would have done if warned is necessarily hypothetical. But if the evidence suggests that the acts or omissions of the defendant would have made no difference to the plaintiff's course of action, the defendant has not caused the harm which the plaintiff has suffered.[6]

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[3] See also *Civil Law (Wrongs) Act 2002* (ACT) s 45(1)(a); *Civil Liability Act 2003* (Qld) s 11(1)(a); *Civil Liability Act 1936* (SA) s 34(1)(1); *Civil Liability Act 2002* (Tas) s 13(1)(a); *Wrongs Act 1958* (Vic) s 51(1)(a); *Civil Liability Act 2002 (WA)* s 5C(1)(a). The 'scope of liability' is considered in Appendix 3.

[4] See also *Civil Liability Act 2003* (Qld) s 11(3); *Civil Liability Act 2002* (Tas) s 13(3); *Wrongs Act 1958* (Vic) s 51(3); *Civil Liability Act 2002* (WA) s 5C(3). The Victorian provision does not include a version of s 5D(3)(b) — evidence by the plaintiff after the event is not admissible.

[5] *Wallace v Kam* (2013) 250 CLR 375, [17] (French CJ, Crennan, Kiefel, Gageler and Keane JJ).

[6] *Chappel v Hart* (1998) 195 CLR 232, [32].

7.3 The materiality of CTE as a subject of warning

In fulfilling its duty of care, a professional SGB must, as a singular entity or through its associated medical staff, investigate and understand the risks of harm in playing its sport and warn its players of any 'material risk' of which it is aware.[7] A material risk is one

> to which a reasonable person … would be likely to attach significance, … [or one] to which [the duty-holder] knows or ought reasonably to know [an affected person] would be likely to attach significance.[8]

Gummow J emphasised in *Rosenberg v Percival* 'that a determination of whether the failure to warn of a risk was causative depends largely on the definition or identification of the risk in question. Central to the identification of the risk are considerations of *degree* and *severity*.'[9] His Honour summarised the path to a conclusion as involving 'consideration of the identification of the risk to the patient, the materiality of that risk, and the causation of the injury she suffered'.[10]

As regards 'degree' and 'severity', most athletes would consider the risk of cognitive damage, destructive behaviour manifestations and ultimately death associated with CTE to be significant. The sequelae of CTE have been catalogued throughout several chapters of this thesis and need not be revisited. Whether athletes cease playing in response to a warning of such risks is a different question.

The material risk is CTE, whether described as such or by any recognised terms — such as *dementia pugilistica* or punch drunk syndrome, as a neurodegenerative disease, or through symptoms and other indicators. The risk of harm, depending on the seriousness of the harm and other circumstances, need only be minor — in *Rogers v Whitaker*[11] a mere 1 in 14,000 chance of materialising was sufficient.

As explained below, much will depend on the fullness of the warning and the extent to which the warning permits the athlete to understand the potentially deadly consequences of repeated head trauma. A warning that is less than complete, or is not understood by the athlete, cannot be accepted as fulfilling the legal obligation.[12]

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7 *Wallace v Kam* (2013) 250 CLR 375, [8].

8 Ibid.

9 *Rosenberg v Percival* (2001) 205 CLR 434, [56]. Emphasis added.

10 Ibid [59].

11 (1992) 175 CLR 479. It should be noted that the plaintiff's repeated concern expressed to her surgeon regarding avoiding adverse risks to her 'good eye' were a major factor in the court's determination.

12 *Rogers v Whitaker* (1992) 175 CLR 479.

7.4 Context: Head trauma yet to be experienced

While accounts of players retiring from contact sport after experiencing what are believed to be the symptoms of CTE or after having been concussed many times are understandable, a different issue of no small deliberative importance is what a player who had *not* suffered symptomatic head trauma would have done in response to a warning of CTE. An SGB could put the question: 'If warned, would you have retired when you first experienced concussive head trauma?' Or: 'You have been concussed once or twice before. If warned, would you have retired there and then?' A negative response may lead to the suggestion that although being warned, the player would have elected to continue playing contact sport and so the failure to warn would not have caused his or her later CTE.

As a matter of reality and practicality, it is very unlikely that a player who reaches the premier division of a contact sport will retire on the mere warning of the risk of CTE without having experienced several symptomatic concussions.[13] In the absence of such experience, the risk of harm is abstract and undefined, and on a personal level may never in fact occur. For a young player convinced of his or her invincibility, the possibility of debilitating cognitive harm may be inconceivable. It is also very unlikely that a player who has experienced one or two (or perhaps more) symptomatic concussions would instantly retire when warned. As explained below, a predictable, reasonable and acceptable response is to rely on 'the science', which indicates that the threshold at which tau begins to leak from the axons of the brain does not occur until the athlete endures multiple concussive blows and, seemingly, experienced hundreds or thousands of sub-concussive episodes.[14] In other words, CTE is a 'does response' to RHT not dissimilar to many practices that may be dangerous depending only when a threshold is exceeded, for example, the taking of many prescription drugs and the athlete is entitled to follow the science.

A more conceptually difficult question is the player's response to a warning that asymptomatic concussion alone presents a risk of progressive cognitive harm. While cognitive damage is not apparent from experiencing asymptomatic head trauma, the player is aware of multiple hits to the body and head causing bruising, damage to facial bones, black eyes, scrapes and cuts, which are all indicative of some level of head trauma. In most cases, an athlete who suffers CTE will have experienced multiple symptomatic concussions — certainly this is the predominant history of those confirmed with CTE at autopsy — and, as logically follows, many more asymptomatic concussions from relatively minor head contact. Tagge et al noted that '[w]hile the preponderance of reported CTE cases include a history of repeated mild TBI

[13] Noted below in several sections are accounts by ex-contact sport athletes who retired after being personally warned by medical staff or others that they should do so, or who retired upon experiencing symptoms of cognitive deficit. This anecdotal account is not, of course, a scientific study.

[14] See Chapter 2: 'The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments'; Appendix 1: 'The Science of Concussion and CTE in Sport'.

[traumatic brain injury], a subset (20% of cases) is notable for absence of prior concussion'.[15] It would seem, though not proven, that incidents of asymptomatic head trauma must number into the many hundreds if not the thousands in an individual to cause the leakage of tau that eventually disrupts brain function. Mike Webster, the National Football League player first found to have tau leakage characteristic of CTE, was reckoned by his legal team to have 'endured twenty-five thousand violent collisions during his career'.[16] Obviously 25,000 head blows does not represent a set threshold after which tau is released from the cells of the brain — a threshold that may well have been reached years before.[17] The legal team's estimate also says nothing of the number of symptomatic concussions Webster may have experienced throughout his career in both junior and premier competitions.

The question is this. Would a plaintiff athlete have retired when warned of the risk of CTE caused by symptomatic and asymptomatic head trauma and, if the player did not retire, should he or she be taken to have accepted the risk of CTE? A court may be influenced in its determination by the fact that as CTE is a disease caused by the *accumulation* of head trauma, it is arguably unreasonable to expect a player to discontinue a professional career until at least one, or more realistically several, symptomatic concussions are experienced. That is, while some research shows a single concussive blow can have long-lasting effects in some people, almost all recover from a single concussive blow within a few weeks.[18] Except perhaps on very rare occasions, the research does not indicate that CTE is caused by less-than-multiple concussive and sub-concussive head traumas.[19] The expectation of retirement in such circumstances is tantamount to expecting a motorist not to drive when warned that a car accident may result in harm.

The argument that it is unreasonable to expect an athlete to retire short of experiencing several concussive events — or when warned by medical staff that the limit of tolerance is approaching — is pertinent to the analysis below.

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[15] CA Tagge et al, 'Concussion, Microvascular Injury, and Early Tauopathy in Young Athletes after Impact Head Injury and an Impact Concussion Mouse Model' (2018) 141(2) *Brain* 422, 424.

[16] JM Laskas, *Concussion* (Random House, 2015) 105.

[17] ML Lipton et al, 'Soccer Heading is Associated with White Matter Microstructural and Cognitive Abnormalities' (2013) 268(3) *Neuroradiology* 850, 854.

[18] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 710.

[19] AT Tysvaer, 'Head and Neck Injuries in Soccer: Impact of Minor Trauma' (1992) 14(3) *Sports Medicine* 200.

7.5 Means of inferring an athlete's response

As a plaintiff's personal account is inadmissible[20] how, then, is a court to determine what an athlete 'would have done if warned of the risk' of CTE a*fter* suffering harm?[21] For example, would the athlete have stopped playing, sought to mitigate the risk of head trauma[22] or, where practicable, sourced remedial treatments in the belief that this would permit continued play?

7.5.1 The circumstances of the 'reasonable' person

A useful though imperfect tool in inferring a plaintiff's response to a warning is to consider the overall circumstances of the plaintiff and determine how a reasonable person placed in the circumstances of the plaintiff would have responded when warned.

Hutchison J in *Smith v Barking, Havering and Brentwood Health Authority* suggested that where facts indicate a reasonable person would have 'assented' to the risk of harm, 'extraneous or additional factors' should then be considered as drivers of that assent, including 'religious or some other firmly held convictions; [or] particular social or domestic considerations justifying a decision not in accordance with what, objectively, seems the right one'.[23] It might be observed that these convictions or values may be tightly held and, on a psychological level, not easily surrendered.

In *Woods v Multi-Sport Holdings*, Gleeson CJ said that the obligation to warn is 'ultimately a question of fact' determined by 'what a reasonable person, in the position of the defendant, would do by way of response to the risk'.[24] McHugh J stated similarly in *Rosenberg v Percival* highlighting several approaches including inference from objective facts, reject evidence of a plaintiff if not credible or where reliability of evidence is too poor to accept, or decide a particular plaintiff may have heeded a warning although others may not. His Honour's statement is worth quoting in full:

> It follows from the test being subjective that the tribunal of fact must always make a finding as to what *this patient* would have done if warned of the risk. In some cases where there is no direct evidence as to what the patient would have done, the judge may infer from the objective facts that the patient would not have undergone the procedure. In exceptional cases, the judge may even reject the patient's testimony as not credible and then infer from the objective facts that the patient would not have proceeded. The judge might find, for example, that the patient was a person whose general

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[20] *Civil Liability Act 2002* (NSW) s 5D(3) (and see equivalent provisions in other jurisdictions (n 4)); see also *Rosenberg v Percival* (2001) 205 CLR 434, [45].

[21] *Rosenberg v Percival* (2001) 205 CLR 434, [25].

[22] For discussion, see BW Benson et al, 'What Are the Most Effective Risk-reduction Strategies in Sport Concussion?' (2013) 47(5) *British Journal of Sports Medicine* 321.

[23] *Smith v Barking, Havering and Brentwood Health Authority* (1994) 5 Med LR 285, 289; referred to in *Rosenberg v Percival* (2001) 205 CLR 434, [89], where Gummow J stated that 'reference to the giving of particular weight by the tribunal of fact to what [Hutchison J] called "the objective assessment" should not be taken too far'.

[24] *Woods v Multi-Sport Holdings* (2002) 186 ALR 145, [39], [41].

> credibility was so poor that no reliance could be placed on that person's oral evidence. Yet, notwithstanding the rejection of the patient's oral testimony, the judge might infer that nevertheless *this patient* would not have undergone the procedure.[25]

Kirby J in *Rosenberg v Percival* spoke of the relevance of a plaintiff's reasonable reaction to warnings:

> If a reasonable person would have undergone treatment, regardless of disclosure, then in the absence of personal characteristics or circumstances which would explain a refusal, it must be difficult for a court to conclude that the plaintiff would have rejected the treatment no matter what the plaintiff now genuinely believes that he or she would have done.[26]

In the same case, Gummow J approved of the trial judge's use of certain objective criteria to determine subjective intent, including 'the low probability of the risk occurring, the fact that the likely consequences were not very severe, the desirability of the treatment[,] … the respondent's [individual] awareness … of the risks inherent in any surgery … [and the absence of] substantial fears or concerns that could counter these objective considerations',[27] no matter what the respondent genuinely believed she would have done.

Nonetheless, as McHugh J remarked:

> What a reasonable person would or would not have done … will almost always be the most important factor … [but that] is not conclusive. If the tribunal of fact … accepts the evidence of the patient as to what he or she would have done, then … that is the end of the matter.[28]

The reasonable person in this case is the reasonable athlete informed of the risk of CTE in playing contact sport. If one looked only at the debilitating harm, then almost certainly a reasonable athlete would retire; but other factors — such as the relative rarity of CTE and, at times, a deterministic attitude of some athletes — must also be considered. Furthermore, financial need may motivate the player to risk contracting CTE; alternatively, depending perhaps on disposition, financial concerns could also impel retirement motivated by the fear being unable to work into middle or old age. These factors are considered in more detail below. Of course, the circumstances of each individual player are — slightly or greatly — different; and, as Gummow J noted, the psychology of reasonable people may also differ, leading to different outcomes. There can also be little doubt that at least in the mind of the general public that the apparent willingness of some contact sport athletes to endure extreme injury and keep

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[25] *Rosenberg v Percival* (2001) 205 CLR 434, [25]. Original emphasis. The case concerned a nurse who underwent a surgical procedure and subsequently suffered from 'severe temporomandibular joint' complications, including 'chronic (ie permanent), severe and disabling pain'. Although the surgeon did not warn of the risk of these complications, he was found not negligent on the matter of causation as the respondent nurse would, according to the judgment, have proceeded with the surgery even if warned.

[26] Ibid [158], quoting Shane S Monks, 'The Concept of Informed Consent in the United States, Canada, England and Australia: A Comparative Analysis' (1993) 17(2) *University of Queensland Law Journal* 222, 233.

[27] *Rosenberg v Percival* (2001) 205 CLR 434, [91].

[28] Ibid [24].

playing indicates, certainly unfairly where the determination is based on an individual response, a preparedness to take a risk.

7.5.2 Inferring the athlete's response through objective indicators

The essence of s 5D(3) of the *Civil Liability Act 2002* (NSW) is to infer subjective intent from objective indicators. In *Commissioner of Main Roads v Jones*, for example, Gleeson CJ drew an inference that the plaintiff speeding motorist would not have slowed down if the existing speed sign had shown a slower speed, because 'he appeared to have ignored speed signs altogether'.[29]

To accurately infer an athlete's response had a warning been given, the assessment must be made as if a dutiful SGB had been the warning players of the risk of CTE since at least the 1970s, if not before. An assessment should not be made as it the player concerned had become aware of the risk for the first time, abruptly and without a culture of concern having been developed over previous decades. The point being that a player who enters a sport environment where knowledge of neurodegeneration from RHT is an established risk accepted by players, and where the symptoms of CTE had been witnessed in afflicted players, is far more likely to respond to accumulating head trauma by retiring.

How then would '*this athlete*', to paraphrase McHugh J in *Rosenberg v Percival*,[30] have responded to a warning by a sporting organisation of the risk of developing CTE in playing contact sport? While the full spectrum of personal response by an athlete had he or she been warned of the risk of CTE is not possible to itemise, several factors may be useful in drawing an inference, including: whether the athlete has a record of hiding symptomatic concussions; the athlete's previous responses to warnings of harm; whether the athlete is a risk-taker in life; whether the athlete has a general concern for his or her health and well-being; the financial position of the athlete; alternative forms of employment available to the athlete and their pay scale; career opportunities available to the athlete at retirement and whether it is important for the athlete to maintain his or her cognitive ability to be able to pursue these; the athlete's family responsibilities, including the number of children and any cultural expectations of caring for extended family; whether the athlete has a record of 'taking one for the team'; whether the athlete has the need to remain attached to a team environment; whether the athlete has a love of attention and fan adulation; the likelihood of representative honours; an absence of other meaningful pursuits; the observable absence of CTE in most long-retired players; the relatively slight risk of CTE; an inability to earn a high income outside professional sport (including payments for endorsements); ongoing financial commitments such as house payments; and, indeed, a complete insouciance as regards the risk of CTE.

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[29] *Commissioner of Main Roads v Jones* (2005) 215 ALR 418, [10].

[30] (2001) 205 CLR 434, [25]: see **[7.5.1]** above.

As the plaintiff athlete must demonstrate what means he or she would have employed to ameliorate the risk of CTE had he or she been warned, it should be noted that as the quantity of head trauma accumulates towards the threshold for CTE, any action short of retirement is unlikely to be successful in preventing the disorder. A similar point can be made with respect to devices thought to offer relief. According to Benson,

> [no] new valid, conclusive evidence was provided [in this review] to suggest the use of headgear in rugby, or mouth guards in American football, significantly reduced players' risk of concussion. No evidence was provided to suggest an association between neck strength increases and concussion risk reduction.[31]

7.5.3 The question of credit

A further matter of no small importance is the relevance of character and credit in the assessment of how a plaintiff would respond if warned. McHugh J noted in *Rosenberg v Percival* the important of a witness's credit, stating further that the inference of a response if warned 'would ordinarily be based not only on the objective facts but also on the tribunal's assessment of the general character and personality of the [plaintiff]'.[32]

Much obviously depends on the judge's assessment of the plaintiff's credibility. Indeed, such may be the overriding consideration. As McHugh J remarked further in *Rosenberg v Percival*:

> If the [plaintiff] is believed, he or she succeeds even though the objective facts point the other way. If the evidence of the [plaintiff] is rejected, he or she carries the heavy evidentiary burden of persuading the court to make a favourable finding on the causation issue solely by reference to the objective facts and probabilities.[33]

Of course, the character of a plaintiff is purely personal and cannot be discerned by reference to any group to which he or she belongs, such as a team or club in the case of an athlete.

7.6 Further indicators from which an inference may be formed

Below we consider contact sport athletes' 'reluctance to report' the experience of concussion and head trauma. The examples provided are recent in publication and so occurred in the post-2010 era when, as proposed, CTE would reasonably be considered a risk of contact sport. In other words, the risk of increasingly deliberating cognitive harm in the form of CTE was known and could weighed by a player against other factors in deciding whether to play on or retire.

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[31] BW Benson et al, 'What Are the Most Effective Risk-reduction Strategies in Sport Concussion?' (2013) 47(5) *British Journal of Sports Medicine* 321, 321.

[32] (2001) 205 CLR 434 [25].

[33] Ibid [44]. The trial judge stated that 'it very rapidly became apparent that the [respondent] was most anxious to tell her story in a way in which she thought would benefit her case and to play down anything that she thought might be to the contrary … and I was far from satisfied that the [respondent] was a reliable witness': at [51].

Could it be argued that the response of post-2010 era players who elected to retire could be said to inform in a general sense of a how a pre-2010 player may have responded if warned of the harm of CTE? Perhaps at best it would inform that the apparent cavalier attitude to injury seen in the pre-2010 era may be put down to a failure to warn of the risk of CTE.

7.6.1 A 'reluctance to report'

How an athlete responds to experiencing symptomatic concussion may be a fact in inferring the athlete's response had he or she been warned on the risk of CTE. Remaining on the field, not reporting head trauma to medicos, not seeking treatment could be argued as a willingness to accept the risk of CTE or at least a cavalier attitude to possible harm. Fastidiousness in following medical advice may indicate a real concern. Again, a reluctant or fastidious response is but one fact that goes towards an inference of 'acceptance or risk' — one of the many 'rays of light' that singularly reveal little but collectively 'clear away the darkness.'[34] As, according to statute, the matter 'is to be determined subjectively in the light of all relevant circumstances' the studies offer no more than an indication of the response of some athletes to warnings of CTE-like harm.

It must also be emphasised that the results of the research papers referred to below are based on groups of athletes acting as 'samples' in a scientific study and do not represent the views of the individual player — the test of response being subjective. Furthermore, they may not represent the prevailing attitudes of contact sport athletes in previous decades.

In the inquiry into the death of Australian Football League (AFL) player Danny Frawley, the Victorian State Coroner referred to a survey conducted by the AFL Players Association, which showed that

> in 2019, 6% of respondents reported experiencing a concussion that they did not report to a medical practitioner; 7.9% of respondents reported continuing to train and/or play after a concussion and not seeking medical attention; while in 2017, 10% of respondents reported continuing to train or play without seeking medical attention after a concussion.[35]

Coroner Spanos made no comment on these statistics and there was no suggestion that Frawley had continued to play and train following a concussion or did not seek medical advice. Had Frawley done so after being warned of the risk of CTE the suggestion could be made of a fact indicative of a willingness to accept that risk.

In the *Journal of Science and Medicine in Sport*, a Sports Medicine Australia publication, Tadmor reported on the results of a survey of 422 Men's and Women's Super League football

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[34] *Belhaven and Stenton Perlerage* (1875) 1 App Cas 278, 279.

[35] In the Coroners Court of Victoria at Melbourne, Finding into Death, Daniel Frawley, Court Reference: COR 2019 4895, 23 February 2021, [90] (Coroner Paresa Spanos) <www.coronerscourt.vic.gov.au/sites/default/files/2021-02/Finding%20D%20Frawley.pdf>. Footnote omitted.

(soccer) players on their attitudes to concussion.[36] The two most common reasons for underreporting concussion were 'didn't want to be ruled out of a match (35%)' and 'didn't want to let down the team' (24%)'.[37] On the other side of the coin, '57% of players were concerned about the potential long-term implications from concussion, and 11% of players would encourage their/family members' children to not play rugby league'.[38] Pearce reported in the international publication *Journal of Sports Sciences* that although some individuals recognised the risk of long-term impairment from a concussive injury, their 'responses contradicted their beliefs, such that they reported that they would risk playing or training with a concussion'.[39]

A study by Delaney in 2018 sought to understand why professional 'grid iron' footballers in Canada '"hid", or decided not to volunteer, their [concussion] symptoms to medical staff'.[40] A total of 454 male players (including 309 — 46.7% of — players in the Canadian Football League) responded to an anonymous questionnaire. The results showed: 'One hundred [and] six of the 454 respondents (23.4%) believed they had suffered a concussion during their previous football season and 87 of the 106 (82.1%) did not seek medical attention for a concussion at least once during that season.'[41] The most common reasons given for not seeking medical attention were: 'Did not feel the concussion was serious/severe and felt you could still continue to play with little danger to yourself' (49 of the 106); 'You felt that you would be removed from the game by the medical staff and you did not wish this to happen' (42 of the 106); 'Fear of letting the team or teammates down by being removed from the game' (40 of the 106); 'Fear that being diagnosed with a concussion would result in your missing future games or practices' (41 of the 106); 'Fear that being diagnosed with a concussion would affect your standing with the current team or future teams' (31 of the 106); and 'Fear of being isolated from the team or teammates by being removed and having to take time off from the team' (15 of the 106).[42] Delaney stated that '[t]he underreporting of concussions is similar to other

[36] D Tadmor et al, 'I Won't Let You Down; Why 20% Of Men's and Women's Super League Players Underreported Suspected Concussions' (2023) 26(12) *Journal of Science and Medicine in Sport* 688. Several of the researchers involved in the study were Australian.

[37] Ibid 688.

[38] Ibid.

[39] Alan Pearce et al, 'Do as I Say: Contradicting Beliefs and Attitudes towards Sports Concussion in Australia' (2017) 35(19) *Journal of Sports Sciences* 1911, 1915.

[40] JS Delaney et al, 'Why Professional Football Players Chose Not to Reveal Their Concussion Symptoms During a Practice or Game' (2018) 28(1) *Clinical Journal of Sport Medicine* 1, 1.

[41] Ibid.

[42] Ibid 5–7.

athlete populations [including ice hockey, soccer and lacrosse] and general emergency department patients'.[43]

Of note, the university student cohort displayed a similar insouciance: 72 of the 92 who believed they had suffered a concussion in the previous year (78.3%) did not report it. The most common reason for not doing so was: 'Did not feel the concussion was serious/severe and felt you could still continue to play with little danger to yourself.'[44]

Can it be argued that the studies cited above represent a common response to the risk of CTE across all eras — including the era prior to *circa* 2010?

Although these studies include players from a different time and from different countries, there appears to be a significant percentage of players in each era who were reluctant to report concussive head trauma to medicos and officials. Nonetheless, to conclude a willingness to accept the risk of CTE on this fact alone would be misplaced. In the era prior to 2010 players were not obliged to report concussive head trauma, records were unlikely to be kept or have been lost, and — most importantly — as the risk of CTE had not been conveyed to players, reluctance to report was not relevant to an acceptance of such a risk. Moreover, as CTE is a disease of multiple concussions rather than one or two, a reluctance to report one concussion does not mean that a player will fail to report subsequent ones. In contrast, from the perspective of this chapter, however, the decision of a player of the pre-2010 era to stay on the field after being concussed or knocked out is meaningless unless they had been informed of the risk of CTE.[45]

7.6.2 Responses of those experiencing multiple incidents of head trauma

An athlete's response to experiencing multiple bouts of head trauma is also a relevant indicator of intent to endure the risk of CTE. The athletes discussed in this section participated in the post-2010 era and been warned of the risk of CTE. Some chose to retire, and others to continue to play. Dozens of examples are available through the media. A few only are chosen here to illustrate a possible reaction to a warning of CTE. And while media reports are not scientific studies based on autopsy evidence, a player's response to the fear of CTE can be found in few

[43] See also JS Delaney et al, 'Why University Athletes Choose Not to Reveal their Concussion Symptoms During a Practice or Game' (2015) 25(2) *Clinical Journal of Sport Medicine* 113; E Kroshus et al, 'Understanding Concussion Reporting using a Model Based on the Theory Of Planned Behavior' (2014) 54(3) *Journal of Adolescent Health* 269; JK Register-Mihalik et al, 'Using Theory to Understand High School Aged Athletes' Intentions to Report Sport-Related Concussion: Implications for Concussion Education Initiatives' (2013) 27(7–8) *Brain Injury* 878.

[44] JS Delaney et al, 'Why Professional Football Players Chose Not to Reveal Their Concussion Symptoms During a Practice or Game' (2018) 28(1) *Clinical Journal of Sport Medicine* 1, 7.

[45] Sport culture at a given time is also relevant Examples include rugby league player John Sattler's decision in 1970 to play almost an entire grand final with his jaw broken in two places, or cricketer Rick McCosker's decision in 1977 to return to bat to save a test match with a jaw that had been broken in the first innings and was being held together with bandages: Alan Whiticker, *Glory Days: The Story of South Sydney's Golden Era* (New Holland Publishers (Australia), 2011) 124.

other places. The purpose of the section, again, is to merely inform as to how a professional athlete may react when confronted with the reality of a serious threat to his or her well-being.

National Rugby League player Liam Fulton, aged 29, retired in 2014 following 'four concussions in six matches', which caused 'headaches, short-term memory loss, [and] moments of confusion'.[46] AFL player Sam Blease, aged 24, retired in 2015 after being concussed three times in 2014 and knocked unconscious the following year; while another AFL player, Matt Maguire, retired the same year at the age of 31 following ongoing symptoms of concussion.[47] Female soccer player Natasha Prior, aged 21, retired 'after suffering her fifth concussion in less than six years', stating that she 'didn't really want to have dementia at the age of 30'.[48] In 2021, while earning $700,000 per season, captain of the Sydney Roosters club and the Australian national rugby league team, Boyd Cordner, aged 29, announced his retirement with two years remaining on his contract 'due to the lasting effects of repeated concussions'.[49]

AFL player Nathan Murphy, aged 24, retired in April 2024 'after getting advice from the AFL's medical concussion panel'. He stated: 'my priority is on my future and ensuring I live a full and healthy life.'[50] The AFL's Max Lynch, also aged 24, retired in August 2023 after a discussion with 'an independent medical panel' due to concussion concerns.[51] Paul Seedsman, another AFL player, retired in August 2023 after being advised to cease all contact sport. Other concussion retirees include AFL players Paddy McCartin, aged 27, and Brad Sheppard, aged 30, 'after multiple concussions posed serious health risk'.[52]

Other contact sport athletes have continued to play despite experiencing repeated head trauma. For example, Australian representative rugby player Berrick Barnes, then aged 24, suffered several head knocks during the 2011 season, stating to the media in April of that year:

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46 N Jeffery, 'Too Many Head Knocks Forced Liam Fulton to Retire from NRL', *The Australian* (Sydney, 11 July 2014).

47 'Lions Defender Maguire Retires after Ongoing Concussion Issues', *AFL* (Web Page) <http://www.afl.com.au/news/2015-06-23/lions-defender-matt-maguire-retires>.

48 P Galloway, 'Australian Research Shows Female Athletes Have a Higher Rate of Concussion and a Prolonged Recovery Time', *ABC News* (online, 2 October 2019) <https://www.abc.net.au/news/2019-10-02/female-athletes-have-higher-rates-of-concussion-than-males-study/11538294>.

49 Nine Wide World of Sports Staff, 'Boyd Cordner Forced into Early Retirement Due to Concussion', *nine.com.au* (online, 13 June 2021) <https://www.nine.com.au/sport/nrl/boyd-cordner-retirement-concussion-sydney-roosters-20210613-p5itaf.html>.

50 'Collingwood Defender Nathan Murphy Retires from the AFL Due to Concussion Concerns', ABC News (online, 16 April 2024) <https://www.abc.net.au/news/2024-04-16/nathan-murphy-retires-due-to-concussion/103728352>.

51 'Hawthorn Ruckman Max Lynch Forced to Retire over Concussion Fears, as Port Faces More Scrutiny over Protocols', ABC News (online, 1 August 2023) <https://www.abc.net.au/news/2023-08-01/max-lynch-retires-from-afl-over-concussion-fears/102675272>.

52 'Brad Sheppard Embraces "Broome Time" as his AFL Career Is Cut Short by Concussions', *ABC News* (online, 26 March 2023) <abc.net.au/news/brad-sheppard-kimberley-move-after-career-ending-concussions/102132132>.

> I have had a couple of concussions in a row, and I've had a history of them. At the moment, I don't feel right. I am not concerned long term at this stage, but it has been brought to the forefront, the risks with head injuries, and it could potentially curtail my career.[53]

A month later, in May 2011, Barnes suffered a further head knock, which was diagnosed as 'footballer's migraine'.[54] In 2016, Barnes, then 29 years old, nevertheless played rugby for the Japanese club Panasonic Wild Knights.[55]

Australian rugby league player Mario Fenech, who played in the 1980s and 1990s, 'admitted publicly [in 2014 that] he had suffered brain damage during his 15 seasons of first grade [rugby]', and that he had been prescribed with the dementia drug Aricept.[56] He said: 'I suffered significant head knocks as a player. I would've been knocked out eight or nine times a season … and I would never leave the field. You know why? Because I was a lunatic.'[57] The introduction of concussion protocols was seen as worthwhile by Fenech, who said:

> If you've had a head knock you should leave the field. I wish this was around when I was playing. … Looking back, I was extremely concerned about my health because I had suffered quite a number of brain injuries. … But it's important the next generation of players do not have to suffer the brain injuries that I have.[58]

Fenech seemed to indicate that information about the risk of CTE may have induced him to take ameliorating action, although retirement was not mentioned. However, in keeping with the rationale behind the rule that self-serving evidence may not be given 'after the event', such a comment may be less than accurate.

7.7 'Running the risk' or not?

What is to be made of athletes who were informed of the risks associated with RHT, but continued to play contact sport? The simple response of a defendant SGB is likely to be: 'The player was prepared to run the risk.' And yet 'running the risk' may not provide the complete

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53 Jamie Pandaram, 'I Don't Feel Right: Concussions Worry Barnes', *Sydney Morning Herald* (online, 6 April 2011) <http://www.smh.com.au/rugby-union/super-rugby/i-dont-feel-right-concussions-worry-barnes-20110405-1d2zb.html>.

54 Jamie Pandaram, 'Hits and Memory Loss but Barnes Clear of Serious Concussion', *Sydney Morning Herald* (online, 25 May 2011) <http://www.smh.com.au/rugby-union/union-news/hits-and-memory-loss-but-barnes-clear-of-serious-concussion-20110524-1f2jl.html>. A 2020 article in *The Complete Soccer Guide* explained: 'The term "Footballer's Migraine" has been around for about a decade, but very little is concretely understood about it. Loosely described as something similar to a cluster headache, Footballer's Migraines are brought on by repeated blows to the head, whether by contact with the ball or other collisions over the course of play': Karoline Gore, 'Footballer's Migraine: What It Is and How to Deal With It', *The Complete Soccer Guide* (online, 9 September 2020) <https://completesoccerguide.com/footballers-migraine-what-it-is-and-how-to-deal-with-it/>.

55 <http://www.rugby.com.au/News/NewsArticle/tabid/1699/ArticleID/17314/Barnes-returns-for-title-fight.aspx>.

56 A Webster, 'Mario Fenech Opens Up about the Brain Damage He Suffered as a Player', *Sydney Morning Herald* (online, 1 February 2016) <https://www.smh.com.au/sport/nrl/mario-fenech-opens-up-about-the-brain-damage-he-suffered-as-a-player-20160201-gmiryn.html>.

57 Ibid.

58 Ibid.

answer. While the issuance of a warning is perhaps the final position of a defendant in avoiding liability, issues such as adequacy of warnings, repetition of warnings over the passage of time and the vulnerability of athletes to the wants of coaching staff and the team, all have the potential to compromise the usefulness of a sporting organisation's warnings.

On this point, a research paper by Cusimano on players aged 13–15 revealed material concerns:

> [C]oaches often instilled fear in their players and expressing any symptoms of concussion were seen as a weakness and hindrance to the team. A parent of an ice-hockey player observed, 'The effect of that coach on these kids … my kids are still struggling with the fear of being yelled at, and the fear of being humiliated and the fear of being degraded if they make a mistake on the ice'.[59]

A player who reports a symptomatic concussion may fear that 'their standing in the team or future teams' would be affected, or that a diagnosis of concussion would result in 'negative repercussions from the coach or coaching staff', or would leave the player 'isolated from the team or teammates', or would mean they would 'miss future games' or be 'letting team mates down'.[60] These fears may overpower concerns regarding cognitive well-being. While a player may wish to report the occurrence of head trauma, or to withdraw from play in the weeks following a severe concussion, the exigencies of the team and the pressures of the coach and fellow players may well overbear such desire.

There can be little doubt that athletes, despite an imperious physical stature, are vulnerable to the influences of important people in their professional lives. Dr John Orchard emphasises the point in the *Medical Journal of Australia* with reference to CTE: 'The problem [of reporting] is compounded by … player reluctance to "let the team down" in acquiescing to being replaced when concussed'.[61] This is reflected in comments of international rugby player Michael Lipman, who was 'was knocked out a staggering 30 times. He noted … that he was part of a culture whereby "if I wasn't completely knocked out, I played on". … [H]e felt bullet-proof, unstoppable on the rugby field.' [62] At age 40, eight years after retirement, Lipman became 'very forgetful, moody, and could get very angry very quickly over trifles'. [63] He was

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[59] MD Cusimano et al, 'Factors Influencing the Underreporting of Concussion in Sports: A Qualitative Study of Minor Hockey Participants' (2017) 27(4) *Clinical Journal of Sport Medicine* 377.

[60] Ibid.

[61] J Orchard, 'Match of the Decade: Risk Management of Concussion Versus High-speed Collisions in the Football Codes' (2015) 203(7) *Medical Journal of Australia* 281, 282.

[62] P Fitzsimons, 'It's At the Stage of Mild Dementia ... and He's Only 40 Years Old', *Sydney Morning Herald* (online, 12 November 2020) <https://www.smh.com.au/sport/it-s-at-the-stage-of-mild-dementia-and-he-s-only-40-years-old-20201111-p56dqf.html>.

[63] Ibid.

diagnosed by Dr Rowena Mobbs of the National Repetitive Head Trauma Initiative with mild dementia.[64]

It must be remembered that athletes now suffering reported symptoms of CTE played in an era when the ethos of contact sport, at least for many, was to 'cop it sweet' and to 'tough it out'. Again, the evidence of this is anecdotal, based on statements by athletes recorded in the media who played in the 1960s through to the early 2000s.[65] The point being that players in that era had not been dissuaded from continued participation in contact sport, despite the risk of further harm, by the prospect of long-term irreversible damage. Players would show such attitudes because they had not been informed — or, more importantly, educated — by the SGB as to the potential effects on their health resulting from repeated head trauma.

7.8 Conclusion

An SGB, as argued, has a duty to take reasonable steps to safeguard the health and well-being of its premier players and is obliged to warn those players of any material risk of harm in playing its contact sport.[66] A material risk is one to which a reasonable person would likely attach significance.[67]

CTE is a disease caused by RHT that results in cognitive and neurological disfunction, leading in most cases to death. A reasonable premier-level athlete would likely attach significance to the risk of contracting such a disease. The privilege of whether to act on a warning of the risk of CTE, or disregard it, is that of the athlete who may be affected by the disease.

The difficulty for a court is determining what the athlete would have done if warned — would he or she continue playing and risk contracting CTE, retire or take other ameliorating steps? The test is subjective but excludes the athlete's evidence as to what his or her response to a warning would have been other than where it is against interest, necessitating resort to objective facts.[68]

As a point of context or perspective, it was suggested in this chapter that it is impractical and unreasonable to expect a player to retire if he or she has not experienced concussive head trauma. In such circumstances the pre-conditions of CTE have not been met and so there is no reason to retire. In similar vein, an athlete is arguably entitled to rely on the science regarding the cause of CTE — multiple symptomatic and asymptomatic incidents of head trauma — and not retire until at least 'a few' concussive blows have been experienced. Obviously, there will

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64 Ibid.

65 Accounts are legion and well known: see, eg, the examples above and at n 45.

66 *Wallace v Kam* (2013) 250 CLR 375, [8].

67 Ibid.

68 Section 5D(3)(b) of the *Civil Liability Act 2002* (NSW); *Chappel v Hart* (1998) 195 CLR 232, [32].

be debate on what number is reasonable, in which case a court will benefit from expert testimony. And yet, given that most contact sport players will suffer concussive head trauma across a career, any argument of an SGB that an athlete who experienced a single concussion, or perhaps a few concussions, should retire is inconsistent with the SGB's broader interests. A premier-level competition has a natural turnover of players each year, some reaching the end of their career others due to injury, and can ill afford to exclude players, both experienced and rookies, on the basis of having experienced concussion.

As an athlete's testimony after suffering harm as to what he or she would have done if warned is not admissible, other evidence must be presented from which the court may infer subjective intent. Suggested objective indicators include the financial position of the player at the time, available alternative employment and family responsibilities, among many others. The character or personality of the player is relevant and may 'cut both ways'. For example, one athlete who is concerned about his or her family may risk contracting CTE to better provide for them financially into the future, while a different athlete may consider that longevity of life is a better means of meeting family needs.

How a reasonable person in the position of the plaintiff athlete would respond to a warning had it been given is a prime consideration in a court's determination. Although the assessment is that of the individual plaintiff athlete, reasonableness implies a level of objectivity. There is difficulty, however, in discerning who exactly is the 'reasonable contact sport athlete'. Logic suggests that a reasonable contact sport athlete who has experienced a number of concussive episodes should retire upon a warning of CTE, and yet many demonstrably do not, or have not.

Again, the response of an athlete had he or she been warned of the risk of CTE is fundamentally tied to the substance and fullness of the warning given by an SGB, and whether the SGB — through its various officials — downplayed the risk. Most importantly, will a court accept an athlete's claim, discerned from the factors discussed above, that he or she would have retired if warned This is, of course, an individual assessment concerned with the athlete's character and evidence supporting the assessment.

Chapter 8:
The Question of 'Generic Capacity': Does the SGB's Contact Sport Cause CTE?

8.1 Introduction

The previous chapter, Chapter 7, concerned the question of factual causation by an SGB's failure to warn of the risk of CTE.

Chapter 8 concerns the question of a plaintiff athlete proving to the legal standard the fact that the contact sport administered by the SGB has the capacity to cause CTE.

The next chapter, Chapter 9, deals with the characterisation of the risk of CTE as 'obvious risk' under state civil liability legislation and other defences.

In a 'failure to warn' case, a plaintiff must prove not only that the defendant's negligence in not warning of a material risk of harm caused the plaintiff to engage in an activity that he or she would have avoided if warned, but also that the activity that *was* engaged in had the capacity to cause the harm suffered by the plaintiff.[1]

As such, a plaintiff athlete must be able to prove on the balance of probabilities that playing the contact sport or type of contact sport that the defendant SGB administers has the 'generic capacity' to cause CTE. Whether a sport has the capacity to do so is a question of fact. Proof requires either direct evidence of causation, such as that revealed at autopsy, or an inference of causation drawn from indirect facts and circumstantial evidence.[2]

This chapter concerns what may be called for descriptive convenience a 'third element' of causation not uncommon in failure to warn cases with a long latency between the stimulus causing harm and the emergence of symptoms of that harm. Stapleton referred to this element

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1 *Amaca Pty Ltd v Booth* (2011) 246 CLR 36.

2 *Holloway v McFeeters* (1956) 94 CLR 470. The facts that go to prove on the balance of probabilities that RHT in playing contact sport causes CTE as discussed as a supplement in Appendix 4.

as the 'generic capacity question'[3] — that is, whether the activity in which the plaintiff engaged can cause the type of injury suffered by the plaintiff. In the present context, whether the 'physical injury' — the symptoms of CTE — are caused by play in the defendant SGB's contact sport (played perhaps years or decades earlier). As Edelman J remarked: '[C]ausation is a concept that establishes a link between a physical event and a physical outcome.'[4]

The relevant 'physical event' is RHT experienced through playing contact sport. The 'physical outcome' is cognitive harm in the form of CTE caused by participation in the governing body's competition.

To begin, for purposes of orientation, with a quotation from the failure to warn case of *Wallace v Kam* heard before the High Court of Australia:

> In a case where a medical practitioner fails to exercise reasonable care and skill to warn a patient of one or more material risks inherent in a proposed treatment, *factual causation* is established if the patient proves, on the balance of probabilities, that the patient has sustained, as a consequence of having chosen to undergo the medical treatment, *physical injury* which the patient would not have sustained if warned of all material risks.[5]

In the passage above two elements are operative in proving 'factual causation'. One, the failure to warn caused the plaintiff to engage in an activity he or she would not have engaged in if warned of the pertinent risk of injury.[6] Two, in engaging in the activity the plaintiff sustained harm — 'physical injury' — that would not have been sustained if he or she had been warned of the risk and had elected not engage in that activity.[7] Clearly, causation of actual harm must be proven, but how to prove where proof of contraction is definitive only at autopsy and the plaintiff is living?

For purposes of certainty, it must be emphasised that proof of the generic capacity to cause harm is a different question to whether the failure to warn caused a plaintiff to engage in an activity that would have been avoided had he or she been warned by the defendant. That is, 'but for' the failure to warn, the plaintiff would not have engaged in the risky activity. This is supplemented with a further 'but for' question: 'But for' the plaintiff's participation in the risky activity, the plaintiff would not have suffered harm. In either case, harm or damage, being the 'gist of negligence', must be proven.[8]

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3 Jane Stapleton, 'Reflections on Common Sense Causation in Australia' in S Degeling and J Edelman (eds), *Torts in Commercial Law* (Thompson Reuters Australia, 2011) 343–4.

4 *Lewis v Australian Capital Territory* (2020) 381 ALJ 375, [151].

5 *Wallace v Kam* (2013) 250 CLR 375, [17]. Emphasis added.

6 Examined in Chapter 7: 'Factual Causation of Harm: Failure to Warn and the Athlete's Response Had a Warning Been Given'.

7 Examined in Chapter 6: 'Proof *In Vivo* of Actual Harm in the Form of CTE: The Question of "Individual Agency"'.

8 *Tabet v Gett* (2010) 240 CLR 537, [23] (Gummow ACJ).

8.1.1 This chapter proceeds as follows

Section 8.2: Failure to warn and the 'but for' test of causation (the generic capacity to cause harm)

This section argues that in a fail to warn case, the question of the causation of physical harm is usually known because it follows clearly and immediately from the impugned event; but where the cause is doubted, the plaintiff must prove physical causation of harm. The recognised legal means of proving the generic capacity of an event to cause harm is through the 'but for' test of causation. It is argued in this chapter that the 'but for' test is applicable to proving causation of CTE through RHT in sport and as a general consequence of RHT.

The section points out that the 'but for' test was utilised by judges of the High Court in *Amaca Pty Ltd v Booth*[9] to determine whether exposure to asbestos was the cause of mesothelioma. As such, this test is argued as appropriate to determining generic causation of CTE through RHT in the playing of contact sport.

Section 8.3: The implications should causation of physical harm *not* have to be proven in a failure to warn case

This section proposes that should the test of causation of physical harm be subsumed into the test of causation by failure to warn, several infelicities in application and injustices would result.

Section 8.4: Multiple causes of CTE and 'material contribution' to harm

This section reviews the application of the doctrine of material contribution to harm in response to an SGB's claim that the symptoms of CTE displayed by a plaintiff athlete were caused by play in a competition other than its own, principally a junior competition or in play in a different premier league. Any competition that exposed the plaintiff to more than a *de minimis* level of RHT will be jointly and severally liable.

This section is supplemented by Appendix 5, which considers facts supporting the player's claim that the SGB's competition was a source of more than a *de minimis* level of head trauma.

Section 8.5: Conclusion

As doubt regarding the cause of a plaintiff athlete's neurological symptoms may be raised given the long latency between the ending of an athlete's career and the initial onset of symptoms, it may be necessary for the plaintiff to prove that playing the SGB's type of contact sport causes CTE. This chapter argued that the accepted methodology for doing so is through the 'but for' test, or, as expressed in legislation, as a 'necessary condition'. The factual means of so proving are discussed at length in Appendix 5.

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9 (2011) 246 CLR 36.

Appendix 5: Multiple Causes of CTE and 'Material Contribution' to Harm

The object of this appendix is to show, primarily by reference to High Court precedent in *Amaca Pty Ltd v Booth*,[10] that in a failure to warn case *the law* pertinent to proving the *generic capacity* of contact sport to cause CTE is the 'but for' test of causation of harm. It is proposed that the scientific methodologies, particularly the Bradford Hill criteria and the general body of medical knowledge, permit a factual assessment to be made that RHT in the playing of contact sport is, on the balance of probabilities, a cause of CTE. The application is reviewed in Appendix 5 as supplementary to the question of law.

8.2 Failure to warn and the 'but for' test of causation (the generic capacity to cause harm)

A plaintiff must be able to prove that the agent claimed to have caused the harm suffered is, in fact, capable of doing so. Proof may be problematic where a long latency in time separates the claimed source of harm from the onset of measurable symptoms. This chapter considers judicial precedent and legal commentary in support of the proposition that the recognised means of doing so is through the application of the 'but for' test of causation, or its legislative equivalent of 'necessary condition'[11] or 'factual causation'.[12]

In the context of CTE, while a necessary element the question of causation of physical harm can be seen as supplementary to the need to prove factual causation due to an SGB's failure to warn its athletes of the risk of CTE.[13] To illustrate the rationale, a plaintiff may be suffering from, say, 'loss of attention and concentration', but displaying those symptoms of CTE does not of itself prove that the harm causing the symptoms was caused by the plaintiff's participation in the activity of which the defendant's failed to warn. As the plaintiff 'always bears the onus of proving … *any fact* relevant to the issue of causation',[14] both forms of causation must be proven by a plaintiff athlete on the balance of probabilities.

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[10] (2011) 246 CLR 36.

[11] *Wallace v Kam* (2013) 250 CLR 375, [16] (French CJ and Crennan, Kiefel, Gageler and Keane JJ). Factual causation is of course a different question to scope of liability.

[12] *Monaghan Surveyors Pty Ltd v Stratford Glen-Avon Pty Ltd* [2012] NSWCA 95, [69].

[13] See Chapter 7: 'Factual Causation of Harm: Failure to Warn and the Athlete's Response Had a Warning Been Given'.

[14] Section 5E of the *Civil Liability Act 2002* (NSW). Emphasis added.

In the failure to warn case of *Wallace v Kam* the High Court of Australia stated:

> The *compensable* damage is … limited to the occurrence and consequences of physical injury sustained by the patient as a result of the medical treatment that is carried out following the making by the patient of a choice to undergo the treatment.[15]

Respected legal scholar Stapleton opined that proof of factual causation 'sometimes … involves a *number of steps*' in addition to establishing that the failure to warn caused the plaintiff to engage in an event that caused injury. Stapleton illustrated the approach through the example of a 'failure to warn' case where proof of causation of physical harm was in issue:

> [I]n a failure-to-warn claim against a pharmaceutical manufacturer the plaintiff typically has to establish that the drug is capable of contributing to the relevant outcome (that is, that it has what we might call 'generic capacity') and that it did so in this individual case (which we might call 'individual agency') before the critical factual cause question can be asked about whether the failure-to-warn contributed to the outcome.[16]

Factual causation due to the failure to warn is, as expressed by Stapleton, a different question to whether the event in which the plaintiff engaged is capable of causing physical harm. In the context of this chapter, in addition to 'factual causation' by failure to warn, the plaintiff must also show that the SGB's sport was 'capable of' causing CTE (referred to as generic capacity) and whether in the plaintiff athlete's case it did cause harm (referred to as individual agency).[17] These 'steps', to use Stapleton's words, are reflected in the approach taken in this chapter to questions of the generic capacity of contact sport to cause CTE.[18]

8.2.1 Proving generic capacity: The 'but for' test of harm

While, as Stapleton said, generic capacity must be proven where the capacity to cause harm is in issue, how, and by what legally accepted means to prove that playing a defendant's contact sport is a cause of the plaintiff's symptoms of CTE.

In the absence of a legally affirmed means by which to judge the generic capacity of an activity to cause harm, a court would be forced to speculate, rely on its own opinion resting on personal notions of causation, or simply accept the word of the most persuasive expert witness. In this chapter the essence of the question concerns the legal means of proving whether the sport administered by a defendant SBG possessed the generic capacity to cause CTE.[19] The

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15 *Wallace v Kam* (2013) 250 CLR 375, [9]. French CJ and Crennan, Kiefel, Gageler and Keane JJ.

16 Jane Stapleton, 'Reflections on Common Sense Causation in Australia' in S Degeling and J Edelman (eds), *Torts in Commercial Law* (Thompson Reuters Australia, 2011) 343–4.. Emphasis added.

17 Discussed in Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"'

18 See Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"'; Chapter 7: 'Factual Causation of Harm: Failure to Warn and the Athlete's Response Had a Warning Been Given'.

19 This is a different question as to whether the activity (even where it has the capacity to cause harm) in fact caused harm to the plaintiff. See Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"'.

established legal method for matters of causation is the 'but for' test, whether as a common law test or transposed in state civil liability legislation to a 'necessary condition'.[20]

In *Amaca Pty Ltd v Booth*,[21] a 'failure to warn' case, judgments of the High Court applied the 'but for' test of causation in determining that the inhalation of asbestos fibres manufactured by the defendants were the cause of the plaintiff's mesothelioma. The same approach is applied in other cases where the physical or medical cause of harm is in doubt. For example, in *Seltsam v McGuiness*, which concerned whether exposure to asbestos was the cause of the plaintiff's renal cell carcinoma, Spigelman CJ phrased the question of physical cause as: 'Is the agent capable of causing the disease? — which is sometimes referred to by epidemiologists as "general causation".'[22] The 'but for' test (necessary condition) is argued here as a judicially approved means of proving that RHT in the playing of contact sport has the generic capacity to cause CTE.

8.2.2 The equivalence of 'but for': 'Necessary condition'

The common law 'but for' test used in cases like *Amaca v Booth* is equivalent to the statutory 'necessary condition' test. In *Adeels Palace v Moubarak*, the High Court stated that 'it is necessary to observe that the first of the two elements identified in s 5D(1) (factual causation) is determined by the "but for" test: but for the negligent act or omission, would the harm have occurred?'[23] Here, of course, the consideration is the generic capacity of RHT in the playing of contact sport to cause CTE and how this capacity in determined as a matter of law by the 'but for' test.

8.2.3 Generic capacity to cause harm is foundational to failure to warn of CTE

In most 'failure to warn' cases the incurrence of harm due to the plaintiff's participation in an event is so immediate and so obviously connected with the event in question that there is no doubt as to the cause of the plaintiff's harm. This immediacy is seen commonly in medical cases such as *Rogers v Whitaker*[24] and 'diving cases' like *Nagle v Rottnest Island Authority*.[25] In some matters, however, the latency in time between the accused activity and the onset of measurable physical harm is so long that a defendant rightly expresses doubt as to whether the activity is the factual cause of the plaintiff's injury. To illustrate, in cases such as *Amaca v*

[20] See s 5D(1) of the *Civil Liability Act 2002* (NSW).

[21] *Amaca Pty Ltd v Booth* (2011) 246 CLR 36.

[22] *Seltsam v McGuiness* [2000] NSWCA 29, [22].

[23] *Adeels Palace Pty Ltd v Moubarak* (2009) 239 CLR 420, [51].

[24] *Rogers v Whitaker* (1992) 175 CLR 479.

[25] *Nagle v Rottnest Island Authority* (1993) 177 CLR 423.

Booth,[26] which concerned the contraction of mesothelioma from exposure to asbestos decades before, and *H v Royal Alexandra Hospital*,[27] which concerned infection with HIV revealed by symptoms years after the plaintiff's treatment with blood products, it was necessary for the court to determine whether the physical injury was caused by the accused activity rather than some other cause. In short, if the physical injury of CTE was not caused by participation in the defendant SGB's contact sport, the defendant SGB cannot be liable for the harm being experienced by the athlete.

The very nature of CTE with its long latency is a major factor necessitating proof of generic capacity. In context, the delay between play in contact sport and the realisation of CTE symptoms is generally measured in years and most usually in decades, fostering challengeable doubt as to whether the plaintiff's harm was in fact caused by his or her participation in the event or an encounter with some other stimulus along the way. In addition, possible intervening causes of harm are more likely to present a line of defensive argument, for example, where an athlete retires from the SGB's sport to participate in a different contact sport such as boxing.

8.2.4 The High Court's use of the 'but for' test in failure to warn cases

As proposed, a plaintiff athlete must, in the face of an SGB's denial of physical causation, prove on the balance of probabilities that participating in the SGB's contact sport or type of contact sport had the generic capacity to cause CTE. This proposition finds authority in the High Court of Australia determination in *Amaca Pty Ltd v Booth*.[28]

The case concerned a motor mechanic who, from 1953 to 1983, was exposed in the course of employment to asbestos fibres from brake linings that the plaintiff argued caused the mesothelioma with which he suffered. Two companies, Amaca and Amaba, had manufactured the brake linings. The defendants questioned the 'sufficiency of the evidence' to support the findings of the trial judge that Booth's exposures to the chrysotile asbestos in the brake linings manufactured by the defendants', Amaca and Amaba, had been causes of his mesothelioma.

The issue to be resolved on appeal in *Amaca v Booth* was not that the defendants' failure to warn exposed the plaintiff to a risk he would otherwise have avoided (argument affirmed at trial), but whether exposure to asbestos manufactured by the defendants was a factual medical cause of actual harm, the 'but for' cause, of the plaintiff's mesothelioma.[29] Strictly speaking the question did not concern the generic capacity of asbestos to cause mesothelioma, which was a known cause, but whether the amount to which the plaintiff was exposed was sufficient

[26] *Amaca Pty Ltd v Booth* (2011) 246 CLR 36.

[27] [1990] 1 Med LR 297. See also *Seltsam Pty Ltd v McGuiness* [2000] NSWCA 29, [93].

[28] *Amaca Pty Ltd v Booth* (2011) 246 CLR 36.

[29] The appeal did not turn on whether asbestos was a cause of mesothelioma, but primarily on whether the degree of exposure was a cause of the disease and a material contribution, which is itself a question of causation.

to have caused the plaintiff's condition. Nonetheless, the inquiry concerned the generic question of whether a given level of exposure to asbestos was sufficient to cause mesothelioma. More to the point, the 'but for' test was utilised as the means of proving cause of harm where doubt is expressed by a defendant.

The defendants' appeal in *Amaca* turned on the proposition that the plaintiff's very limited exposure to asbestos 'did not support an inference that asbestos exposure was a *factual cause* of the deceased person's fatal lung cancer.'[30] However, neither the New South Wales Court of Appeal nor the High Court found that the trial judge had erred in law, upholding his Honour's utilisation of the 'but for' test in determining causation of actual harm.

French CJ found that:

> an inference of *factual* causation, as against both Amaca and Amaba, was *open on the evidence* before the primary judge. … [and] The cumulative effect mechanism, accepted by his Honour, implicated the products of both Amaca and Amaba in the development of Mr Booth's disease. The primary judge's interpretation of the expert evidence and his conclusions from it, were open as a matter of law.[31]

Immediately below are statements from the High Court judgments referring to the need to prove causation of physical harm and which affirm the 'but for' test as the operative means of determining the generic capacity of an event of long latency. In fact, the words 'causation' and 'but for' are used in the judgment in respect to actual harm in what was, as said, a failure to warn case.

French CJ made multiple references to the application of factual causation in respect to exposure to asbestos throughout his judgment, all of which support the proposition that proof of causation of actual physical or medical harm is an essential element of a plaintiff's claim of a negligent failure to warn. For example, his Honour stated that,

> Factual causation which can be established by the application of the "*but for*" test is the threshold test for determining whether a particular act or omission qualifies as a cause of the damage sustained.[32]

In *Amaca v Booth*, reference was made to *Amaca Pty Ltd v Ellis*,[33] where factual causation was not proven but where the 'but for' test was applied in the court reaching its determination. The High Court in *Ellis* noted without demurrer that it was the plaintiff's case that a specific state of affairs existed, and '[t]he "specific state of affairs" alleged to exist in this case was "but for" causal connection between the relevant defendant's exposing Mr Cotton to respirable

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30 *Amaca Pty Ltd v Booth* (2011) 246 CLR 36, [40]. Emphasis added.

31 *Amaca Pty Ltd v Booth* (2011) 246 CLR 36, [51]. Emphasis added. The 'cumulative effect mechanism' is examined at length in Appendix 5: 'Multiple Causes of CTE and "Material Contribution" to Harm'.

32 Ibid [47].

33 (2010) 240 CLR 111.

asbestos fibres and his contracting the lung cancer from which he died'.[34] Further, in *Amaca v Booth*, French CJ said:

> Generally speaking, that causal connection is established if it appears that the plaintiff would not have sustained his or her injuries had the defendant not been negligent. … Factual causation does not require that the propounded cause be one link in a chain of causative factors or events.[35]

In *Amaca v Booth*, the causal connection between exposure to the defendants' products and the plaintiff's mesothelioma was sufficiently clear that French CJ stated: 'It is not necessary in this case to consider the application of any modified concept of causation of the kind developed in *Fairchild v Glenhaven Funeral Services Ltd*.'[36] Statements also ensued as to the difference between '[t]he risk of an occurrence and the cause of the occurrence';[37] and, affirming a need to establish causation in addition to that associated with the failure to warn, '[t]he mere existence of such an association or correlation does not justify a statement, relevant to factual causation in law'.[38] In conclusion, French CJ stated:

> The present case proceeds upon the foundation of findings, based on evidence before the primary judge … [that Mr Booth's exposure to asbestos] causally contributed to its development and continuation.[39]

The joint judgment of Gummow, Hayne and Crennan JJ also (as expected) dealt with the issue of whether exposure to asbestos in the defendants' products was to be determined as a matter of 'but for' causation. Relevant statements include the following: 'An inference of fact, concerning the contraction of disease by Mr Booth, … was reasonably open on the evidence';[40] and, with reference to medically assessed causation of harm, 'many issues of causation … lie outside the realm of common knowledge and experience'.[41] Clearly, a medical assessment involves an assessment of whether the plaintiff's disease is associated with the proffered cause of asbestos exposure and not an assessment of a defendant's obligation to warn. Further, 'the general onus which lies upon the plaintiff where the issue of causation lies outside the realm of common knowledge and experience'.[42] Their Honours also stated: 'The "but for" criterion of causation proved to be troublesome in various situations in which multiple acts or events led

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34 Ibid [15].

35 (2011) 246 CLR 36, [47], quoting *March v E & MH Stramare Pty Ltd* (1991) 171 CLR 506, 514.

36 (2011) 246 CLR 36, [48], referring to *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32.

37 (2011) 246 CLR 36, [41].

38 Ibid [43].

39 Ibid [53].

40 Ibid [63].

41 Ibid [67].

42 Ibid [69].

to the plaintiff's injury.'[43] These comments are explicable on the basis that the proffered disease is not apparent from the nominated stimulus or where the defendant challenges the plaintiff on proof.

Heydon J, in a dissenting judgment, also referred to the need to prove physical causation of harm; for example, '[o]ne causation difficulty created by these facts is that …';[44] 'to prove causation against Amaca … the plaintiff had to prove two things';[45] and '[the defendants] contended, as they had to, that there was no evidence to support [a particular] finding, which was an essential step to the trial judge's conclusion that the plaintiff's exposure to the asbestos in brake linings materially contributed to his mesothelioma'.[46]

The statements of his Honour refer not to participation in an activity that the plaintiff would have avoided if warned, but to proof of the physical manifestation of mesothelioma as the physical result of that failure to warn.

8.3 The implications should causation of physical harm *not* have to be proven in a failure to warn case

Where a defendant demands a plaintiff prove harm connected to the activity the plaintiff undertook because he or she was not warned by the defendant of a risk of harm that materialised, it is necessary that proof be provided.[47] In such cases any acceptance of causation based on mere participation in the suspected event alone would be founded on an assumption, and assumption is not, of course, a recognised legal standard of proof.

Moreover, if a failure to warn alone established causation of physical harm, there would be no need to independently prove that the plaintiff in fact suffered physical harm. Consider for purposes of illustration the infelicities and tactical adjustments that would arise in a case like *Amaca Pty Ltd v Ellis*,[48] in which the sole inquiry before the court concerned the cause of lung cancer where the plaintiff had been exposed to both asbestos in cement pipes during employment and tobacco smoke as a smoker. The plaintiff sued the manufacturer of the asbestos pipes. The High Court stated that the issue was not the existence of a duty of care nor breach, but 'whether it had been shown to be more probable than not that exposure to asbestos was a cause of Mr Cotton's death'.[49] The plaintiff's case consisted mainly of epidemiological

[43] Ibid [70]. This question is dealt with in Appendix 5: 'Multiple Causes of CTE and "Material Contribution" to Harm'.

[44] Ibid [102].

[45] Ibid [103].

[46] Ibid [105].

[47] Section 5E of the *Civil Liability Act 2002* (NSW).

[48] (2010) 240 CLR 111.

[49] *Amaca Pty Ltd v Ellis* (2010) 240 CLR 111, [11]. Ellis was the executor of Mr Cotton's estate.

studies to establish asbestos as a probable causative agent. The court found against the plaintiff, stating:

> The issue here is whether one substance that can cause injury did cause injury. … Knowing that inhaling asbestos can cause cancer does not entail that in this case it probably did.[50]

Suppose also that, under a new law, there was no requirement to prove physical causation where the sole accusation was a failure to warn of a risk of harm. Suppose further that the plaintiff amended his claim to plead only the defendant's failure to warn of the risk of mesothelioma. Proof of the generic capacity of asbestos dust to cause mesothelioma under this new law is now superfluous. In this illustrative example, the necessity of proving that the substance argued to have caused injury *did* in fact cause injury, to use a word operative in the High Court decision in *Ellis*, is not required. The proposition that proof of physical harm may be dispensed with produces a clear injustice.

8.4 Multiple causes of CTE and 'material contribution' to harm

This section briefly considers a defendant's argument that the cause of a plaintiff athlete's CTE was not play in the contact sport administered by the defendant SGB, but in a different competition; for example, in a school competition or for a different SGB. This argument claims that the other competition was the sole cause of the plaintiff athlete's contraction of CTE. The counter-argument proposed in this section is that each contact sport in which the plaintiff participated made, as recognised in law, a 'material contribution', such that all SGBs are jointly and severally liable.

Appendix 5 provides a full explanation of the facts underpinning the application of material contribution to a plaintiff athlete's case against an SGB.

'Material contribution' (excluding the 'exceptional case') must be proven by a plaintiff as a 'necessary condition'.[51] In *Merck Sharp & Dohme (Australia) Pty Ltd v Peterson*,[52] the plaintiff claimed that the anti-inflammatory drug Vioxx caused a heart attack. Several other risk factors were listed by the court, including obesity and hypertension. Allsop P found causation had not been proven, stating that

> [t]he Court must proceed on the footing that for [the plaintiff] to show that the consumption of Vioxx materially contributed to his MI [myocardial infarction] … he is obliged to show that his consumption of Vioxx was a *necessary condition* for the occurrence of the heart attack. … To say that the consumption of Vioxx was, for example, 'in the mix' of possible causes is not enough in this regard.[53]

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[50] Ibid [68]. Original emphasis.

[51] See the following quote of Allsop P.

[52] (2011) 284 ALR 1.

[53] Ibid [104]. Emphasis added.

Material contribution was the basis of the House of Lords determination of liability in *Bonnington Castings Ltd v Wardlow*.[54] The relevant facts centred on the accumulation of silica particles from two different sources that each contributed to the plaintiff's harm, an arguable parallel to CTE caused by the accumulation of RHT by the material contribution of playing contact sport in more than one competition.

In *Bonnington*, the plaintiff was employed by the defendant on the factory floor over an eight-year period, during which he contracted pneumoconiosis from inhaling minute particles of silica. Two sources of silica leakage were identified: (1) from 'swing grinders' with blocked extractors, for which the employer would be liable in negligence; and (2) from pneumatic hammers, for which the employer would not be liable: 'As there was no known means of collecting or neutralising this dust, and as it is not alleged that these machines ought not to have been used, there was no breach of duty on the part of the [defendant] in allowing this dust to escape into the air.'[55] It should be noted that the harm to the plaintiff was not categorised as due to 'multiple sufficient causes' (where any cause is sufficient in itself to cause the total harm); rather, in the words of Lord Keith of Avonholm,

> the particles inhaled are acting cumulatively, and I think the natural inference is that had it not been for the cumulative effect the [plaintiff] would not have developed pneumoconiosis when he did and might not have developed it at all.[56]

Lord Reid recognised similarly that 'pneumoconiosis is caused by a gradual accumulation in the lungs of minute particles of silica inhaled over a period of years'; consequently, the disease 'is caused by the whole of the noxious material inhaled and, if that material comes from two sources, it cannot be wholly attributed to material from one source or the other'.[57]

In continuing, his Lordship revealed the logic to the concept of 'material contribution':

> It appears to me that the source of [the plaintiff's] disease was the dust from both sources, and the real question is whether the dust from the swing grinders *materially contributed* to the disease. What is a material contribution must be a question of degree. A contribution which comes within the exception *de minimis non curat lex* is not material, but I think that any contribution which does not fall within that exception must be material. I do not see how there can be something too large to come within the *de minimis* principle but yet too small to be material.[58]

Notably, his Lordship considered that the contribution of the swing grinders to causing a 'guilty atmosphere'[59] exceeded the *de minimis* and was thereby material:

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[54] [1956] AC 613.

[55] Ibid 615.

[56] Ibid 626.

[57] Ibid 621.

[58] Ibid. Emphasis added.

[59] Ibid 617.

> It may be that, of the noxious dust in the general atmosphere of the shop, more came from the pneumatic hammers than from the swing grinders, but I think it is sufficiently proved that the dust from the grinders made a substantial contribution.[60]

An athlete's case of 'material contribution' rests on a medical fact: CTE is a product of cumulative head trauma over many years. As the years of play pass, the athlete moves from junior to senior ranks accumulating multiple concussive and sub-concussive blows to the head. There is little point in revisiting the many studies cited in this and in other chapters that affirm CTE as a disease caused by RHT. The research studies discussed in Appendix 3, unlike those of previous chapters, do not focus on CTE as a disease, but on the force and power exerted on the head in a contact sport played at all levels.

A 'material contribution' to the causation of the plaintiff's harm occurs where a defendant's negligence contributes, along with the negligence of another or several defendants to the harm suffered by the plaintiff. Liability is not dependent on proof of one defendant's negligence.[61]

This brief section provided a revealing and sufficient explanation of the operation of the law in respect to 'material contribution' and how the doctrine will apply in favour of a plaintiff athlete.

8.5 Conclusion

In a claim of negligence by failure to warn of a risk of harm, causation requires that if a warning had been given the plaintiff would have avoided the harm suffered. In most failure to warn cases the resultant harm is so directly connected to the activity undertaken by the plaintiff that there is no doubt that the activity participated in was the cause of the plaintiff's harm. However, in cases where there is doubt as to whether the accused activity could cause the harm, the plaintiff must provide proof of generic capacity. As considered in detail above, the High Court in *Amaca Pty Ltd v Booth*,[62] a fail to warn case, determined whether exposure to asbestos dust produced by the defendants was the cause of the plaintiff's mesothelioma by applying the 'but for' test of causation.

In terms of a claim that an SGB's failure to warn of the risk of CTE caused a plaintiff athlete's participation in the SGB's premier-level competition, the plaintiff must prove that he or she would not have so participated if warned of the risk of CTE, *and*, if put to proof by a defendant SGB, must also prove that participation in the SGB's type of contact sport had the generic capacity to cause CTE. The accepted means of doing so, as applied in *Amaca v Booth*, is the 'but for' test of causation.[63]

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60 Ibid 622.

61 *Strong v Woolworths* (2012 246 CLR 182, [22].

62 (2011) 246 CLR 36.

63 Discussion of the scientific means by which generic capacity may be proven at law on the balance of probabilities is found in Appendix 4, as a supplement to this chapter.

Chapter 9:
CTE and the Characterisation of 'Risk of Harm': Dangerous Recreational Activity, Assumption of Risk and Other Defences

9.1 Introduction

The previous chapter, Chapter 8, concerned the question of the generic capacity of RHT in the playing of contact sport to cause CTE.

Chapter 9 concerns the characterisation of CTE as an obvious risk of contact sport. An obvious risk, depending on the specific statutory provision applied, excludes a tortfeasor from liability. How a risk is characterised, narrowly, broadly or by type, will determine whether an SGB that failed to warn of the risk of CTE is to be excused of liability.

This chapter also addresses the question as to the date (month and year) that CTE will be deemed to have become an obvious risk of playing contact sport as determined by a court. A plaintiff athlete whose premier-level career began after that date will be taken to have accepted the risk of CTE.

The next chapter, Chapter 10, is the concluding chapter of the thesis.

Premier-level athletes[1] who claim that their SGB was negligent in failing to warn of the risk of CTE or to initiate other concussion-ameliorating protocols will almost certainly be confronted

[1] For examples of current or proposed litigation in which the governing bodies of the relevant contact sports are listed as defendants, see *Rooke v Australian Football League* (Supreme Court of Victoria, S ECI 2023 00969, commenced 14 March 2023); 'Dementia and Sport: Rugby Players Launch Legal Action against Governing Bodies', *BBC Sport* (online, 25 July 2022) <https://www.bbc.com/sport/rugby-union/62290045>.

with a defendant's claim that CTE is an 'obvious risk' of contact sport.[2] Under state civil liability legislation, 'obvious risks' of harm are generally 'liability defeating'.[3]

Two arguments are proposed as central to resolving the question of what constitutes 'obvious risk' in respect of CTE caused in the playing of premier-level contact sport: (1) The first argument relates to the point in time (yet to be determined) at which harm in the form of CTE is deemed by a court to be an obvious risk of contact sport. It must be accepted as a matter of logic that the 'risk of harm' in the form of CTE is today an 'obvious risk' to premier-level contact sport athletes. There was, though, a time when it was not — argued in this thesis as prior to *circa* 2010. The date of this cross-over from ignorance to awareness is crucial to a plaintiff athlete's case. Under civil liability legislation, a claim of negligence by an athlete who had not played premier-level contact sport prior to this date will, all things being equal, be defeated. (2) The second argument relates to the characterisation of the risk of CTE as either 'general' or 'specific' — the more general the characterisation, the more likely it is that the risk of harm will be deemed 'obvious'.

To stress the importance of point (2): should it be accepted by a court that the relevant risk of harm is to be characterised in general terms as 'concussion', 'concussion-related concerns', 'head trauma' or some other form of broad description, there is every likelihood that a defendant SGB will be found not liable in its failure to warn players of the risk of CTE.[4] This chapter argues, however, that such descriptors are too general to properly characterise the true risk of harm in the form of CTE, and that the correct application is one focusing specifically on CTE and its symptoms or — still within the narrow band — a neurodegenerative disease or a close equivalent.

9.1.1 This chapter proceeds as follows

Section 9.2: Legislative provisions: Obvious risk, dangerous recreational activity and assumption of risk of harm

This section considers and explains the operation of state civil liability legislation relevant to the characterisation of CTE as an obvious or not-obvious risk of harm in playing contact sport.

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[2] Indeed, such seems to be a key defensive stance of the Australian Football League (AFL) in the current Max Rooke class action. See *Jarad Maxwell Rooke v Australian Football League* (Supreme Court of Victoria, S ECI 2023 00969, commenced 14 March 2023) Defence, cl 16(b), (d)(i), discussed briefly at **[9.6.1]** below. 'Contact sport' is used as a general term to cover collision sports such as rugby, along with contact and semi-contact sports such as football (soccer) or field hockey.

[3] *Goode v Angland* (2017) 96 NSWLR 503, [185] (Leeming JA).

[4] The basis of an argument that an SGB has a duty to care for the health and well-being of its premier-level athletes is found in the obligation of the SGB to be informed of the risk of CTE and to warn its athletes of that risk. For discussion, see David Thorpe, 'Concussion, 'Chronic Traumatic Encephalopathy, and the Legal Obligation of Sporting Organisations to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks' (2021) 28 *Journal of Law and Medicine* 760.

Section 9.3: An emphasis on ‘obvious risk’

This section explains that emphasis in this chapter is placed on the application of ‘obvious risk’ rather than entering debate on the non-contentious issue of whether a contact sport such as rugby or AFL are classifiable as a ‘dangerous recreational activity’.

Section 9.4: Base questions in contention

This section first explains that the application of ‘obvious risk’ must, as a matter of logic, be confined to a date prior to when a court would deem that the risk of CTE become obvious to a premier-level contact sport athlete in Australia. Second, it explains that the more general the characterisation of the risk of CTE, the more likely it is that the risk of the disease will be deemed ‘obvious’ by a court and defeat an athlete’s claim. Conversely, the more narrow the characterisation of the risk of CTE, the more likely it is that it will be declared ‘not obvious’, and so advantage the plaintiff athlete.

Section 9.5: Dating when the risk of CTE became obvious

This section presents the argument that CTE became an obvious risk to premier-level contact sport athletes in Australia around the year 2010, noting that this date could reasonably be said to have been somewhat earlier or somewhat later without affecting the foundation of the argument. Those whose premier-level career began from the date declared by a court as to when CTE became ‘obvious’ will, under state civil liability legislation and all things being equal, be unable to successfully claim they did not accept the risk of contracting CTE.

The section also points out, importantly, that an SGB with a duty to take reasonable steps to care for the health and well-being of its premier-level athletes is obliged to take proactive steps to monitor for extant and emerging risk of harm, noting that the risk of CTE was recorded in medical journals throughout most of the twentieth century, thereby giving SGBs the capacity to inform themselves of the risk and to warn their premier players of that risk.

Section 9.6: Defining harm: General and specific applications

This section discusses the difference in application of ‘obvious risk’ as between a general or specific characterisation. It argues that CTE is most appropriately characterised as a specific risk or type of risk.

The section also presents an extensive argument that although a risk of harm is more usually described by the conduct that led to the harm, such as falling off a horse, in some cases the correct identification of the risk requires it to be described in terms of the resulting harm caused by the conduct engaged in by the plaintiff, particularly where harm is unlikely to have been foreseen by a plaintiff, such as quadriplegia due to falling while running down a sand dune.

Section 9.7: Obvious to a reasonable person in the position of that person (the victim)

This section considers the application of ‘obvious risk’ with respect to the legal requirement that it be adjudged from the ‘reasonable person in the position of the plaintiff’. Focus is directed

to the reasonable player of premier-level contact sport in Australia, including their age and experience, and how such factors may influence the legal application of 'obvious risk'.

The section also considers the question of 'voluntariness' with respect to legislative provisions concerned with 'assumption of awareness of obvious risk' in the form of CTE.

Section 9.8: CTE as a *not* 'obvious risk of harm' to a reasonable premier-level contact sport athlete

This section argues that the risk of CTE was not 'obvious' (for the purposes of state civil liability legislation) to premier-level contact sport athletes prior to around 2010. Consideration is given to an athlete's subjective perception of 'obvious risk', as well as the following:

- that the risk of CTE is an entirely different risk to the risk of symptomatic concussion — which *is* an obvious risk of playing contact sport;
- that the athlete would not fully appreciate the risk of CTE without notification of that risk, and that an awareness of symptomatic concussion does not equate to an awareness and appreciation of the progressive illness and death that result from CTE; and
- that the risk that CTE may eventuate was not consciously adverted to by the athlete.

Section 9.9: Contributory negligence

This section considers the possible, although unlikely, defence of contributory negligence. The section argues that as contributory negligence applies where a plaintiff foresees a risk of harm, and consistent with the position taken throughout this thesis, the defence does not apply on the basis that a plaintiff athlete was not aware and ought not to be aware of the risk of CTE in the pre-2010 era.

Section 9.10: Conclusion

This section concludes that CTE, given that it is both medically and descriptively unique from symptomatic concussion, is most appropriately classified as a unique disease in its own right, or as a neurological disease or tauopathy.

9.2 Legislative provisions: Obvious risk, dangerous recreational activity and assumption of risk of harm

The term 'obvious risk' is applicable to several defences under the civil liability legislation:[5] (1) a defendant is not liable for harm resulting from the materialisation of an 'obvious risk' of

[5] See *Glad Retail Cleaning Pty Ltd v Alvarenga* [2013] NSWCA 482 (Sackville AJA).

a 'dangerous recreational activity' engaged in by the plaintiff;[6] (2) under the 'presumption of awareness of obvious risk' provisions, a defendant is not liable in negligence for the harm caused to a person from an 'obvious risk';[7] and (3) a defendant does not owe a duty of care to another person to warn of an obvious risk.[8] Quite clearly, the application of the statutory 'obvious risk' provisions and the breadth or narrowness of their characterisation by a court is a foundational determinant in a claim that an SGB negligently failed to warn its premier-level athletes of the risk of CTE.

The relevant provisions of the *Civil Liability Act 2002* (NSW), recorded below, are largely — though not completely — representative of those of other states:[9]

> **Division 4 Assumption of risk**
>
> **5F Meaning of 'obvious risk'**
>
> (1) For the purposes of this Division, an 'obvious risk' to a person who suffers harm is a risk that, in the circumstances, would have been obvious to a reasonable person in the position of that person.
>
> (2) Obvious risks include risks that are patent or a matter of common knowledge.
>
> (3) A risk of something occurring can be an obvious risk even though it has a low probability of occurring.
>
> (4) A risk can be an obvious risk even if the risk (or a condition or circumstance that gives rise to the risk) is not prominent, conspicuous or physically observable.

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6 *Civil Liability Act 2002* (NSW) ss 5F, 5K, 5L; *Civil Liability Act 2003* (Qld) ss 13, 14, 17, 18, 19; *Civil Liability Act 2002* (Tas) ss 15, 16, 20; *Wrongs Act* 1958 (Vic) ss 53, 54; *Civil Liability Act 2002* (WA) ss 5H, 5E. All jurisdictions except Queensland and Tasmania exclude the word 'sport' from the definition of 'recreational activity'. Nonetheless, sport played for 'enjoyment, relaxation or leisure' may still be a 'dangerous recreational activity' in Queensland and Tasmania; the effect is to exclude professional sport from the definition in those jurisdictions. The New South Wales Court of Appeal found that the 'dangerous recreational activity' provisions cover 'professional' sport: see *Singh v Lynch* [2020] NSWCA 152. For background, see D Thorpe and L Houston, 'Game Changer? Professional Sport and Dangerous Recreational Activity: Revisiting the Ruling in *Dodge v Snell*' (2016) 11(1) *Australian and New Zealand Sports Law Journal 75*.

7 *Civil Liability Act 2002* (NSW) s 5G; *Civil Liability Act 2003* (Qld) s 14; *Civil Liability Act 1936* (SA) s 37; *Civil Liability Act 2002* (Tas) s 16; *Wrongs Act 1958* (Vic) s 54; *Civil Liability Act 2002* (WA) s 5N. This is related to the defence of *volenti non fit injuria* and is often referred to as the plaintiff's 'assumption of risk'.

8 *Civil Liability Act 2002* (NSW) s 5H; *Civil Liability Act 2003* (Qld) s 15; *Civil Liability Act* 1936 (SA) s 38; *Civil Liability Act 2002* (Tas) s 17; *Civil Liability Act 2002* (WA) s 5O. In respect of para 2(c) of s 5H of the New South Wales Act, the exception does not apply to a professional and the risk is of the death of or personal injury to the plaintiff. The exception does not apply to medical doctors in Queensland and Tasmania. In South Australia, the exception is limited to health-care services.

9 See *Civil Liability Act 2003* (Qld) ss 13–15; *Civil Liability Act 1936* (SA) ss 36–38; *Civil Liability Act 2002* (Tas) ss 15–17; *Wrongs Act 1958* (Vic) ss 53–54; *Civil Liability Act 2002* (WA) ss 5M–5O. There are no equivalent provisions in the Australian Capital Territory or the Northern Territory.

5G Injured persons presumed to be aware of obvious risks

(1) In proceedings relating to liability for negligence, a person who suffers harm is presumed to have been aware of the risk of harm if it was an obvious risk, unless the person proves on the balance of probabilities that he or she was not aware of the risk.

(2) For the purposes of this section, a person is aware of a risk if the person is aware of the type or kind of risk, even if the person is not aware of the precise nature, extent or manner of occurrence of the risk.

5H No proactive duty to warn of obvious risk

(1) A person ('the defendant') does not owe a duty of care to another person ('the plaintiff') to warn of an obvious risk to the plaintiff.

(2) This section does not apply if:

(a) the plaintiff has requested advice or information about the risk from the defendant, or

(b) the defendant is required by a written law to warn the plaintiff of the risk, or

(c) the defendant is a professional and the risk is a risk of the death of or personal injury to the plaintiff from the provision of a professional service by the defendant.

(3) Subsection (2) does not give rise to a presumption of a duty to warn of a risk in the circumstances referred to in that subsection.

Division 5 Recreational activities

5J Application of Division

(1) This Division applies only in respect of liability in negligence for harm to a person ('the plaintiff') resulting from a recreational activity engaged in by the plaintiff.

(2) This Division does not limit the operation of Division 4 in respect of a recreational activity.

5K Definitions

In this Division —

'dangerous recreational activity' means a recreational activity that involves a significant risk of physical harm.

'obvious risk' has the same meaning as it has in Division 4.

'recreational activity' includes —

(a) any sport (whether or not the sport is an organised activity), and

(b) any pursuit or activity engaged in for enjoyment, relaxation or leisure …

5L No liability for harm suffered from obvious risks of dangerous recreational activity

(1) A person ('the defendant') is not liable in negligence for harm suffered by another person ('the plaintiff') as a result of the materialisation of an obvious risk of a dangerous recreational activity engaged in by the plaintiff.

…

5M No duty of care for recreational activity where risk warning

(1) A person ('the defendant') does not owe a duty of care to another person who engages in a recreational activity ('the plaintiff') to take care in respect of a risk of the activity if the risk was the subject of a risk warning to the plaintiff. …

As provided by s 5G (regarding the plaintiff's 'assumption of risk'), the defence associated with 'obvious risk' will not apply where an injured person — the plaintiff — can prove that he or she was not aware of the risk of harm. In contrast, under ss 5F ('Meaning of "obvious risk"') and 5H ('No proactive duty to warn of obvious risk'), the test is objective on the basis that the risk would have been obvious to a reasonable person in the position of the injured person.

The determination of an 'obvious risk' is prospective, that is, the assessment of obviousness is forward looking rather than retrospective Note that the term 'obvious risk' has the same meaning whether it applies to 'dangerous recreational activity' or 'assumption of risk'.[10] Note further that the term 'risk' is not defined within civil liability legislation.[11] Nor is the word 'obvious' defined, although Tobias JA in *Fallas v Mourlas* adopted the following meaning from a previous judgment of his own:

> 'Obvious' means that both the condition and the risk are apparent to and would be recognised by a reasonable man, in the position of the [plaintiff], exercising ordinary perception, intelligence and judgment.[12]

Although s 5G also relieves the defendant of the burden of proving that the plaintiff was aware of the risk of harm at the time he or she was injured, it remains for the defendant to prove that the plaintiff possessed 'some conscious advertence to the possibility that the known risk might eventuate, and [made] a decision to proceed with the conduct regardless'.[13]

The New South Wales and Western Australia Acts provide that a defendant does not owe a duty of care to another person who engages in a recreational activity 'to take care in respect of a risk of the activity if the risk was the subject of a risk warning'.[14] The warning applies generally to recreational activities, not only activities that carry a level of danger. It is a contention of this thesis that the SGBs of major contact sport in Australia did not provide risk warnings regarding CTE to their premier players until *circa* 2010 or later. It should also be noted that a defendant is not entitled to rely on a risk warning unless it was given by or on behalf of a defendant, thus eliminating any reliance on comments within specialised

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[10] See *Civil Liability Act 2002* (NSW) ss 5F, 5K.

[11] *Chapman v Hearse* (1961) 106 CLR 112, 120–1.

[12] *Fallas v Mourlas* [2006] NSWCA 32, [100], quoting *Wyong Shire Council v Vairy; Mulligan v Coffs Harbour City Council* [2004] NSWCA 247, [161].

[13] *Carey v Lake Macquarie Council* [2007] NSWCA 4, [27] (McClellan CJ).

[14] *Civil Liability Act 2002* (NSW) s 5M; *Civil Liability Act 2002* (WA) s 5I. Section 17 of the *Civil Liability Act 2002* (Tas) provides that there is no duty to warn of an obvious risk of recreational activity; however, the word 'sport' was removed from the definition of 'recreational activity' in s 19 in 2019, such that the provision now applies to 'any pursuit or activity engaged in for enjoyment, relaxation or leisure', thereby excluding professional sport.

publications such as medical journal reports. As such, this provision will not be considered further.

9.3 An emphasis on 'obvious risk'

Given that the major contact sports in Australia — such as Australian football, rugby league and rugby — undoubtedly involve 'a significant risk of physical harm', there is little point in engaging in protracted discourse on whether these types of sports, or aspects of them, meet the definition of a 'dangerous recreational activity'.[15] Accordingly, this chapter mainly focuses on discussion of the characterisation of 'obvious risk' as applied to harm in the form of CTE.[16] Where a risk is found to be 'obvious' — subjectively or objectively, depending on the relevant provision — a defendant is absolved of liability. There is, importantly, a difference, between what may be obvious to a defendant SGB that is duty-bound to inform itself of risks of harm material to its players, as against any expectation that a plaintiff athlete will seek out information on such risks.

Although head contact leading to concussion is an obvious risk of contact sport, the risk of CTE in the playing of contact sport was *not*, as argued here, obvious to a premier-level athlete in Australia until around 2010, when information and publicity associated with the publication of medical journal reports in 2005 and 2009 informing (or 're-informing') of the risk of CTE in contact sport was the subject of SGB warnings, adverted to and absorbed.[17]

Once a risk of harm is identified and characterised, it must then be determined whether the risk in question satisfies the elements that it was 'obvious', 'dangerous' and a 'materialisation of that risk' under s 5L of the *Civil Liability Act 2002* (NSW) or its equivalents (regarding dangerous recreational activities), or whether it was an 'obvious risk' under s 5G of the New South Wales Act or its equivalents (regarding the presumption of awareness of obvious risks).

9.4 Fundamental questions in contention

Two main contentions regarding obvious risk of harm in the form of CTE are considered in this chapter. The first relates to the discernment of a date (a year and month) when CTE would be deemed by a court to be an obvious risk of contact sport. From this date onwards, newcomers

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[15] In *Campbell v Hay* [2014] NSWCA 129, Ward J observed at [116]: '"Significant risk" has been said to mean more than trivial and does not import an "undemanding" test of foreseeability (*Fallas* [*v Mourlas* [2006] NSWCA 32] per Ipp JA at [14]); it does not mean a risk that is likely to occur (*Fallas* per Ipp JA at [16]) but lays down a standard lying somewhere between a trivial risk and a risk likely to materialise (*Fallas* per Ipp JA at [18]); and as a general guide, it means a risk that is "not merely trivial, but generally speaking, one which has a real chance of materialising" ([*Fallas* per] Tobias JA at [90]–[91], this being the test his Honour applied later in *Jaber v Rockdale City Council* [2008] NSWCA 98 at [54]).'

[16] For further discussion of what constitutes a 'dangerous recreational activity', see D Thorpe and P Stewart, 'Not To Be Too Pedantic ... But What Exactly Is a Dangerous Recreational Activity?' (2006) 1(1) *Australian and New Zealand Sports Law Journal* 121.

[17] The exact date is not crucial. What is important in terms of analysis is that the risk of CTE changed from being not known by players to an 'obvious risk' around the year 2010.

to a premier-level competition who sue their SGB for failing to warn them of the risk of CTE would encounter the 'liability defeating'[18] 'obvious risk' provisions of the civil liability legislation. However, provided the head trauma incurred in the playing of contact sport was more than *de minimis*, those who played at least part of their career prior to the cut-off date retain a factual basis upon which to argue that the SGB's failure to warn caused their exposure to CTE-inducing head trauma — in theory, a player who was warned could have retired before RHT reached the threshold at which tau protein leaked from the brain's axons to initiate CTE.[19]

The second main contention regarding obvious risk of harm in the form of CTE concerns whether the risk of CTE is to be characterised in a general form, such as 'concussion' or 'head trauma', or as a specific disease or specific type of disease. This determination bears upon how obvious the risk is to a reasonable contact sport athlete — the more general the risk, the more obvious the risk. A characterisation that the relevant risk of harm is 'concussion' or 'head trauma' advantages a defendant, given that these conditions are clearly obvious to a premier-level contact sport athlete, and serves to relieve the defendant of liability. This chapter argues, however, that the symptoms, sequelae and prognosis of CTE necessitate a specific characterisation of CTE as a type of harm designated as a neurodegenerative disease or tauopathy.

9.5 Dating when the risk of CTE became obvious

The rationale of including the term 'obvious risk' in the civil liability legislation is that a 'person ordinarily will be expected to exercise sufficient care by looking where they are going and perceiving and avoiding obvious hazards'.[20] Clearly, a plaintiff athlete's ability to 'perceive' the risk of CTE is critical to the determination of obvious risk.

A risk of harm cannot be 'obvious' under the civil liability legislation unless it 'would have been obvious to a reasonable person in the position of [the plaintiff]'.[21] CTE — whether referred to as *dementia pugilistica* or punch drunk syndrome, or through its symptoms and sequelae — has been recognised in medical circles since at least 1928, with Dr Harrison Martland's publication in the *Journal of the American Medical Association*.[22] Although dozens of medical journal articles reported on the risk of CTE in boxing and then other contact sports from the 1970s, it was not until the publication of Dr Bennet Omalu et al's study 'Chronic Traumatic Encephalopathy in a National Football League Player' in 2005 that the disease

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[18] *Goode v Angland* (2017) 96 NSWLR 503, [185] (Leeming JA).

[19] See, eg, ML Lipton et al, 'Soccer Heading Is Associated with White Matter Microstructural and Cognitive Abnormalities' (2013) 268(3) *Neuroradiology* 850, 854.

[20] *Brodie v Singleton Shire Council; Ghantous v Hawksbury City Council* (2001) 206 CLR 512, 581 (Gaudron, McHugh and Gummow JJ).

[21] See, eg, *Civil Liability Act 2002* (NSW) s 5F.

[22] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

received wide, or at least renewed, public attention.[23] The study was downplayed by the National Football League and largely ignored by Australian SGBs until Dr Ann McKee et al's meta-study, published in 2009, revealed CTE-causing 'hyperphosphorylated tau-positive aggregates', or 'p-tau', in the autopsied brains of players of several different contact sports.[24]

This calendar of discovery raises the question of *when* the risk of CTE became obvious to a reasonable premier-level contact sport athlete in Australia. As argued, only athletes who participated in premier-level competitions prior to the date when CTE became an obvious risk will be unhampered by the defence of obvious risk. From that date on, as argued in this chapter, the SGB is not duty-bound to warn of the obvious risk of CTE. Those athletes who began their career after the cut-off point will need to consider alternative forms of SGB negligence associated with CTE, such as a failure to enforce concussion mitigation programs.[25]

Consequently, the date at which it became reasonable to expect that athletes would be aware of the risk of CTE is foundational to the cases of both plaintiff and defendant. While a precise date is a matter yet to be determined by a court, based on fact and expert evidence,[26] it is suggested that after the publicity resulting from McKee et al's CTE study in 2009, followed by the introduction of more stringent concussion mitigation protocols by SGBs (such as the AFL and National Rugby League), the known purpose of which was to reduce the risk of CTE, a premier-level athlete in Australia could be reasonably expected to be aware that CTE was an obvious risk of contact sport. Indeed, by 2011 the media in Australia was reporting pointedly on the issue. For example, the *Herald Sun* reported in April 2011 that 'it emerged yesterday that former [AFL] Melbourne defender Daniel Bell was seeking compensation after being diagnosed with brain damage linked to multiple concussions suffered during his playing career'.[27] A relevant date, given some uncertainty, is perhaps best described as '*circa* 2010'. Nonetheless, irrespective of a precise date, what is important is the realisation that a court will find that at 'some date' in the 'post-Omalu era', the risk of CTE became obvious to reasonable premier-level athletes in Australia.

A different question concerns the date when an SGB knew or ought to have known that CTE was a risk to its premier players. An SGB cannot be liable for failing to warn of the risk of CTE — or, indeed, failing to implement other protocols — unless it ought reasonably to

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[23] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[24] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

[25] Claims of a failure to follow or enforce concussion prevention protocols are based on a failure to perform a stated obligation. See, eg, *Watson v British Boxing Board of Control; World Boxing Organisation Inc* [2001] QB 1134.

[26] *H v Royal Alexandra Hospital* [1990] 1 Med LR 297.

[27] Scott Gullan, 'Cats Gamble on Selwood as KO Storm Erupts in a Wing and a Prayer', *Herald Sun* (Melbourne, 1 April 2011).

have known of that risk.[28] For clarity, a risk that ought to have been known by an SGB may not be obvious to a reasonable athlete. An SGB holding a duty of care is obliged to take reasonable steps to be informed of extant and developing risks to the health and well-being of its premier players. As such, debate as to whether an SGB ought to have foreseen the risk of CTE revolves around four possible dates across time:

- from the time of Dr Martland's 1928 study; or
- by extrapolating from the plethora of research into CTE in boxing to other collision and contact sports; or
- through examination of medical journal articles dating from the 1970s that implicated non-pugilistic sports in the contraction of CTE; or
- through consideration of Australian reports touching on CTE from the 1990s

However, these dates are not likely to be relevant to the question of whether a reasonable premier-level athlete was aware of the risk of CTE in playing contact sport.[29]

As considered in detail below, for an athlete to know of the risk of concussion is not, of itself, to know of the risk of CTE. Knowledge of the risk of the transitory effects of concussion is not knowledge of the debilitating, ongoing and commonly fatal consequences of CTE.

9.6 Defining harm: General and specific applications

The generality with which a risk of harm, and resultant injury, is characterised bears upon the 'obviousness' of a risk to a reasonable person in the plaintiff's position. As Bryson JA said regarding 'obvious risk': 'Much depends, in the application of provisions dealing with obvious risk, upon the degree of generality or precision with which the risk is stated.'[30] For example, if all segments of the game of field hockey present as obvious risks, the plaintiff will be taken to have accepted 'the obvious' and a defendant cannot be liable in negligence. On the other hand, if the only segment of the game that poses an obvious risk is being struck with the ball, the defendant is exposed to a charge of negligence in all other segments of the game. When considering the harm that results from an SGB's breach of duty by failure to warn, a fair assessment of the risk of CTE necessitates that the harm be characterised with a high degree of specificity over generality.

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[28] *Roe v Minister of Health* [1954] 2 QB 66.

[29] National Health and Medical Research Council, *Football Injuries of the Head and Neck* (1994); Australian Law Reform Commission, *Essentially Yours: The Protection of Human Genetic Information in Australia* (Report No 96, 2003) vol 2 chapter 38 'Sport'; Nathan Gibbs, 'Common Rugby League Injuries' (1994) 18(6) *Sports Medicine* 438.

[30] *CG Maloney Pty Ltd v Hutton-Potts* [2006] NSWCA 136, [173] (Beazley JA agreeing).

9.6.1 CTE: A general or specific characterisation of risk?

With regard to the 'assumption of risk' — that is, that the plaintiff is presumed to be aware of a risk where the risk would have been obvious to a reasonable person in the position of the plaintiff — according to McClellan CJ, '[a] plaintiff cannot rebut the presumption by claiming that even though he or she was aware of the general risk of harm, he or she was not aware of all its possible manifestations, including the one that eventuated'.[31] Consequently, as it is unnecessary for a 'reasonable man to foresee damage of a precise and particular character'[32] and 'it is sufficient if the kind or type of injury was foreseeable',[33] a defendant SGB may well argue that the risk of CTE is to be characterised generally as, say, 'head trauma' or 'concussion'.

Whether CTE as a risk of harm is characterised generally or specifically will largely determine the availability of an 'obvious risk' defence to an SGB. For example, if CTE were included as a subset of the general term 'concussion', an SGB would likely avoid liability on the basis that concussion is an obvious risk of contact sport — and has been for centuries.[34] On the other hand, if CTE is characterised as a specific disease, it is not likely to be deemed 'obvious' prior to *circa* 2010. Of course, given the wide publicity on the cause and harm of CTE and education programs provided to contact sport athletes, CTE is likely to be considered an obvious risk of harm from, as proposed, *circa* 2010.

In fact, just such a general characterisation of the risk of harm appears to be a key a defensive approach taken by the AFL in the current Jarad Rooke class action. In that action, the AFL defence states inter alia that:

- 'head knocks', 'concussions', 'head injury', 'concussion injury', 'injury' and 'injuries' were 'reasonably foreseeable to any reasonable person throughout the relevant period, including to the Plaintiff';[35]
- 'the risk of the Plaintiff suffering from a concussion and/or head knocks during a match or training was a risk that was obvious to a reasonable person in the Plaintiff's position;'[36]

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[31] *Carey v Lake Macquarie City Council* [2007] NSWCA 4, [90].

[32] *Chapman v Hearse* (1961) 106 CLR 112, 120–1, referenced in *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* (2022) 273 CLR 454, [108].

[33] *Rosenberg v Percival* (2001) 205 CLR 434, [64], referenced in *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* (2022) 273 CLR 454, [109].

[34] See ST Casper, 'Concussion: A History of Science and Medicine, 1870–2005' (2018) 58 *Headache* 795.

[35] *Jarad Maxwell Rooke v Australian Football League* (Supreme Court of Victoria, S ECI 2023 00969, commenced 14 March 2023) Defence, cl 16(b), (d)(i).

[36] Ibid cl 65.

- 'the Plaintiff freely and voluntarily, with awareness of the risk of suffering from a concussion and/or head knocks during a match or training impliedly agreed to incur that risk;'[37]

- '[f]urther and in the alternative, … any such harm resulted from the materialisation of an inherent risk within the meaning of section 55 of the *Wrongs Act 1958* (Vic).'[38]

9.6.2 Obvious risk: Conduct and harm

Commonly, a risk of harm is described by means of the conduct that caused injury to the plaintiff; for example, sustaining injury by falling off a horse due to a broken saddle strap. In many cases, it is proposed, there is little to be gained in describing the actual harm suffered in the calculation of obvious risk because, from common experience, almost all harm that results from well-known events — such falling off a horse — is within the ambit of foreseeable and observable harm: perhaps a broken arm, a broken hip or spinal damage, among many other forms of predictable injury.

However, some forms of harm are so unpredictable, novel or peculiar that any fair assessment of the obviousness of a risk must include the actual harm that results from the breach. This point was also made by Perry, who opined that a generic description of harm is sufficient 'so long as this generic description does not bring to mind the prospect of harm being sustained of a materially different kind or severity to that which occurred', and proposed that actual harm should 'be read as implied and made explicit'.[39]

It is argued here that to correctly distinguish risks that are obvious from those that are not, emphasis must be placed upon harm in the specific form. As such, the applicable 'risk of harm' is the risk of CTE specifically, or if by 'type', as a neurodegenerative disease or tauopathy and, as regards the mechanism of harm, that of RHT.

Consider the reasoning in *Ackland v Stewart*,[40] which identified the actual harm as the central element in determining that the risk of harm in question was not obvious. The plaintiff attended an amusement park, where he used an attraction known as a 'jumping pillow' — a large inflatable pillow about 20 metres by 10 metres in size that visitors, including children, used like a trampoline to jump up and down, and perform somersaults and backflips. In attempting a back somersault, the plaintiff (who was an adult) landed heavily on his head resulting in quadriplegia (referred as tetraplegia in the appeal). He claimed the defendants were negligent in not instructing in the safe use of the jumping pillow, not warning of the risk of

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[37] Ibid cl 67.

[38] Ibid cl 68. Section 55(1) of the *Wrongs Act 1958* (Vic) provides: 'A person is not liable in negligence for harm suffered by another person as a result of the materialisation of an inherent risk.'

[39] Gabriel Perry, 'Obvious Risks of Dangerous Recreational Activities: How Is Risk Defined for *Civil Liability Act* Purposes?' (2016) 23(1) *Torts Law Journal* 56, 72, referring to the judgment of Beech-Jones J in *Collins v Clarence Valley Council (No 3)* [2013] NSWSC 1682.

[40] [2014] ACTSC 18.

neck injury and not prohibiting back somersaults or other inverted manoeuvres, among several other claims. The defendants claimed relevantly that the injury arose from the plaintiff's participation in a dangerous recreational activity within the meaning of the *Civil Liability Act 2002* (NSW).

Burns J found that the activity of performing a back somersault posed 'a significant risk of physical harm' under the 'dangerous recreational activity' provisions. The assessment of 'obvious risk' was, though, the main point of contention. His Honour characterised 'obvious risk' as 'the risk that a person might suffer a serious neck injury if an inverted manoeuvre was not properly performed'.[41] Much depended on the degree to which a jumping pillow was comparable with a trampoline (a device with which the plaintiff had experience) in terms of softness of surface or yielding force.

His Honour took into account factors that a reasonable plaintiff would have assessed in deciding whether a back somersault posed an obvious risk of harm: that young children were performing similar manoeuvres without any apparent attempt by staff to stop them; that a reasonable person would be entitled to reason that if there was a danger in performing inverted manoeuvres on the jumping pillow, a warning would be given to young children, and staff would take steps to stop children performing such manoeuvres; and that those who best knew the properties of the jumping pillow — its owners — apparently permitted young children to perform somersaults on it.[42]

Other factors included: that the surface of the jumping pillow was yielding; that the jumping pillow was full of air; that the plaintiff had extensive experience in performing back somersaults; that the jumping pillow performs very much like a trampoline; that the plaintiff used the jumping pillow to land on his stomach and back without any injury or apparent discomfort (and landing on the stomach and back provides a better appreciation of the 'give' in the surface than landing on the feet); that the equipment was called a 'pillow', suggesting something soft and supportive; and that the plaintiff had previously landed awkwardly on the jumping pillow after attempting a back somersault and had sustained no injury or discomfort.[43]

The instructive determination of Burns J is worth quoting in full:

> On balance, I am not persuaded that it would have been obvious to a reasonable person in the position of the plaintiff that there was a risk of serious neck injury in attempting to perform a back somersault on the jumping pillow. A reasonable person would have acknowledged, as the plaintiff did, that there is a risk of some minor harm if they failed to perform the manoeuvre properly. But *perception of risk of minor harm is not the equivalent of perception of risk of a serious neck injury*.[44]

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41 Ibid [296].

42 Ibid [300].

43 Ibid.

44 Ibid [304]. Emphasis added.

On appeal, Walmsley JA agreed with the trial judge stating:'[I]t was a relevant matter that the jumping pillow was there for [the respondent's] enjoyment, in particular, to jump on, and was very much under the appellants' control and I do not accept the proposition that it was obvious that it was potentially catastrophic for a manoeuvre to fail on that surface.'[45]

Walmsley JA also considered whether the risk of *any injury* as opposed to *a serious neck injury* was the relevant question.[46] In other words, does the application require differentiating a 'risk of a minor injury from a major one'?[47] His Honour stated that 'the clear meaning of the section must be to save a potential tortfeasor harmless from an action arising from a risk which has come home rather than one which has not'.[48] A risk that 'comes home' is a knowable risk — an obvious risk. In this case, the risk of quadriplegia was not an obvious risk.[49]

The Court of Appeal in *Queensland v Kelly*[50] utilised a similar approach in determining whether the risk in question was 'obvious'. The plaintiff, a 22-year-old tourist, was rendered a partial quadriplegic when he ran down a sand dune towards a lake on Frazer Island, Queensland. About a metre from the water's edge, he lost his footing, probably because the sand gave way beneath him, and he plunged inadvertently into shallow water, striking his head. In the 17 years prior, 18 serious injuries to tourists had been recorded at the same lake, 'many of which involved serious spinal injuries'.[51] The gravamen of Kelly's case was described in *Stewart v Ackland* as follows: 'The outcome in favour of the plaintiff depended on how the risk was classified.'[52]

Referring to the degree of risk, Henry J in *Kelly* said: 'It is obvious that running down a sand dune into a lake involves a risk of some injury. … Whether running down a sand dune into a lake involves an obvious risk of serious injury will very much depend upon the individual circumstances of the case.'[53] The circumstances going to the obviousness of risk included, according to his Honour, the 'forgiving surfaces' of 'sandy slopes and water'.[54]

Fraser JA saw the risk of harm as minor: 'running down the sand dunes towards the lake was dangerous in the sense that it involved the risk of some injury, such as a sprain or bruising

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45 *Stewart v Ackland* (2015) 10 ACTLR 207, [147] (Court of Appeal).

46 Ibid [135].

47 Ibid [148].

48 Ibid [150].

49 Robinson JA agreeing at [160].

50 [2015] 1 Qd R 577.

51 Ibid [17].

52 *Stewart v Ackland* (2015) 10 ACTLR 207, [151].

53 *Queensland v Kelly* [2015] 1 Qd R 577, [62].

54 Ibid.

as a result of a fall consequent upon looseness of the sand'.[55] His Honour added that the circumstances, including the presence of warning signs, did 'not necessarily require the conclusion that the risk of serious injury in that activity would have been obvious to a reasonable person'.[56] Philippides JA determined similarly that the 'risk of injury that materialised, that is, the risk of *serious* injury from an accident caused by running down the sand dune into the lake, was not a risk that in the circumstances would have been obvious to a reasonable person in the position of the respondent'.[57]

To emphasise the point, in *Kelly* the risk of quadriplegia was not an obvious risk to a plaintiff who had no experience in the activity of running down sand dunes and yet, to the defendant, the risk was foreseeable and not insignificant, given that it had received previous reports of spinal injuries resulting from just that recreational activity.

The judgments in *Kelly* draw a distinction between a risk of harm such as 'a sprain' and that of 'serious injury', in that case quadriplegia. Clearly the distinction between non-serious and serious harm is evident from the injury suffered by the plaintiff. In the context of head injuries, one could draw a similar distinction between non-serious forms of harm such as concussion, and serious forms of harm such as CTE. Moreover, a determination that distinguishes between the serious and non-serious forms of harm could well, as a matter of principle, draw finer distinctions — including the differentiation of harm by the disease or type of disease suffered by a claimant.

The High Court majority in *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* characterised the risk of harm through reference to the degree of potential injury: '[T]he risk should be characterised as the *substantially elevated risk of physical* injury by falling from a horse that slipped by reason of the deterioration of the surface of the arena.'[58] The Victorian Court of Appeal in *Erickson v Bagley* reflected this approach stating that, '[n]ecessarily, the risk must be defined taking into account the particular harm that materialised, and the circumstances in which that harm occurred'.[59]

Tobias JA adopted a similar approach in *Jaber v Rockdale City Council*:

> [T]he focus of the enquiry is not upon the putative tortfeasor but upon the person who has been injured or, more accurately, a reasonable person in his position. Whether or not a risk is 'obvious' may well depend upon the extent to which the probability of its occurrence is or is not readily apparent to the reasonable person in the position of the plaintiff.[60]

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55 Ibid [48].

56 Ibid [50].

57 Ibid [58]. Emphasis added.

58 *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* (2022) 273 CLR 454, [125]. Emphasis added.

59 *Erickson v Bagley* [2015] VSCA 220, [33].

60 *Jaber v Rockdale City Council* [2008] NSWCA 98, [35].

A statement to the same purport was made in *Wyong Shire Council v Vairy*:

> 'Obvious' means that both the condition and the risk are apparent to and would be recognised by a reasonable man, in the position of the [plaintiff], exercising ordinary perception, intelligence, and judgment.[61]

Emphasis is placed on the knowledge of the reasonable victim in the circumstances. It is not only knowledge that a risk might materialise but also the probability of its materialisation that goes to the assessment of 'obvious'. In many cases, knowledge of the probability of harm cannot be realised unless the type of harm is also known. In fact, Tobias JA indicated as much in *Jaber*:

> '[C]ondition' refers to the factual scenario facing the plaintiff. Thus in a diving case the condition might typically be the fact that the plaintiff is faced with water of unknown depth. Under such a condition the risk would be that diving into the water (while the depth remains unknown) might result in (serious) injury. This risk would be considered obvious if, in the context of the case, it was perceptible to a reasonable person in the position of the plaintiff that if you do not know the depth of a body of water into which you are about to dive, then to dive into such water under such conditions inevitably brings with it the risk of injury.[62]

To put into terms of CTE and frank concussion: The 'condition' a contact sport athlete faces is head trauma, or even RHT, in the playing of contact sport. Drawing on the example given by Tobias JA, just as the assessment of 'risk' (obvious or otherwise) within the circumstances of that condition depends on knowledge that diving into water of unknown depth brings a risk of injury, so too does the assessment of 'obvious risk' in respect of CTE depend on an athlete's knowledge of that disease and its means of occurrence. Without knowledge of that specific risk of CTE, the relevant risk of harm cannot be obvious.

The *Jaber* judgment included passages of cross-examination of the plaintiff that highlight the importance of knowledge of harm prior to the event that caused injury in assessing obvious risk.

> Q. When you dived into this water, you didn't know what depth it was, did you? A. Not exactly. Q. And you knew when you dived in, not knowing exactly the depth, that you could become very badly injured if you hit your head on the bottom, didn't you? A. I thought it was deep enough but. Q. You knew that if you hit your head on the bottom you could be very badly injured, didn't you? A. Well I didn't think that I'm going to hit my head at the bottom. Q. You knew that if you hit your head on the bottom? A. Yes. Q. You could be very badly injured? A. Yes. Q. Perhaps even killed? A. Yes. Q. End up in a wheelchair? A. Yes.[63]

This exchange of words reinforces a point made earlier[64] — that a general description of harm is useful where it describes the range of predictable and known injuries associated with a

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61 *Wyong Shire Council v Vairy* [2004] NSWCA 247, [161] (Tobias JA and Mason P agreeing).

62 Ibid [162].

63 *Jaber v Rockdale City Council* [2008] NSWCA 98, [37].

64 See **[9.6.2]**: 'Obvious risk: Conduct and harm'.

given event, but it does not serve the purpose of the 'obvious risk' provisions where the harm that materialises is not, by common experience, realistically connected to the event in question. Clearly, the point of the cross-examination was to illicit an admission that the plaintiff knew of the risk of 'ending up in a wheelchair'. Consider the response to a question transposed from *Jaber* above to the circumstances of a contact sport athlete: 'You knew you would be very badly injured … Perhaps ending up with dementia, parkinsonism, being violent to family members, suffering depression, suicidality, loss of impulse control, confusion and poor judgment?'[65] The response to the question in cross-examination for those who participated in contact sport pre-*circa* 2010 would be clear: 'No, I did not know I was exposed to the risk of contracting such a serious disease.'

A The relevance of the statutory 'general principles' establishing breach

The statutory 'general principles' of the civil liability legislation[66] require in the determination of 'negligence' that an assessment of foreseeable harm be examined by the court; that is, the probability of harm occurring, the likely seriousness of the harm, the burden of taking precautions to avoid harm, and the social utility of the activity that creates the risk of harm. The relevant provisions of the *Civil Liability Act 2002* (NSW), recorded below, is identical to the corresponding provisions of other state jurisdictions.[67]

> **Division 2 Duty of care**
>
> **5B General principles**
>
> (1) A person is not negligent 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,

[65] Paraphrased from 'Frequently Asked Questions: What Is CTE?', *Boston University, Center for the Study of Traumatic Encephalopathy* (Web Page) <https://www.bu.edu/cte/about/frequently-asked-questions/>.

[66] See, eg, *Civil Liability Act 2002* (NSW) s 5B.

[67] See *Civil Liability Act 2002* (ACT) ss 42, 43.; *Civil Liability Act 2003* (Qld) s 9; *Civil Liability Act 1936* (SA) ss 31, 32; *Civil Liability Act 2002* (Tas) s 11; *Wrongs Act 1958* (Vic) s 48; *Civil Liability Act 2002* (WA) s 5B. The Northern Territory has no equivalent provision, such that the common law continues to apply in respect of breach of duty.

(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.

In *Tapp v Australian Bushmen's Campdraft & Rodeo Ltd*, the High Court majority stipulated that 'obvious risk' under s 5L is the 'same risk as that with which s 5B is concerned' and involves the same basic facts that establish breach of duty.[68] In essence, the degree or type of harm must be identified to assess the above-mentioned elements, which are determinative of whether a risk of harm was foreseeable, whether it was not insignificant, and whether a reasonable person would have taken precautions. In short, the more serious the harm faced by a plaintiff, the better the precautions that must be taken by a defendant to mitigate the risk of harm. While steps taken to ameliorate the risk of concussion serve the purpose of mitigating the risk of CTE, it is the risk of CTE that drives the assessment of the adequacy of precautions. Moreover, the means of occurrence and the catastrophic nature of CTE necessitate the precaution of warnings, given that non-participation is the only sure way to avoid RHT in contact sport.

In discussing the relationship between contributory negligence and general negligence under s 5B, Basten JA (Meagher and Emmett JJA agreeing) in *Hallmark Construction Pty Ltd v Harford* made a point that further illustrates the need to be aware of actual physical harm when assessing obvious risk: '[I]n order to identify "the risk of *that* harm", it is necessary to have regard to the harm in fact suffered in the accident. Because the relevant precautions will depend to a significant extent on the nature of the risk, it is not sufficient to identify the risk as the possibility of personal injury [that is, a general description].'[69] A contradiction arises where it is accepted that to properly assess contributory negligence, a plaintiff must be aware of the specific risk of harm, but not in assessing obvious risk.

Can it be said that 'head trauma', as the common cause of frank concussion and CTE, is sufficient to create a 'type' of risk? Where a common form of conduct leads to harm that is not or cannot be anticipated, it is arguable that a there is no commonality of type.

B Borrowing from the medical cases

The concept of 'obvious risk' permits an individual to accept the risk of harm and, in doing so, bear personal responsibility should that harm materialise. Within the decision-making process is a weighing up of the chances of harm and the seriousness of harm should it materialise. *Ceteris paribus*, a reasonable individual, if warned, is more willing to accept a risk of minimal harm compared to serious harm or certain types of harm. Clearly the acceptance of a risk of harm necessitates knowledge of the possible seriousness of the potential harm the individual faces.

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[68] *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* (2022) 273 CLR 454, [116].

[69] *Hallmark Construction Pty Ltd v Harford* [2020] NSWCA 41, [27]. Original emphasis.

In context, an athlete who is not warned of the risk of progressive and permanent cognitive disfunction due to RHT may well decide to accept the consequences of concussion, or indeed many concussions. Of course, the decision-making process is very different where the athlete is informed of the life-altering consequences of CTE. Only through the provision of information regarding the symptoms, sequelae and prognosis of CTE can the premier-level athlete assess whether to run the risk CTE or not — without this information, the purpose behind the concept of 'obvious risk' is devoid of meaning. In short, knowledge of the mechanism of how a disease is caused is of no use to a plaintiff without knowledge of the debilitating effects of that disease.

The medical cases convey the necessity, as recognised in judicial determinations, of supplying individuals with specific knowledge of risks to their health as a predicate to making an informed decision. Surgery, for example, may involve many risks, ranging from the minor to the major to the catastrophic. To merely describe a surgical procedure generally as 'carrying risks of harm' does not permit an individual to weigh whether the risk is worth taking. Only when the risk is conveyed specifically in terms of type and degree of harm can the decision-maker elect the best course to follow. As the *Ipp Report* stated:

> The more narrowly a risk is defined, the less likely it is that a person will have been aware of it [and, accordingly, the less likely it is that the risk was 'obvious']. For instance, a person may be aware of the risk of suffering bodily injury as a result of engaging in a particular activity. But the person may not be aware of the risk of suffering bodily injury in a particular way.[70]

Consider in this respect the case of *Rogers v Whitaker*.[71] The plaintiff, Mrs Whitaker, was almost entirely blind in her right eye as the result of a penetrating injury she sustained as a child. She was advised by Dr Rogers that the removal of scar tissue on her right eye would probably restore much of its sight. As it transpired, the operation made little difference to her right eye but resulted in sympathetic ophthalmia in her left eye, from which she lost all sight. The chances of this occurring were around one in every 14,000 procedures. No doubt some warnings had been issued to Mrs Whitaker prior to the operation. None, however, seemingly conveyed the risk of serious and irreparable blindness associated with sympathetic ophthalmia. Herein lies the difference between a general warning of possible physical harm and a warning that brings home the stark specific consequences of serious and permanent debilitating harm.

In *H v Royal Alexandra Hospital*,[72] the plaintiff, a minor suffering from haemophilia, was infected with HIV through a blood products transfusion in March 1982 or September 1983. Badgery-Parker J found that a general warning that the plaintiff might get infections from blood products was, at the time, a 'completely adequate' discharge of the defendant's responsibility. However, when a 'new and different' risk in the form of HIV emerged in September 1983 — a disease which was 'clearly recognisable by physicians who had kept in touch with

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[70] *Review of the Law of Negligence* (Final Report, September 2002) [8.28].

[71] (1992) 175 CLR 479.

[72] [1990] 1 Med LR 297 (Supreme Court of New South Wales).

developments both in the United States and Australia' — this required that the plaintiff's parents 'should have been warned in appropriate terms of the AIDS risk; and the fact that no such warning was given constituted negligence'.[73]

The 'devastating consequences' of AIDS as against a relatively minor infection featured heavily in Badgery-Parker J's determination. His Honour quoted Lord Bridge: '[D]isclosure of a particular risk was so obviously necessary to an informed choice on the part of the patient that no reasonably prudent medical many would fail to make it.'[74]

Badgery-Parker J considered the obligation to warn in terms of probability and consequences: 'The greater the chance that the risk will eventuate, the more obviously will disclosure be necessary, even though the consequences of the happening of the risk may not be disastrous. Conversely, where the possible consequence is disastrous, disclosure may be "obviously necessary" even though the risk be quantified as tiny.'[75] As such, after September 1983 the fact that the risk of contracting AIDS was small was irrelevant with regard to the obligation to warn. Required at that time was a specific warning informing blood product recipients that 'in a very small number of cases in the United States … AIDS had developed in patients who had been treated' with the blood product in question.[76] That the number of cases was only 12 out of a haemophiliac population between 15,000 and 20,000 did not, given the horrendous consequences of contracting AIDS, eliminate the duty to warn.[77]

While a general characterisation may be sufficient to capture a risk of harm in some cases, in others the harm is so removed from common experience that it cannot 'be presumed that the person against whom the defence is pleaded was actually aware' of it or that it could be assumed to fall within 'the type or kind of risk' that eventuated.[78] So it is argued in respect of CTE. Neither knowledge of concussion nor the mechanism of how concussion occurs enables an athlete to knowingly accept the risk of harm in the form of CTE.

9.6.3 A methodology to characterise the 'risk of harm'

Given the influence of the generality with which a risk of harm is characterised in determining obvious risk, how, then, is the correct measure of the spectrum of that generality to be judged?

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[73] Ibid 299.

[74] Ibid 324, quoting Lord Bridge in *Bolam v Friern Hospital Management Committee* [1957] 1 WLR 582.

[75] *H v Royal Alexandra Hospital* [1990] 1 Med LR 297, 324.

[76] Ibid 325.

[77] Although the warnings of the risk of harm were inadequate, the defendant was found not liable because his Honour considered that had the parents been properly warned, they would nonetheless have assented to the treatment, given the medical needs of their haemophiliac son and the low chances of infection. Badgery-Parker J's judgment also included the finding, on the balance of probabilities, that the plaintiff was infected in March 1982 — a date when the risk of blood contamination with HIV was not recognised.

[78] *Review of the Law of Negligence* (Final Report, September 2002) [8.30]–[8.31] ('*Ipp Report*').

In *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd*,[79] the majority High Court judgment of Gordon, Edelman and Gleeson JJ provided a methodology by which to characterise 'obvious risk' at the appropriate level of generality.[80] The essence of the majority opinion was that '[t]he risk with which s 5L [of the *Civil Liability Act 2002* (NSW)] is concerned is … the same risk as that with which s 5B is concerned'[81] and 'involve[s] the essential facts' that go to establish breach of duty.[82] The majority presented three matters of consideration.

One: '[T]he risk to which s 5L refers should be characterised at the same level of generality as it is characterised when assessing whether the defendant has breached a duty of care under s 5B.'[83] That is, the harm that materialised and is claimed to be an 'obvious risk' by the defendant must be the same harm that resulted from the breach under s 5B and expressed with the same level of generality (or precision). In context, the harm that materialised and is claimed by the defendant to be an obvious risk is CTE or, if characterised by type, a neurodegenerative disease or tauopathy.[84] In terms of aligning with 'breach', the plaintiff claims that the SGB failed to warn of the risk of CTE, or more by type, the risk of a neurodegenerative disease or tauopathy. The perspective from which the materialised harm is said to be 'obvious' is, of course, that of the reasonable premier-level athlete.

Two: '[T]he characterisation of the risk for the purpose of s 5L should be at the same level of generality as the risk considered for the purposes of the breach of duty which caused the harm to the plaintiff.'[85] Or, as otherwise expressed, 'the characterisation of the risk must include the "general causal mechanism of the injury sustained" which "gave rise to the potential for the harm for which the plaintiff seeks damages"'.[86] In context, the causal mechanism is exposure to RHT sufficient to cause CTE — or, if characterised by type, exposure to sufficient

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79 (2022) 273 CLR 454.

80 Following agreement by the parties as to the base mechanism of harm and the approximate resulting harm (should disagreement be an issue), debate may ensue as to the characterisation of harm and the mechanism of harm with respect to its generality or specificity. *Ceteris paribus*, the plaintiff pushes for a specific form of mechanism and harm to advance the argument that the 'obvious risk' doctrine is not applicable, while the defendant argues for a general characterisation to bring 'obvious risk' into application. For example, where it is agreed that the base mechanism of harm is falling off a horse rather than, say, being kicked by a horse, contention may extend to whether this general application is correct or something more specific is appropriate, such as a fall due to the deterioration of the ground on which the horse strode.

81 *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* (2022) 273 CLR 454, [116].

82 Ibid [119].

83 Ibid [112].

84 Their Honours continued (ibid): 'Since s 5L operates upon established liability based on duty, breach, and causation of harm, the risk to which s 5L refers must be the same risk that has "materialised" as a result of the "harm" for which "liability in negligence" would arise. That is the risk to which s 5B refers in the context of the requirement to establish a breach of a duty of care, upon which, in turn, the requirement of causation depends.'

85 Ibid [114].

86 Ibid, quoting *Perisher Blue Pty Ltd v Nair-Smith* (2015) 90 NSWLR 1, [98] (Payne JA).

RHT to cause a neurodegenerative disease or tauopathy — which would have been avoided had the plaintiff athlete been warned the risk of harm by his or her SGB.

Three: '[J]ust as it is unnecessary in the characterisation of risk for the purposes of assessing a breach of the duty of care for the plaintiff to show the reasonable foreseeability of the "precise manner in which [the] injuries were sustained", so too for the purposes of s 5L it is unnecessary for the defendant to show the precise manner in which the injuries were sustained for the purpose of characterising the risk.'[87] The manner by which CTE is sustained is, as argued and confirmed by multiple scientific studies since Dr Martland in 1928, RHT experienced in the playing of the SGB's contact sport. It is not, as argued, the experience of a single or a few symptomatic concussions.

The majority's methodology was paraphrased by McWilliam J in *Glover v Fuller (No 2)* as follows:

> The risk should be characterised at the same level of generality as it was characterised when assessing breach under s 5B of the *Civil Liability Act*, including the same facts as those that established the risk under s 5B and without descending to the precise detail of the mechanism by which an injury was suffered, if that detail was unnecessary to establish a breach of duty.[88]

The application offered in *Tapp* is best illustrated through the facts of the case. The majority found that the characterisation of the risk of harm (applicable to breach, causation and obvious risk) was a 'substantially elevated risk of physical injury by falling from a horse that slipped by reason of the deterioration of the surface of the arena'.[89] The defendant Rodeo Association, as a 'reasonable person', should have taken 'the immediate precaution of stopping the event in response to the substantially elevated risk of contestants being injured from falling from a horse that slipped on the deteriorated surface of the arena'.[90] On the factual evidence of 'four falls, all occurring in the hour leading up to Ms Tapp's incident',[91] 'but for' the Rodeo Association's failure to take that precaution, Ms Tapp would not have suffered harm.[92] On the factual evidence that a reasonable person in Ms Tapp's position could not have been aware of the risk of harm posed by 'substantially elevated risk of physical injury by falling from a horse that slipped by reason of the deterioration of the surface of the arena', the risk of harm was not an 'obvious risk'.[93]

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87 *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* (2022) 273 CLR 454, [115], quoting *Perisher Blue Pty Ltd v Nair-Smith* (2015) 90 NSWLR 1, [100]–[101] (Payne JA).

88 *Glover v Fuller (No 2)* [2023] ACTSC 12, [107].

89 *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* (2022) 273 CLR 454, [125].

90 Ibid [126]. While a somewhat general characterisation, it should be noted that this passage narrows the scope of the relevant risk by referencing both the degree of harm and the mechanism of its occurrence.

91 Ibid [22].

92 Ibid [144].

93 Ibid [151].

9.6.4 Characterising CTE as the risk of harm: How general is too general?

Section 5G(2) of the *Civil Liability Act 2002* (NSW) 'Injured persons presumed to be aware of obvious risks' provides:

> For the purposes of this section, a person is aware of a risk if the person is aware of the type or kind of risk, even if the person is not aware of the precise nature, extent or manner of occurrence of the risk.[94]

That the 'obvious risk' defence remains operative where a plaintiff lacked awareness of the specific harm but possessed knowledge of the general type of harm begs the question of how general must knowledge of harm be before it bears too little resemblance to the materialised harm to be deemed 'obvious'.

A 'general' risk is not a risk at large or without parameters — a characterisation of risk can be 'far too broad' and 'not include the essential facts that constituted the alleged breach of duty'.[95] As said by the Victorian Court of Appeal in *Erickson v Bagley*: '[W]hat must be reasonably foreseeable is the nature of the particular harm that ensued, or, more relevantly, the nature of the circumstances in which that harm was incurred.'[96]

J Forrest J stated in *Ultra Thoroughbred Racing Pty Ltd v Those Certain Underwriters* that 'it is important to remember that the High Court has on a number of occasions reminded lower courts that it is necessary to identify with some precision the nature of the foreseeable risk in determining both the question of duty and breach at common law'.[97] His Honour noted further that the '[i]dentification of the risk enables a court to determine whether the risk is, indeed, foreseeable and then (if applicable) to identify the relevant considerations in relation to breach'.[98]

According to Leeming JA in *Menz v Wagga Wagga Show Society Inc*, while it is accurate to say that 'obvious risk is in principle to be specified with a degree of generality', nonetheless 'the specification of the obvious risk must be sufficiently precise as to capture the harm which resulted from its materialisation on the facts of the particular case'.[99] His Honour stated further

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94 See n 7 for the equivalent provisions in the other states.

95 *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* (2022) 273 CLR 454, [122].

96 *Erickson v Bagley* [2015] VSCA 220, [33].

97 *Ultra Thoroughbred Racing Pty Ltd v Those Certain Underwriters* [2011] VSC 589, [290].

98 Ibid.

99 *Menz v Wagga Wagga Show Society Inc* [2020] NSWCA 65, [70]–[71] (Payne and White JJA agreeing).

in respect of the mechanism of harm: 'If it is too generally expressed, such that the nature of what occurred is mischaracterised, then a greater degree of specificity is required.'[100]

Noting that the High Court majority in *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd*[101] required correspondence between obvious risk, breach and causation, and in line with the comments of Leeming JA above, this chapter argues that a general characterisation of the risk of harm as, say, 'concussion', 'head trauma' or similar forms of injury description are inapt in capturing the harm of CTE. In short, concussion is not CTE.

In *Benic v New South Wales*, Garling J stated that '[i]t is essential in considering the first step [that must be taken to establish a breach of duty under the *Civil Liability Act 2002* (NSW); that is, the identification of a risk of harm] to carefully identify the particular risk of harm to which the later steps will be applied'.[102] His Honour continued: '[B]efore any consideration of causation as provided for in s 5D of the *Civil Liability Act*, it is sufficient if the risk of harm is described as a class of injury, as distinct from the particular injury actually suffered by the plaintiff.'[103]

Notwithstanding, it is proposed here that the correct characterisation of CTE as a 'risk of harm' requires a degree of specificity in respect of both the mechanism of harm and the actual harm that results from that mechanism. That is, CTE is to be characterised specifically as the disease 'CTE' or, alternatively, as a 'type' of harm best described as a 'neurodegenerative disease', which includes Alzheimer's disease, Creutzfeldt–Jakob disease, dementia and parkinsonism,[104] or alternatively as a 'tauopathy' that is defined 'by the presence of tau protein aggregates in the brain' and includes 'Alzheimer's disease, progressive supranuclear palsy, corticobasal syndrome, [and] some frontotemporal dementias'.[105] Correspondingly, the plaintiff athlete will claim that the harm suffered is CTE (or, by type, a tauopathy or neurodegenerative disease), which was not an obvious risk to the reasonable premier-level athlete in the pre-*circa* 2010 era. The mechanism of harm is RHT in playing contact sport.

9.6.5 CTE: The necessity for specification

A defendant SGB claiming no liability must show that the harm that materialised was caused by an obvious risk. In contrast, a plaintiff athlete can only claim compensation where the risk

[100] Ibid [71].

[101] (2022) 273 CLR 454.

[102] *Benic v New South Wales* [2010] NSWSC 1039, [82], referring to the judgment of Gummow J in *Roads and Traffic Authority of NSW v Dederer* (2007) 234 CLR 330, [59]–[61].

[103] [2010] NSWSC 1039, [83].

[104] M Bruzova et al, 'Autopsy-diagnosed Neurodegenerative Dementia Cases Support the Use of Cerebrospinal Fluid Protein Biomarkers in the Diagnostic Work-up' (2021) 11 *Scientific Reports* 10837.

[105] M Orr et al, 'A Brief Overview of Tauopathy: Causes, Consequences, and Therapeutic Strategies' (2017) 38(7) *Trends in Pharmacological Science* 637, 637.

of harm was not obvious. As such, the proposals of a defendant and a plaintiff will differ where either the risk of harm is general as against specific, or where the harm that materialises is not obvious; for example, consider the difference between harm expressed generally as 'head trauma' as compared to a specific description such as 'neurodegenerative harm', or harm described as 'Alzheimer's disease' as against 'CTE'.

In following on from the previous section, this section argues that harm characterised as, say, concussion or head trauma lacks the precision necessary to capture the harm of CTE. Several rationales are discussed below, largely reflecting the formulation of the majority judgment in *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd*.[106] In essence, the characterisation of the risk of harm must correspond across the areas of obvious risk, breach and causation. Several of the sub-sections below consider these elements in various configurations in explaining why the proper characterisation of harm is one of specificity (CTE, neurodegenerative disease or tauopathy) to the exclusion of harm described in general terms (concussion or head trauma). Given a commonality of facts, some overlap between various considerations is expected.

A Obvious risk and breach to be characterised at the same level of generality

As said by the High Court majority in *Tapp* and reiterated by McWilliam J in *Glover v Fuller (No 2)*,[107] 'obvious risk' is characterised at the same level of generality as that applied to the assessment of breach of duty under s 5B of the *Civil Liability Act 2002* (NSW) and its equivalents. In *Shaw v Oakdale Junior Motorcycle Club Inc*, Cavanagh J said similarly: '[O]n any variation of the risk of harm to encompass the circumstances of what actually occurred, the risk was foreseeable within the meaning of s 5B(1)(a) and not insignificant within the meaning of s 5B(1)(b) of the [*Civil Liability Act*].'[108]

Accordingly, in assessing the generality with which the risk of harm is to be characterised, a court is to consider foreseeability, the insignificance of the risk, and the response of a reasonable person given the circumstances. In the context of a claimed breach by failure to warn of the risk of CTE, these three factors would translate as follows: (1) The risk of a player contracting CTE if not warned was or ought to have been foreseen by the SGB. (2) The risk of CTE to the player was not insignificant. (3) In the circumstances, a reasonable person would have taken precautions given the likely seriousness of harm in the form of CTE, the minor burden involved in issuing a warning, and the slight cost in social utility. Considering the 'general principles' of s 5B, the defendant SGB is argued to have been negligent in failing to warn the plaintiff athlete of the risk of CTE (or, if classified by type, a tauopathy or neurodegenerative disease).

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[106] (2022) 273 CLR 454.

[107] [2023] ACTSC 12, [107], extracted at **[9.6.3]** above.

[108] *Shaw v Oakdale Junior Motorcycle Club Inc* [2020] NSWSC 180, [224].

B Obvious risk and causal mechanism and the level of generality

The medical cause of CTE is exposure to RHT, including both concussive and sub-concussive trauma, of sufficient quantity and force to liberate the protein tau from the axons of cerebral neurones. The legal cause is the athlete's participation in the SGB's competition, which the athlete would have avoided if warned of the risk of CTE.[109]

C The subject matter of the failure to warn of CTE

The claimed negligence of the SGB concerns its failure to warn its premier-level athletes of the risk of CTE, the natural corollary of which is the question: If warned of the risk of frank concussion (with no mention of CTE-like harm), would a plaintiff athlete have avoided playing the SGB's contact sport in the SGB's premier-level competition? Leaving aside the indisputable fact that the cause and consequence of concussion would have been witnessed by premier-level athletes for years and that concussion is thereby classifiable as an obvious risk, it is doubtful that a warning of concussion in isolation from a warning of CTE-like harm would induce an athlete to retire from a sport where severe or even life-threatening injuries are a demonstrable and accepted hazard of play. After all, the fact remains that prior to the modern revelations of CTE, premier-level athletes would have witnessed multiple times their colleagues suffering the effects of symptomatic concussion only to resume normal play a week or two later apparently none the worse.

Other possible descriptors of concussion-like harm (as opposed to CTE) are also inadequate to characterise the risk of CTE in legal terms, including variations such as 'the risk of being tackled and being injured', or 'the risk of tackling or being tackled and suffering head trauma', or 'the risk of suffering multiple concussions'. While concussion and multiple concussions are forms of harm, neither discloses the symptoms and sequelae of CTE and therefore neither adequately warns of the true materiality of harm.

As such, this chapter argues that a description of the risk of CTE requires a higher level of particularity that characterises CTE specifically as the risk of harm or, by type, as a 'neurodegenerative disease' or 'tauopathy'.

D Causal connection between harm and risk

Leeming JA in *Menz v Wagga Wagga Show Society Inc* noted that s 5L only defeats a plaintiff's claim 'where the harm is suffered "as a result of" the materialisation of an obvious risk',[110] and stated that the phrase 'as a result of' in that section 'requires a close causal connection between the harm and the risk which materialises. … If the risk is obvious, it may be expected that there is a close causal connection between the risk which materialises and the harm.'[111] A causal

[109] In a 'failure to warn' case, the legal cause of harm is the plaintiff's participation in an event that he or she would have avoided if warned by the defendant. In addition, it may also have to be proven that participation in the event caused the harm for which the plaintiff seeks compensation. See *Amaca Pty Ltd v Booth* (2011) 246 CLR 36.

[110] *Menz v Wagga Wagga Show Society Inc* [2020] NSWCA 65, [60] (Payne and White JJA agreeing).

[111] Ibid [73].

connection is established where the risk that materialises flows from, or is associated with, exposure to the mechanism of foreseeable risk. For example, consider the scenario in which a defendant does not give a warning that, due to a shallow rock platform in a public pool, there is a risk of quadriplegia to those who dive into the pool. A causal connection between the harm (quadriplegia) and the risk that materialised is established where a plaintiff, not having been warned, dives into the pool, strikes his head on the shallow rock platform and suffers quadriplegia. The risk said to be obvious must correspond with the materialised harm. In this case, the materialised harm is quadriplegia (or, more generally by type, spinal injury). The question to be asked is: In the circumstances, was quadriplegia (or spinal injury) an obvious risk to a reasonable person in the position of the plaintiff?

The harm that materialised in the instant discussion is CTE. The causal connection is exposure to RHT, which is the only known cause of CTE. If one wished to view causation in terms of foresight, exposing a contact sport athlete to RHT risks harm in the form of CTE.

E Specifying the causal mechanism

The High Court majority commented in *Tapp* that 'the characterisation of the risk must include the "general causal mechanism of the injury sustained" which "gave rise to the potential for the harm for which the plaintiff seeks damages"'.[112] In context, did the mechanism of RHT to which the plaintiff athlete was exposed give rise to the potential for CTE?

As argued, and without going into the cerebral processes, the mechanism of medical causation is *repetitive* head trauma (including both concussion and asymptomatic concussion) of such quantity that the protein tau is released from the axons of the brain to form abnormal hyperphosphorated tau that clumps together to disrupt the cells' transport system, slowly spreading 'over decades to involve widespread regions of the neocortex, medial temporal lobe, diencephalon, basal ganglia, brainstem and spinal cord'.[113] On the other hand, the causal mechanism of 'concussion' in the playing of contact sport is a blow to the head or body of sufficient force to cause the brain to contort, make contact with the inner skull and produce the symptoms of concussion.[114] This description does not include asymptomatic concussion.

F The harm for which the plaintiff seeks damages

With reference to 'the potential for the harm for which the plaintiff seeks damages',[115] the stimulus causing harm is RHT in the playing of contact sport. The harm for which the plaintiff athlete seeks damages is CTE (or, by type, a neurodegenerative disease or tauopathy). Noting

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[112] *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* (2022) 273 CLR 454, [114], quoting *Perisher Blue Pty Ltd v Nair-Smith* (2015) 90 NSWLR 1, [98] (Payne JA).

[113] Ann McKee et al, "The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43, 60.

[114] G Symons et al, The Neurological Consequences of Engaging in Australian Collision Sport, (2020) 27 *Journal of Neurotrauma*, 792.

[115] *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* (2022) 273 CLR 454, [114], quoting *Perisher Blue Pty Ltd v Nair-Smith* (2015) 90 NSWLR 1, [98] (Payne JA).

that the proper characterisation of the relevant risk is 'fact-dependant',[116] the risk of harm is exposure to RHT. The potential for harm (and the harm that arose) is CTE. It is CTE for which the athlete seeks damages.

For the purposes of s 5L, 'it is unnecessary for the defendant to show the precise manner in which the injuries were sustained for the purpose of characterising the risk'.[117] As such, there is, as noted above, a defensive argument that the foreseeability of concussion or head trauma is sufficient to establish CTE as an 'obvious risk'. However, quite clearly, the harm endured by an athlete suffering the symptoms of CTE-related dementia is not concussion, but CTE.[118]

As regards the harm suffered by the plaintiff athlete, a comparison is made between the symptoms, sequelae and prognosis of symptomatic concussion as against those of CTE. A concussion is

> defined as a traumatically induced transient disturbance of brain function that involves a complex pathophysiological process. Concussion is a subset of mild traumatic brain injury which is classified based on acute injury characteristics at the less severe end of the brain injury spectrum. The clinical signs and symptoms of concussion cannot be otherwise explained by drug, alcohol, medication use, or other injuries (such as cervical injuries or peripheral vestibular dysfunction) or other comorbidities (psychological or medical conditions).[119]

Furthermore, although symptoms of concussion include various cognitive effects, 'most resolve within 7–14 days'.[120] It can also be noted that a suit based on a failure to warn of concussion, given its ubiquity, would be indeterminant.

The harm of symptomatic concussion usually manifests as 'non-specific symptoms such as headaches, mood changes, "fogginess", dizziness, visual changes, fatigue and neck pain'.[121]

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[116] *Menz v Wagga Wagga Show Society Inc* [2020] NSWCA 65, [74].

[117] *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* (2022) 273 CLR 454, [115].

[118] It should be stressed that where a breach of duty concerns a failure to warn, the failure to warn must have caused the plaintiff to engage in conduct that would otherwise have been avoided, and such engagement must have resulted in harm: *Wallace v Kam* (2013) 250 CLR 375. CTE is taken to refer also to *dementia pugilistica* or by its symptoms and sequelae.

[119] KG Harmon et al, 'American Medical Society for Sports Medicine Position Statement on Concussion in Sport' (2019) 4(53) *British Journal of Sports Medicine* 213, 214. A further informative definition is 'a type of traumatic brain injury (TBI) that results in temporary impairment of mental function. Concussions are sometimes referred to as mild traumatic brain injuries (mTBI) to distinguish them from more serious types of brain injuries. … The main treatment for concussion is rest, both physical and mental, usually for one to two days following the injury': 'Facts Sheets: Concussion', *Yale Medicine* (Web Page) <https://www.yalemedicine.org/conditions/brain-concussion>.

[120] Richard Bunworth, 'Egg-shell Skulls or Institutional Negligence? The Liability of World Rugby for Incidents of Concussion Suffered by Professional Players in England and Ireland' (2016) 16 *International Sports Law Journal* 82, 83. Bunworth notes that some symptoms may persist for weeks or months, or may in some cases be lifelong.

[121] KG Harmon et al, 'American Medical Society for Sports Medicine Position Statement on Concussion in Sport' (2019) 4(53) *British Journal of Sports Medicine* 213, 213. Other symptoms include 'vertigo, nausea, vision and speech disturbances, and — less commonly — difficulty concentrating, sensitivity to noise or light, behavioural or emotional changes, feeling dazed and fatigue': 'Concussion', *Health Direct* (Web Page) <https://www.healthdirect.gov.au/concussion>.

The symptoms of concussion are distressing and discomforting but, as stated above, generally resolve within a few days. Although a blow to the head may lead to 'persistent post-concussive syndrome' that may last several months, this malady is not one for which contact sport athletes in Australia appear to be seeking compensation. Moreover, as a professional premier-level athlete is usually paid while convalescing, a claim for damages in respect of lost earnings is redundant.[122]

In contrast, the harm for which a plaintiff athlete seeks compensation is the 'insidious' cognitive and behaviour disfunction of CTE, which includes 'profound loss of attention and concentration, executive dysfunction, language difficulties, explosivity, aggressive tendencies, paranoia, depression, gait and visuospatial difficulties, impulsivity, dysarthria and parkinsonism' — sequelae that over time make work of all forms impossible — and which leads ultimately to death.[123]

Chapter 2 and Appendix 1engaged in a deep discussion of the characteristics of CTE and examined its cause: RHT, leading to the release of tau from the axons of the brain to accumulate as phosphorated tau in neurons and neurites, to then form into neurofibrillary tangles in the depths of the sulci and to spread throughout the brain over a period of years. The chapter and appendix considered the symptoms, sequelae and behavioural manifestations of CTE, for which there is no known cure, along with a prognosis that is fatal. To highlight the issue: 'CTE is a definite neuropathological entity with quite characteristic histological features and relatively protean clinical manifestations',[124] and is not a variation of concussion.

In considering the appropriate degree of 'generality' or 'particularity' in respect of the risk of harm, it is inescapable that the harm in question is CTE or, by type, a neurodegenerative disease or tauopathy, or by whatever nomenclature or cluster of symptoms and sequelae are utilised in description. In summary, an SGB claiming that concussion, head trauma or head knocks are obvious risks of contact sport is secure in its argument; however, understandably, the risk of concussive trauma is not the basis of the plaintiff athlete's claim of breach and would not avoid the operation of the obvious risk provisions.

While both CTE and frank concussion affect the brain and cognition, the symptoms and sequelae noted above disclose that they are not only different maladies, but also different *types* of maladies. Relevantly, McKee et al described the pathology of CTE as 'unique', with 'a

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[122] One could consider the commonly fatal condition of 'second impact syndrome', a malady for which little evidence exists, which is doubted in some medical circles and is extremely rare in any case: T Bey and B Ostick, 'Second Impact Syndrome' (2009) 10(1) *Western Journal of Emergency Medicine* 6, 6. The article drew upon comments from Paul McCrory in 'Does Second Impact Syndrome Exist?' (2001) 11(3) *Clinical Journal of Sports Medicine* 144.

[123] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43. See Chapter 2 'Concussion, Chronic Traumatic Encephalopathy and Sport in a Legal Setting'.

[124] Roger Byard et al, 'Chronic Traumatic Encephalopathy (CTE) — Features and Forensic Considerations' (2023) 19(4) *Forensic Science, Medicine and Pathology* 620, 623.

distinctive molecular structural configuration of p-tau fibrils that is unlike the changes observed with aging, Alzheimer's disease, or any other tauopathy'.[125]

G In summary

To return to a basic definition: Meagher JA in *Liverpool Catholic Club Ltd v Moor* described an obvious risk as 'one which is clearly apparent or easily recognised or understood'.[126] Tobias JA in *Wyong Shire Council v Vairy* said that '"obvious" means that both the condition and the risk are apparent to and would be recognised by a reasonable man, in the position of the [plaintiff], exercising ordinary perception, intelligence and judgment'.[127] As argued, knowledge of the obvious risk of concussion or head trauma does not indicate that the permanent disfunction of CTE is clearly apparent, easily recognised or understood.

9.6.6 The inefficacy of concussion as a descriptor of the risk of harm

Consider how a characterisation that looked simply at 'concussion' or 'head trauma' (as opposed to CTE) as the relevant risk of harm would be applied if argued as an obvious risk: As claimed by the plaintiff athlete, the defendant SGB possessed a duty to warn its premier players of the risk of concussion. As claimed by the plaintiff, the defendant breached that duty by not warning its premier-level athletes of the risk of concussion. As counter-claimed by the defendant, concussion is an 'obvious risk' in the playing of its contact sport. As the risk of concussion is indeed obvious, the defence would apply to defeat the SGB's liability. The athlete faces the further frustration that a suit based on a failure to warn of concussion would likely be considered indeterminant in avoiding an obligation of care.[128]

There is of course a temptation for a plaintiff athlete to argue: 'Had I been warned of the risk of concussion, I would not have played the SGB's contact sport and therefore I would not have suffered CTE.' Where concussion is not linked to a more serious condition, the plaintiff's argument is similar to saying: 'Had I been warned of the risk of a ruptured knee, I would not have played the SGB's contact sport and therefore I would not have suffered CTE.' In this there are parallels to the argument of the plaintiff in *Wallace v Kam*.[129] Mr Wallace had surgery on his lumbar spine. He was not warned of the risk, which eventuated, of 'bilateral femoral neurapraxia' caused by lying face down on the operating table for an extended period. He had, however, been warned of the one-in-20 chance of permanent and catastrophic paralysis caused by damage to his spinal nerves. Mr Wallace claimed that if he had been warned of the risk of

[125] Ann McKee et al, 'Chronic Traumatic Encephalopathy (CTE): Criteria for Neuropathological Diagnosis and Relationship to Repetitive Head Impacts' (2023) 145(4) *Acta Neuropathologica* 371, 371.

[126] *Liverpool Catholic Club Ltd v Moor* [2014] NSWCA 263, [23].

[127] *Wyong Shire Council v Vairy* [2004] NSWCA 247, [161].

[128] *Perre v Apand* (1999) 198 CLR 180.

[129] (2013) 250 CLR 375.

neurapraxia, he would not have undergone the operation and would not therefore have suffered neurapraxia. At trial, Harrison J found Mr Wallace would have undergone the surgery even if warned of the risk of neurapraxia, and stated further that 'the "legal cause" of the neurapraxia "could never be the failure to warn of some other risk that did not materialise"'.[130] Before the High Court the question was considered utilising an example provided by Lord Hoffman in *Banque Bruxelles SA v Eagle Star* of a mountaineer who is negligently advised by his doctor that his knee is fit for a difficult climb. He undertakes the climb but is injured in an avalanche. The court affirmed that the injury is a 'foreseeable consequence of mountaineering but has nothing to do with his knee'.[131]

There is an assumption implicit in the above discussion that an athlete warned of concussion would retire from contact sport from contact sport. It can be rightly argued, however, that the risk of a relatively minor injury such as concussion (without reference to the serious disease of CTE) is unlikely to convince a professional athlete to retire from his or her career. It would seem demonstrably, if anecdotally, that those who retire from contact sport following RHT do so where they have been warned of CTE-like harm or upon experiencing unresolved cognitive deficits.[132] It can also be noted that where discussion of permanent cognitive harm in the media and within interested persons, the topic is not concussion but of CTE.

In *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd*,[133] the High Court majority approved of a characterisation of a risk of harm that quantified the degree of harm. According to McCallum JA in the New South Wales Court of Appeal, the correct description was not 'the risk of injury as a result of falling from a horse that slipped' but 'the *substantially elevated* risk of physical injury by falling from a horse'.[134] Clearly, when one compares the consequences of CTE against those of concussion, there is a 'substantially elevated risk of physical injury'. Having said that, it is nonetheless argued below that correct characterisation requires a more precise description — that of 'CTE' itself or of a 'neurodegenerative disease' or 'tauopathy'.

Reflective of the rules noted in *Tapp*, Lord Reid stated in *Watson v Fram Reinforced Concrete Co (Scotland) Ltd* that '[t]he ground of any action based on negligence is the concurrence of breach of duty and damage'[135] — in context, breach being a failure to warn of

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[130] *Wallace v Kam* (2013) 250 CLR 375, [3], quoting *Wallace v Ramsay Health Care Ltd* [2010] NSWSC 518, [96].

[131] *Wallace v Kam* (2013) 250 CLR 375, [24], referring to *Banque Bruxelles Lambert SA v Eagle Star Insurance Co Ltd* [1997] AC 191, 213 (also known as *South Australia Asset Management Corp v York Montague Ltd*). See also *Kenny & Good Pty Ltd v MGICA (1992) Ltd* (1999) 199 CLR 413.

[132] There are multiple examples of this response reported in the media and referred to throughout this thesis.

[133] (2022) 273 CLR 454.

[134] Ibid [125], quoting *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* [2020] NSWCA 263, [166]. Emphasis added.

[135] *Watson v Fram Reinforced Concrete Co (Scotland) Ltd* (1960) SC HL 92, 109.

CTE and damage being the contraction of CTE. In *Khan v Meadows*[136] a patient was wrongly advised by her doctor that she was not a carrier of haemophilia. On this advice, she decided to have a child. The child was born with haemophilia and autism (which was not caused by the haemophilia). The medico admitted to negligence regarding the failure to **co**nduct genetic tests regarding haemophilia, but the court found he was not responsible for the autism. The Supreme Court of the United Kingdom in *Manchester Building Society v Grant Thornton UK LLP* said: '[O]ne looks to see what risk the duty was supposed to guard against and then looks to see whether the loss suffered represented the fruition of that risk.'[137]

9.6.7 The bind: Foreseeable risk of harm and obvious risk

Bearing in mind that a 'type' of harm is sufficient to ground breach and obvious risk, and that the level of generality used to describe breach and obvious risk must correspond,[138] a characterisation of the risk of harm as simple concussion or 'head trauma' presents potential difficulties in consistency for plaintiff athletes.

For an affected athlete, the argument of a breach of duty is easily made in respect of a failure to warn of the risk of frank concussion. In basic terms, the risk was foreseeable and (perhaps) not insignificant. The risk of concussive trauma is probable in any given week of playing contact sport. The burden of taking precautions — at least as regards the issuance of a warning — is light, and the social utility of a sport is unlikely to be affected by players suffering a single concussion.

The bind arises because of the common 'risk of harm' applicable to breach and obvious risk. A plaintiff athlete is (initially) advantaged in claiming that concussion, head trauma or a head knock is the relevant risk and was foreseeable to a reasonable SGB. In doing so, however, the plaintiff is effectively identifying a risk of harm that is 'obvious' and, for the purposes of the statute, is 'liability-defeating'.[139] Symptomatic concussion is demonstrably common in contact sports and, indeed, is manifestly visible. The proposition that the risk of concussion is sufficiently foreseeable that an SGB must be aware of it, but at the same time not obvious to premier-level athletes, deals in unreality. Clearly the risk of concussion is an obvious risk and, further, an obvious risk of a dangerous recreational activity.[140]

Even if the risk of concussion could somehow be classified as not obvious, the athlete encounters the problem that while concussion is highly distressing, it is generally transient and

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[136] [2021] UKSC 21.

[137] *Manchester Building Society v Grant Thornton UK LLP* [2021] UKSC 20, [17].

[138] *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd* (2022) 273 CLR 454, [110]. And also bearing in mind that 's 5L operates upon established liability based on duty, breach, and causation of harm': [112].

[139] *Goode v Angland* (2017) 96 NSWLR 503, [185]. Per Leeming JA.

[140] A similar 'interplay' between breach and obvious risk was noted by Beech-Jones J in *Collins v Clarence Valley Council* [2013] NSWSC 1682, [113].

not serious.[141] Even if liable, what meaningful compensation could the athlete seek in damages from suffering a single concussion unlinked to serious harm?

Consequently, a characterisation of CTE as a 'type' of concussion or the very broad 'head trauma' is ineffective from a plaintiff athlete's perspective in securing compensation. Claims of negligent failure to warn in the period before *circa* 2010 must, as a matter of logic, concern the fact that CTE is a different type of harm to concussion and was not, as such, an obvious risk to the reasonable player of contact sport at the time.

9.7 Obvious to a reasonable person in the position of that person (the victim)

The 'obvious risk' element of the defence of obvious risk raises the questions of if, when and how the plaintiff athlete is supposed to have become aware of such a risk: '[A] person who suffers harm is presumed to be aware of the risk of harm if it is an obvious risk. The focus in on a reasonable person in the position of the *plaintiff*.'[142]

The relevant application of obvious risk was discussed in the section above, 'Obvious risk: Conduct and harm' and need not be considered extensively here, other than to point out that the personal traits of the plaintiff are to some degree incorporated into those of the reasonable person, including 'the age, experience and personal characteristics of the person',[143] and, as above, the reasonable person exercises 'ordinary perception, intelligence, and judgment' in discerning an obvious risk.[144] In addition,

> one is required to have regard to the particular circumstances in which the [plaintiff] suffered the relevant harm … [A]ll of the surrounding circumstances which occurred immediately prior to the [plaintiff's] suffering the relevant harm must also be identified for the purpose of determining whether the risk which materialised was 'obvious'.[145]

In brief compass, premier-level contact sport athletes are young, perhaps beginning their career in their early 20s and continuing through to their early 30s, with the limited judgment and perception of risk that youth implies, along with many other age-related traits. Immaturity of age, coupled with experience in the playing of contact sport at premier level, is likely to imbue such athletes with a particular psychological disposition as regards an appreciation of risks of harm and related concerns. They will have personal knowledge of risks of harm gained through years of play. They may have acquired many insights associated with CTE and cognitive disfunction in contact sport athletes, or there may be an element of apparent or

[141] G Symons et al, The Neurological Consequences of Engaging in Australian Collision Sport, (2020) 27 *Journal of Neurotrauma*, 792, 793.

[142] *Menz v Wagga Wagga Show Society Inc* [2020] NSWCA 65, [56]. Emphasis added.

[143] *Carey v Lake Macquarie City Council* [2007] NSWCA 4, [97] (McCLellan CJ).

[144] *Wyong Shire Council v Vairy* [2004] NSWCA 247, [161].

[145] *Fallas v Mourlas* (2006) 65 NSWLR 418, [98].

believed innocuity regarding CTE. Personal characteristics will include those touching on their capacity to perceive CTE as an obvious risk. To illustrate, in *Jaber v Rockdale City Council* Tobias JA emphasised that a determination 'may well depend upon the extent to which the probability of [the risk's] occurrence is or is not readily apparent to the reasonable person in the position of the plaintiff'.[146]

Consider the application of obvious risk through the example of the 19-year-old plaintiff who suffered quadriplegia when she fell from her horse while warming up for a 'campdrafting' event in *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd*.[147] The cause of the fall was at least partly due to the 'substantial deterioration' of the arena's surface.

The defendant event organiser claimed the risk was obvious to the plaintiff. The High Court gave three reasons why the deteriorated service was not an obvious risk: (1) The plaintiff did not have the opportunity to examine the condition of the ground before competing.[148] (2) '[A] reasonable person in [the plaintiff's] position would not have had any concerns about the condition of the ground from observations of other contestants or information about other contestants', and she had not observed any falls.[149] (3) A reasonable person in the plaintiff's position would rely on the committee overseeing the event for an assessment of the arena. Here the plaintiff's age was emphasised: teenagers are 'likely to be less attuned to the risks that would be more obvious to a more experienced' member of the community.[150]

There are, in this description, some base parallels to the premier-level athlete who relies upon his or her SGB to inform of risks that cannot be known by the athlete. Other descriptors of the reasonable athlete may refer to his or her age, experience, and level of training and education (including the course undertaken), whether he or she is a professional athlete, and whether (depending on the sport) knowledge of 'punch drunk syndrome' (in boxing, for example, knowledge is arguably greater) can be attributed.

Noting that '[t]he obviousness of the risk is to be considered from the perspective of the plaintiff, prior to the incident occurring',[151] the timeline as to when information on CTE entered the consciousness of the 'objective athlete' is central to whether the risk of CTE was 'obvious'. In context: was the risk of CTE an obvious risk to a reasonable person playing the professional contact sport in question at the time? Bearing in mind that, with few exceptions, athletes diagnosed with CTE or said to be suffering CTE are predominantly in their 40s, 50s and 60s, and on the basis that the major contact sport codes in Australia did not introduce 'concussion

[146] *Jaber v Rockdale City Council* [2008] NSWCA 98, [35].

[147] (2022) 273 CLR 454.

[148] Ibid [152].

[149] Ibid [153], [154].

[150] Ibid [155].

[151] *Menz v Wagga Wagga Show Society Inc* [2020] NSWCA 65, [72].

protocols' or otherwise advise their players of the risk of long-term cognitive disfunction until around 2010, the likelihood that 'knowledge' was 'obvious' to these plaintiffs is remote. To take a rudimentary example, suppose an AFL player on his 52nd birthday in 2024 is diagnosed with CTE. If he played in the premier league between the ages of 20 and 30, he was exposed to the risk of CTE with his premier club between the years 1992 and 2002. Given the timeline above, would a reasonable player in his position have been aware of CTE as an obvious risk during his years of play? Today of course the risk of CTE is obvious to the reasonable contact sport athlete, which partly explains why some athletes retire from their sport after several concussions, at an indication of prolonged cognitive distress or when advised by medicos. Previously, the risk of CTE was not obvious, and seemingly there were no concerns in athletes continuing to play safe in the belief that they would recover from concussions, irrespective of the number experienced.

The inquiry, though, is not whether the plaintiff athlete was aware that head trauma or concussion occurs in contact sport, but whether the risk of CTE-like harm was obvious to the reasonable premier-level athlete playing in the SGB's contact sport prior to *circa* 2010. There is, as argued, a clear difference between an awareness of concussion and the debilitating harm of the neurodegenerative disease CTE.

Much will also depend on the calendar years in which the plaintiff participated in the SGB's premier-level competition; in particular, the degree of information — formal and informal — reasonably available at the time. Considering the timeline formulated from the three notable studies of CTE-like harm listed in Chapter 2 and the lower-impact Australian articles of the 1990s and early 2000s, several possibilities present:

- A reasonable person in the position of the plaintiff athlete would have read, or otherwise informed themselves in sufficient detail of, Dr Bennet Omalu et al's research published in 2005,[152] and would have known that CTE-like harm was an obvious risk in playing their sport.
- A reasonable person in the position of the plaintiff athlete would have read, or otherwise informed themselves in sufficient detail of, Dr Ann McKee et al's meta-research published in 2009.[153]
- A reasonable person in the position of the plaintiff athlete would have read, or otherwise informed themselves in sufficient detail of, Dr Harrison Martland's 'Punch Drunk' article published in 1928.[154]

[152] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[153] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

[154] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

- A reasonable person in the position of the plaintiff athlete would have read, or otherwise informed themselves in sufficient detail of, the hundreds of research papers on CTE-like harm published in multiple journals in the period between Martland's 1928 'Punch Drunk' article and the studies of Omalu et al or McKee et al in 2005 and 2009 respectively.
- A reasonable person in the position of the plaintiff athlete would have become aware of the risk of CTE at a point in time when the risk of harm was sufficiently publicised or when the SGB warned them.

The SGB's defence is prima facie made out should any of these scenarios have occurred. It may be submitted, however, that the degree of knowledge required of a 'reasonable contact sport athlete' to establish 'some conscious advertence' — that playing contact sport presents a risk of long-term, progressive cognitive harm in the form of CTE — is untenable. It is unlikely that, by 'exercising ordinary perception, intelligence and judgment', the risk of CTE would be 'apparent to' and 'recognised by' a reasonable contact sports athlete.[155]

Nonetheless, given the statutory 'obvious risk' provisions, there is reason to consider the establishment of a duty of care in the period *circa* 2010, when the risk of long-term cognitive harm in playing contact sport would be reasonably obvious to premier-level athletes.

9.7.1 The voluntary element of assumption of risk

Under s 5G of the *Civil Liability Act 2002* (NSW) ('Injured persons presumed to be aware of obvious risks'), a plaintiff is presumed to be aware of obvious risks of harm unless the plaintiff can prove that he or she was not aware of that risk.[156] An 'obvious risk' is a risk that 'would have been obvious to a reasonable person in the position of that person' — the injured person.[157]

The inference of the section is that a reasonable person aware of an obvious risk must, all things being equal, have volunteered or consented to that risk and any materialised harm.[158] Importantly, the civil liability legislation introduced the following additional provision:

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[155] *Laoulach v Ibrahim* [2011] NSWCA 402, [120] (Tobias JA).

[156] *Civil Liability Act 2002* (NSW) s 5G(2). See n 7 for the equivalent provisions in the other states. In application, the 'presumption' is often referred to as the plaintiff's 'assumption of risk'.

[157] See, eg, *Civil Liability Act 2002* (NSW) s 5F.

[158] Discussion of s 5G of the *Civil Liability Act 2002* (NSW) is placed in this position within the chapter for ease of reference to the factors regarding 'athlete knowledge' set out in **[9.7]** above: 'Obvious to a reasonable person in the position of that person (the victim)'.

> For the purposes of this section, a person is aware of a risk if the person is aware of the type or kind of risk, even if the person is not aware of the precise nature, extent or manner of occurrence of the risk.[159]

Again, the application turns on characterisation. If, for example, CTE is characterised as a form of symptomatic concussion or head trauma, plaintiff athletes would have difficulty proving they were unaware of the risk, particularly given that awareness of the 'precise nature, extent or manner of occurrence of the risk' is not necessary. However, as argued throughout this chapter and supported by multiple scientific reports referenced in this thesis, CTE is not a type of symptomatic concussion — by reason of its medical nature, the extent of harm endured and the manner of its occurrence; it is, rather, a type of neurodegenerative disease or tauopathy.

In this section, the argument regarding the 'presumption of awareness of obvious risk' follows that expressed throughout this chapter: CTE was not an obvious risk to contact sport athletes before *circa* 2010. The question with respect to the context of this section is whether a plaintiff athlete suffering the neurodegenerative symptoms of CTE can be found to have voluntarily assumed the risk of contracting CTE.

The defence under s 5G does not apply in several situations: where the risk of harm would not have been obvious to a reasonable person in the position of the plaintiff; where, although a risk is found to be obvious the injured person can prove on the balance of probabilities that he or she was not aware of the risk; and where the defendant cannot prove the plaintiff possessed 'some conscious advertence' that the risk might materialise and proceeded in 'the conduct regardless'.[160] Or, as otherwise expressed, the plaintiff must have 'fully appreciated' the risk.[161]

9.7.2 Discerning an assumption of risk

Consider in illustration the case of *Carey v Lake Macquarie Council*.[162] The plaintiff was injured when he struck a bollard that was in the middle of the concrete pathway along which he was cycling. He was aware of the bollard, as he often walked along a nearby track on his way to the beach, but he gave the obstruction no thought on this occasion. The defendant claimed that being aware of the bollard, the plaintiff had voluntarily assumed the risk of harm. The plaintiff maintained that as the 'bollard was not an impediment on his path to the beach,

[159] *Civil Liability Act 2002* (NSW) s 5G(2). The equivalent Victorian provision — s 54 of the *Wrongs Act* 1958 (Vic) — does not include the words 'nature, extent or manner of occurrence'. In addition, under s 54(2) of the Victorian Act there is no presumption of awareness of obvious risks in claims (relevantly to the present discussion) concerned with professional and health services, or work done by one person for another. In South Australia, an additional provision requires that the risk was one that a reasonable person would have taken steps to avoid, and that the plaintiff failed to take those steps: *Civil Liability Act 1936* (SA) s 37(3).

[160] *Carey v Lake Macquarie Council* [2007] NSWCA 4, [27] (McClellan CJ).

[161] *Randwick City Council v Muzic* [2006] NSWCA 66, [48].

[162] [2007] NSWCA 4. As the application in law and many of the factual points in issue were addressed throughout this chapter, this section is considered succinctly.

… he had not previously focused on it or appreciated the danger it could present to a cyclist'.[163] McClellan CJ stated:

> The question is not simply whether the plaintiff freely and voluntarily decided to embark upon a course of conduct that involved a risk of which he or she was aware. There must also be some conscious advertence to the possibility that the known risk might eventuate, and a decision to proceed with the conduct regardless. It is not enough that the plaintiff knows of the physical facts or circumstances that constitute the risk and exposes him or herself to them ...[164]

In addition to the plaintiff being aware of the obvious risk of harm or the type of harm (or being unable to establish that he or she was not aware of the obvious risk), McClellan CJ said:

> [I]t is apparent that to make out the defence of *volenti* the defendant must prove three elements:
>
> 1. That the plaintiff perceived the existence of the danger;
>
> 2. That he or she fully appreciated it;
>
> 3. That he or she freely and voluntarily agreed to accept the risk.[165]

9.8 CTE as a *not* 'obvious risk of harm' to a reasonable premier-level contact sport athlete

The application of obvious risk was canvassed extensively in the sections above, 'Obvious risk: Conduct and harm' (at **[9.6.2]**) and 'Obvious to a reasonable person in the position of that person (the victim)' (at **[9.7]**), and need not be again considered in detail. Nonetheless, to again orientate the issue: athletes who played before *circa* 2010 (which is virtually all of those athletes who today have been diagnosed with CTE or are said to be suffering the symptoms of CTE) could not have been aware of the risk of CTE as required under s 5G(1) of the *Civil Liability Act 2002* (NSW) or its equivalents.

9.8.1 Subjective perception of the obvious risk

That a risk of harm is objectively obvious does not mean it was necessarily subjectively obvious to a plaintiff. Should it be found (contrary to the position taken in this chapter) that CTE was an obvious risk prior to *circa* 2010, a claim of negligence will not be defeated where a plaintiff athlete can prove that he or she was not aware that CTE posed a risk of harm. Facts that establish or infer that the athlete was not aware of the risk of CTE are relevant. Of course, an unknown risk cannot be perceived.

9.8.2 Awareness of the type or kind of risk is sufficient

Awareness of the 'type' or 'kind' of harm is sufficient to establish awareness, and this does not require the plaintiff to be aware of the 'precise nature, extent or manner of occurrence of the

[163] Ibid [27].

[164] Ibid [107].

[165] Ibid [85].

risk': s 5G(2). Irrespective of the generality that this provision seeks to incorporate into the assessment of obvious risk, CTE is entirely different — in terms of nature, the extent of harm suffered and the manner by which it occurs — to symptomatic head trauma. An obvious risk must be identified with sufficient specificity to permit the risk to be calculated. In the words of McClellan CJ in *Carey v Lake Macquarie Council*, 'what risks are the provisions directed to? It serves no purpose to answer that they are directed to "obvious risks," because no conclusion can be reached as to whether or not a risk is obvious until the risk itself is identified.'[166] The possible scenarios by means of which information may have come the way of premier-level athletes presented in the section at **[9.7]** above, 'Obvious to a reasonable person in the position of that person (the victim)', are relevant to this discussion of awareness of a risk of harm.

Can it be said, then, that CTE is a type of symptomatic concussion or head trauma for the purposes of the 'assumption of risk' provisions? Arguments throughout this thesis regarding the infelicity of characterising CTE as a form of concussion or head trauma (that is, a true obvious risk) rather than as a neurodegenerative disease or tauopathy are pertinent here. At a basic level, could an SGB point to warnings issued by it prior to 2010? Could it be expected that a professional contact sport athlete would be a reader of medical journals in general and, more particularly, those specialising in neurology? There can be no credible suggestion that pre-*circa* 2010, an athlete who was aware of the risk of symptomatic concussion was necessarily aware of the entirely different and debilitating condition of CTE.

9.8.3 Full appreciation of the danger

A defendant SGB must prove that the plaintiff athlete 'fully appreciated' the risk of CTE.[167] Two points are relevant here: (1) There can be no appreciation of a risk of harm where that harm was not known to the plaintiff. (2) A plaintiff athlete aware of the risk of symptomatic concussion cannot be taken to be aware of the progressive debilitation and death that result from CTE.

As said by McClellan CJ, it is not necessary to prove that a plaintiff 'believed that a risk would materialise'; rather, '[a]ll that must be proved is that the plaintiff voluntarily accepted a chance that a perceived and fully appreciated risk would materialise'.[168] For example, in taking the field it need only be proven that the plaintiff athlete accepted and appreciated that there was a chance that a risk would materialise. However, the defence is negatived where a plaintiff holds a 'genuine belief' that the risk in question would not materialise;[169] for example, where an athlete is informed of a risk of cross infection through exposure to opponents' blood, but does not believe it can occur. Irrespective, a risk cannot be appreciated without knowledge of the risk. Full appreciation of CTE is not established by a plaintiff athlete being fully aware of

[166] Ibid [91].

[167] *Randwick City Council v Muzic* [2006] NSWCA 66, [48].

[168] *Carey v Lake Macquarie Council* [2007] NSWCA 4, [84].

[169] Ibid.

the risk of symptomatic concussion; nor does an awareness that some contact sport athletes suffer from mental disfunction prove that a plaintiff athlete appreciated that the risk of the CTE is associated with RHT in playing contact sport.

9.8.4 Freely and voluntarily agreed to accept the risk

The 'assumption of risk' provisions do not displace the common law requirement that a plaintiff must have volunteered to risk the harm in question. Einstein J remarked that 'the plaintiff must not only consent to the risk but must also assume the risk freely and voluntarily'.[170] Therefore, the defendant must prove on the balance of probabilities that the plaintiff volunteered to risk contracting CTE.

Again, a plaintiff must know of the risk of harm and fully appreciate the risk for there to be a finding that he or she voluntarily assumed the risk. McClellan CJ remarked in *Carey v Lake Macquarie Council*:

> There must also be some conscious advertence to the possibility that the known risk might eventuate, and a decision to proceed with the conduct regardless. It is not enough that the plaintiff knows of the physical facts or circumstances that constitute the risk and exposes him or herself to them, although proof of this will in many cases be sufficient to support an inference that the plaintiff voluntarily assumed the risk.[171]

9.9 Contributory negligence

Contributory negligence refers to the failure of a person to take reasonable care for his or her safety, which contributes to his or her own harm. The principles applicable to determining whether a plaintiff has been contributorily negligent are the same as those for general negligence and, in conformity with the common law, import an objective standard.[172] An object of the doctrine 'is to allow for the apportionment of responsibility for the injury by a reduction in the damages recoverable … "to such an extent as the Court thinks just and equitable"'.[173] A finding of contributory negligence is a finding of fact.[174]

The standard of care to avoid contributory negligence is that of a reasonable person in the position of the injured person. A finding is 'determined on the basis of what that person knew or ought to have known' of the risk of harm at the relevant time.[175] For this reason, a plaintiff athlete is unlikely to be found contributorily negligent prior to *circa* 2010 (the date proposed in this thesis as a rough estimate of when CTE became 'obvious', for the purposes of the statute,

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[170] *Oran Park Motor Sports Pty Ltd v Fleissig; Teamfox Pty Ltd v Fleissig* [2002] NSWCA 371, [104]–[105].

[171] *Carey v Lake Macquarie Council* [2007] NSWCA 4, [107].

[172] See, eg, *Civil Liability Act 2002* (NSW) s 5R. See also *Le v Rawson* [2009] NSWCA 332.

[173] *City of the City of Taree v Wells* [2010] NSWCA 147, [106].

[174] *Pennington v Norris* (1956) 96 CLR 10; *Roads and Traffic Authority of NSW v Dederer* (2007) 234 CLR 330.

[175] *Collins v Clarence Valley Council* [2015] NSWCA 263, [188]. See also, eg, *Civil Liability Act 2002* (NSW) s 5R.

to premier-level contact sport athletes in Australia).[176] Nonetheless, for reasons of completeness the possibility is briefly considered.

Section 5R of the *Civil Liability Act 2002* (NSW) provides:[177]

> **5R Standard of contributory negligence**
>
> (1) The principles that are applicable in determining whether a person has been negligent also apply in determining whether the person who suffered harm has been contributorily negligent in failing to take precautions against the risk of that harm.
>
> (2) For that purpose:
>
> (a) the standard of care required of the person who suffered harm is that of a reasonable person in the position of that person, and
>
> (b) the matter is to be determined on the basis of what that person knew or ought to have known at the time.

As said by Mason J in *Commissioner of Railways v Ruprecht*:

> Contributory negligence differs from negligence. There is no duty of care owed to another person … and contributory negligence involves conduct which exposes the actor to the risk of injury without necessarily exposing others to risk.[178]

The questions posed by s 5R(2) are 'essentially the same questions as those which arise in determining the issue of "obvious risk"', such that when the questions have been resolved against the plaintiff, 'a finding of contributory negligence almost follow[s] as a matter of course'.[179] This means, of course, that action undertaken by the plaintiff with full awareness of an obvious risk of harm may indicate a failure, or partial failure, of the plaintiff to act in his or her best interests.

Section 5R requires a court to apply ss 5B and 5C in determining contributory negligence; these provisions are directed to breach and the standard of care. Beazley P in *Grills v Leighton Contractors Pty Ltd* referred to the 'conceptual difficulty' of applying these sections, which primarily deal with a breach of duty owed by one person to another person rather than whether a person has taken reasonable care for their own safety.[180]

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[176] Again, this date is, as argued, an estimate but may well move forward or backward by date depending on a court's judgment.

[177] See also *Civil Liability Act 2003* (Qld) s 23; *Civil Liability Act 2002* (Tas) s 23; *Wrongs Act 1958* (Vic) s 62; *Civil Liability Act 2002* (WA) s 5K. The South Australian provision is worded differently: *Civil Liability Act 1936* (SA) ss 3, 44. There are no equivalent provisions in the Australian Capital Territory or the Northern Territory.

[178] *Commissioner of Railways v Ruprecht* (1979) 142 CLR 563, 570.

[179] *Collins v Clarence Valley Council* [2015] NSWCA 263, [188].

[180] *Grills v Leighton Contractors Pty Ltd* [2015] NSWCA 72, [162].

9.9.1 What the plaintiff athlete ought to have known

A claim of contributory negligence is determined by reference to what the plaintiff 'knew or ought to have known'.[181] Consideration is also to be given to the 'general principles' statutory provisions relating to foreseeability of a risk of harm, whether the risk was not insignificant, and whether a reasonable person in the circumstances would have taken precautions (see, for example, *Civil Liability Act 2002* (NSW) s 5B), including the factors to be considered that are listed in the probanda of these sections (such as s 5B(2)). Beazley P noted in *Grills v Leighton Contractors Pty Ltd* that 'it is to be borne in mind' that s 5B(2) is not limited to the factors there listed and, 'pursuant to s 5R(2), the standard of care is that of a reasonable person in the position of the plaintiff and the matter is to be determined on the basis of what the person knew'.[182] Her Honour emphasised the point by stating that the applicable standard is that 'of a reasonable person, with the state of his *actual knowledge*'.[183]

It goes without saying that the knowledge of a defendant in respect of a risk of harm will often be superior to that of a plaintiff and, more importantly, often a defendant 'ought to know' about risks of harm in the performance of his or her obligations of care. In short, applying the principles in ss 5B and 5C with regard to the contributory negligence of a plaintiff and the breach of duty by a defendant, 'the exercise to be undertaken is different in each case':[184]

> A critical difference between the assessment of negligence and the assessment of contributory negligence is that the purpose of the latter assessment is to allow for an apportionment of responsibility for the injury by a reduction in the damages recoverable by the plaintiff 'to such extent as the Court thinks just and equitable having regard to the claimant's share in the responsibility for the damage'.[185]

In *Council of Greater Taree v Wells*,[186] the plaintiff cyclist, Mr Wells, was injured when the front wheel of his bike struck a metal chain that had been strung across the path leading into a park, catapulting him over the handlebars. Mr Wells did not see the chain until he was 10–12 feet away, and was unable to stop in time. He had ridden down the path a week previously, but was unaware of the placement of the chain only two days before. The defendant council claimed that the plaintiff was contributorily negligent due to a 'failure to exercise reasonable care and skill'.[187]

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[181] *Collins v Clarence Valley Council* [2015] NSWCA 263, [188]. See also, eg, *Civil Liability Act 2002* (NSW) s 5R(2)(b).

[182] *Grills v Leighton Contractors Pty Ltd* [2015] NSWCA 72, [162] (Barrett and Gleeson JJA agreeing).

[183] Ibid [178]. Emphasis added.

[184] *Council of the City of Greater Taree v Wells* [2010] NSWCA 147, [106]

[185] Ibid [107].

[186] Ibid.

[187] Ibid [32].

Basten JA commented:

> [An] important difference in approach in assessing the negligence of the defendant, as against the contributory negligence of the [plaintiff], involves the degree of precision by which the activity, including relevant states of knowledge and understanding, is to be identified. … Although the ultimate question is what a reasonable person in the cyclist's position would have known and done, it is inevitable that the evidence will focus upon the knowledge, understanding and actions of the plaintiff himself, shortly prior to the accident, in part to determine whether he exercised reasonable care, but also to assess what would be reasonable care in the specific circumstances.[188]

His Honour referred to the judgment of Mason J in *Commissioner of Railways v Ruprecht*:

> [T]he standard of care in contributory negligence, like the standard of care in negligence, depends on foreseeability and is that of the reasonable and prudent man, so that a [plaintiff] is guilty of contributory negligence if he ought reasonably to have foreseen that, if he did not act as a reasonable and prudent man, he would expose himself to risk of injury …[189]

Mr Wells failed to see the chain in time even though he was exercising reasonable care for his own safety. Basten JA found him not guilty of contributory negligence, noting that while some individuals may have detected the presence of the chain, some would not have done so, and Mr Wells fell into the latter category.[190]

Where it is accepted that knowledge of CTE caused in the playing of contact sport became generally or publicly available after the publications of Dr Omalu et al or Dr McKee et al in 2005 and 2009 respectively, then it must also be accepted that at some point, argued in this thesis as *circa* 2010, players of contact sport became aware of the risk of CTE. To labour the point and to avoid any confusion: the risk of CTE is known to contact sport athletes today, but at some time in the past it was not known; therefore, as a matter of logic, there is a point in time at which the risk of CTE will be deemed by a court to have become known to such athletes. The precise date does not really matter — the same legal analysis will apply (although in practical terms, for sufferers of CTE seeking compensation it clearly will matter) but is not likely to involve a spread of more than a few years.

The inability of a player to be cognisant of and to research the risk of CTE is applicable to the question of contributory negligence. For example, it is unlikely that a court will find a plaintiff professional athlete was obliged to have read Dr McKee et al's 2009 meta-study on CTE published in *The Journal of Neuropathy & Experimental Neurology*,[191] or to otherwise have absorbed a range of medical journal papers published prior to *circa* 2010.

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[188] Ibid [108].

[189] Ibid [110], quoting *Commissioner of Railways v Ruprecht* (1979) 142 CLR 563, 770.

[190] *Council of the City of Greater Taree v Wells* [2010] NSWCA 147, [115].

[191] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

There can be no doubt that the question of a plaintiff athlete's contributory negligence is resolved by a finding that a reasonable person with the athlete's 'actual knowledge' did not know, and ought not to have known, of the risk of CTE in the playing of the SGB's contact sport prior to *circa* 2010.

9.10 Conclusion

The governing body of a major contact sport in Australia confronted with allegations that it negligently failed to warn its players of the risk of CTE or failed to initiate other mitigation programs will predictably claim in its defence that concussion, head trauma or CTE-like harm are 'obvious risks' of play to deny liability under state civil liability legislation.

The most common means of defining harm is through the conduct or mechanism by which harm occurred, such as falling from a horse. In cases where a multitude of different forms of harm may materialise there is little point in listing each possible form and still hazard the chance that some harm will fall outside the list of the imagination. However, identifying harm by this means is argued as inapplicable where the harm that materialised would not, by type or severity, ordinarily be perceived by a reasonable plaintiff. In such a case the relevant harm is identified most effectively by the damage (harm) sustained by the plaintiff.

The term 'obvious risk' applies through the statutory provisions of state civil liability legislation to three defences: 'dangerous recreational activity'; the 'presumption of awareness of obvious risk'; and no duty to another person 'to warn of an obvious risk'. These defences were argued to depend on two matters: (1) the date that at which CTE had become an obvious risk of the SGB's contact sport; and (2) whether the risk of harm in the form of CTE is characterised by a court in either general or specific terms.

From the day CTE become an obvious risk in the playing of contact sport all participants in the SGB's competition will, *ceteris paribus*, be deemed to have accepted the risk of harm. It was proposed as a logical and convenient estimate as *circa* 2010, a date by which publicity on CTE had expanded into the media and by which major SGBs had introduced early forms of concussion-prevention protocols into contact sport in Australia and in so doing either expressly or impliedly warned of the risk of CTE.

It was proposed that a broad characterisation on CTE such as 'concussion', 'concussion-related concerns' or 'head trauma' — clearly obvious risks of contact sport — would activate the statutory defences noted above. A narrow characterisation as 'CTE' specifically, or by type as a 'neurodegenerative disease' or 'tauopathy' would work in the athletes' favour to exclude a finding of obvious risk.

In cases where there is a long latency period between the harm causing event and the onset of symptoms it was argued, with reference to precedent, that an accurate assessment of 'obvious risk' requires a specific application whereby characterisation is focused on CTE itself or, if by type, a neurodegenerative disease or tauopathy. Facts were presented to reinforce this view, most pertinently that a broad characterisation such as 'concussion' or 'head trauma',

from which people recover within a week or two, does not describe the material condition of CTE which is marked by ongoing and increasingly severe cognitive disfunction leading to death. There can be no expectation that a premier-level contact sport athlete prior to *circa* 2010 would have consulted medical journals or possessed the skills to understand the meaning of implications their content.

A narrow characterisation of the risk of harm in the form of CTE was, as argued, supported by the High Court judgment in *Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd*,[192] where Gordon, Edelman and Gleeson JJ required that obvious risk be characterised at the same level of generality as risk for the purposes of assessing breach, and to also correspond with the 'general causal mechanism' along with the form of harm that materialised. The breach, as argued, is a failure of the SGB to warn of the risk of CTE. The harm for which the plaintiff athlete seeks damages is CTE. The means of harm is exposure to RHT of sufficient force and quantity over sufficient time to cause the protein tau to leak from the axons of the brain and to gather as hyperphosphorated tau around the brain's neurones increasingly disturbing cognitive function. In no instance is the risk of harm 'concussion' or some unspecified 'concussion related concern'.

It was also argued that there is no utility in an athlete arguing that the risk of harm is concussion, which is not only an obvious risk to premier players but, given that concussion is recovered from within a week or two, without any serious compensatory damage.

Under the 'assumption of risk' provisions, an injured plaintiff must prove he or she was not aware of the type or kind of risk. A plaintiff athlete's case is, as discussed, favoured where CTE is given a narrow classification by type or kind as a neurodegenerative disease or tauopathy. Clearly a classification that identified the very broad risk as concussion or head trauma, would likely see the plaintiff athlete's argument fail. In addition, a plaintiff who was uniformed of CTE could not possibly discern the real risk of harm, appreciated the danger, or be taken to have volunteered to risk the contraction of a disease that was personally unknown.

A claim that a player of contact sport should be taken as contributory negligent, which requires foresight of the risk of CTE, cannot be liable in the absence of knowledge of the disease.

In summary, there is a case to be made that a premier-level contact sport athlete playing in an SGB's competition prior to *circa* 2010 could not know that CTE was an obvious risk of contact sport. As such, the defence of obvious risk applicable to dangerous recreational activity, assumption of risk of harm, and no duty to warn of an obvious risk of harm will not apply to athletes who played in that period.

[192] (2022) 273 CLR 454.

Chapter 10:
Conclusion

10.1 Conclusion to thesis

This thesis has systematically and comprehensively examined the potential liability in negligence of SGBs in Australia concerning CTE. The thesis explored elements of negligence law as they apply to the unique circumstances of CTE, a debilitating brain disorder caused by RHT. The central proposition is that SGBs owe a duty of care to their premier-level athletes to take reasonable steps to be informed of the risk of CTE and to warn those athletes of that risk.[1] The primary argument of the thesis is directed towards affirming this proposition.

The approach taken by this thesis is that of an interdisciplinary research methodology integrating legal analysis with scientific and medical evidence. New applications of law are considered, yet the emphasis is unavoidably on emerging facts — an emphasis that stems from the nature of applying law to the novel issue of CTE in the context of sports-related negligence. The law is the framework by which facts are legally analysed. This thesis presents a unique set of facts to which the law has not yet been applied. The thesis identifies, analyses, and applies the relevant legislation and case law to the pertinent emerging facts of CTE.

10.2 A review of key arguments and findings

This thesis contended that the application of the law of negligence to the failure of an SGB to be informed and to warn of the risk of CTE cannot be understood unless one possesses a sound knowledge of CTE as a neurological disease and of its aetiology. This understanding involves such concerns as: the discovery of CTE as a disease, the date when it was discovered, how it is caused, developing medical knowledge over time, the nature of symptoms and sequelae, the delayed onset of symptoms, how it is characterised as a disease, how it is different from other neurological diseases, among many other facts bearing upon the legal application of an SGB's failure to know and to warn.

Chapter 2: 'The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments' was augmented by Appendix 1, which concerned medical facts argued as underpinning a claim that Australian SGBs failed to warn their premier players of the risk of CTE. The chapter introduced CTE as a cognitive disease, describing its physical cause, the slow development of symptoms and the apparent uncertainty of diagnosis other than at autopsy. Each feature influences the course of a negligence claim in several interrelated ways.

[1] The obligation of an SGB to take reasonable steps to be informed of the risk of CTE coincides with a general obligation to be informed of extant and emerging risk of harm in the playing of a contact sport like the SGB's.

The chapter discussed at length the processes by which accumulated head trauma causes the brain protein 'tau' to leak from the axon walls of brain cells and form phosphorated tau, which builds up over years and decades to cause increasingly severe cognitive disfunction and ultimately death. One of the most informative statements is that of Lipton, quoted several times in this thesis: while 'it is well established that most patients with mild TBI recover fully over time', the body's 'repair mechanisms may be unable to keep pace with the cumulative injury that occurs beyond a degree of exposure'.[2] This unknown and often distant threshold is a basis to the obligation of SGBs to warn at-risk players before the buildup of tau reaches a critical level.

Chapter 2 argued that three landmark studies drew a connection between RHT and CTE, and provide points in time by which to judge the development of knowledge of CTE: the 1928 research of Dr Martland informing of 'punch drunk syndrome' affecting boxers;[3] that of Dr Omalu et al in 2005, revealing that ex-NFL player Mike Webster was afflicted with CTE;[4] Dr McKee et al's 2009 article that reported the occurrence of CTE across a range of contact sports, including those common in Australia.[5] Several Australia cases from the 1990s were argued, at least collectively, to have perhaps revealed sufficient reason for concern to have promoted dutiful SGBs to have initiated inquiries into the risk of CTE to their premier players.

The date of these studies, and those extending from the 1970s reporting CTE-like harm linked to a range of common contact sports such as rugby, AFL and soccer, are critical in determining when Australian SGBs ought to have known of the risk of CTE and warned their players of a risk of neurological disease through RHT in playing contact sport.

Chapter 3: 'A Duty Relationship Between an SGB and the Athletes Who Play Contact Sport' argued that a governing body of a contact sport in Australia possesses a duty of care to the athletes who play in its premier competition. It was argued that the 'no duty' determination by the High Court of Australia in *Agar v Hyde*[6] is not, as some commentators suggest, applicable to the relationship between a governing organisation in Australia and its players for two reasons: (1) *Agar v Hyde* is not a 'governing body' case; and (2) it is distinguishable on its facts from those cases that characterise the relationship of an Australian governing body with its premier players.

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2 ML Lipton et al, 'Soccer Heading is Associated with White Matter Microstructural and Cognitive Abnormalities' (2013) 268(3) *Neuroradiology* 850, 854.

3 Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

4 Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

5 Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

6 (2000) 201 CLR 552.

A ‘salient features’ approach was argued as the correct means by which to establish a governing body’s duty of care. Several salient features were argued to apply, though special emphasis was placed on ‘control’, ‘reliance’ and ‘vulnerability’. This application arises from the governing bodies in Australia exercising, through contractual mandate, almost total and independent control over their sport, its rules and its protocols, including those that affect the health and well-being of their premier-level athletes. The violent nature of contact sport throws upon a governing body — as the sole determiner of rules and protocols, and as a financial beneficiary of the sport — an obligation to monitor the sport for emerging risks of harm and to warn their premier players where those risks are material.

It was argued that a salient features approach was in fact, though not in name, applied in the principal sport cases of *Agar v Hyde*,[7] *Green v Country Rugby Football League of New South Wales Pty Ltd*[8] and *Watson v British Boxing Board of Control; World Boxing Organisation Inc*.[9] A duty of care was established in both *Green* and *Watson*, though not in *Agar*. The salient features reviewed by the courts in these cases align with those collated from High Court decision by Allsop P in *Caltex Refineries (Qld) Pty Ltd v Stavar*.[10] As argued, the presence or absence of a salient feature, such as control or reliance, either affirms or disaffirms a duty of care. This chapter is supplemented by Appendix 3: ‘Brief consideration of the scope of liability.’

Chapter 4: ‘CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care’ considered the application of law pertinent to establishing that a governing body of a major sport in Australia is obliged to inform itself of the risk of CTE and, where that risk is material, to warn the premier players in its competition of that risk. The chapter engaged in brief discussion of the relationship between a sporting organisation and the medicos appointed to attend to the health needs of premier players. The chapter argued that the ‘medical model’ applied to the duty to be informed and to warn based primarily on the fact that contact sport involved inherent risks of personal injury that can only be reasonably protected through an ongoing monitoring of extant and emerging risks of harm.

The broad content of the duty to be informed and to warn of material risks was demonstrated through the decisions in three cases: (1) a defendant’s conduct is ‘only judged in the light of knowledge which then was or ought reasonably to have been possessed’;[11] (2) the test of when a warning is to be given is not when there is certainty of a given risk, but rather when ‘mere suspicions would necessarily have been elevated in the minds of responsible Australian

7 (2000) 201 CLR 552.

8 [2008] NSWSC 26.

9 [2001] QB 1134.

10 (2009) 75 NSWLR 649, [103].

11 *Roe v Minister of Health* [1954] 2 QB 66, 92.

physicians … into a strong apprehension';[12] and (3) the obligation of a duty-holder to be informed is proactive: to take 'steps to find the relevant literature, to read it, to understand it and to discuss it'.[13] The third of these cases, that of *South Eastern Sydney Area Health Service v King*, also noted that where an obligation is delegated, the duty-holder must 'take reasonable care to ensure that someone else actively searched for material which was relevant to the task he was to undertake'.[14] The chapter concluded that the governing bodies of Australian contact sport did not, as duty-holders, meet the standards applied in these cases with respect to the duty to inform themselves of emerging risks of harm and the duty to warn their premier players of the risk of CTE.

Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times' reviewed information on the risk of CTE-like harm and whether it was reasonably available to the SGBs of major sport, principally through their medical associates, prior to *circa* 2010.[15] A point argued was that athletes who were exposed to RHT prior to these dates must be able to prove that the governing body of their sport had reasonable access to information on the risks of CTE at the time they played. That is, the organisation 'ought reasonably to have been possessed'[16] of knowledge of the risk of CTE and warned their athletes of that risk.

A difficulty faced by plaintiff athletes is that Martland's 1928 'Punch Drunk' article[17] concerned boxers, suggesting that the violence and trauma associated with boxing may be too severe to 'ignite the imagination' of a governing body to enable it to draw a parallel with the sport it administers. While this is a realistic concern, the chapter discussed at length other avenues through which access to knowledge of CTE and its causes was reasonably available, pointing out that: the cause of CTE in both boxing and other contact sports is RHT; to know what causes concussion in general is to know what causes concussion in contact sport; and from the 1970s medical journals had reported that players of contact sports such as football (soccer), rugby and wrestling were at risk of contracting CTE. The chapter concluded that sufficient information on the risk of cognitive disfunction associated with RHT was available decades before Omalu et al's 2005 study and obviously prior to *circa* 2010, which a reasonable governing body of a contact sport in Australia could access. It could then assess the risk of harm and warn its premier players of that risk. The possibility that governing bodies were

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12 *H v Royal Alexandra Hospital* [1990] 1 Med LR 297, 311.

13 *South Eastern Sydney Area Health Service v King* [2006] NSWCA 2, [67].

14 Ibid [60].

15 Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128; Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

16 *Roe v Minister of Health* [1954] 2 QB 66, 92.

17 Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

informed of the risk of CTE by their associated medicos but chose for unknown reasons not to issue warnings, was also considered.

This chapter was supplemented by Appendix 2: 'CTE and "Negativing Influences": Do They Determine Liability?', which addressed the question of whether published information that was said by some commentators to have diminished or downplayed the risk of CTE in contact sport was so significant that it excused SGBs from warning its premier-level athletes on the basis that the risk was not real or foreseeable — an argument referred to by Dr Buckland as 'CTE denialism'. The thesis argued, however, that these publications (Consensus Statements of the CISG and comments of Dr Paul McCrory) did not diminish the obligation of Australian SGBs to warn their premier players of the risk of neurological disease caused by RHT. Several reasons were provided including: the relevant articles had not been published in the era when most present-day sufferers of CTE played contact sport, that the risk of CTE was not outright denied but referred to methodological shortfalls in the scientific standard, that the scientific standard for judgment as to the obligation to warn of a risk of harm is not the legal standard, that the publications at times acknowledged the risk of CTE in playing contact sport, and that many of the commentators who denied a clear scientific between RHT and CTE held positions with major sporting organisations thereby indicating a conflict of interest. The sociologically based claim that CTE had been 'medicalised' was argued as inapplicable to the obligation of an SGB to warn its premier players of the risk of CTE.

Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"' examined whether a plaintiff can prove that the symptoms suffered are those of CTE rather than a different cognitive disease, the crux of the issue being that '[a] cause of action is not complete until the plaintiff first suffers actual loss or damage'.[18] Because CTE is only definitively diagnosable at autopsy, a living athlete must prove by inference that the symptoms suffered are those of CTE. The chapter focused on traumatic encephalopathy syndrome (TES), as 'the clinical disorder associated with neuropathologically diagnosed CTE'. The TES methodology considers the strength of set 'primary' and 'supportive criteria' including exposure to RHT, cognitive impairment, and dementia. Proof of CTE *in vivo* is established on the balance of probabilities. An analytical illustration was provided by utilising the *in vivo* medical history of AFL player Danny Frawley who was later confirmed at autopsy as having been afflicted with CTE. The chapter concluded that an inference of CTE *in vivo* can be proven on the balance of probabilities by plaintiff athletes who display the TES criteria and other indicators. This chapter is supplemented by Appendix 5: 'Multiple Causes of CTE and 'Material Contribution' to Harm.'

Chapter 7: 'Factual Causation of Harm: Failure to Warn and the Athlete's Response Had a Warning Been Given' considered the question of factual causation of CTE under the statutory equivalent of the 'but for' test; that is, was the failure to warn a 'necessary condition' of the plaintiff athlete's contraction of CTE. The argument centres on the question of the athlete's response to a warning of the risk of CTE by his or her SGB in the pre-*circa* 2010 era. Liability

[18] *Scarcella v Lettice* (2000) 51 NSWLR 302, 306.

does not attach if the athlete would have disregarded the warning and continued playing. Although the test is directed to the individual's response, the athlete faces the difficulty of not being permitted to give evidence as to what he or she would have done if warned unless it is against interest. Clearly the risk of a disease causing severe cognitive disfunction and death is 'material'.

The chapter argued that the assessment of a player's hypothetical response must be made as if the SGB had been warning its premier players of the risk of CTE for decades. A response such as retirement is far more likely where a culture of acceptance of such a decision had been created with the playing groups of the sport and in particular through the encouragement of its SGB. Retirement is also more likely where the cause of cognitive decline is witnessed in past players who suffered RHT.

Because the question is individualised and hypothetical, only a broad listing of facts useful in inferring the response of players is possible. On the basis that CTE is a disease caused by the accumulation of head trauma, it was argued that an athlete could not be expected to retire from a professional position on the mere warning of the risk of CTE without experiencing several symptomatic concussions or when warned by medical staff that the threshold at which tau is released from the axons of the brain may be approaching. Although a finding is directed to the plaintiff, it was noted that courts will consider the objective reasonableness of an individual's response, the relative rarity of CTE as a disease, the severity of the disease once contracted.

Personal matters from which an inference can be made were argued to include the credibility of the athlete plaintiff, personal characteristics like alternate job opportunities, money owed, family or community responsibilities, previous cavalier responses to physical risk and a reluctance to report health issues to medical staff, preparedness to 'take one for the team' among many others.

Chapter 8: 'The Question of "Generic Capacity": Does the SGB's Contact Sport Cause CTE?' addressed the means by which a plaintiff athlete, who is required in law to prove all matters concerned with causation of harm,[19] is able to produce evidence that playing the type of contact sport that the SGB administers causes CTE. The chapter drew largely on the High Court determination in *Amaca Pty Ltd v Booth*,[20] a mesothelioma case where failure to warn was in issue, to show that the 'but for' test of causation is a recognised means of doing so. State civil liability legislation used the term 'necessary condition' as equivalent to the 'but for' test. A 'necessary condition' is established by the application of the 'but for' test; that is, 'but for' the governing body's negligence the athlete would not have contracted CTE.

Appendix 4: 'Factual Means of Assessing Generic Capacity to Cause CTE' was a factual supplement to Chapter 8, revealing how proof of the generic capacity of RHT in the playing of

[19] See, eg, s 5E of the *Civil Liability Act 2002* (NSW).

[20] (2011) 246 CLR 36.

contact sport to cause CTE may be provided through the 'general body of medical knowledge' and the utilisation of the 'Bradford Hill criteria', a medically recognised methodology of establishing causation of disease. Medical researcher Nowinski applied the Bradford Hill criteria and affirmed that CTE was caused by RHT.[21] Noting that the standard of proof is on the balance of probabilities, the assessment of Nowinski, along with the opinions expressed by 'other means', support the conclusion that CTE is caused by RHT in the playing of contact sport.

Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences' concerned the defence under civil liability legislation that excuses a tortfeasor of liability where a risk of harm was, depending on the specific statutory provision, either objectively obvious, or subjectively obvious. The argument that CTE was an 'obvious risk' in the playing of contact sport present a clear threat to a plaintiff athlete's claim of negligence against an SGB. How the risk of CTE is characterised will, as argued, largely determine whether it presents an 'obvious risk' for purposes of alleviating the SGB of responsibility in negligence. This chapter proposed, however, that CTE is accurately characterised as a specific disease, or as a type of tauopathy or neurodegenerative disease, rather than the clearly obvious risk of concussion.

The chapter also argued that due to the growing publicity following the publication of Dr McKee et al's 2009 study nominating several contact sports commonly played in Australia, along with major sports beginning to introduce 'concussion protocols' that CTE would be declared by a court as becoming an 'obvious risk' of RHT in the playing of contact sport for the purposes of defences available under state civil liability legislation.

10.3 Recommendations

The object of this thesis is to consider whether SGBs of contact sport in Australia were negligent in law by failing to inform themselves of the risk of CTE and in failing to warn their premier players of that risk. The question of liability is directed to athletes who played in premier competitions prior to *circa* 2010 — when, as argued, CTE became an 'obvious risk' of contact sport for purposes of law.[22] Recommendations in the context of this thesis would refer to possible current or future actions. Although such recommendations fall outside the strict purpose of this thesis, brief discussion regarding matters of fact and law pertinent to the contraction of CTE in contact sport is not out of place. Any practical recommendation is, however, constrained by two overriding facts: (1) head trauma is unavoidable in contact sport; and (2) the law denies compensation where the risk of CTE is objectively obvious to a premier

[21] Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1.

[22] See Chapter 9 at **[9.2]**: 'Legislative provisions: Obvious risk, dangerous recreational activity and assumption of risk of harm'.

player of a contact sport.[23] Recommendations mutually relevant to both SGBs and players are briefly considered below.

10.3.1 Evaluation of existing measures

Recommendations as to future actions must first evaluate the usefulness of steps already taken by an SGB to minimise the risk of CTE, and determine whether those steps are, from a legal perspective, adequate. In the post-2010 era, many SGBs introduced rule changes and protocols designed to limit the risk of RHT and thereby CTE. These steps included: the removal of a player from the field to conduct a head injury assessment where concussion is suspected; the appointment of independent medicos to monitor games to avoid home-team bias; mandated suspension following a concussion; and penalising offenders should they make head contact or tackle an opponent in the air.[24] None of these steps actually prevent head trauma, and most only come into operation *after* a player has suffered concussion. These measures are, however, to be applauded in diminishing the risk of head trauma accumulating to the point that tau is released from the brain's axons, beginning the irreversible processes of CTE, and should, where effective, be maintained.[25]

10.3.2 The unavoidability of head trauma in contact sport

Those who play contact sport face the harsh reality that head trauma cannot be eliminated from any sport that permits hard physical contact between opponents or a head strike by a fast-moving ball. The speed and ferocity of premier contact sport remains an 'obviously' dangerous undertaking — a fact witnessed by spectators and players of sports such as rugby league, AFL and test match cricket.[26] As commented by Qi et al, '… elite athletes exhibited a higher prevalence of CTE compared to amateur athletes' … which indicates that 'the prevalence of CTE increased with the level of competition.'[27]

As such, the only recommendation that will work to eliminate the risk of CTE is to ban hard physical contact — in effect, transforming sports such as rugby league into touch football and cricket into a game of underarm bowling.[28] Obviously, this option is unrealistic and untenable

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[23] See Chapter 8: 'The Question of "Generic Capacity": Does the SGB's Contact Sport Cause CTE?'; Chapter 9 at **[9.2]**: 'Legislative provisions: Obvious risk, dangerous recreational activity and assumption of risk of harm'.

[24] See Chapter 4 at **[4.5.2]**: 'The (collateral) rules that SGBs have volunteered to adjust'. See generally Rugby Australia, *Concussion Management Procedure Update Summary* (Web Document, February 2025) <4e9a60c3-d1d7-41aa-4bae-08dd4c795fd2.pdf>.

[25] See generally Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"'; ML Lipton et al, 'Soccer Heading Is Associated with White Matter Microstructural and Cognitive Abnormalities' (2013) 268(3) *Neuroradiology* 850.

[26] See Chapter 9 at **[9.2]**: 'Legislative provisions: Obvious risk, dangerous recreational activity and assumption of risk of harm'.

[27] Bote Qi et al, 'Prevalence of Chronic Traumatic Encephalopathy in Athletes with Repetitive Head Impacts: A Systematic Review and Meta-Analysis' (2025) 35 *Scandinavian Journal of Medicine & Science in Sports* 11, 8.

[28] See Chapter 8: 'The Question of "Generic Capacity": Does the SGB's Contact Sport Cause CTE?'

for the administrators, players and spectators of premier-level sport, not to mention the commercial interests of the billion-dollar contact sport industry in Australia. Moreover, there is no legal obligation to take this step following the High Court's recognition in *Agar v Hyde* that athletes are free to participate in sports involving a risk of serious injury and, indeed, where 'danger is part of [a sport's] attraction'.[29] *Agar v Hyde* not only represents the law as it stands today, but also reflects centuries of criminal and civil precedent legitimising unintentional harm through violence in sport.[30]

Given that head trauma in playing contact sport is unavoidable, to minimise the risk of litigation it can be recommended that SGBs regularly advise their players that the only way to prevent CTE is not to play contact sport, such advice being augmented through communication with respected medical professionals. The more thorough the education of a player, the more likely he or she is to accept a recommendation to retire after experiencing multiple concussions with prolonged symptoms. Concomitantly, it can be recommended that where the threat of CTE is a medical concern, athletes heed the advice of informed clinicians.

10.3.3 The ongoing obligation to be informed and to warn

Although 'obvious risks' are 'liability defeating',[31] the fact that SGBs have taken it upon themselves to take reasonable steps to protect their premier players by minimising RHT throws upon them a duty to continually inquire into developments affecting both the risk and mitigation of CTE. There are two aspects to this recommendation. One, where the risk of RHT is mitigated through newly discovered methods that do not change the foundational nature of the sport, it is recommended that these means be implemented.[32] Two, where a newly discovered risk cannot be reasonably ameliorated, it is eminently arguable under present law that an SGB's responsibility does not extend beyond making the risk 'obvious' by warning its premier players of that risk.

10.3.4 Changing the collateral rules

As argued in this thesis, there is no legal obligation imposed on an SGB to alter the foundational rules of its sport, including those that expose a player to head trauma.[33] It can be recommended, however, that to minimise the risk of CTE an SGB adjust rules and protocols that do not change the basic nature of the sport. For example, a scrum in rugby (an integral part of the game that is known for head clashes and quadriplegia following a collapse) has been made safer by an alteration of the on-field rules requiring front rows to go through the routine of 'crouch, bind,

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[29] *Agar v Hyde* (2000) 201 CLR 552, [89] (Callinan J). See the extensive discussion in Chapter 4 at **[4.5]**: 'Rule changes and the standard of care: Safety measures — voluntary or duty?'

[30] See, eg, *R v Bradshaw* (1878) 14 Cox CC 83; *R v Coney* (1882) 8 QBD, 534.

[31] *Goode v Angland* (2017) 96 NSWLR 503, [185] (Leeming JA).

[32] See Chapter 4 at **[4.5.1]**: 'Is there a legal obligation to change the rules of play to make a sport safer?'

[33] Ibid.

set' before engaging. Off-field safety protocols, similarly, do not generally alter the basic fabric of a sport, and serve to assist an SGB in claiming it has done all that is reasonable to ameliorate the risk of CTE.

10.3.5 Forced retirement at the threshold of CTE-like harm

Can it be recommended that an SGB force a player who experiences intense and prolonged symptoms of concussion into early retirement? One the one hand is the player's right to make decisions as to his or her future. On the other is an SGB's concern for the well-being of the player and, perhaps predominantly, its concern at damage to the image of its sport where a player is knocked unconscious or staggers from the field of play after relatively minor contact. At the core of the debate is the medical fact that although an individual player may experience several concussions, affliction with CTE over coming decades is far from certain. Moreover, at the time of early directed retirement, the player is not suffering from CTE.[34] An overreaction by an SGB or an overly cautious assessment by a medico could force a player who may never suffer CTE out of his or her chosen profession, with few other means of financial support. In addition, forced early retirement from sport has been found to have life-altering consequences and put the player at risk of suffering mental health disorders.[35]

What recommendations can be made from a legal perspective where such an athlete demands the right to continue playing? As the law stands today, provided the SGB, through its medical associates, fully explains the risks of permanent neurological harm should the athlete continue to play, it can rely on the 'obvious risk' provisions of state-based legislation to avoid liability.[36]

As the SGB is the dominant contracting party, it can be recommended that it incorporate a term into its formal arrangements with players that where, on expert medical advice, a player's neurological health is reasonably considered to be at risk, the SGB will retire to player from the sport. However, while outwardly simple, a term mandating early retirement 'is plainly a fetter on the right of a player to seek and engage in employment' and a restraint of trade 'contrary to public policy'.[37] The only legal response by a covenantor to a restraint of trade is

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[34] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 709.

[35] C Reardon et al, 'Mental Health in Elite Athletes: International Olympic Committee Consensus Statement' (2019) 53 *British Journal of Sports Medicine* 667; C Chang et al, 'Mental Health Issues and Psychological Factors in Athletes: Detection, Management, Effect on Performance and Prevention: American Medical Society for Sports Medicine Position Statement — Executive Summary' (2020) 54 *British Journal of Sports Medicine* 54.

[36] See Chapter 9 at **[9.2]**: 'Legislative provisions: Obvious risk, dangerous recreational activity and assumption of risk of harm'.

[37] *Buckley v Tutty* (1971) 125 CLR 353, 373.

to contend that the impost is 'reasonable' with reference to 'the interests of the parties' and 'reasonable' with reference to 'the interests of the public'.[38]

Furthermore, while such a restraint may operate to secure a benefit to the SGB, a confounding issue is that a player is not an agent for the 'best interests of the game', such that the player's interests are not to be subsumed by those of the sport.[39]

As may also be argued, the best judge of a player's overall best interests is the adult player him or herself, taking into account several factors ranging from their personal beliefs as to the state of their health, their financial future and the effect upon their families. On this point, comments of Callinan J resonate: 'Each participant in the match was adult and must be taken to have consented to the application of physical force in accordance with the laws of the game.'[40]

A restraint of trade must be shown to protect a legitimate interest of the SGB.[41] The question is this: does retiring a player from his or her profession against the player's will, serve a legitimate interest? In the SGB's favour is a recognition that 'the categories of legitimate interest are not closed'.[42] As noted above, it is likely that forced retirement in these circumstances does protect a legitimate interest of the SGB. In particular, the SGB has an interest in securing the image of the sport and in protecting the health and well-being of the player — the latter fact being argued in this thesis as determinative in establishing the SGB's duty of care.

To be enforceable, a restraint of trade must also be reasonable; that is, as noted above, 'reasonable' with reference to 'the interests of the parties' and reasonable with reference to 'the interests of the public'.[43] Does the restraint go further than is necessary to protect the legitimate interests of the sport? In essence, while there is a legitimate interest in an SGB securing the image of the sport and protecting players from CTE, is it reasonable to retire players who have not contracted CTE but are thought to be in danger of contracting it, but who may in fact never suffer from the disease?

Consideration of reasonableness begins with a fact noted several times in this thesis: a threshold may be reached where the body's 'repair mechanisms may be unable to keep pace with the cumulative injury that occurs beyond a degree of exposure', at which point tau is

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38 *Nordenfelt v Maxim Nordenfelt Guns and Ammunition Co Ltd* [1894] AC 535, 565.

39 *Adamson v New South Wales Rugby League Ltd* (1991) 103 ALR 319, 324.

40 *Agar v Hyde* (2000) 201 CLR 552, [88].

41 *Petrofina (Great Britain) Ltd v Martin* [1966] 1 All ER 126, 139.

42 *Infomax International Pty Ltd v Clarius Group Ltd* [2011] FCA 183, [26].

43 *Nordenfelt v Maxim Nordenfelt Guns and Ammunition Co Ltd* [1894] AC 535, 565.

released from the axons of the brain.[44] It is not in the interests of an SGB charged with taking steps to protect the health and well-being of its premier players[45] and to promote the image of its sport that ex-players — perhaps in their 40s or even 30s — enter neurodegenerative decline leading ultimately to death. Moreso, where the symptoms and sequala of CTE, ranging from violence to suicidality to dementia and death, are pronounced and obvious to the public.

Is there a legitimate interest in an SGB seeking to attract or maintain players, sponsors and spectators to its game, thereby securing financial solvency? Certainly, on a broad scale these objects have been judicially recorded. In *Buckley v Tutty*,[46] the High Court recognised a legitimate interest in the SGB attracting the support of the public through maintaining competitive balance among the district clubs; and in *De Belin v Australian Rugby League Commission Ltd*,[47] the Federal Court recognised a legitimate interest in securing sponsorship such that it was reasonable to stand down a player who was accused of a serious crime. Of course, the question remains as to whether securing these interests through forced early retirement is reasonable.

Foundational to an SGB case is the production of evidence justifying forced retirement based on the health of an individual player. As said by Wilcox J in *Adamson v New South Wales Rugby League Ltd*: '[It is not] permissible to give [the New South Wales Rugby League] the benefit of any doubt. … If the court is left uncertain as to justification, it must find for the [players].'[48] That is, to successfully exclude a player, an SGB must be able to produce supporting factual evidence.

Medical commentators regularly advise athletes to retire from contact sport where the effects of concussion become increasingly severe and prolonged; and while these comments seem logical, there is an absence of evidenced-based research by which to justify such a proposal.[49] Makdissi et al conducted a meta-study of online medical journals using the words 'concussion', 'sports' and 'retirement' — the word 'retirement' being broadly defined to capture all possibilities. More than 8,000 articles were reduced to 93 for the purposes of the study, but the authors found that 'none of the studies directly examined the issue of retirement

[44] ML Lipton et al, 'Soccer Heading Is Associated with White Matter Microstructural and Cognitive Abnormalities' (2013) 268(3) *Neuroradiology* 850, 854. See Chapter 2: 'The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments'; Appendix 1: 'The Science of Concussion and CTE in Sport'.

[45] See Chapter 3: 'A Duty Relationship Between an SGB and the Athletes Who Play Contact Sport'.

[46] (1971) 125 CLR 353.

[47] [2019] FCA 688.

[48] *Adamson v New South Wales Rugby League Ltd* (1991) 103 ALR 319, 356.

[49] Bote Qi et al, 'Prevalence of Chronic Traumatic Encephalopathy in Athletes with Repetitive Head Impacts: A Systematic Review and Meta-Analysis' (2025) 35 *Scandinavian Journal of Medicine & Science in Sports* 11.

and/or discontinuation from contact collision sports'.[50] This study could suggest, in the words of Wilcox J above, that a court 'is left uncertain' as to the justification of the restraint.

Nonetheless, Makdissi et al found that while precise guidance on when an athlete should retire is lacking, several factors common across the meta-study were relevant in making the determination: a history of playing contact sport and experiencing concussion; a record of repeated symptomatic concussions; the effects of concussion having become more severe; prolongation of recovery times; concussion being more easily or readily sustained; sleep disturbance, or any mental health or behavioural issues; the level of play; and the views of independent specialist medicos.[51] The degree of autonomy to be granted an athlete who displays ongoing and severe symptoms of concussion as against the mandate of his or her SGB to retire is a related subject of further legal research.

While courts take account of a medico's professional opinion, the views expressed are not determinative of the reasonableness of a restraint of trade. The enforceability of a restraint of trade is a matter of law, to be decided by a court of law.[52] In a broad sense — and paraphrasing Carter with respect to the 'tension' between competing interests under the restraint of trade doctrine — courts find these issues 'difficult to resolve'.[53]

10.3.6 Further legal and medical research

This thesis comprehensively examined the legal and factual issues concerned with a claim that SGBs in Australia were negligent in failing to inform themselves of the risk of CTE and failing to warn their premier players of that risk. The element of negligence and the facts relevant to its application have been researched and conclusions drawn. With respect to many of the proposed applications of law to fact, little more can be added other than (should it come to it) a judicial determination or, at least, a peer review and challenge.

Nonetheless, some proposals in this thesis are, or may be, contrary to the views of other researchers — contrary views being the very purpose of peer review. As such, further research into certain applications of law to fact will serve to explore alternative possibilities and provide possible clarity to a debateable issue and therefore can be recommended. Several further research proposals are listed below.

This thesis proposed 'material contribution' as a means to resolve an SGB's defensive claim that it was the plaintiff's participation in junior contact sport that caused the RHT that led to his or her CTE, rather than play in the SGB's premier competition. It was also suggested that, where harm is due to the presence of 'multiple sufficient causes', causation of CTE may be

[50] M Makdissi et al, 'When Should an Athlete Retire or Discontinue Participating in Contact or Collision Sports Following Sport-related Concussion? A Systematic Review' (2023) 57 *British Journal of Sports Medicine* 882, 884.

[51] Ibid.

[52] *Buckley v Tutty* (1971) 125 CLR 353, 380.

[53] JW Carter, *Contract Law in Australia* (8th ed, 2023, JW Carter Publishing) 575.

proven through the 'exceptional case' provisions of state civil liability legislation.[54] Others have said that such an application is unavailable in law, suggesting a need, at least from the perspective stated above, for further research.[55]

Supporting the argument regarding an SGB's duty of care in this thesis was analysis of the three-way relationship between an SGB, the medicos who practise within the auspices of the SGB, and the athletes who play in the SGB's premier competition.[56] While this tripartite arrangement is well recognised, formal research into the dynamics between the parties will serve to resolve legal issues regarding the relative degree to which a medico serves the interests of the SGB as against those of the player, and the scope of the duty that each owes to the player.

Based on the concept of 'obvious risk', it was argued in this thesis that an SGB is not obliged in law make a sport safer by adjusting rules of play that will change the very nature of the sport.[57] There is little doubt as to the logic of this position, and yet some commentary takes a contrary position — or at least indicates the need for legislative imposition to make contact sport safer. The extent to which, if at all, negligence could be claimed based on a failure to change the rules of play, or whether a parliament would consider such an intrusion, is a subject for further research.[58]

Legal commentator Opie suggested in 2000 that the High Court decision in *Agar v Hyde*[59] 'left open' the possibility that an SGB owed a duty of care 'to amend the rules so as to make the sport safer'.[60] More recently, this notion has been supported by other commentators.[61] Although this thesis has argued that the judgments in *Agar v Hyde* did not support an 'open door', further research into this suggested application is warranted.

An area of further research likely to influence the legal approach to CTE concerns the revelations of medical-scientific discovery. Although CTE in the form of 'punch drunk syndrome' has been the subject of hundreds of medical journal articles since Martland's 1928

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[54] See Chapter 8 at **[8.4]**: 'Multiple causes of CTE and 'Material Contribution' to Harm'; Appendix 5: 'Multiple Causes of CTE and "Material Contribution" to Harm'.

[55] General academic discourse.

[56] See Chapter 4 at **[4.2]**: 'The link between the SGB and medicos'.

[57] Chapter 4 at **[4.5]**: 'Rule changes and the standard of care: Safety measures — voluntary or duty'.

[58] See A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024).

[59] (2000) 201 CLR 552.

[60] H Opie, 'The Sport Administrator's Charter: *Agar v Hyde*' (2001) 9 *Torts Law Journal* 131, 143, referring to *Agar v Hyde* (2000) 201 CLR 552.

[61] Henrietta Gelber, 'Suing the AFL for Negligence: An Analysis of the Process and Prospects of a Potential Class Action Proceeding Based on Concussion-related Injuries Sustained in AFL Football' (2021) 14(1) *Australian and New Zealand Law Journal* 108; A Greenhow, 'Concussion Policies of the National Football League: Revisiting the "Sport Administrator's Charter" and the Role of the Australian Football League and National Rugby League in Concussion Management' (2011) *Bond University Sports Law eJournal* 13, 16.

publication in the *Journal of the American Medical Association*,[62] it was not until the staining methods employed by Omalu et al in 2005 that CTE could actually 'be seen' and become accepted as a disease.[63] In this sense, CTE is a relatively recent subject of scientific research and, where new discoveries touch on the cause, progression and diagnosis of CTE, some may well impact on the application of law.

Some commentators have argued that CTE is, or was, a non-medical problem that became 'medicalised', which was said to create friction between groups interested in the cause of CTE.[64] This thesis argued that 'medicalisation' does not apply to CTE, and that such a position is untenable and unsupported through the historical record. Nonetheless, the trope of 'medicalisation' remains operative in some circles, suggesting a need for future research.[65]

Of great benefit to a living athlete will be the development of a method to definitively diagnose CTE *in vivo*. At present, *in vivo* diagnosis is by methodologies such as traumatic encephalopathy syndrome (TES), which require a judicial determination by inference — leaving open the possibility that a court will be unconvinced that the symptoms suffered are not those of, say, age-related dementia.

Commentary in scientific and medical journals often notes shortfalls in the research methodologies examining the cause, progression and diagnosis of CTE. Many of these shortfalls relate to imperfections in the collection of data, which in turn have their origins in the impermissibility of deliberately exposing human beings to RHT over an extended period. Among the shortfalls observed is an accurate concern at biased sampling on the basis that only those who suffer clinical symptoms of CTE are likely to have their brains examined after death, suggesting that the risk of CTE may be overstated, and therefore neither obvious nor giving rise to a duty of care.[66] As research broadens the scope of autopsy examination over time, it is likely that defensive arguments based on biased sampling will diminish. As improvements in medical research methodologies advance, there is a clear need to conduct research into how these advances impact on the legal entitlements of plaintiff athletes.

A related concern is the presence in the brains of some who are diagnosed at autopsy with CTE of other neurodegenerative conditions, such as Alzheimer's disease and frontotemporal dementia. Although CTE is identified by different indicators to the other diseases, the argument

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62 Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

63 Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

64 See, eg, Dominic Malcolm, 'Concussion, Chronic Traumatic Encephalopathy, and the Medicalization of Sport' in M Ventrusca and M McDonald (eds), *Sociocultural Examinations of Sports Concussions* (Routledge, 2019); A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024).

65 A Greenhow and L Gowthorp, 'Head Injuries and Concussion Issues' in N Schulenkorf and S Frawley (eds), *Critical Issues in Global Sport Management* (Routledge, 2017) 258.

66 N Schwab and L Hazrati, 'Assessing the Limitations and Biases in the Current Understanding of Chronic Traumatic Encephalopathy' (2018) 64(4) *Journal of Alzheimer's Disease* 1067.

could be made that many of the symptoms for which compensation is sought are not those of CTE but of the accompanying disease.

Most potentially impactful would be a scientific breakdown of the relative contribution to CTE of symptomatic and asymptomatic concussive head trauma. It cannot be doubted — on the basis of research stretching back a century or more — that RHT is the physical cause of CTE; and that almost without exception, sufferers of CTE report a history of multiple concussions, with many experiencing knockout blows. The thesis proposed, however, that should the cause of CTE be proven to be exclusively asymptomatic head trauma, the plaintiff athlete's case will be more difficult, given that the connection between CTE and asymptomatic head trauma was less obvious.[67]

Against the notion that new discoveries into CTE could derail a plaintiff athlete's case in negligence against an SGB is the fact that unless scientific research produces evidence completely contradicting the present state of knowledge, it is unlikely to disturb the legal analysis presented in this thesis. This comment is based primarily on the argument presented in this thesis that the standard by which to judge matters such as causation of CTE is the legal standard of the 'balance of probabilities', not a scientific standard approaching some level of certainty.[68] The legal standard addresses basic matter of causation, such as whether RHT is the cause of CTE, whether an individual athlete is afflicted with CTE, and whether a particular sport has the generic capacity to cause CTE. Only if it were found that CTE was a disease purely of genetic origin would the application of the legal standard be incorrect — a revelation verging on the impossible, given the amount of scientific research that has already verified the cause of CTE as RHT. Nonetheless, a continual monitoring a scientific journal reports into CTE, and a mapping of potential legal responses necessitates ongoing research.

10.4 Concluding remarks

The successful outcome of a plaintiff athlete's case that the governing body of his or her contact sport breached its duty by not informing itself of the risk of CTE or not warning the athlete is obviously less than certain. Unlike cases where the elements of negligence are established and recognised — say, those regarding a manufacturer's duty of care to a product user — none of the elements necessary to prove such a case has yet been recognised in a court of law.

For a brief overview of the complexity of the challenges in law argued in this thesis to face the plaintiff athlete, consider a few non-exhaustive observations. A duty of care as between a governing body of a sport and its premier players is not a recognised duty and must be established by a 'salient features' approach. An obligation of an SGB to be informed and to warn premier players of risks of harm in the form of CTE has not been established in a court of law. A judicial determination is yet to find that medical journal articles are sufficient to prove that CTE is incurred through the playing of contact sport, or that an SGB is duty-bound

[67] See Chapter 2 at **[2.5.1]**: 'The significance of sub-concussive head trauma'.

[68] See Chapter 6 at **[6.5]**: 'The standard of proof and inference of CTE *in vivo*'.

to access such articles. Proof that RHT through playing contact sport causes CTE has not been accepted by a court of law; nor whether the competition administered by a particular governing body of a contact sport caused harm in the form of CTE.

Throughout this thesis, the damaging cognitive effects of CTE on athletes have been documented. Often, though, the burden of caring for those suffering the mental and physical breakdown caused by CTE falls naturally on family and friends. The following media comments reveal a level of distress rarely contemplated as a consequence of RHT endured in playing contact sports:

> But what about the women? Their wives or partners, the ones placing Airtags on their husbands in case they go missing again and can't work out how to get home?
>
> The ones toileting their 50-something-year-old husbands? How do they feel?
>
> 'Nobody asked me about that,' the wife of one former league great said. 'Nobody asked me if I would do it all again, because I certainly wouldn't.'
>
> The wife, who remains anonymous, said she had been looking forward to a retirement of travelling and enjoying time with the family, but that was now impossible. She was now his full-time carer, like most women are with a husband with early onset dementia.
>
> … [S]o many women who are dipping into their superannuation and left financially crippled because their husband can't work. There are women who have to leave the marriages of 30 years because their husbands' behaviour has become intolerable — violent and manipulative.[69]

For many athletes struck down with CTE, or who will experience the symptoms of CTE in coming years, the disease could have been avoided through a simple step taken by their SGB — providing a risk warning. This is not to that say a warning would have been accepted by everyone, or that personal steps would have been taken in mitigation. Indeed, as intimated in the comments of the wife of a former rugby league player noted above, and the words of an ex-player who said 'I'd do it all again',[70] some athletes may keep playing even if warned. Such a choice, however, when informed of the risk is entirely different to one made in ignorance of the possible consequences. Once warned, a player can assess different forms of risk mitigation. Certainly, as CTE is a result of repeated head trauma over many years, retirement may not be necessary until concussive head trauma and the facial tenderness indicative of asymptomatic concussion reach levels of concern; or when, say, a player is advised by medicos that his or her slow recovery from concussive trauma suggests that a threshold of tolerance may be near.

It is important to realise that the bravado or the rationalisations of a player who now suffers from early-onset CTE, and who can do little to stifle its natural debilitating course, may have been very different had he or she been well-schooled in the risks of RHT leading to CTE, prior to and throughout their playing careers.

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[69] Jessica Halloran and Wendy Carlisle, 'Wally Lewis: The Tragic Cost of a Glorious Rugby League Career', *The Australian* (Sydney, 31 July 2023).

[70] Ibid.

There is also a cultural aspect to contact sports that is pertinent to how an athlete may react if warned. Had athletes been warned of the risk of CTE in, say, the 1980s, the culture of contact sport may well have developed from one that diminished those who did not 'take one for the team' or who retired early following several concussive events, to be superseded some 40-odd years later by a culture of acceptance. It may be that contact sports would have reached a point where SGBs took active steps to discourage athletes who had been concussed multiple times, or whose symptoms were long-lasting, from continuing in their playing career — perhaps, for example, to be offered alternative employment within the administration of the sport.

Dr Rowena Mobbs, a neurologist specialising in treating CTE, reported that 'she is treating more than 40 rugby league players for brain injuries':

> As one neurologist alone, I have seen over 100 in trouble, another 100 patients under watch, mainly through playing footy for 20–30 years … 'If we extrapolate to those in the community with over 10 years of footy at higher levels, then even a small percentage affected could mean tens of thousands of Australians with CTE.[71]

A point made in Chapter 2 seems accurate: CTE is like a 'ticking time bomb'.[72]

Although there are indisputable difficulties in proving the elements of a claim in negligence, this thesis nonetheless concludes that on the balance of probabilities the governing bodies of contract sport in Australia did indeed owe their premier-level athletes a duty of care that was breached in failing to introduce protocols to ameliorate the risk of harm and, principally, by failing to inform themselves of the risk of harm in the form of CTE and failing to warn their athletes of that material risk.

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[71] Ibid.

[72] Stephanie Convery, 'Damage Found after Late AFLW Player Jacinda Barclay's Brain Donated for Concussion Research', *The Guardian* (London, 30 April 2021).

Bibliography

A Articles/Books

Adams, ID, 'Brain Damage in Sport' (13 March 1976) *The Lancet* 585

Adeney, E, 'The Challenge of Medical Uncertainty: Factual Causation in Anglo-Australian Toxic Tort Litigation' (1993) 19 *Monash University Law Review* 23

Alosco, ML et al, 'Developing Methods to Detect and Diagnose Chronic Traumatic Encephalopathy during Life: Rationale, Design, and Methodology for the DIAGNOSE CTE Research Project' (2021) 13(1) *Alzheimer's Research & Therapy* 136

Aubry, M et al, 'Summary and Agreement Statement of the First International Conference on Concussion in Sport, Vienna 2001' (2002) 36 *British Journal of Sports Medicine* 6

Bachynski, Kathleen, 'Tolerable Risks? Physicians and Youth Tackle Football' (2016) 374(5) *New England Journal of Medicine* 405

Bailes, Julian, 'Role of Subconcussion in Repetitive Mild Traumatic Brain Injury: A Review' (2013) 119 *Journal of Neurosurgery* 1235

Barclay, 'Equestrian Sports' (1978) 240(17) *Journal of the American Medical Association* 1892

Barret, RS et al, 'The Dynamic Loading Response of Surfaces Encountered in Beach Running' (1998) 1(1) *Journal of Science and Medicine in Sport* 3

Becker, Robert, 'Does Traumatic Brain Injury Hold the Key to the Alzheimer's Disease Puzzle?' (2018) 14 *Alzheimer's & Dementia* 431

Benson, BW et al, 'What Are the Most Effective Risk-reduction Strategies in Sport Concussion?' (2013) 47(5) *British Journal of Sports Medicine* 4

Bey, T and B Ostick, 'Second Impact Syndrome' (2009) 10(1) *Western Journal of Emergency Medicine* 5

Bieniek, KF, 'The Second NINDS/NIBIB Consensus Meeting to Define Neuropathological Criteria for the Diagnosis of Chronic Traumatic Encephalopathy' (2021) 80(3) *Journal of Neuropathology & Experimental Neurology* 210

Blennow, K et al, 'The Neuropathology and Neurobiology of Traumatic Brain Injury' (2012) 76 *Neuron* 886

Bradford Hill, Austin, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965)

Bruzova, M et al, 'Autopsy-diagnosed Neurodegenerative Dementia Cases Support the Use of Cerebrospinal Fluid Protein Biomarkers in the Diagnostic Work-up' (2021) 11 *Scientific Reports* 10

Calandrillo, Steve, 'Sports Medicine Conflicts: Team Physicians vs Athlete-patients' (2005) 50 *St Louis University Law Journal* 185

Cantor, RC, 'Cerebral Concussion in Sport: Management and Prevention' (1992) 14(1) *Sports Medicine* 64

Casper, ST, 'Concussion: A History of Science and Medicine, 1870–2005' (2018) 58 *Headache* 795

Casper, ST et al, 'Toward Complete, Candid, and Unbiased International Consensus Statements on Concussion in Sport' (2021) 49(3) *Journal of Law, Medicine & Ethics* 372

Cherry, JD et al, 'CCL11 Is Increased in the CNS in Chronic Traumatic Encephalopathy But Not in Alzheimer's Disease' (2017) 12(9) *PLoS One*

Clark, M et al, 'Effects of Career Duration, Concussion History, and Playing Position on White Matter and Functional Neural Recruitment in Former College and Professional Football Athletes' (2018) 286 (3) *Radiology* 973

Corsellis, J, 'The Aftermath of Boxing' (1973) 3 *Psychological Medicine* 270

Critchley, M, 'Medical Aspects of Boxing, Particularly from a Neurological Standpoint' (1957) 1(5015) *British Medical Journal* 357

Daly, PJ, 'Ice Hockey Injuries: A Review' (1990) 10(3) *Sports Medicine* 122

Davies, M and I Malkin, *Torts* (LexisNexis Butterworths, 7th ed, 2015)

Davis, Gavin et al. 'Neurodegeneration and Sport' (2015) 76(6) *Neurosurgery* 655

Dietrich, J and I Field, 'The "Reasonable Tort Victim": Contributory Negligence, Standard of Care and the "Equivalence Theory"' (2017) 41(2) *Melbourne University Law Review* 602

Dryden, DM et al, 'Personal Risk Factors Associated with Injury among Female Recreational Ice Hockey Players' (2000) 3(2) *Journal of Science and Medicine in Sport* 3

Echlin, PS et al, 'A Prospective Study of Physician-observed Concussions during Junior Ice Hockey: Implications for Incidence Rates' (2010) 29(5) *Neurosurgery Focus* E4

Eggleston, Richard, *Evidence, Proof and Probability* (Weidenfeld and Nicholson, 2nd ed, 1983)

Engelhardt, J et al, 'Second Impact Syndrome. Myth or Reality?' (2021) 67(3) *Neurochirugie* 265

Erlanger, DM, 'Neuropsychology of Sports-related Head Injury: Dementia Pugilistica to Post Concussion Syndrome' (1999) 13(2) *Clinical Neuropsychologist* 193

Fain, E, 'Take a Stand: The New World of Sports Safety' (2021) *Georgia State University Law Review* 38

Fainaru-Wada, Mark and Steve Fainaru, *League of Denial* (Three Rivers Press, 2014)

Falcon, Benjamin, 'Novel Tau Filament Fold in Chronic Traumatic Encephalopathy Encloses Hydrophobic Molecules' (2019) 568 *Nature* 420

Fleming, JG, *The Law of Torts* (LBC Information Services, 9th ed, 1998)

Foster, JB, 'Brain Damage in National Hunt Jockeys' (8 May 1976) *The Lancet* 981

Freckelton, I 'Epilogue: Dilemmas in Proof of Causation' in Danuta Mendelson and Ian Freckelton (eds), *Causation in Law and Medicine* (Taylor & Francis Group, 2002)

Gardner, A et al, 'A Systematic Review and Meta-analysis of Concussion in Rugby Union' (2014) 44 *Sports Medicine* 1717

Gavett, BE et al, 'Chronic Traumatic Encephalopathy: A Potential Late Effect of Sport-related Concussive and Sub-concussive Head Trauma' (2011) 30 *Clinical Journal of Sports Medicine* 20

Gelber, Henrietta, 'Suing the AFL for Negligence: An Analysis of the Process and Prospects of a Potential Class Action Proceeding Based on Concussion-related Injuries Sustained in AFL Football' (2021) 14(1) *Australian and New Zealand Sports Law Journal* 108

Gibbs, N, 'Common Rugby League Injuries' (1994) 18(6) *Sports Medicine* 438

Gordy, L, 'Concussion of the Brain' (1955) *ABA Section of Insurance, Negligence and Compensation Law Procedure* 437

Gordy, Sara, 'The NFL's Head Cases: Why the League Faces a Big Concussion Problem' (2013) 28(3) *Marquette Sports Law Review* 491

A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024)

Greenhow, A, 'Concussion Policies of the National Football League: Revisiting the "Sport Administrator's Charter" and the role of the Australian Football League and National Rugby League in Concussion Management' (2011) *Bond University Sports Law ejournal*

Greenhow, A, 'Mitigating the Concussion Crunch: League Governance, Passive Actors and Regulators of Risk' (2021) 14(1) *Australian and New Zealand Sports Law Journal* 36

Greenhow A and Gowthorp L, 'Head Injuries and Concussion Issues' in N Schulenkorf and S Frawley (eds), *Critical Issues in Global Sport Management* (Routledge, 2017) 258.

Gronwall, D and P Wrightson, 'Cumulative Effect of Concussion' (22 November 1975) *The Lancet* 995

Grossman, AD, 'Bodily Injury Torts, The Restatement (Second) of Torts, and The Pathways of the First Hundred Years' (2014) 27(2) *Tort Trial and Insurance Practice Law Journal* 563

Halstead, ME and KD Walter, 'Sport-related Concussion in Children and Adolescents' (2010) 126 *Paediatrics* 597

Hart, J, 'Neuropathology of Traumatic Brain Injury' (2016) 43(1) *Legal Pathology* 269

Healey, D, 'Governance in Sport: Outside the Box?' (2009) 23(3) *Economic and Labour Relations Review* 39

Hirad, AA et al, 'A Common Neural Signature of Brain Injury in Concussion and Subconcussion' (2019) 5(8) *Science Advances* 1

Hooker, ER et al, 'Combat Blast-related Mild Traumatic Brain Injury Is Associated with Large Acute and Chronic Brain Lesions: A 1H-Magnetic Resonance Spectroscopy Study' (2018) 1(1) *EBioMedicine* 41

Horst, F, 'Blast-related Traumatic Brain Injury: Experience from the German Armed Forces (2002–2011)' (2016) 43(2) *Legal Pathology* 291

Hoskins, W et al, 'Injury in Rugby League' (2006) 9 *Journal of Science and Medicine in Sport* 24

Hu, X et al, 'Assessment of the Genetic Basis of Chronic Traumatic Encephalopathy' (2016) 70(3) *Neurology* 229

Hu, X et al, 'Elevated Levels of Cerebrospinal Fluid Biomarkers in Chronic Traumatic Encephalopathy Associate with Functional Impairment in Former NFL Players: An Ecological Approach' (2016) 63(7) *Alzheimers & Dementia* 805

Hume, PA et al, 'A Comparison of Cognitive Function in Former Rugby Union Players Compared with Former Non-Contact-Sport Players and the Impact of Concussion History' (2017) 51(2) *Sports Medicine* 200

Iacono, D et al, 'Chronic Traumatic Encephalopathy: Known Causes, Unknown Effects' (2017) 28 *Physical Medicine and Rehabilitation Clinics of North America* 301

Iverson, GL, 'Chronic Traumatic Encephalopathy: How Serious a Sports Problem Is It?' (2016) 70(9) *Clinical Journal of Sport Medicine* 167

Iverson, GL et al, 'Chronic Traumatic Encephalopathy Neuropathology Might Not Be a Real Disease' (2019) 74(4) *Behavioral Neurology of Dementia* 313

Iverson, GL et al, ‘Chronic Traumatic Encephalopathy Research: Perceptions and Recommendations of the Broader Scientific Community’ (2021) 75(7) *Journal of Alzheimer's Disease* 21

Iverson, GL et al, ‘Examining Later-in-life Health Risks Associated with Sport-related Concussion and Repetitive Head Impacts: A Systematic Review of Case-control and Cohort Studies’ (2003) 57 *British Journal of Sports Medicine* 26.

Iverson, GL et al, Chronic traumatic encephalopathy neuropathologic change is uncommon in men who played amateur American football, (2023) 19 *Front Neurol* 1.

Jessee, Thomas L, ‘Football Injury Statistics, the Study, and its Results’ (1959) 1(3) *Journal of the International College of Surgeons* 295

Kassirer, MD, ‘Journals in Bits and Bytes: Electronic Medical Journals’ (1992) 326(3) *New England Journal of Medicine* 195

Kemp, SPT et al, ‘The Epidemiology of Head Injuries in English Professional Rugby Union’ (2015) 49(1) *Physician and Sportsmedicine* 52

Kerr, ZY et al, ‘Association between History of Multiple Concussions and Health Outcomes among Former College Football Players: 15-Year Follow-up from the NCAA Concussion Study (1999–2001)’ (2019) 53(2) *American Journal of Sports Medicine* 987

Kersey, RD et al, ‘Risk of Suicide after Concussions: A Systematic Review and Meta-analysis’ (2018) 72(11) *JAMA Neurology* 1301

Kirkendall, DT et al, ‘Heading and Head Injuries in Soccer’ (2001) 31(5) *Sports Medicine* 369

Laird, MD et al, ‘Characterization of the Microglial Response to Chronic Traumatic Encephalopathy’ (2021) 10(12) *Journal of Neurotrauma* 1331

Lampert, PW and JM Hardman, ‘Morphological Changes in Brains of Boxers’ (1984) 251(20) *Journal of the American Medical Association* 27

Leclerc, S et al, ‘Permanent Consequences of Head Injury in Children’ (2001) 131 *Archives de Pédiatrie* 674

Lewis, N et al, ‘Co-existing Chronic Traumatic Encephalopathy and Alzheimer’s Disease Pathology in Aged University Athletes’ (2017) 55(7) *Acta Neuropathologica Communications* 1

Lindsay, KW, ‘Serious Head Injury in Sport’ (1980) 281 *British Medical Journal* 789

Lynch, ME, ‘The Development of Neuropathology in Sports: A Review’ (2004) 6 *Medicine & Science in Sports & Exercise* 47

Madden, M and S Syed-Khuzzan, 'Football and American Football Injuries' (1995) 2 *Sports Medicine* 223

Magnusson, E, 'Statistical Proof of Causation' in Danuta Mendelson and Ian Freckelton (eds), *Causation in Law and Medicine* (Taylor & Francis Group, 2002)

Makdissi M et al, 'When Should an Athlete Retire or Discontinue Participating in Contact or Collision Sports Following Sport-related Concussion? A Systematic Review' (2023) 57 *British Journal of Sports Medicine* 882.

Malcolm D, 'Concussion, Chronic Traumatic Encephalopathy, and the Medicalization of Sport' in M Ventrusca and M McDonald (eds), *Sociocultural Examinations of Sports Concussions* (Routledge, 2019).

Mannix, RC et al, 'Effect of Cognitive Activity Level on Duration of Post-concussion Symptoms' (2016) 88 *Pediatrics* 1

Marshall, SW and R Spencer, 'Concussion in Rugby: The Hidden Epidemic' (2001) 36(3) *Journal of Athletic Training* 334

Martland, Harrison, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103

Mavroudis, I et al, 'A Review of the Most Recent Clinical and Neuropathological Criteria for Chronic Traumatic Encephalopathy' (2023) 11(12) *Healthcare* 37

Mawdsley, C and FR Ferguson, 'Neurological Disease in Boxers' (1963) (19 October 1963) *The Lancet* 795

May, T et al, 'Second Impact Syndrome' (3 July 2022) *StatPearls [Internet]*

McCrory, P, 'Does Second Impact Syndrome Exist?' (2001) 11(3) *Clinical Journal of Sports Medicine* 144

McCrory, P, 'When to Retire after Concussion? (2002) 5(3) *Journal of Science and Medicine in Sport* 42

McCrory, P et al, 'Consensus Statement on Concussion in Sport: The 3rd International Conference on Concussion in Sport Held in Zurich, November 2008' (2009) 12(3) *Journal of Science and Medicine in Sport* 342

McCrory, P et al, 'Consensus Statement on Concussion in Sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012' (2013) 47(5) *British Journal of Sports Medicine* 250

McCrory, P et al, 'Consensus Statement on Concussion in Sport — The 5th International Conference on Concussion in Sport held in Berlin, October 2016' (2017) 51(11) *British Journal of Sports Medicine* 838

McCrory, P, 'Sports Concussion and the Risk of Chronic Neurological Impairment' (2011) 21(6) *Clinical Journal of Sports Medicine* 6

McCrory, P et al, 'Summary and Agreement Statement of the 2nd International Conference on Concussion in Sport, Prague 2004' (2005) 39(4) *British Journal of Sports Medicine* 196

McDonald, CG, 'The Sequelae of Head Injuries' (11 January 1936) *Medical Journal of Australia* 1

McGraw, Ken, 'Concussion, the NFL, and Major Personal Injury Lawsuits' (2012) 15 *University of Pennsylvania Journal of Constitutional Law* 1009

McKee, A et al, 'Chronic Traumatic Encephalopathy (CTE): Criteria for Neuropathological Diagnosis and Relationship to Repetitive Head Impacts' (2023) 145(4) *Acta Neuropathologica* 54

McKee, A et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709

McKee, A et al, 'The First NINDS/NIBIB Consensus Meeting to Define Neuropathological Criteria for the Diagnosis of Chronic Traumatic Encephalopathy' (2016) 131(75) *Acta Neuropathologica* 85

McKee, A et al, 'The Neuropathology of Chronic Traumatic Encephalopathy' (2015) 25 *Brain Pathology* 2

McKee, A et al. 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2012) 366(23) *New England Journal of Medicine* 2278

McLendon, LA et al, 'The Controversial Second Impact Syndrome: A Review of the Literature' (2016) 62 *Paediatric Neurology* 9

Mez, J et al, 'Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football' (2017) 318(4) *Journal of the American Medical Association* 360

Molnar, GF et al, 'Effect of Cognitive Activity Level on Duration of Post-concussion Symptoms' (2016) 88 *Pediatrics* 1

Montenigro, PH et al, 'Clinical Subtypes of Chronic Traumatic Encephalopathy: Literature Review and Proposed Research Diagnostic Criteria for Traumatic Encephalopathy Syndrome' (2014) 6(5) *Alzheimer's Research & Therapy* 39

Morrison, RG, 'Medical and Public Health Aspects of Boxing' (1986) 255(18) *Journal of the American Medical Association* 27

Mountjoy, M, 'Causation in Sport Injury: A Look at the "Blame Game"' (2013) 47(5) *British Journal of Sports Medicine* 221

Mountjoy, M, 'The Basis for Canada's Position on Banning Heading the Ball in Soccer for Children 10 Years and Younger' (2017) 2016(22) *Canadian Medical Association Journal* E111

Murch, AR et al, 'Computerized Neuropsychological Screenings in Amateur and Professional Rugby League Players: A Longitudinal Study' (2008) 15(5) *Journal of Head Trauma Rehabilitation* 489

Nagahiro, S et al, 'Magnetic Resonance Imaging in Chronic Traumatic Encephalopathy: Systematic Review' (2019) 63 *Psychiatry and Clinical Neurosciences* 40

Nemetz, PN et al, 'Traumatic Brain Injury and Time to Onset of Alzheimer's Disease: A Systematic Review' (2014) 37 *Neuroepidemiology* 135

Nowinski C, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1

Omalu, Bennet et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128

Omalu, Bennet et al, 'Chronic Traumatic Encephalopathy in a National Football League Player: Part II' (2006) 59(5) *Neurosurgery* 1086

Omalu, Bennet et al, 'Chronic Traumatic Encephalopathy, Suicides and Parasuicides in Professional American Athletes: The Role of the Forensic Pathologist' (2010) 134(6) *American Journal of Forensic Medicine and Pathology* 977

Omalu, Bennet et al, 'Emerging Histomorphologic Phenomena of Brain Damage in Football Players: Implications for Clinical Diagnosis' (2010) 79(6) *Neurosurgery* 662

Orchard, J, 'Comparison of Injuries in Elite Senior and Junior Australian Football (1998) 2 *Journal of Science and Medicine in Sport* 82

Orchard, J, 'Match of the Decade: Risk Management of Concussion Versus High-speed Collisions in the Football Codes' (2015) 203(7) *Medical Journal of Australia* 1

Petersen, RC et al, 'Mild Traumatic Brain Injury: A Clinical Approach' (2014) 83 *Mayo Clinic Proceedings* 1234

Pierce, JE et al, 'Effects of Concussive and Subconcussive Brain Trauma on Alzheimer's Disease Pathology in C57Bl/6 Mice' (2019) 8(3) *Scientific Reports* 12845

Quarrie KL, 'The New Zealand Rugby Injury and Performance Project. VI: A Prospective Cohort Study of Risk Factors for Injury in Rugby Union Football' (2001) 35 *British Journal of Sports Medicine* 157

Qi B et al, Prevalence of Chronic Traumatic Encephalopathy in Athletes With Repetitive Head Impacts: A Systematic Review and Meta- Analysis, (2025) 35 *Scandinavian Journal of Medicine & Science in Sport* 1.

Rasmussen, C, 'How Dangerous Are Youth Sports for the Brain? A Review of the Evidence' (2018) 7 *Berkeley Journal of Entertainment and Sports Law* 67

Reams, N et al, 'Pathological Changes in the White Matter after Concussion' (2018) 75(3) *Archives of Pathology & Laboratory Medicine* 332

Robbins, CA et al, 'Chronic Traumatic Encephalopathy in Athletes: Lessons from the Boston University Flag Football Experience' (2014) 12(4) *Neurosurgery* 572

Roberts, AH, *Brain Damage in Boxers* (Pitman, 1969)

Rowson, S et al, 'Biomechanical Perspectives on Concussion in Sport' (2012) 47 *Sports Medicine* 459

Rowson, S et al, 'Can Helmet Design Reduce the Risk of Concussion in Football?' (2014) 129 *Journal of Neurosurgery* 919

Russell, ER, 'Association of Field Position and Career Length with Risk of Neurodegenerative Disease in Male Former Professional Soccer Players' (2021) 78(9) *JAMA Neurology* 1057

Sameer, S et al, 'Cognition and Mental Health Later in Life' (2017) 74 *JAMA Neurology* 909

Sancar, J, 'Subconcussive Football Hits May Leave a Telltale Signature of Brain Damage' (2019) 322(16) *Journal of the American Medical Association* 1537

Saulle, M et al, 'Chronic Traumatic Encephalopathy: The Hidden Epidemic in Our Midst' (2012) 45(6) *Journal of the American Osteopathic Association* 22

Schneider, KJ et al, 'Sports Injuries and Illnesses During the London Summer Olympic Games 2012' (2013) 47(7) *British Journal of Sports Medicine* 407

Schwab N and L Hazrati L, 'Assessing the Limitations and Biases in the Current Understanding of Chronic Traumatic Encephalopathy' (2018) 64(4) *Journal of Alzheimer's Disease* 1067.

Schwenk, TL et al, 'Epidemiology of Concussions among United States High School Athletes in 20 Sports' (2017) 45(3) *American Journal of Sports Medicine* 715

Sercl, M and O Jaros, 'The Mechanisms of Cerebral Concussion in Boxing and their Consequences' (1962) 3 *World Neurology* 27

Shenton, ME et al, 'A Review of Magnetic Resonance Imaging and Diffusion Tensor Imaging Findings in Mild Traumatic Brain Injury' (2012) 68(8) *Brain Imaging and Behavior* 77

Society for the Study of Trauma and Dissociation, 'Child Dissociative Checklist (CDC-CH)' (2019)

Stern, RA et al, 'Clinical Presentation of Chronic Traumatic Encephalopathy' (2013) 121 *Neurology* 1121

Stewart, P and A Stuhmcke, *Australian Principles of Tort Law* (The Federation Press, 5th ed, 2022)

Storli, OV and NI Bachen, 'Soccer Injuries to the Brain: A Neurologic and Electroencephalographic Study of Former Players (1989) 80(2) *Acta Neurologica Scandinavica* 151

Strich, SJ, 'Shearing of Nerve Fibres as a Cause of Brain Damage Due to Head Injury' (26 August 1961) *The Lancet* 17

Talavage, TM et al, 'Functionally-detected Cognitive Impairment in High School Football Players Without Clinically-diagnosed Concussion' (2013) 108(47) *Journal of Neurotrauma* 15

Tator, CH, 'Concussions and Their Consequences: Current Diagnosis, Management and Prevention' (2016) 88(3) *Canadian Medical Association Journal* 185

Teasdale, G et al, 'Neurological Outcome after Severe Head Injury: A Study of 502 Patients' (1979) 2(8151) *Journal of Neurology, Neurosurgery and Psychiatry* 234

Thorndike, A, 'Serious Recurrent Injuries of Athletes' (1952) 247 *New England Journal of Medicine* 554

Thorpe, David, 'Concussion, Chronic Traumatic Encephalopathy, and the Legal Obligation of Sporting Organisations to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks', (2021) 28 *Journal of Law and Medicine* 63

Thorpe, David, 'Concussion, Chronic, Traumatic Encephalopathy and Proof of Causation by Material Contribution' (2022) 15(1) *Australian and New Zealand Sports Law Journal* 63

Thorpe, David, 'Concussion, Chronic Traumatic Encephalopathy and Sport in a Legal Setting' (2020) 27 *Journal of Law and Medicine* 63

Thorpe, David, 'Knowledge of Chronic Traumatic Encephalopathy and Concussion Cannot Support a Negligence Suit against major Sporting Organisations by Athletes. Or Can It?' (2022) 29(4) *Journal of Law and Medicine* 1052

Thorpe, David, 'Proof In Vivo of Actual Harm in the Form of CTE: The Question of "Individual Agency"' (2024) 17(1) *Australian and New Zealand Sports Law Journal* 64

Thorpe, David et al, *Sports Law* (Oxford University Press, 3rd ed, 2017)

Thorpe, David et al, *Sports Law* (Oxford University Press, 4th ed, 2022)

Tierney, RT et al, 'Epidemiology of Concussions in High School Football: Implications for Data Collection' (2005) 39 *Clinical Journal of Sport Medicine* 206

Tierney, RT et al, 'Sex Differences in Head Acceleration during Heading while Wearing Soccer Headgear' (2008) 20(2) *Journal of Athletic Training* 107

Turk, KW, 'A Comparison between Tau and Amyloid-B Cerebrospinal Fluid Biomarkers in Chronic Traumatic Encephalopathy and Alzheimer Disease' (2022) 14(28) *Alzheimer's Research & Therapy* 65

Turner, RC et al, 'Concussion in the National Football League: An Evidence-Based Commentary' (2016) 7 *CNS Spectrums* 113

Tysvaer, AT and EA Lochen, 'Soccer Injuries to the Brain: A Neuropsychologic Study of Former Soccer Players' (1991) 19(1) *American Journal of Sports Medicine* 22

Tysvaer, AT and J Lokke, 'Football Injuries to the Head and Face' (1991) 25 *Clinical Sports Medicine* 235

Unger, D, *George Marshall: A Biography* (Harper-Collins, 2014)

Villa, Dominic, *Annotated Civil Liability Act 2002 (NSW)* (Lawbook Co, 2004)

Warren, A et al, 'High Acute: Chronic Workloads Are Associated with Injury in England & Wales Cricket Development Programme Fast Bowlers' (2018) 21 *Journal of Science and Medicine in Sport* 40

Wetjen, NM et al, 'Second Impact Syndrome: Concussion and Second Injury Brain Complications' (2010) 211(4) *Journal of the American College of Surgeons* 553

Williams, AM et al, 'Do Concussion Laws Reduce Head Injury Rates in High School Sports?' (2021) 55(5) *American Journal of Sports Medicine* 1132

Wilson, JC et al, 'Player and Game Characteristics and Head Impacts in Female Youth Soccer Players' (2017) 52(7) *Orthopaedic Journal of Sports Medicine* 232

Windholz, E, 'Professional Sport, Work Health and Safety Law and Reluctant Regulators' (2015) *Bond University Sports Law ejournal* 7

Windholz, E, 'Team-based Professional Sporting Competitions and Work, Health and Safety Law: Defining the Boundaries of Responsibility' (2015) 43 *Australian Business Law Review* 303

Windle, WF, RA Groat and CA Fox, 'Experimental Structural Alterations in the Brain During and After Concussion' (1944) 79 *Surgery, Gynecology and Obstetrics* 561

Wortzel, HS et al, 'Effects of Repeated Subconcussive Trauma on the Neurobehavioral Profile in High School Football Players' (2014) 28(6) *Journal of Neurotrauma* 1049

B Cases

Adeels Palace Pty Ltd v Moubarak (2009) 239 CLR 420

Agar v Hyde (2000) 201 CLR 552

Amaca Pty Ltd v Booth (2011) 246 CLR 364

Amaca Pty Ltd v Ellis (2010) 240 CLR 111

Bale v Seltsam Pty Ltd [1996] QCA 288

Bank of New South Wales v Commonwealth (1948) 76 CLR 124

Banque Bruxelles Lambert SA v Eagle Star Insurance Co Ltd [1997] AC 191

Belhaven and Stenton Peerage (1875) 1 App Cas 278

Benic v New South Wales [2010] NSWSC 1039

Bolton v Stone [1951] AC 850

Bonnington Castings v Wardlaw [1956] AC 613

Bradshaw v McEwans Pty Ltd (1951) 217 ALR 1

CAL No 14 Pty Ltd v Motor Accidents Insurance Board (2009) 239 CLR 390

Capital and Counties Plc v Hampshire County Council [1997] 3 WLR 331

Caparo Industries Plc v Dickman [1990] 2 AC 605

Cartledge v E Jopling & Sons Ltd [1963] 1 All ER 341

Caswell v Powell Duffryn Associated Collieries Ltd [1940] AC 152

Caterson v Commissioner of Railways (NSW) (1973) 128 CLR 99

Chamberlain v The Queen (1984) 153 CLR 521

Chapman v Hearse (1961) 106 CLR 112

Chappel v Hart (1998) 195 CLR 232

Collins v Clarence Valley Council [215] NSWCA 263

Commissioner of Railways v Ruprecht (1979) 142 CLR 563

Crimmins v Stevedoring Industry Finance Committee (1999) 200 CLR 1

CSR Ltd v Young (1998) 16 NSWCCR 56

Doherty v New South Wales [2010] NSWSC 450

Donoghue v Stevenson [1932] AC 562

EMI (Australia) Ltd v BES [1970] 2 NSWR 238

Erickson v Bagley [2015] VSCA 220

Fairchild v Glenhaven Funeral Services Ltd [2003] 1 AC 32

Graham Barclay Oysters Pty Ltd v Ryan [2002] HCA 54

Grills v Leighton Contractors Pty Ltd [2015] NSWCA 72

Harriton v Stephens (2006) 226 CLR 52

H v Royal Alexandra Hospital (1990) 1 Med LR 297

Holloway v McFeeters (1956) 94 CLR 47

Jaber v Rockdale City Council [2008] NSWCA 98

Jones v Dunkel (1959) 101 CLR 298

Kenny & Good Pty Ltd v MGICA (1992) Ltd (1999) 199 CLR 413

Laoulach v Ibrahim [2011] NSWCA 402

Le Lievre v Gould [1893] 1 QB 491

Mallonland Pty Ltd v Advanta Seeds Pty Ltd [2004] HCA 25

March v E & MH Stramare Pty Ltd (1991) 171 CLR 506

Maxwell v National Football League (Superior Court of California, County of Los Angeles, BC 465842, 19 July 2011)

Menz v Wagga Wagga Show Society Inc [2020] NSWCA 65

Modbury Triangle Shopping Centre Pty Ltd v Anzil (2000) 205 CLR 254

Nagle v Rottnest Island Authority (1993) 112 ALR 393

Paul v Cooke (2012) NSWSC 840

Perre v Apand Pty Ltd (1998) 198 CLR 180

Perrett v Collins [1998] 2 LL Rep 255

Pollard v Trude [2008] QSC 119

Powney v Kerang and District Health (2014) 43 VR 506

Pyrenees Shire Council v Day (1998) 192 CLR 330

Reay v British Nuclear Fuels (1994) 5 Med LR 1

Roads and Traffic Authority of NSW v Dederer (2007) 234 CLR 330

Roe v Minister of Health [1954] 2 QB 66

Rogers v Whitaker (1992) 175 CLR 479

*Rooke v Australian Football Leagu*e (Supreme Court of Victoria, S ECI 2023 00969, commenced 14 March 2023)

Seltsam Pty Ltd v McGuinness [1997] NSWCA 292

Shaw v Thomas [2010] NSWCA 169

Shepherd v The Queen (1990) 170 CLR 573

South Australia Asset Management Corp v York Montague Ltd [1996] UKHL 10

South Eastern Sydney Area Health Service v King [2006] NSWCA 2

Stewart v Ackland (2015) 10 ACTLR 207

Strong v Woolworths (2012) 246 CLR 182

Stuart v Kirkland-Veenstra (2009) 237 CLR 215

Sullivan v Moody (2001) 207 CLR 562

Swinton v China Mutual Navigation Co Ltd (1951) CLR 553

Tabet v Gett (2010) 240 CLR 537

Tame v New South Wales (2002) 211 CLR 317

Tapp v Australian Bushmen's Campdraft & Rodeo Association Ltd (2022) 273 CLR 454

TNT Management Pty Ltd v Brooks (1979) 53 ALJR 267

Vairy v Wyong Shire Council (2005) 223 CLR 422

Wallace v Kam (2013) 250 CLR 375

Watson v British Boxing Board of Control; World Boxing Organisation Inc [2001] QB 1134

Western Suburbs Hospital v Currie (1987) 9 NSWLR 511

Wyong Shire Council v Shirt (1980) 146 CLR 40

Zabic v Alcan Gove (2015) 292 FLR 413

Zantuck v Richmond Football Club [2022] VSC 405

C Legislation

Civil Law (Wrongs) Act 2002 (ACT)

Civil Liability Act 1936 (SA)

Civil Liability Act 2002 (NSW)

Civil Liability Act 2002 (Tas)

Civil Liability Act 2002 (WA)

Civil Liability Act 2003 (Qld)

Personal Injuries (Liabilities and Damages) Act 2003 (NT)

Wrongs Act 1958 (Vic)

D Media/Other

Alwyn, Michael and Andy Bull, 'Rugby World Cup Winner Steve Thompson Reveals He Has Dementia and Joins Landmark Legal Case', *The Guardian* (London, 8 December 2020)

Barrett, C, 'Lawyer Keen to Act for NRL Players on NFL-style Concussion Lawsuits', *Sydney Morning Herald* (Sydney, 10 November 2014)

Branch, J, 'Brain Trauma Extends to Soccer Field', *The New York Times* (New York, 26 February 2014)

Bruce, J, 'Rugby League Reveals Eye-watering Spike in Revenue after 2021 NRL Season', *NCA Newswire* (online, 28 February 2022) <https://www.pilbaranews.com.au/news/rugby-league-reveals-eye-watering-spike-in-revenue-after-2021-nrl-season-c-5865841>

'Concussions Force Retirement', *The Australian* (Sydney, 9 November 2012)

Convery, S, 'Damage Found after Late AFLW Player Jacinda Barclay's Brain Donated for Concussion Research', *The Guardian* (London, 30 April 2021)

Convery, S, 'Slowly Tortured by His Brain': Family Urges Action on Head Trauma at Concussion Inquiry', *The Australian* (Sydney, 13 February 2023)

'Dementia and Sport: Rugby Players Launch Legal Action against Governing Bodies', *BBC Sport*, *BBC* (online, 25 July 2022) <https://www.bbc.com/sport/rugby-union/62290045>

Elder, John, '"Existential Threat to Professional Sport": Brain Disease Could Affect One in 10 Footballers', *The New Daily* (online, 7 March 2021)

'Former NRL Player Suing over Brain Disease from Repeated Head Knocks', *The Australian* (Sydney, 19 August 2017)

'Graham "Polly" Farmer is the First Australian Rules Player Diagnosed with Chronic Traumatic Encephalopathy', *ABC News* (online, 27 February 2020) <https://www.abc.net.au/news/2020-02-27/australian-rules-great-graham-polly-farmer-diagnosed-with-cte/12005508>

Grand Prix: The Killer Years (BBC Knowledge, 2008)

Halloran, Jessica, 'Don't Listen to the People Trying to Put a Veil over This', *The Australian* (Sydney, 1 September 2023)

Halloran, Jessica and Wendy Carlisle, 'The Tragic Cost of a Glorious Rugby League Career — Wally Lewis', *The Australian* (Sydney, 31 July 2023)

Harari, Fiona, 'Out Cold', *Weekend Australian* (Sydney, 27 August 2022)

Ingle, S, 'Steve Smith's Test Concussion Reignites Debate about Safety, *The Guardian* (London, 19 August 2019)

'James McManus's Concussion Compensation Case against NRL Club Newcastle Settled in Favour of the Knights', *ABC News* (online, 18 September 2021) <https://www.abc.net.au/news/2021-09-18/former-knight-mcmanus-misses-nrl-concussion-compensation/100473992>

Kent, P, 'NRL Should Heed US Lessons over Dangers of Repeated Head Knocks', *news.com.au* (online, 8 September 2012) <https://www.news.com.au/sport/nrl/nrl-should-heed-us-lessons-over-dangers-of-repeated-head-knocks/news-story/e9a1778b5e51bad6a0e6b5f999c807da>

Kieza, G, 'Fears of Football Injury Time Bomb across All Codes', *The Courier-Mail* (Brisbane, 22 September 2012)

Kilgore, A, 'Aaron Hernandez Suffered from the Most Severe CTE Ever Found in a Person His Age', *The Washington Post* (Washington DC, 9 November 2017)

Marshal, K, 'The Concussion Class Action that Could Punch a Hole in the AFL', *The Age* (online, 16 March 2019) <https://www.theage.com.au/sport/afl/the-concussion-class-action-that-could-punch-a-hole-in-the-afl-20190311-p513ay.htm>

Masters, R, 'World Rugby's Medical Chief Doubts Breakthrough CTE Findings', *Sydney Morning Herald* (Sydney, 30 June 2019)

'NRL Wants to Widen Research into Concussion', *The Australian* (Sydney, 3 August 2011)

Pandaram, Jamie, ‘Hits and Memory Loss but Barnes Clear of Serious Concussion’, *Sydney Morning Herald* (online, 25 May 2011)

Pandaram, Jamie, ‘I Don’t Feel Right: Concussions Worry Barnes’, *Sydney Morning Herald* (online, 6 April 2011)

Popham, Alix, ‘Ex-Wales Flanker on Early Onset Dementia Diagnosis’, *BBC Sport*, *BBC* (online, 8 December 2020) <https://www.bbc.com/sport/rugby-union/55208227>

‘Rugby League Legend Wally Lewis Diagnosed with Early Signs of Brain Disease CTE’, *The Australian* (online, 31 July 2023)

Reid, A, ‘“Can’t Think of Things”: Rugby League Legend’s Devastating Diagnosis’, *Yahoo Sport Australia* (online, 1 June 2021) <https://au.sports.yahoo.com/nrl-2021-steve-mortimer-dementia-rugby-league-great-diagnosis-225810122.html>

Sheehan, P, ‘Concussion a Concern from Elite to Schools’, *Sydney Morning Herald* (Sydney, 28 February 2013).

Simers, TJ, ‘No-brainer: Al Toon Will Attest that Symptoms Can Remain for Years’, *Los Angeles Times* (Los Angeles, 20 October 1995)

Walsh, C, ‘St Kilda’s Koby Stevens Succumbs to Concussion Symptoms’, *The Australian* (Sydney, 24 July 2018)

Weston, J, ‘Love and Pain: An AFL Legend Looks Back at the Damage Done’, *The Standard* (online, 20 October 2020) <https://www.theswinstandard.net/2020/10/20/love-and-pain-an-afl-legend-looks-back-at-the-damage-done/>

Woods, M, ‘NRL Taking Concussion Seriously: Greenberg’, *ESPN*, 8 July 2019, <https://www.espn.com/nrl/story/_/id/27143857/greenberg>

Writer, L, *Bumper: The Life and Times of Frank ‘Bumper’ Farrell* (Hachette, 2011)

Zita, D, ‘Who the F**k Are You?’: Ex-AFL Player Explains Brutal Toll of Concussions that Ended Career’, *Fox Sports Australia* (online, 27 August 2021) <https://www.foxsports.com.au/afl/afl-news-2021-jack-frost-concussion-long-term-impact-interview-girlfriend-mental-health/news-story/1633cec888cd4e99b5da7d1518c3d365>

Appendix 1:
The Science of Concussion and CTE in Sport

1.1 Introduction

The application of law relevant to a claim of negligence by a plaintiff athlete against an SGB due to failure to warn cannot be properly understood unless 'the disease processes' of CTE are comprehended. The progression of CTE — initially asymptomatic, to early symptoms of cognitive disfunction to increasingly severe disability, and ultimately to death — along with its causes, discovery, history and scientific development are critical facts pertinent to the operation of law. Accordingly, this appendix examines the features of CTE argued as relevant to such a negligence claim.

This appendix is supplemental to the main body of the thesis. The facts relevant to the discovery, cause and progression of knowledge of CTE are the basis to this appendix and directed to matters relevant to legal discourse in the body of the thesis.

As a detailed supplement to Chapter 2, the appendix provides additional information regarding the factual foundation of claims of a breach of duty within a broad legal context.

1.1.1 This appendix proceeds as follows

Section 1.2: The US NFL and NHL Litigations general body of medical knowledge

Players of American football in the National Football League (NFL) in the United States who believed they suffered symptoms of CTE, sued the governing body of their sport for failing to take reasonable steps to mitigate the risk of CTE, including a failure to warn. The case and terms of settlement are briefly discussed in this section and contrasted with that of Australian contact sport athletes.

Section 1.3: Financial arrangements between SGBs and district clubs

The district clubs, the usual employer of contact sport athletes in Australia, are at best minimally profitable. For this reason, athletes who seek compensation after suffering symptoms of CTE, must seek redress from their relevant SGB. This section discusses the financial arrangements between SGBs and the district clubs to highlight why these clubs are not the natural defendant in a litigation suit by employee athletes.

Section 1.4: Tau accumulation and contact sport

Through reference to medical research, this section examines the connection between RHT in the playing of contact sport and the release of tau from the axons of the brain to form abnormal hyperphosphorylated tau (p-tau) in the neurons and astroglia distributed around small blood vessels, the ultimate result of which is symptoms of CTE.

Section 1.5: Contact sport and the bio-mechanism of damage

This section reviews literature concerned with symptomatic concussion and how the mechanical forces of repeated trauma to the brain damages axons leading to structural changes to the brain itself.

Section 1.6: The progression of CTE

Once the level of head trauma reaches a particular, but unknown threshold, the processes of CTE begin. CTE is marked by several distinct stages of increasing disability leading ultimately to death. As compensation is only awarded once harm is detected and material, this section discusses the relevance of the symptom's characteristic of each stage of CTE.

Section 1.7: Second impact syndrome

This section discusses the rare but nonetheless documented fatal condition of Second impact syndrome, caused when an individual suffers a second concussion within a short time period. Focus is on the reasonable response of a SGB to this condition.

1.2 The US NFL and NHL litigations

Legal action by Australian athletes, actual or threatened, generally emulates that of players of American football in the National Football League (NFL) in the United States in the sense that both concern an SGB of a contact sport which, as contended, possessed a duty of care to players who had since been diagnosed with or suspected of suffering from CTE argued to have been caused by participation in that SGB's competition. Other than where the facts and elements of law are relevant, this thesis is not concerned with the application of the US tort-based legal system and clearly places no reliance upon these studies or makes any suggestion that they might influence Australian judicial decision-making. This section is a summary for purposes of context and overview.

Beginning with *Vernon Maxwell et al v National Football League*[1] in 2011, the first US suits were wide ranging, stating typically that the NFL

> owed a duty to protect plaintiffs on the playing field; owed a duty to Plaintiffs to educate them and other players in the NFL about CTE and/or concussion injury; owed a duty to Plaintiffs to educate trainers, physicians and coaches about CTE/or concussion injury; owed a duty to design rules and penalties for players who use their head or upper body to hit or tackle; to design rules to eliminate

[1] US District Court for the Central District of California, October 11, 2011, 2:2011cv08394.

> the risk of concussion during games and/or practices; to promote research into and cure for CTE and the effects of concussion injury over a period of time.[2]

By 2013 'more than 4,800 player-plaintiffs' and more than 1,000 player spouses had brought '242 concussion-related lawsuits' against the NFL.[3] The complaints were consolidated to be heard before the District Court of the Eastern District of Pennsylvania. The class action claimed in part that

> the NFL has held itself out as the guardian and authority on the issue of player safety and has unilaterally shouldered for itself a common law duty to provide players with rules and information that protect them as much as possible from short-term and long-term health risks.
>
> …
>
> Despite the NFL's assumption of this responsibility, the NFL … was negligent and failed to carry out this duty in that it failed to inform NFL players of the risks associated with MTBI [mild traumatic brain injury][4] and/or it was wilfully blind to the medically proven fact that repetitive MTBI would lead to neuro-cognitive injuries in many NFL players, including the Plaintiffs [and was] aware of the health risks associated with repetitive blows producing sub-concussive and concussive results and the fact that some members of the NFL player population were at significant risk of developing long-term brain damage and cognitive decline as a result.[5]
>
> Among the many claims brought by the NFL players, a feature common in ensuring all litigatory possibilities are covered, are several concerned with the failure to warn. For example:
>
> > For decades, the NFL failed to warn NFL players of the medical risks associated with repetitive head impacts during NFL games and practices' …

And

> 'Despite its knowledge and controlling role in governing player conduct on and off the field, the NFL turned a blind eye to the risk and failed to warn and/or impose safety regulations governing this health and safety problem.[6]

The 'failure to warn' claim is also seemingly recognised by Greenhow in an article appearing in a *Bond eJournal*. Listed under 'breach' in reference to the Maxwell litigation are

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2 Ibid. See also *Easterling et al v National Football League et al*, Pennsylvania Eastern District Court 2011, Case No 2:11-cv--05209-AB; *Evans et al v National Football League*, Pennsylvania Eastern District Court 2012, Case No 2:12-cv-02682.

3 'Plaintiffs/Former Players', *NFL Concussion Litigation* (Web Page) <http://nflconcussionlitigation.com/?page_id=274>.

4 'Mild traumatic brain injury (mTBI), or concussion, is the most common type of traumatic brain injury. With mTBI comes symptoms that include headaches, fatigue, depression, anxiety and irritability, as well as impaired cognitive function': K McInnes et al, 'Mild Traumatic Brain Injury (mTBI) and Chronic Cognitive Impairment: A Scoping Review' (2017) 12(4) *PLoS One* <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5388340/>.

5 *In Re National Football League Players' Concussion Injury Litigation*, No 2:12-md-02323-AB, MDL No 2323, Plaintiffs' Amended Master Administrative Long-Form Complaint, [6]–[7]–[8]–[9]; District Court of the Eastern District of Pennsylvania. Footnote added.

6 Ibid [102], [9].

the words: 'Failure to properly inform players of the health risks associated with concussive injury' and 'Failure to warn of the harm of repetitive concussion injury'.[7]

A settlement agreement was reached in June 2014, amended on 13 February 2015 and certified by the United States Court of Appeals on 22 April 2015 as 'a fair, reasonable and adequate compromise'.[8] Under the settlement, claimants were divided into three classes, comprising 'retired NFL players', authorised representatives of 'deceased or legally incapacitated' retired NFL players, and 'close family members' of retired NFL players. The settlement incorporated three primary elements: a 'Monetary Award Fund' to pay compensation according to a 'qualifying diagnosis'; an optional 'Baseline Assessment Program' to diagnose cognitive deficits; and an 'Education Fund' to 'support education programs promoting safety and injury prevention with respect to football players'.[9] The level of compensation was aligned to the type of condition suffered and the number of years the player participated in the NFL; for example, an ex-player who suffers Alzheimer's disease under 45 years old is entitled to US$3.5 million, while one aged 65–69 receives US$620,000.[10] As of January 2023, 20,570 retired player and representative claimants had registered for compensation and US$1,080,526,669 'payable money awards' had been made.[11] The settlement includes the statement that the NFL and related parties

> expressly deny that they … have violated any duty to, breached any obligation to, committed any fraud on, or otherwise engaged in any wrongdoing … and expressly deny the allegations asserted in the Class Action Complaint and Related Lawsuits, and deny any and all liability related thereto.[12]

A similar suit to that of the NFL was brought by retired ice hockey players of the North American National Hockey League (NHL). A settlement in 2018, prompted by 26 initial claims, totalled US$18 million. It granted US$7 million to players who 'opted in' to the scheme, and included an agreement to fund neuropsychological testing of players, and a 'common good' fund of US$2.5 million to support retired players' health and welfare.[13] The settlement, far more modest than that of the NFL, is said to reflect a pool of potential sufferers

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7 A Greenhow, 'Concussion Policies of the National Football League: Revisiting the "Sport Administrator's Charter" and the role of the Australian Football League and National Rugby League in Concussion Management' (2011) *Bond University Sports Law ejournal*, referring to Maxwell et al v National Football League (Superior Court of California, County of Los Angeles, BC 465842, 19 July 2011), 1, Complaint against the National Football League [110].

8 *Turner v NFL (In re NFL Players Concussion Injury Litigation)* 307 FRD 351 (ED Pa 2015).

9 *In Re National Football League Players' Concussion Injury Litigation*, No 2:12-md-02323-AB, MDL No 2323, Class Action Settlement Agreement, <https://www.nflconcussionsettlement.com/Docs/Amended%20Class%20Action.pdf>, 'Article III: Settlement Benefits for Class Members'.

10 Ibid Exhibit A-3.

11 Ibid 'Article XXIV: Denial or Wrongdoing, No Admission of Liability',

12 Ibid.

13 Settlement Agreement between the NHL and Plaintiffs' Counsel (NHL/NHL Retired Player Settlement Agreement), 5 November 2018, <https://www.nhlconcussionlitigation.com/assets/htmldocuments/NHL-SettlementAgreement.pdf>, Section 2: 'Settlement Benefits'.

in the hundreds rather than thousands, and a sport that generates 'somewhere between US$4 billion and US$5 billion a year', compared to the NFL's US$14 billion per year.[14] The NHL, in step with the NFL, stated: 'Neither this Agreement nor any exhibit, document or instrument delivered hereunder … is intended to be or shall be construed as … an admission or concession by the NHL or any Released Persons of any fault, liability [or] wrongdoing …'.[15]

1.3 Financial arrangements between SGBs and district clubs

District clubs of a major sport in Australia are at best minimally profitable, and may in fact experience a financial loss over the course of a season. For this reason, an SGB is the 'defendant of choice' in matters where damages payments are in the realm of multiple millions of dollars.

A district club's internal income is principally that of gate receipts and sponsorship, while the bulk of its total revenue is provided through monetary grants from the sport's SGB, and payments generated via alcohol and gambling through its local 'licensed club'.[16] It is 'clear', said the court in *News Ltd v Australian Rugby Football League Ltd*, that 'in general, the [district] clubs have been very heavily dependent upon grants (in one form or another) from their associated Leagues clubs. These derive their revenue principally from the sale of food and liquor and from poker machines.'[17]

Expediency does not permit a detailed examination of the interconnected financial arrangements between the SGBs and the district clubs, nor of the clubs' private sources of revenue. But this appendix will now briefly consider one example — that of the National Rugby League's (NRL's) Eastern Suburbs District Rugby League Football Club Limited (the 'Company'), a not-for-profit company limited by guarantee that administers the premier district rugby league team (the Roosters) and several other entities. The Company's 'stated purpose … is to assist generally in the promotion, conduct and propagation of Rugby League Football in the Rugby League Football District of the Eastern Suburbs of Sydney or elsewhere'.[18] The Company receives revenue from the entities that it controls — principally the 'licensed club',

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[14] Michael McCann, 'The Wins and Losses of the NHL's Tentative Concussion Lawsuit Settlement', *Sports Illustrated* (online, 12 November 2018, <https://www.si.com/nhl/2018/11/12/nhl-concussion-lawsuit-settlement-wins-losses-former-players>.

[15] Settlement Agreement between the NHL and Plaintiffs' Counsel (NHL/NHL Retired Player Settlement Agreement), 5 November 2018, Section 13, 'No Admission of Liability', <https://www.nhlconcussionlitigation.com/assets/htmldocuments/NHL-SettlementAgreement.pdf>.

[16] While the district clubs are permitted to engage with sponsors, the terms of their contracts with their SGBs does not permit them to engage commercially with a sponsor that is also the sponsor of the SGB. See further David Thorpe, 'Athlete Product Endorsement as a False and thereby Unreasonable Restraint of Trade' (2012) 9 *Macquarie Journal of Business Law* 181.

[17] *News Ltd v Australian Rugby Football League Ltd* (1996) 139 ALR 193, 233.

[18] Eastern Suburbs District Rugby League Football Club Limited, *2019 Annual Report* (Report 2019) 37.

an independent not-for-profit company that secures most of its income through alcohol and gambling.

According to the Company's 2019 Annual Report,

> [the] net profit of the Company before tax for the year amounted to $8,863 compared with $715,054 for the prior year. This result was achieved after charging $115,162 (2018: $105,710) for depreciation and amortisation and receiving $2,100,000 (2018: $3,280,000) in grants from [the licensed club].[19]

The licensed club and the Company are, of course, separate legal entities such that the provision of grants (except under contractual arrangement) may cease where, for example, those funds would be directed to a purpose other than that intended. Such a purpose could conceivably include the payment of compensation to players for harm caused by the negligence of the Company.

In contrast, the national SGB, as the controller of a sporting enterprise, has access to levels of revenue unavailable to district clubs. It is from these revenue streams that grants to clubs are made. For example, NRL Chief Executive Andrew Abdo said in 2021 that despite the COVID-19 pandemic, '[r]evenues returned to over $550m and we have secured long-term broadcast agreements which will see broadcast investment increase to over $400m a year from 2023'.[20] Just prior to the commencement of the 2022 season, the Chairman of the Australian Rugby League Commission, Peter V'landys, said that rugby league 'recorded a $43.1m surplus last season' and that the sport's 'revenue surged to $575.1m last year, 37 per cent or $155.4m higher than 2020'.[21] Importantly, the SGB provides the bulk of funds to the clubs and players: '[A]n extra $9m in payments was paid to the 16 NRL clubs in October 2021. The total amount of cash grants distributed to the clubs was $239.6m last year.'[22]

To give some context, a comparison can be made between the expense of caring for a CTE sufferer and caring for a person who has incurred spinal cord injury — both being forms of harm that often require long-term care. According to SpinalCord.com, '[m]edical expenses for spinal cord injuries are often well in excess of a million dollars'.[23] Of course, the level of expense reflects the severity of injury and debilitation:

- For the first year, people with high tetraplegia can expect to pay about a million dollars for care. Low tetraplegia produces about $769,000 in medical expenses, while paraplegia costs

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19 Ibid 36.

20 Jasper Bruce, 'Rugby League Reveals Eye-Watering Spike in Revenue after 2021 NRL Season', *NCA Newswire* (online, 28 February 2022) <https://www.pilbaranews.com.au/news/rugby-league-reveals-eye-watering-spike-in-revenue-after-2021-nrl-season-c-5865841>.

21 Ibid.

22 Ibid.

23 'What's the Real Spinal Cord Injury Cost in 2023?', *SpinalCord.com* (Web Page) <https://www.spinalcord.com/blog/what-is-the-real-spinal-cord-injury-cost>.

about $518,000. Injuries that produce incomplete motor function at any level cost an average of $347,000.

- After the first year, costs tend to go down. Spinal cord injury patients with high tetraplegia incur costs of about $184,000 annually, compared to $113,000 for people with low tetraplegia. Paraplegia costs about $69,000 each year …[24]

Also relevant are figures provided by the Australian Government on 'the lifetime cost of each traumatic brain injury in Australia [which] is estimated at $2.5 million for moderate and $4.8 million for severe injuries'.[25] Of course, many who suffer spinal cord injuries live productive commercial lives. In contrast, while CTE sufferers in the early stages of the disease remain highly functional, with increasing debilitation over the latter stages, close and ongoing care is required.[26]

The figures in this section reveal not only the incapacity of the premier district rugby league clubs to meet damages claims for harm such as that caused by CTE, but also the unpredictability of such clubs' profits from year to year and their dependence on the financial support of licensed leagues club.

1.4 Tau accumulation and contact sport

When Dr Bennet Omalu first observed the brain of retired NFL star Mike Webster in 2005, there were no immediate medical indications as to why Webster's mental and physical condition had declined so profoundly since his playing days, approximately 12 years earlier. Much of the brain appeared 'unremarkable' and 'there was no external evidence of recent trauma'.[27] It was not until sections of Webster's brain were stained and viewed microscopically some days later that tau protein was found to have entangled the footballer's neurones and their surrounding blood vessels.[28] Omalu et al stated of the results of Webster's autopsy: 'Chronic traumatic encephalopathy was evident with many diffuse amyloid plaques as well as sparse neurofibrillary tangles and positive neuritic threads in neocortical areas.'[29] The source of Webster's uncharacteristic behaviour, including disappearing for days at a time and attempting

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[24] Ibid.

[25] '$50 Million for Traumatic Brain Injury Medical Research', *Ministers: Department of Health and Aged Care* (Web Page) <https://www.health.gov.au/ministers/the-hon-greg-hunt-mp/media/50-million-for-traumatic-brain-injury-medical-research>.

[26] See Appendix 1 at **[1.6]**: 'The progression of CTE'.

[27] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128, 129

[28] Perhaps of interest, the revelation occurred in Omalu's flat. Omalu habitually stored brain tissue he was studying in his home refrigerator. See Jeanne Marie Laskas, *Concussion* (Random House, 2015), 114.

[29] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128, 131.

to implant lost teeth with superglue,[30] is apparent when one considers that the neocortex is the site of higher brain functions:

> **neocortex**
>
> The newest part of the cerebral cortex, involved in higher functions such as sensory perception, generation of motor commands, spatial reasoning, conscious thought, and in humans, language; consists of gray matter surrounding the deeper white matter of the cerebrum; accounts for approximately 23% of the neurons in brains of human males and 19% of the neurons in human females.[31]

It is worth noting Omalu et al's reference to 'neurofibrillary tangles', the presence of which characterise sport-related encephalopathy and, revealingly, several debilitating non-sport brain disorders such as Alzheimer's disease and parkinsonism. As stated by Hof et al in an article pre-dating Omalu et al's:

> Head trauma has been associated with the occurrence of Alzheimer's disease and plays a clear role in the etiopathogenesis of the boxers encephalopathy referred to as dementia pugilistica. Neurofibrillary tangles (NFT), one of the hallmarks of Alzheimer's disease are observed in very high densities in the brains of former professional boxers suffering from dementia pugilistica.[32]

While tau clusters, as noted, are common to a range of diseases affecting cognitive function, Hof et al continued:

> [T]heir location in CTE is unique — near small blood vessels, and most often near the bottom of sulci, the deep folds in the brains cortex.[33]

Although expressed in somewhat technical medical language, McKee et al's and Omalu et al's descriptions of the appearance of tau in affected brains of athletes, and the processes by which tau forms, are reasonably understandable, at least in respect to the entanglement and restricting of cerebral apparatus. McKee et al described the appearance of tau entanglements in her 2009 meta-study:

> Microscopically, there are extensive tau immunoreactive neurofibrillary tangles, astrocytic tangles, and spindle-shaped and threadlike neurites throughout the brain.[34]

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[30] Jeanne Marie Laskas, *Concussion* (Random House, 2015), 94–7. For full discussion, see Chapter 6: 'CTE and Proof of Actual Harm (Damage)'.

[31] 'Neocortex', *Medical Dictionary* (Web Page) <https://medical-dictionary.thefreedictionary.com/neocortex>.

[32] PR Hof et al, 'Differential Distribution of Neurofibrillary Tangles in the Cerebral Cortex of Dementia Pugilistica and Alzheimer's Disease' (1992) 85 *Acta Neuropathologica* 23, 23.

[33] Ibid 23.

[34] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 709. Astrocytes are '[s]tar-shaped connective tissue cells of the nervous system that link nerve cells to blood vessels and, by wrapping round brain capillaries, help to form the blood-brain barrier … Their function is believed to be nutritional and structural and to be involved in the clearance of neurotransmitters from within the synaptic cleft': 'Astrocytes', Medical Dictionary (Web Page) <https://medical-dictionary.thefreedictionary.com/astrocytes>. Neurofibrillary tangles are 'accumulations of tau protein that are seen

In a later 2013 study, McKee et al described further the damage to the microtubules of axon cells and the release of tau, which 'may become abnormally phosphorylated, misfolded, aggregated and proteolytically cleaved by calpains and caspases, all of which are associated with neurotoxicity'.[35]

The connection between repeated head trauma and an accumulation of CTE-inducing tau had been detailed by Omalu et al in 2005:

> The pathological mechanisms for … delayed post traumatic changes are thought to be biochemical cascades that are induced by cumulative effects of repeated low-grade concussive brain injury … It has been suggested that repeated axonal injury, vascular injury, and ischemia trigger a cascade of molecular events involving derangement of neuronal cytoskeletal metabolism and accumulation of abnormal cytoskeletal proteins; increased expression of amyloid precursor protein; and a subsequent increase in the amyloid fragment, which is deposited in amyloid plaques.[36]

McKee et al stated similarly in a 2016 'medical consensus' article:

> The results [of a blind evaluation by seven neuropathologists of 25 cases of various tauopathies] demonstrated that there was good agreement among the neuropathologists who reviewed the cases … and even better agreement between reviewers and the diagnosis of CTE … Based on these results, the panel defined the pathognomonic lesion of CTE as an accumulation of abnormal hyperphosphorylated tau (p-tau) in neurons and astroglia distributed around small blood vessels at the depths of cortical sulci and in an irregular pattern.[37]

According to Tagge et al, there is a 'mechanistic link' initiated by trauma to the brain which damages micro-blood vessels, causing the leakage of blood and biochemicals that accumulate as phosphorylated tau leading ultimately to CTE:

> Neuropathological examination of teenage athlete brains in the acute-subacute period [one to four months] after closed-head impact injuries[38] and histopathological and ultrastructural analyses of brains from impact-injured mice suggested traumatic microvascular injury and blood–brain barrier

in neurodegenerative diseases': 'Neurofibrillary Tangles', *Neuroscientifically Challenged* (Web Page) <https://neuroscientificallychallenged.com/glossary/neurofibrillary-tangles>.

[35] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43, 60.

[36] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128, 131. 'Amyloid' is defined as 'resembling starch; characterized by starch-like staining properties '[o]ne of a range of proteins deposited in the brain in spongiform encephalopathies': 'Amyloid', Medical Dictionary (Web Page) <https://medical-dictionary.thefreedictionary.com/amyloid>.

[37] Ann McKee et al, 'The First NINDS/NIBIB Consensus Meeting to Define Neuropathological Criteria for the Diagnosis of Chronic Traumatic Encephalopathy' (2016) 131(75) *Acta Neuropathologica* 85, 86.

[38] 'Closed head injury is [a] form of traumatic brain injury where the skull remains intact … [A] closed brain injury … means that nothing directly touches the brain … A closed brain injury can come from a sharp blow in the head that shakes and traumatize the brain. A common example is a concussion. Since it is closed, the extent of the injury cannot be identified immediately by the naked eye': 'Closed Head Injury', *Brain Injury Institute* (Web Page) <https://www.braininjuryinstitute.org/closed-head-injury>.

> disruption as candidate mechanisms linking closed-head impact to acute brain injury and chronic post-traumatic sequelae.[39]

At some point, as yet not known,[40] the number of blows to the brain reaches a threshold where irregular and excessive tau accumulation occurs. Lipton suggests that while 'it is well established that most patients with mild TBI recover fully over time', the body's 'repair mechanisms may be unable to keep pace with the cumulative injury that occurs beyond a degree of exposure'.[41] Lipton's statement is referred to several times in this thesis. It is particularly informing of the gradual process by which CTE develops and affirms the need for athletes to be warned as soon as knowledge is available and, hopefully, before the CTE threshold is reached.

CTE is also associated with several physical changes to the structure of the brain itself, in particular atrophy or 'shrinking': 'Neuropathologically, CTE is characterized by atrophy of the cerebral hemispheres, medial temporal lobe, thalamus, mammillary bodies, and brainstem, with ventricular dilatation and a fenestrated cavum septum pellucidum.'[42]

Of passing interest, the methodologies of staining brain tissue for microscopic examination used by Omalu in 2005 were not available in 1928 when Dr Harrison Martland's explanation for 'punch drunk syndrome' looked to physical harm to the brain, such as 'concussion hemorrhages' and 'hydraulic shock to the neurones by distention of the perineuronal spaces'.[43] Nonetheless, Martland indicated, perhaps intuitively, that the aetiological origins of punch drunk syndrome may not be solely attributable to physical injury to the anatomical structures of the brain, and suggested some yet-to-be-detected cause. He stated:

> [A] replacement gliosis or even a progressive degenerative lesion may be the late manifestations of these former hemorrhages. The late manifestations of punch drunk will be seen chiefly in the neurologic clinics and asylums, and such material will practically fall to the neuropathologist connected with such institutions.[44]

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[39] CA Tagge et al, 'Concussion, Microvascular Injury, and Early Tauopathy in Young Athletes after Impact Head Injury and an Impact Concussion Mouse Model' (2018) 141 *Brain* 422, 436. Footnote added.

[40] ML Lipton et al, 'Soccer Heading is Associated with White Matter Microstructural and Cognitive Abnormalities' (2013) 268(3) *Neuroradiology* 850, 854.

[41] Ibid.

[42] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 709.

[43] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103, 1103, 1105.

[44] Ibid 1107. A gliosis is '[o]vergrowth of the astrocytes in an area of damage in the brain or spinal cord': 'Gliosis', *Medical Dictionary* (Web Page) < https://medical-dictionary.thefreedictionary.com/gliosis>.

1.5 Contact sport and the bio-mechanism of damage

Almost all athletes suffering from CTE, or who had the disease confirmed at autopsy, report multiple episodes of symptomatic concussion, and often several bouts of unconsciousness following blows to the head. Not to underplay the potential seriousness of concussive harm, it should be realised that on some level the word 'concussion' is a convenient means of referring to head trauma in general, particularly in media reports, while medical reports concerned with the cause of CTE use terms such as 'head trauma', 'low-grade concussive brain injury' or 'sub-concussive impacts' rather than frank concussion.

Concussion is

> a disturbance in the brain's ability to acquire and process information. The reduced function of the brain represents damage to nerve cells (neurons). The neurons can be damaged by a direct blow to the head, which causes the brain to rotate and/or move forward and backward.[45]

Recent research, however, suggests that injury to the brain under the site of a blow to the head may also produce detrimental neurological effects. Tagge et al exposed mice to closed-head impact and found that 'impact produces focally-concentrated point loading at the impact site'.[46]

It has long been accepted that concussion is not due to the force of the blow at the point of contact, but rather by the rapid acceleration and deceleration of the head, which causes the brain, 'floating' in cerebral fluid, to strike the sides of the hard, internal skull and to distort in shape, causing 'stretching and tearing of the brain tissue'.[47] The main form of harm is called 'diffuse axonal injury (DAI), which is due to acceleration/deceleration forces that lead to shearing of axons'.[48] Hirad reports that '[t]he acute brain pathology produced by the forces in mTBI and RSHI [repetitive sub-concussive head impacts] is considered to be diffuse axonal injury (DAI), which affects diverse regions of the brain'.[49] These diverse regions of the brain include the brainstem, which incorporates the midbrain. The brainstem is 'biomechanically

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[45] 'Concussion Management Policy', *Brisbane Netball Association*, adopted 9 September 2013 (Web Page) <https://www.pdhpe.net/wp-content/uploads/2015/12/Brisbane-Netball-Association-Concussion-Management-Policy.pdf>.

[46] CA Tagge et al, 'Concussion, Microvascular Injury, and Early Tauopathy in Young Athletes after Impact Head Injury and an Impact Concussion Mouse Model' (2018) 141 *Brain* 422, 451.

[47] K Blennow et al, 'The Neuropathology and Neurobiology of Traumatic Brain Injury' (2012) 76 *Neuron* 886, 888.

[48] Ibid.

[49] Adnan Hirad et al, 'A Common Neural Signature of Brain Injury in Concussion and Subconcussion' (2019) 5(8) *Sci Adv* 1 <https://www.science.org/doi/10.1126/sciadv.aau3460>. Hirad et al noted that their findings were 'agnostic' as to whether 'CTE may start in the frontal lobe', as previous research indicated: Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43.

vulnerable to injury from head hits due to its narrow dimensions and differential sensitivity to rotational loading'.[50]

Tau may accumulate at sites removed from the point of impact. According to McKee et al, the location of tau and astrocytic entanglements 'suggests that the distribution is related to direct mechanical injury from blows to the side or top of the head … Damage to the blood–brain barrier and release of local neurotoxins might explain the tendency toward perivascular nests of tau-immunoreactive [tangles].'[51] This observation was confirmed by later studies, including one by Tagge et al, which stated: '[F]unctional effects of head injury, including neurophysiological responses underpinning concussion, are capable of potentiating structural changes and secondary alterations that lead to further brain injury and neurological sequelae.'[52] Tagge et al found tauopathy in both hemispheres of the cerebral cortex of mice five-and-a-half months after a unilateral impact, commenting: '[T]au proteinopathy detected at this remote time point was present not only at the primary locus of acute brain injury, but also at distant sites in both hemispheres of the brain.'[53]

A key feature of CTE, disturbingly in respect of repetitive MTBI in sport, is that the disease continues to progress decades after the activity that produced traumatic injury has stopped. 'It is most likely that multiple pathological cascades continue to exert their effects throughout the individual's lifetime once they are triggered by repetitive trauma.'[54] Importantly for those sports that are 'contact' (such as basketball), rather than 'collision' (such as rugby), concussion and low-grade brain trauma may nonetheless result without a blow to the head: an indirect impact 'elsewhere on the body with an "impulsive" force that is transmitted to the head' may cause concussion.[55]

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[50] Adnan Hirad et al, 'A Common Neural Signature of Brain Injury in Concussion and Subconcussion' (2019) 5(8) *Science Advances* 1 <https://www.science.org/doi/10.1126/sciadv.aau3460>.

[51] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 731.

[52] CA Tagge et al, 'Concussion, Microvascular Injury, and Early Tauopathy in Young Athletes after Impact Head Injury and an Impact Concussion Mouse Model' (2018) 141 *Brain* 422, 436.

[53] Ibid 447.

[54] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 732.

[55] P McCrory et al, 'Consensus Statement on Concussion in Sport' (Conference Paper, International Conference on Concussion in Sport, Zurich, November 2008) 37. See also 'Concussion Management Policy' adopted 9 September 2013, *Brisbane Netball Association* (Web Page) <https://www.pdhpe.net/wp-content/uploads/2015/12/Brisbane-Netball-Association-Concussion-Management-Policy.pdf>.

1.5.1 Damage to axons

McKee et al's 2013 study confirmed that repetitive brain trauma also damages the axons — the structures through which the nerve cells of the brain communicate — and found that axon damage was present at all stages of CTE development. McKee et al stated:

> [I]t is likely that axonal dysfunction and loss contribute to the production of clinical symptoms, especially in the early stages of CTE when tau pathology is focal and unlikely to account for the headache, attention and concentration loss, and memory difficulties experienced by subjects with stage I or II disease.[56]

McKee et al's research reveals that following tau release, CTE 'begins focally' and spreads 'slowly over decades to involve widespread regions of the neocortex, medial temporal lobe, diencephalon, basal ganglia, brainstem and spinal cord'.[57] Unlike Alzheimer's disease or frontotemporal dementia, 'the clinical course of CTE is slow, progressing at a rate of 11–14 years between pathological stages'.[58]

1.6 The progression of CTE

In almost all cases CTE develops over years and decades as multiple sub-concussive blows accumulate to emerge as symptoms, often well beyond an athlete's retirement age. The delayed development of CTE presents difficulties for litigants in identifying the proper defendant from among the multiple clubs and organisations for which a typical athlete has played over his or her career. To illustrate: what number of blows occurred when the athlete was playing for a junior club, or for his or her first premier club, or for subsequent premier clubs, or when on representative duties? Or, given that CTE is only definitively diagnosable at autopsy, delayed onset also raises the difficulty of proving that, as the athlete ages, symptoms are not those of another cognitive malady, such as dementia.[59] Nonetheless, as argued in Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"' , *in vivo* indicators are now able to prove harm in the form of CTE on the balance of probabilities.

1.6.1 The onset of CTE

CTE is described as possessing 'an insidious onset and gradual course'.[60] With respect to the subjects in Gravett et al's study, while CTE usually emerges later in life, on average eight years after the cessation of play, 'approximately one-third of athletes were reportedly symptomatic

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[56] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43, 61.

[57] Ibid 60.

[58] Ibid 61.

[59] K Pierre et al, 'Chronic Traumatic Encephalopathy: Update on Current Clinical Diagnosis and Management' (2021) 9 *Biomedicines* 414, 415.

[60] BE Gravett et al, 'Potential Late Effect of Sport-related Concussive and Sub-concussive Head Trauma' (2011) 30 *Clinical Journal of Sport Medicine* 179, 183.

at the time of retirement'.[61] There are, however, cases where symptoms of CTE occur during active participation or shortly after retirement. One such case, described as presenting 'very unusual findings in an individual of this age',[62] is that of 27-year-old ex-NFL player Aaron Hernandez, who hanged himself with a bed sheet while serving a life sentence for murder. Boston University's Center for the Study of Traumatic Encephalopathy described Hernandez as suffering from stage III CTE, along with 'early brain atrophy' and other deleterious changes to the structure of the brain.[63]

At the onset of the disease, McKee et al's 2013 study[64] found that memory loss and executive disfunction were the most common clinical symptoms. With regard to 13 subjects with stage IV CTE,

> all developed severe memory loss with dementia during [the course of the disease]. Most subjects also showed profound loss of attention and concentration, executive dysfunction, language difficulties, explosivity, aggressive tendencies, paranoia, depression, gait and visuospatial difficulties. Less common symptoms were impulsivity, dysarthria and parkinsonism; 31% were suicidal at some point [during the course of the disease]. Two of the 13 subjects developed symptoms of MND [motor neurone disease] years after developing cognitive and behavioural abnormalities.[65]

Of course, given that CTE is associated with RHT, the later an athlete retires, the more likely he or she is to develop symptoms while playing. The research does not, however, indicate that a player who retires young because of the effects of concussions or asymptomatic concussion will necessarily develop CTE.

1.6.2 The stages of CTE

In their 2013 study, McKee et al divided CTE into four stages of severity according to criteria such as neurofibrillary tangles, astrocytic tangles and amyloid plaque, along with physical distortions to the brain such as atrophy, changes in colour and alteration to structural appearance. Stained slices of brain matter became increasingly dark at each progressive stage of CTE. McKee et al then associated the stages of CTE with clinical symptoms, including behavioural manifestations.

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[61] Ibid.

[62] Ann McKee MD, quoted in A Kilgore, 'Aaron Hernandez Suffered from the Most Severe CTE Ever Found in a Person His Age', *The Washington Post* (Washington DC, 9 November 2017).

[63] 'BU CTE Center Statement on Aaron Hernandez', *Boston University, Center for the Study of Traumatic Encephalopathy* (Web Page, 21 September 2017) <https://www.bumc.bu.edu/busm/2017/09/21/bu-cte-center-statement-on-aaron-hernandez/>. It may, nonetheless, be noted that Hernandez encountered difficult life experiences that may have contributed to CTE. 'Hernandez grew up a football star in Connecticut and fell in with a rough crowd at age 15, after his father died unexpectedly during a routine operation. He starred at the University of Florida even as off-field trouble, in the form of drugs and violence, dogged him': A Kilgore, 'Aaron Hernandez Suffered from the Most Severe CTE Ever Found in a Person His Age', *The Washington Post* (Washington DC, 9 November 2017).

[64] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43.

[65] Ibid 59.

Eighty-five subjects with a history of repetitive MTBI were examined post-mortem. All but one were males. The subjects ranged in age from 17 to 98 with a mean of 59.5 years, and comprised '80 athletes (22 of whom were also military veterans), three military veterans with no history of contact sports, one civilian who had experienced multiple falls and one individual who engaged in self-injurious repetitive head-banging behaviour'.[66] Evidence of CTE was found in 68 subjects. Their clinical status 'leading up to death [was] determined through post-mortem interviews with next of kin'.[67] In the analysis of 'the clinical symptoms associated with CTE, case selection was restricted to those cases with post-mortem semi-structured family interviews and pathological diagnoses of CTE'.[68] Eighteen age- and gender-matched individuals without a history of repetitive MTBI provided a comparison control group and did not reveal evidence of CTE on autopsy.[69]

According to McKee et al, '[a]lthough the data suggest that CTE pathology is progressive, it remains to be determined whether some individuals are relatively resilient with static or even reversible pathology'.[70] Whether the progression of CTE can be halted or is peculiar to individuals is a matter of legal relevance. For example, a club or governing body may be justified in excluding an athlete from play who experiences early-stage indications of CTE.

As stated by McKee et al, 'the spectrum of hyperphosphorylated tau pathology ranged in severity … thereby allowing a progressive staging of pathology from stages I–IV'.[71] Consequently, the main behavioural traits and clinical symptoms of each CTE stage — those manifestations of CTE most debilitating to the individual sufferer — are recorded below. As changes to the brain, such as the presence of tau entanglements, increase in each progressive stage of CTE it is not necessary to report each variation in precise medical terms.

A Stage I CTE

Seven subjects revealed stage I CTE, marked by 'low densities of neurofibrillary tangles' and 'scattered distorted axonal varicosities in frontal cortex' and 'immunopositive neurites … in the frontal subcortical white matter'.[72] The frontal cortex is an area of the brain engaged in higher level thinking and executive control.

Family interviews and medical record reviews were available in relation to six of the seven subjects. One subject was asymptomatic. Four of the six reported headaches, and loss of

[66] Ibid 48.

[67] Ibid 45.

[68] Ibid 45.

[69] Ibid 43.

[70] Ibid 61.

[71] Ibid 43.

[72] Ibid 51.

attention and concentration. Three reported short-term memory difficulties, aggressive tendencies and depression. Two reported executive disfunction and explosivity. Two suffered from diagnosed post-traumatic stress disorder.[73]

B Stage II CTE

Three of the 14 subjects with stage II CTE were asymptomatic. Depression or mood swings, headaches and short-term memory loss were reported symptoms. Three subjects presented with symptoms of MND. Other symptoms included 'mood lability, explosivity, loss of attention and concentration, short-term memory loss and headache. Less common symptoms included executive disfunction, impulsivity, suicidality and language difficulties.'[74]

C Stage III CTE

Stage III CTE was found in 15 subjects. Family interviews and medical record reviews were available in relation to 12 of these.

> The most common presenting symptoms were memory loss, executive dysfunction, explosivity and difficulty with attention and concentration. Other symptoms frequently found … were depression or mood swings, visuospatial difficulties and aggression. Less common symptoms include impulsivity, apathy, headaches and suicidality. Seventy-five per cent of subjects were considered cognitively impaired. Two subjects developed symptoms of MND after the onset of cognitive or behavioural abnormalities, another developed cognitive changes after the onset of MND.[75]

D Stage IV CTE

Fifteen subjects were considered to have stage IV CTE. As would be expected, this most debilitating form exhibited severe changes to the brain:

> Macroscopic brain changes included atrophy of the cerebral cortex and white matter and marked atrophy of the medial temporal lobe, thalamus, hypothalamus and mammillary body. Mean brain weight was significantly smaller than lower stage CTE … Severe p-tau abnormalities were found widely distributed throughout the cerebrum, diencephalon, basal ganglia, brainstem and spinal cord.[76]

Family interviews and medical record reviews were available in relation to 13 of these subjects, revealing that they were 'uniformly demented' and that most showed signs of paranoia.[77]

McKee et al's four stages of CTE are supported and referenced in many research articles on the condition. Nonetheless, and while emphasising that CTE is a progressive disease, the Second NINDS/NIBIB Consensus Meeting believed that four stages of progression were difficult to discern with precision and recommended that

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[73] Ibid 52.

[74] Ibid 55.

[75] Ibid 56.

[76] Ibid 58.

[77] Ibid 60.

> CTE p-tau pathological severity be designated as 'High CTE' or 'Low CTE.' Low CTE cases are characterized by cortical pathology, whereas high CTE cases have pathology widely distributed in hippocampus, amygdala, entorhinal cortex, thalamus, mammillary bodies and cerebellar dentate nucleus.[78]

There is, though, no legal significance in classifying CTE according to four stages or two stages provided a causation according to law can be established. Obviously, the progression from the initial symptoms to the final macroscopic changes remains the same, while the dividing point between symptoms is merely allocated to a different more definite symptoms — not unlike dividing a cake into two or four pieces does not alter the size or content of the cake.

1.6.3 Death and CTE: Footballers and boxers

McKee et al's 2009 study revealed the age of death, along with the mean and with standard deviation (SD), of subjects suffering from CTE, as verified at autopsy, according to the sport played:

> Five [American] football players … [a]ll died suddenly in middle age (age at death, 36–50 years; mean, 44.0 years; SD, 5.0) and were younger at the time of death compared with boxers with CTE (age at death, 23–91 years; mean, 60.0 years; SD, 15.2). The duration of symptomatic illness was also shorter in the football players (range, 3–10 years; mean, 6.0 years; SD, 2.9) compared with the boxers (range, 5–46 years; mean, 20.6 years; SD, 12.3) … Furthermore, 4 of the 5 experienced tragic deaths, that is, 2 from suicide … one during a high-speed police chase … and another from an accidental gunshot while cleaning his gun.[79]

Gravatt has noted that, '[i]f the affected individual does not die of other causes, full-blown clinical dementia may occur late in the course of the disease'.[80]

With regard to the subjects of McKee et al's 2013 study, the '[m]ean age at death for former National Football League players diagnosed with CTE was 67.1 ± 16.6 years (range 38–98 years)'.[81] Of particular concern, 'CTE usually begins with behaviour and personality changes at mid-life (mean age of onset 44.3 years, range 17–83 ± 12.1 years)'.[82] Notably, 11 per cent of individuals with CTE were asymptomatic, 'with a mean age at death of 59 years (range 26–

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[78] KF Bieniek, 'The Second NINDS/NIBIB Consensus Meeting to Define Neuropathological Criteria for the Diagnosis of Chronic Traumatic Encephalopathy' (2021) 80(3) *Journal of Neuropathology & Experimental Neurology* 210. (The NINDS is the National Institute on Neurological Disorders and Stroke and the NIBIB is the National Institute of Biomedical Imaging and Bioengineering.) The object of the consensus meeting (at 210) was to propose 'a working protocol including a minimum threshold for CTE diagnosis and an algorithm for the assessment of CTE severity as "Low CTE" or "High CTE" for use in future clinical, pathological, and molecular studies'.

[79] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 710.

[80] BE Gravett et al, 'Potential Late Effect of Sport-related Concussive and Sub-concussive Head Trauma' (2011) 30 *Clinical Journal of Sport Medicine* 179, 183.

[81] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43, 61.

[82] Ibid.

87 years)'.[83] In summary, McKee et al 'analysed the brains of 85 individuals with a history of repetitive mild traumatic brain injury and found evidence of CTE in 80%; all males, ranging in age from 17 to 98 years (mean = 59.5 years)'.[84]

1.7 Second impact syndrome

Where an athlete suffers a symptomatic concussion, the administrators of the game, including the referee, coach and other officials in attendance, are duty-bound to take reasonable steps to prevent further harm to an injured player. This is specifically necessary to avoid the fatal condition known as 'second impact syndrome' (SIS). The duty is pronounced where a player is unable to reason as regards his or her best interests, or feels a duty to the team to remain on the field.

SIS 'results from acute, usually fatal, brain swelling that occurs when a second concussion is sustained before complete recovery from a previous concussion that causes vascular congestion and increased intracranial pressure, which may be difficult or impossible to control'.[85] As differently described: 'Second impact syndrome occurs when an individual suffers a symptomatic head injury resulting in concussion symptoms, then before recovering sustains a second impact, developing altered mental status and sometimes loss of consciousness within seconds to minutes of the second hit.'[86] Moreover, the 'second impact can be of smaller magnitude and not directly to the head … and may occur in a routine tackle'.[87] There is some debate as to whether SIS is a distinct medical condition or an exacerbation of an existing concussive state.[88]

The syndrome primarily affects athletic children and young adults.[89] It was first reported in 1973, when two young athletes 'who experienced initial concussive syndromes … subsequently died after a relatively minor second head injury'.[90] The term 'second impact syndrome' was coined in a 1984 description of 'a 19-year-old college [American] football

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[83] Ibid.

[84] Ibid.

[85] 'Brain Injury in Sports', *Brain Injury Resource Center* (Web Page) <http://www.headinjury.com/sports.htm#guidelines>.

[86] LA McLendon et al, 'The Controversial Second Impact Syndrome: A Review of the Literature' (2016) 62 *Paediatric Neurology* 9, 9.

[87] Ibid 9, 12.

[88] Ibid.

[89] P McCrory, 'Does Second Impact Syndrome Exist?' (2001) 11(3) *Clinical Journal of Sport Medicine* 144, 149.

[90] NM Wetjen et al, 'Second Impact Syndrome: Concussion and Second Injury Brain Complications' (2010) 211(4) *Journal of the American College of Surgeons* 553, 553, quoting RL Saunders and RE Harbaugh, 'The Second Impact in Catastrophic Contact-sports Head Trauma' (1984) 252(4) *Journal of the American Medical Association* 538.

player who suffered a head injury with brief loss of consciousness, returned to play, reported a headache, and on the 4th day collapsed, became unresponsive, and died'.[91]

McCrory[92] argued that two distinct mechanisms may cause post-traumatic brain swelling, although their 'precise contribution to the morbidity and mortality of closed head injury remains unknown':

> The first of these is thought to be due to increased cerebral blood volume that in turn is secondary to a failure of cerebral vascular autoregulatory mechanisms … [The second] is due to true cerebral edema. The characteristic feature of this entity is that the cut brain at autopsy 'wept fluid'.[93]

A meta-study conducted by McLendon considered 17 cases — aged 13–23 years — that met the criteria of SIS. Twelve of the 17 individual subjects, all younger than 20 years of age, 'had poor outcomes ranging from death to permanent disability'.[94] Age was a relevant factor: 'four of the five cases with good outcomes were older than 19 years.'[95] McCrory noted that

> [i]n children, given the apparent higher risk of cerebral swelling after minor head injury and the propensity for structural injury (e.g., subdural hematomas) with relatively mild injury, some authors have argued that there may be a different cerebral autoregulatory response to trauma than in adults.[96]

There is medical disagreement as to the exact time that a concussed child remains vulnerable to further head trauma. One proposition is that SIS only eventuates 'when the second hit occurs within a maximum of ten days from the first hit';[97] another holds that 'children may be at risk for second impact syndrome as long as post-concussive symptoms persist after the first hit'.[98] If it is accepted that an athlete, whether child or adult, professional or amateur, wants to get back on the field of play as soon as possible after a head injury, the period of time until he or she is cleared to do so is medically relevant and thereby legally relevant. On the first theory the player may return to play on the 11th day following a concussion, despite continuing to display symptoms of concussion. For those with a purported duty of care — such as a coach,

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91 Ibid.

92 P McCrory, 'Does Second Impact Syndrome Exist?' (2001) 11(3) *Clinical Journal of Sports Medicine* 144.

93 Ibid 146.

94 LA McLendon et al, 'The Controversial Second Impact Syndrome: A Review of the Literature' (2016) 62 *Paediatric Neurology* 9, 11.

95 Ibid.

96 P McCrory, 'Does Second Impact Syndrome Exist?' (2001) 11(3) *Clinical Journal of Sports Medicine* 144, 147.

97 T Bey and B Ostick, 'Second Impact Syndrome' (2009) 10(1) *Western Journal of Emergency Medicine* 6; reported in LA McLendon et al, 'The Controversial Second Impact Syndrome: A Review of the Literature' (2016) 62 *Paediatric Neurology* 9, 12.

98 ME Halstead and KD Walter, 'Sport-related Concussion in Children and Adolescents' (2010) 126 *Paediatrics* 597; reported in LA McLendon et al, 'The Controversial Second Impact Syndrome: A Review of the Literature' (2016) 62 *Paediatric Neurology* 9, 12.

a medico or a governing organisation — the question of a safe to return to play is a matter for consideration and concern.

It can be noted, however, that the incidence of SIS in Australian contact sport appears low. An article in the *Western Journal of Emergency Medicine* called SIS

> a condition so rare that even the frequency of its occurrence is in question. From 1980 to 1993 … 35 probable cases among American football players [were identified]. However, this incidence is called into question by the lack of similar reports from Australian football, despite the high participation rate and a concussive injury rate approximately eight times that of American football.[99]

McCrory referenced an inquiry into SIS in Australian football conducted by the State of Victoria between 1968 and 1999. A total of 25 player deaths were recorded: 'Nine were due to neurotrauma and the remaining 16 deaths were due to other causes.'[100] Of the deaths caused by neurotrauma, none were associated with SIS: 'The nine player deaths due to brain injury were predominantly due to either intracerebral hematoma or traumatic subarachnoid hemorrhage due to vertebral artery trauma.'[101] From the figures compiled by the Victorian study, McCrory concluded:

> For registered players, the crude risk of death from all causes in Australian football is approximately 22 per 240 million player games or 1 per 11 million player games. The risk of death from brain injury is therefore 1 per 30 million player games.[102]

A number of studies, including that of McCrory, propose that SIS is not a distinct condition but rather explained by the 'rare catastrophic complication of single brain impact, namely diffuse cerebral swelling'.[103] In support, McCrory referred to the sport of boxing, where multiple head impacts in a single match do not elevate the risk of SIS. Nonetheless, McCrory does not advocate returning to play until post-concussion symptoms have resolved, on the basis that in contact sports such as football or high-risk sports such as motor racing, the player is a greater risk of injury given diminished reaction times following concussion.[104]

Doubts that SIS is a distinct medical phenomenon persist, and indeed studies continue to quote McCrory's 2001 research while nonetheless expressing a need for vigilance. For example, Engelhardt stated in 2021: 'Taking into account the scarcity of this syndrome, one may question whether athlete-intrinsic features may be involved in at least some cases of SIS.

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[99] T Bey and B Ostick, 'Second Impact Syndrome' (2009) 10(1) *Western Journal of Emergency Medicine* 6, 6. The article drew upon comments from Paul McCrory in 'Does Second Impact Syndrome Exist?' (2001) 11(3) *Clin J Sport Med* 144, 146.

[100] P McCrory, 'Does Second Impact Syndrome Exist?' (2001) 11(3) *Clinical Journal of Sports Medicine* 144, 146. The other causes were 'predominantly unrecognized ischemic or congenital heart disease'.

[101] Ibid 146.

[102] Ibid 146.

[103] Ibid 147. For full discussion on this point, see also LA McLendon et al, 'The Controversial Second Impact Syndrome: A Review of the Literature' (2016) 62 *Paediatric Neurology* 9.

[104] P McCrory, 'Does Second Impact Syndrome Exist?' (2001) 11(3) *Clinical Journal of Sports Medicine* 144, 147.

On a pathophysiological level, many explanations remained unsatisfactory because they were unable to explain all the clinical phenomena and observed lesions.'[105] May's 1922 study observed: 'The management of second impact syndrome is limited due to the limited understanding of the condition, paucity of research, and the controversial nature of the condition', but also stated: 'Though it is a relatively rare condition, physicians should be aware of SIS, and educate patients who have experienced or are at risk of experiencing a head injury, as the syndrome is often deadly.'[106]

The apparent uncertainty regarding the reality of SIS, together with an absence of evidence of mortality in contact sport in Australia, suggest prima facie that as 'an ordinary careful man does not take precautions against every foreseeable risk',[107] a sport may well argue it is 'not negligent in failing to take precautions against a risk of harm'[108] in the form of SIS. Be that as it may, McCrory closed on a note of caution with respect to the care of young people suffering from concussive injury:

> Our current limited knowledge suggests that children and adolescents are at higher risk, and increased clinical vigilance and perhaps more aggressive investigational strategies may be necessary after all head injuries in this age group. Certainly the presence of post-concussive symptoms should be an important factor prompting further medical assessment.[109]

1.8 Conclusion

This appendix introduced the medical condition known as CTE, the aetiology and indicators of which are relevant to claims of negligence brought by athletes who play contact and collision sports in their governing organisation's premier competitions, and who, later in life come to suffer cognitive impairment.

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[105] J Engelhardt et al, 'Second Impact Syndrome. Myth or Reality?' (2021) 67(3) *Neurochirugie* 265.

[106] Todd May et al, 'Second Impact Syndrome' (July 2022) *StatPearls [Internet]*, <https://www.ncbi.nlm.nih.gov/books/NBK448119/>.

[107] *Bolton v Stone* [1951] AC 850, 863 (Lord Oaksey).

[108] See, eg, *Civil Liability Act 2002* (NSW) s 5B(1).

[109] P McCrory, 'Does Second Impact Syndrome Exist?' (2001) 11(3) *Clin J Sport Med* 144, 147. Dr McCrory has been found guilty of plagiarism and several of his articles have been retracted from various journals. The 2022 inquiry into Dr McCrory's plagiarism by the AFL noted this article was subject to an accusation of 'self-plagiarism' by a blog of a Nicholas Brown. The inquiry, for its purposes, ignored the claim stating that it 'we have excluded from this review instances of self-plagiarism.' See *Independent Review of the Work Undertaken by Associate Professor Paul McCrory For or At the Request of the Australian Football League* (17 October 2022) [279]–[281] ('*Quinn Inquiry*').

Appendix 2:
CTE and 'Negativing Influences':
Do They Determine Liability?

2.1 Introduction

The requirement of foreseeability begs the question: was there sufficient information prior to *circa* 2010 that playing contact sport (or the SGB's contact sport) posed a risk of CTE? Or, as this appendix discusses, was there information downplaying or denying CTE as a risk of playing contact sport that an SGB would have been justified in law to have ignored CTE as a risk that ought not have been foreseen?

This appendix is necessary because some reports or articles, principally those published by the Concussion in Sport Group (CISG) and Dr Paul McCrory, a neurologist associated with the Australian Football League (AFL), perhaps indicating that the risk of CTE was exaggerated or implied that there was no risk at all. Consideration is also necessary because this view appears to be favoured by legal commentator, Annette Greenhow, who believes that an 'intra-and inter-professional debate' about 'the extent to which repetitive concussions and sub-concussive impacts contribute to long term harm'[1] arose in the 2000s and represents an argument that cannot be left unaddressed by plaintiff athletes. To revisit the base legal principle as stated in *Roe v Minister of Health*: 'There is no duty of care owed to a person when you could not reasonably foresee that he might be injured by your conduct.'[2]

2.1.1 This appendix proceeds as follows

Section 2.2: CTE 'denialism'

This section gives an overview of claims that CTE is not a 'real' disease, along with general responses to such claims by medicos associated with the detection and treatment of CTE.

Section 2.3: The historical record countering an overreaction to doubt

This section introduces the argument that denials that RHT in playing Australian contact sports causes CTE should be met with scepticism and doubt

1 A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024) 108.

2 [1954] 2 QB 66, 94.

Section 2.4: The CISG Consensus Statements

The influential CISG is said by some commentators to have questioned the reality of CTE or at least to have downplayed its risk. This section examines these claims, with a view to showing that parsing of the CISG's publications indicates no such denial.

Section 2.5: Dr McCrory: Expressing doubt as to CTE

Dr Paul McCrory, a neurologist with the AFL, is said to have denied that CTE is a real disease. This section explores this claim to show that McCrory's statements, while 'carefully worded', cannot be said to repudiate the actuality of CTE.

Section 2.6: The 'medicalisation' of CTE: A sociological view

What can be referred to as sociological commentary is said by some legal commentators to have been 'medicalised', creating a 'friction' between different entities responsible for the detection and treatment of CTE. This section questions the validity of such claims.

Section 2.7: Conclusion

This appendix concludes by suggesting that a claimed denial of CTE by some commentators is overstated and cannot support a claim that CTE though RHT was not foreseeable.

2.2 CTE 'denialism'

The term 'CTE denialism' is used by Dr Michael Buckland to describe the concept that 'despite the very large body of evidence linking [RHT] to later life decline, significant scepticism and confusion remains, particularly around CTE and its relationship to sports-related [RHT]'.[3] The comments of Buckland and others serve to add credence to the argument that sections of the CISG Consensus Statements, some comments of Dr McCory and other commentators that questioned the reality of CTE as a disease or the manner of its medical causation through RHT does not, considering the historical reporting of CTE, give warrant for SGBs to ignore the obligation to warn of the risk posed by CTE to their premier players.

Buckland said the 'debate' was similar to the controversy and ensuing doubt, promoted by tobacco companies with the intention of protecting their businesses, that tobacco use was a cause of lung cancer. With CTE, the debate is said to originate with lucrative sports where a

[3] Michael Buckland et al, 'Chronic Traumatic Encephalopathy as a Preventable Environmental Disease' (2022) 13 *Frontiers in Neurology* 1, 4. Associate Prof Dr Buckland is the Executive Director and Founding Director of the Australian Sports Brain Bank which was established in 2018 by the Neuropathology Department at RPA Hospital Sydney in partnership with the Brain and Mind Centre at the University of Sydney, and the Concussion Legacy Foundation in the USA. He is also a senior neuropathologist and the Head of the Department of Neuropathology at Royal Prince Alfred Hospital, Head of the Molecular Neuropathology Program at the Brain & Mind Centre, University of Sydney, and Co-Director of the Multiple Sclerosis Research Australia Brain Bank: 'About Us', *Australian Sports Brain Bank* (Web Page) <https://www.brainbank.org.au/about-us/>.

threat to the health of players 'threatens personal and commercial interests'.[4] Continuing, Buckland said that views that transmit denialism

> do not seriously consider the possibility that RHI (repetitive head injury) has the potential to result in serious damage, and that CTE may contribute significantly to this. The possibility that prominent, popular, and lucrative sports might pose a risk to health threatens personal and commercial interests.[5]

Casper also commented on the myopia of CTE denialism apparent in the CISG Consensus Statements: the 'process creating these documents has been narrow, compromised, and flawed' and 'almost certainly avoided the candour required for informed consent to be complete and frank'. Further, these were 'dominated by individuals with close relationships to professional and amateur sports organizations' and 'sought to emphasize all that we do not yet know rather than all that we do know, a pattern that was first established in concussion research for sports by the [National Football League Mild Traumatic Brain Injury] Committee'.[6]

Another neurologist concerned with the treatment of CTE, Dr Rowena Mobbs, responded as follows at a public meeting in 2024 to the question, 'What do you say to those who say CTE is not real?':

> CTE symptoms, or traumatic encephalopathy syndrome under research terminology, I believe is an identifiable condition during life. It shows unique characteristics clinically and radiologically that allow us to follow a process of ruling in the features of CTE as well as ruling out other subtypes of dementia or mimics. It's a slow, inexorable path of decline. I see CTE patients day in day out and treat the symptoms of memory loss and emotional dysregulation like I would any subtype of dementia. Anyone who says it doesn't exist should spend a day in my clinic.

Proof of infliction of actual harm is a question of fact and inference on the balance of probabilities.[7] Any doubt as to the reality of the risk of CTE as a disease caused by RHT is difficult to comprehend objectively. In simple terms, CTE has long been a known outcome of RHT in boxing, in contact sports including rugby and football, as a consequence of 'head banging syndrome', of repeated military blast exposure, and most of all a disease confirmed at autopsy.[8]

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4 Michael Buckland et al, 'Chronic Traumatic Encephalopathy as a Preventable Environmental Disease' (2022) 13 *Frontiers in Neurology* 1, 4.

5 Ibid.

6 ST Casper et al, 'Toward Complete, Candid, and Unbiased International Consensus Statements on Concussion in Sport' (2021) 49(3) *Journal of Law, Medicine & Ethics* 372.

7 For full discussion, see Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"'.

8 Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43; Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709, 811.

2.3 The historical record countering an overreaction to doubt

For those familiar with the plethora of medical reports linking CTE with RHT in boxing, and then from the 1970s revealing CTE as a risk across a range of contact sports, an absolute denial that RHT in, say, AFL could be a cause of CTE should be met with scepticism and doubt.[9] The common link of repetitive blows to the head in all sports is not to be explained away by claims of 'methodological flaws' where the SGB, perhaps informed through its associated medical practitioners, is notified of athletes who display symptoms of CTE, whether described as 'punch drunk' or by some other nomenclature. In this sense the pre-1990s to 2000s international studies on CTE provide a bulwark against an overreaction to the CISG Consensus Statements. For the historically informed, the statements of McCrory and the CISG provide no cause to withdraw or shrink from taking reasonable steps to be fully informed and to provide warnings or protocols to ameliorate the risk of CTE to premier players. The origin of commentary dismissing CTE as a legal risk of contact sport may well arise where the researcher starts at the McCrory and CISG articles without the benefit of a deep knowledge of the disease, its causes and progression which is provided through the historic record, and the balanced reasoning of comparative research.

2.4 The CISG Consensus Statements

The Consensus Statements of the CISG are said to be an influential voice downplaying the risk of CTE through head trauma in sport.[10] This is certainly true with respect to the diagnosis and treatment of symptomatic concussion and related concerns, most tellingly in the dissemination of the CISG Sport Concussion Assessment Tool. Whether from a legal perspective the Consensus Statements can be said to have overwhelmed SGBs with doubt to the point that the risk of CTE in playing contact sport was denied, is an entirely different question.

In short, the Consensus Statements cover several areas associated with head trauma, and some, such as CTE, are given scant treatment whereby somewhat statements are substituted for analysis. In addition, each Consensus Statement does not assign equal weight in expressing doubt as to CTE — in fact, some Consensus Statements clearly indicate that CTE is a factual risk of playing contact sport.

The Consensus Statements are published reports of the 'Concussion in Sports Symposium', which was initially organised by the International Ice Hockey Federation, the Fédération Internationale de Football Association Medical Assessment and Research Centre, and the

[9] For further discussion, see Chapter 2: 'The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments'; Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times'.

[10] A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024) chapter 4.

International Olympic Committee Medical Commission.[11] Six Consensus Statements (two of which are strictly Summary and Agreement Statements) have been published in easily accessible professional journals, including the *British Journal of Sports Medicine*, the *Australian Journal of Science and Medicine in Sport* and the *Journal of Clinical Neuroscience*. McCrory, a neurologist who was then associated with the AFL, co-authored the first five Consensus Statements, but was not an author of the sixth.[12]

2.4.1 What Consensus Statements were published within the critical time frame?

Six Consensus Statements were published between 2002 and 2022. An initial point is that many contact sport athletes, if not most, who today report symptoms of CTE played prior to the publication of the first CISG Consensus Statement in 2002. As such, a downplaying of the risk of CTE in this publication cannot have influenced an SGB prior to 2002 to ignore the risk of CTE. Nor, in turn, can it have caused SGBs to fail to warn of the risk of CTE. As this thesis is primarily concerned with a risk of CTE prior to a judicial decision as to when it became 'obvious' for purposes of state civil liability legislation (suggested as *circa* 2010) no Consensus Statement after that date is material in downplaying or denying the risk of CTE.

2.4.2 As against the historical record, are the Consensus Statements truly influential in law?

A study of the development of understanding CTE, through medical journals dating back to Martland's 1928 study in *The Journal of the American Medical Association*[13] up until Omalu et al's[14] and McKee et al's[15] studies in 2005 and 2009 respectively, provides a lens through which to question the efficacy of the CISG Consensus Statements. As old as Martland's publication is, it is part of the provenance of CTE and is remarkably in sync with the modern descriptions of CTE and its cause. For decade after decade, hundreds of medical publications affirmed the essential nature of CTE and its causes. So pronounced is the historical record that the later expressions of doubt in the Consensus Statements (and those of McCrory, discussed further below in **[2.5]**) should have little impact on an SGB's realisation of the need to warn its premier players of the risk of CTE or to introduce other ameliorating steps, irrespective of the

[11] M Aubry et al, 'Summary and Agreement Statement of the First International Conference on Concussion in Sport, Vienna 2001' (2002) 36(1) *British Journal of Sports Medicine* 6.

[12] His non-authorship followed a plagiarism scandal: see **[2.5]** below.

[13] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

[14] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[15] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

less-than-concrete scientific evidence expressed in some Consensus Statements.[16] The legal standard of care is a proactive obligation to warn where a risk of harm is foreseeable, rather than certain.[17]

2.4.3 The Consensus Statements' use of qualifying words

The Consensus Statements almost always use qualifying or non-committal words that do not amount to an outright denial of CTE. These words should not, therefore, be taken to have conveyed an outright denial of the risk of CTE in contact sports, and cannot thereby excuse the failure of an SGB to inquire into the risk of CTE and to warn of that risk.

For example, the 2001 Consensus Statement refers to the gene apolipoprotein E e4 (ApoE4) as being a 'risk factor' for the development of CTE in boxers. Thus, while isolated to boxing, a disease called CTE is nonetheless expressed as real in that Consensus Statement.[18]

2.4.4 Is there really disagreement?

Despite claims that the Consensus Statements deny CTE as a risk of contact sport, many passages within the Statements in fact affirm such a risk of harm, albeit at times qualified.

The 2008 Consensus Statement, for example, records CTE as a distinct disease. Under the heading 'Chronic Traumatic Encephalopathy', it states:

> Epidemiological studies have suggested an association between repeated sports concussions during a career and late life cognitive impairment. Similarly, case reports have noted anecdotal cases where neuro-pathological evidence of chronic traumatic encephalopathy was observed in retired football players. Panel discussion was held and no consensus was reached on the significance of such observations at this stage. Clinicians need to be mindful of the potential for long-term problems in the management of all athletes.[19]

Epidemiological studies are a recognised form of legal evidence. As said by Spigelman CJ: 'In my opinion, evidence of possibility, including epidemiological studies, should be regarded as circumstantial evidence which may, alone or in combination with other evidence, establish causation in a specific case.'[20] Moreover, his Honour stated that the science of epidemiological causation is not that of legal causation: 'When assessing expert evidence on causation, the legal concept of causation requires the court to approach the matter in a distinctively different

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[16] For discussion, see Chapter 4: 'CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care'.

[17] See Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"'.

[18] M Aubry et al, 'Summary and Agreement Statement of the First International Conference on Concussion in Sport, Vienna 2001' (2002) 36(1) *British Journal of Sports Medicine* 6, 8.

[19] Ibid. 41.

[20] *Seltsam Pty Ltd v McGuiness* [2000] NSWCA 29, [89].

manner from that which may be appropriate in either philosophy or science, including the science of epidemiology.'[21]

To be 'mindful of the potential for long-term problems' is far from a denial. Nonetheless the words '*suggested* an association' and 'anecdotal cases' are qualifications — and also accurate descriptors.

The 2012 Consensus Statement records CTE as a distinct disease and states that the question of cause and effect is open:

> [I]t was agreed that chronic traumatic encephalopathy (CTE) represents a distinct tauopathy with an unknown incidence in athletic populations. It was further agreed that a cause and effect relationship has not as yet been demonstrated between CTE and concussions or exposure to contact sports. At present, the interpretation of causation in the modern CTE case studies should proceed cautiously.[22]

Under the heading 'What is the evidence for chronic concussion-related changes?' are the following qualifying words:

> At present, there are no published epidemiological, cohort or prospective studies relating to modern CTE. … As such, the speculation that repeated concussion or sub-concussive impacts cause CTE remains unproven. The extent to which age-related changes, psychiatric or mental health illness, alcohol/drug use or co-existing medical or dementing illnesses contribute to this process is largely unaccounted for in the published literature.[23]

Although described as 'speculation', the question of a causative connection between RHT and CTE is left open, and the possibility of other causes of symptoms is said to be 'largely unaccounted for'; furthermore, this latter phrase is itself is narrowed to the 'published literature'. For those familiar with the decades of medical publications of the twentieth century, the symptoms of 'modern CTE' (a term used in the 2012 Consensus Statement) coincide with many of the symptoms and sequelae reported in the twentieth century.

Not without apparent irony, the CISG possesses doubts that CTE is caused by RHT in playing contact sport because 'there are no published epidemiological, cohort or prospective studies relating to modern CTE',[24] and yet has no problem 'agreeing', in the absence of similar reports, that CTE is 'not related to concussions alone or simply exposure to contact sport'.[25]

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21 Ibid [142].

22 P McCrory et al, 'Consensus Statement on Concussion in Sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012' (2013) 47(5) *British Journal of Sports Medicine* 250, 257.

23 Ibid 257.

24 Ibid.257

25 Ibid 258.

If 'disagreement' is taken to mean opposing positions on a given point, rather than an expression of doubt, then it is not likely that the 2016 Consensus Statement[26] can be taken as denying that CTE is caused by RHT incurred in playing contact sport. This Statement records that '[t]he literature on neurobehavioral sequelae and long-term consequences of exposure to recurrent head trauma is inconsistent'.[27] It then suggests that clinicians should be mindful of potential long-term problems such as cognitive impairment, but goes on to state that '[h]owever' — a word used to express an alternative possibility or a contrasting view — 'there is much more to learn about the potential cause-and-effect relationships of repetitive head-impact exposure and concussions'.[28] Here, RHT is recognisable as a 'potential' cause of CTE, although this is not expressly stated. The Statement (then says that CTE 'must be a consideration, as this condition appears to represent a distinct tauopathy with an unknown incidence in athletic populations'.[29] It goes on to state that a 'cause-and-effect relationship has not yet been demonstrated', such that 'the notion that repeated concussion or sub-concussive impacts cause CTE remains unknown'.[30] These statements, are again, not inaccurate.

To find areas of agreement between those argued to deny or qualify the risk of CTE in contact sport and those who do not, minimises the claim that SGBs were not required to warn of the risk of CTE because 'influential reports', such as the CISG Consensus Statements, denied such a risk. In fact, what is said to be 'a friction' or 'tension' between the 'Concussion in Sport Group (CiSG) affiliated with sporting organisations, and other research groups with no connections to sporting organisations'[31] underpins legal commentator Greenhow's contention of 'intra- and inter-professional debate'.

The 2016 Consensus Statement is not oppositional or in disagreement with, say, McKee et al's stated position regarding RHT as a cause of CTE.[32] For example, McKee et al noted in the journal *Brain* that several areas required further investigation and had not been settled, including ascertainment bias, confounding drug abuse, and the degree to which pathology is responsible to clinical presentation. Irrespective of possible confounding events, McKee et al explained that 'CTE is a unique neurodegenerative condition that is associated with repetitive mild traumatic brain injury'.[33]

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[26] P McCrory et al, 'Consensus Statement on Concussion in Sport — The 5th International Conference on Concussion in Sport held in Berlin, October 2016' (2017) 51(11) *British Journal of Sports Medicine* 838.

[27] Ibid 844.

[28] Ibid.

[29] Ibid.

[30] Ibid.

[31] A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024) 106.

[32] Ann McKee et al, 'The Spectrum of Disease in Chronic Traumatic Encephalopathy' (2013) 136 *Brain* 43.

[33] Ibid 62.

The 2001 Consensus Statement recorded:

> Published studies have demonstrated that ApoE4 is a risk factor for adverse outcome following moderate-to-severe brain injury. Similarly, ApoE4 has been shown to be a risk factor for the development of chronic traumatic encephalopathy in boxers. The significance of ApoE4 in concussion risk or injury outcome is unclear.[34]

In 2004, the Consensus Statement said in words almost identical to the previous report:

> Published studies have shown that ApoE4 is a risk factor for adverse outcome after all levels of brain injury. Similarly ApoE4 has been shown to be a risk factor for the development of chronic traumatic encephalopathy in boxers. The significance of ApoE4 in sports concussion risk or injury outcome is unclear.[35]

Again, this is not a denial of CTE as a possible risk factor.

Although multiple studies had recorded the risk — and actuality — of CTE in contact sport across decades (as noted many times in this thesis) the CISG has confined itself to terms such as 'anecdotal' and 'no consensus'. Again, the Consensus Statements quoted above, while not endorsements of a risk of CTE, indicate that there is such a disease and must be taken as an affirmation.

A revealing feature of the 2008 Consensus Statement, despite the uncertainty with which CTE is characterised, are two references to Dr Omalu et al's 2005 and 2006 research into CTE.[36]

The 2012 Consensus Statement, under the heading 'Chronic Traumatic Encephalopathy', states:

> Clinicians need to be mindful of the potential for long-term problems in the management of all athletes. However, it was agreed that chronic traumatic encephalopathy (CTE) represents a distinct tauopathy with an unknown incidence in athletic populations. It was further agreed that a cause and effect relationship has not as yet been demonstrated between CTE and concussions or exposure to contact sports. At present, the interpretation of causation in the modern CTE case studies should proceed cautiously. It was also recognised that it is important to address the fears of parents/athletes from media pressure related to the possibility of CTE.[37]

This statement is a direct admission that CTE is 'real' and a possible risk of harm in contact sport.

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[34] M Aubry et al, 'Summary and Agreement Statement of the First International Conference on Concussion in Sport, Vienna 2001' (2002) 36(1) *British Journal of Sports Medicine* 6, 9.

[35] P McCrory et al, 'Summary and Agreement Statement of the 2nd International Conference on Concussion in Sport, Prague 2004' (2005) 39(4) *British Journal of Sports Medicine* 196, 201.

[36] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128; Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player: Part II' (2006) 59(5) *Neurosurgery* 1086;

[37] P McCrory et al, 'Consensus Statement on Concussion in Sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012' (2013) 47(5) *British Journal of Sports Medicine* 250, 258.

In summary, the CISG Consensus Statements do not provide permission to an SGB to avoid the implementation of CTE-amelioration protocols.

2.5 McCrory: Expressing doubt as to CTE

Dr Paul McCrory is 'a neurologist, general physician and sports physician with particular expertise in the area of traumatic brain injuries'.[38] From 2011 he was a member of the AFL's Concussion Working Group, the purpose of which was to consider 'the current suite of concussion projects, and identifying which further steps were necessary to ensure [the AFL Research Board is] doing all that [it] can to consolidate the current best practice approach'.[39] The interest here, however, is in the period prior to *circa* 2010 — an approximate date from which CTE is argued to have become an obvious risk of contact sport.

As an introductory note, McCrory in 2002 published an article in the *Journal of Science and Medicine in Sport* titled 'When to retire after concussion?',[40] which made extensive use of the term 'neuromythology'. This article is discussed extensively in this section.

This publication was retracted by the British Medical Journal Group in 2022 because the 'article contains inaccuracies which in our view undermine the reliability of the work.'[41] The fault was a 'an inaccurate quotation in this article' of a 1952 article by Thorndike. McCrory's offence was to use these words:

> [A]fter experiencing "three concussions, which involved loss of consciousness for any period of time, the athlete should be removed from contact sports for the remainder of the season."

In fact, Thorndike did not use these words but said:

> Patients with cerebral concussion that has recurred more than three times or with more than momentary loss of consciousness at any one time should not be exposed to further body-contact trauma.[42]

Curiously, however, as at November 2024 McCrory's article was accessible on the *Journal of Science and Medicine in Sport* website. Furthermore, the article was not listed as 'retracted' on the monitoring service 'Retraction Watch' (https://retractionwatch.com/) as at that date. Certainly, McCrory has had many medical journal articles retracted or withdrawn by their

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[38] *Independent Review of the Work Undertaken by Associate Professor Paul McCrory For or At the Request of the Australian Football League* (17 October 2022) [5] ('*Quinn Inquiry*').

[39] Ibid [200].

[40] P McCrory, 'When to Retire after Concussion?' (2002) 5 (3) *Journal of Science and Medicine in Sport* 169.

[41] 'Retraction: When to Retire after Concussion?', *British Journal of Sports Medicine* (Web Page) <https://bjsm.bmj.com/content/early/2022/10/13/bjsports-35-6-380ret?rss=1>.

[42] A Thorndike, 'Serious Recurrent Injuries of Athletes: Contraindications to Further Competitive Participation' (1952) 247(15) *New England Journal of Medicine* 554, 556.

publishers — up to 17, according to one report — and many more had 'expressions of concern' placed on them.[43]

Irrespective of the retraction, it may be argued by an SGB in attempting to use McCrory's 'neuromythology article' to excuse its failure to warn of the risk of CTE that the article was not retracted until 2022 and was thereby operative in downplaying the risk of CTE in the pre-2010 period.

2.5.1 Are McCrory's comments sufficiently substantive to dismiss CTE a 'real risk' and excuse an SGB's failure to be informed and to warn?

An electronic search of 'chronic traumatic encephalopathy' in the *Journal of Science and Medicine in Sport* yields 16 results, only one of which was published prior to Omalu et al's 2005 article[44] — that of McCrory's '2002 Refshauge Lecture': 'When to Retire after Concussion?'.[45] At a time when studies of concussive harm in the United States were ongoing, deep and extensive, according to McCrory knowledge of how to manage players with concussion in 'Australian sports' remained 'anecdotal'[46] and claims of long-term harm were unpersuasive:

> It is often stated dogmatically in many published papers on concussion, that sustaining several concussions over a sporting career will *necessarily* result in permanent damage. … In fact, the scientific evidence for such a statement in sporting concussive injuries is not compelling.[47]

While in no way defending any possible cryptic subtext to McCrory's statement, the words that several concussions do not 'necessary' result in permanent damage is true — then, as today. In fact, an alert researcher parsing the words of the sentence will understand that the sentence leaves open the possibility of permanent damage.

Consider the article's position on several critical propositions; such as the proposition that one concussion increases the risk of repeat concussions:

> It has become a widely held belief that, having sustained a concussive injury, one is then more prone to future concussive injury. The evidence for this contention is limited at best. … Significant methodological problems flaw this study. Not least is the fact that the authors included cases of

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[43] 'Concussion Researcher McCrory up to 17 Retractions', *Retraction Watch* (Web Page) <https://retractionwatch.com/2024/05/09/concussion-researcher-mccrory-up-to-17-retractions/>.

[44] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[45] P McCrory, 'When to Retire after Concussion?' (2002) 5(3) *Journal of Science and Medicine in Sport* 169. A second article was published in 2009: P McCrory et al, 'Consensus Statement on Concussion in Sport: The 3rd International Conference on Concussion in Sport Held in Zurich, November 2008' (2009) 12(3) *Journal of Science and Medicine in Sport* 342.

[46] P McCrory, 'When to Retire after Concussion?' (2002) 5(3) *Journal of Science and Medicine in Sport* 169, 169.

[47] Ibid 169. Emphasis added. Referring to P McCrory et al, 'Evidence Based Review of Sport Related Concussion — Basic Science' (2001) 11 *Clinical Journal of Sport Medicine* 160.

> catastrophic brain injury. … The player's risk of injury may be increased by utilising dangerous game strategies and illegal tackling techniques.[48]

The subject matter here is concussion — not CTE; and the application is narrowed by the word 'limited' as well as other qualifications.[49]

On the proposition that repeat concussions may cause cumulative damage, McCrory states:

> Apart from boxing-related head injuries, the most widely cited studies of the cumulative effects of concussion have studied patients with injuries sustained in motor vehicle accidents that were severe enough to warrant presentation to hospital. Generally, concussive injuries suffered in collision sports such as football involve lesser degrees of acceleration–deceleration forces than experienced in motor vehicle accidents.[50]

Again, the application is limited. McCrory does not discuss the risks accumulated in 'football', but instead compares injuries sustained in football to those incurred in motor vehicle accidents.

Regarding a retrospective study of retired Scandinavian soccer players with cognitive deficits, McCrory said there were

> significant methodological problems flaw the results. These problems include the lack of pre-injury data, selection bias, lack of observer blinding and inadequate control subjects.[51]

McCrory also found the usefulness of other retrospective studies to be suspect because they involved

> loss of consciousness [that] was associated with evidence of permanent change in fine motor control. The significance of this symptom in isolation from other cognitive domains is questionable.[52]

While McCrory offered the view that the significance of the risk of CTE should be discounted because of 'methodological problems', the fact that the human subjects nonetheless experienced cognitive deficits was real and was linked to two sports, boxing and soccer, where RHT is common.

On the proposition of recovery of neuropsychological function following concussion in sport, McCrory stated:

> In general terms there appears to be clear evidence of neuropsychological deficits during the first week following mild concussive injury … While ongoing *neuropsychological deficits have been reported by some researchers at one-month other workers have reported normal neuropsychological performance*. … [I]n both symptomatic and non-symptomatic concussed individuals when tested

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48 P McCrory, 'When to Retire after Concussion?' (2002) 5(3) *Journal of Science and Medicine in Sport* 169, 171–2. See also S Gerberich et al, 'Concussion Incidences and Severity in Secondary School Varsity Football Players' (1983) 73 *American Journal of Public Health* 1370.

49 P McCrory, 'When to Retire after Concussion?' (2002) 5(3) *Journal of Science and Medicine in Sport* 169, 171–2.

50 Ibid 172–3.

51 Ibid 172.

52 Ibid 173.

> between one and three years' post-injury, 'no overall gross differences on tests of attention and information processing' exist.[53]

This was contrasted with other studies that reported deficits in attentional and information-processing tasks between nine and 22 months post-injury; however, McCrory considered that there were

> a number of methodological issues that may underlie the inconsistencies reported between studies including test selection, different mechanisms of injury and different injury severity.[54]

2.5.2 What did McCrory mean by 'neuromythology'?

Important in assessing of McCrory's article is his well-publicised use of the word 'neuromythology'. Neuromyths are 'commonly held misconceptions about the brain — often generated by a misunderstanding of scientifically established facts'.[55] The word 'neuromythology' first appears in McCrory's article under the heading 'Repeated concussion in an *asymptomatic* athlete'.[56] Here, 'neuromythology' — while perhaps considered by some as a denial of CTE — is technically confined by its context:

> There is no scientific evidence that sustaining several concussions over a sporting career will necessarily result in permanent damage. Part of the neuromythology surrounding concussion is the concept of the 'three strike rule'; namely if an athlete has three concussions then he or she is ruled out of competition for a period of time. On occasions their sport participation is permanently curtailed. It is important to remember that this approach has no scientific validity yet continues to be the rationale underpinning most of the current return to play guidelines.[57]

The word 'necessarily' qualifies the risk of 'permanent damage' — a condition that is not denied, and is used later in the same passage to refer to concussion that 'involved a loss of consciousness'.[58] Interesting, but seemingly without a confirmed reason for its use, is McCrory's comment that a player excluded from all contact sport following three concussions 'may consider a medico-legal appeal that would be impossible to defend in a court of law' in the absence of documented objective evidence.[59]

Further qualifying words limit the impact of the following statement:

> Although difficult for many clinicians to embrace, the current consensus is that an athlete participating in a contact or collision sport may receive a large number of concussive injuries during

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53 Ibid 173–4. Emphasis added.

54 Ibid 174.

55 E Bei et al, 'Neuromyths: Misconceptions about Neurodevelopment by Italian Teachers' (2024) 34 *Trends in Neuroscience and Education* 100219, 1.

56 P McCrory, 'When to Retire after Concussion?' (2002) 5(3) *Journal of Science and Medicine in Sport* 169, 175.

57 Ibid. Emphasis added.

58 Ibid.

59 Ibid.

> his or her career yet there is no evidence that he or she will *necessarily* sustain any neuropathological, clinical or neuropsychological damage.[60]

Knowledge, and indeed the possibility of CTE is recognised in this statement:

> The unstated fear behind the approach above is that an athlete suffering repeated concussions will suffer a gradual cognitive decline similar to the so-called 'punch drunk' syndrome or chronic traumatic encephalopathy (CTE) seen in boxers.[61]

McCrory goes on to state, however:

> Based on published evidence, this fear is largely unfounded and recent developments suggest that the risk of CTE in this setting may be largely genetically based rather than simply a manifestation of repeated concussive injury.[62]

McCrory said that boxing should not be used as a model for cumulative head injury in other contact sports

> since it presents unique risks to the athlete in terms of the frequency of repetitive head trauma. … [S]pecific genetic abnormalities have been reported as the major risk factor for the development of boxing-related chronic traumatic encephalopathy.[63]

This is true, though it is at least arguable (although perhaps doubtful) that the known reality of CTE caused through RHT in boxing should have given an alert duty-holder pause to consider a risk of CTE through RHT in *any* contact sport.

2.6 The 'medicalisation' of CTE: A sociological view

The term 'medicalisation' refers to

> the process through which nonmedical problems become defined and treated as medical problems often requiring medical treatment.[64]

In essence, therefore, the concept of medicalisation in the context in which it is used refers to the process of framing a non-medical issue as a medical issue. But is CTE a non-medical issue? As explored in Chapter 2: 'The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments', CTE is a well-documented neurological disease with

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60 Ibid 176. Emphasis added.

61 Ibid 175.

62 Ibid.

63 Ibid 172.

64 'Medicalization', *ScienceDirect*, (Web Page), <https://www.sciencedirect.com/topics/social-sciences/medicalization>. The term is often used pejoratively, as in the 'medicalisation of weight loss' or the 'medicalisation of aging'. On the positive side, it may be applied when research discovers that a condition thought to be benign is in fact a symptom of a disease, such that the medicalisation of a condition may result in research, diagnostic techniques and treatment.

identifiable symptoms and sequelae, which leads to death. To propose otherwise is, paradoxically, 'medicalisation in reverse'.

The notion that CTE had been 'medicalised' in a form of *legal* context seems to originate (and not extend beyond) publications of legal commentator Annette Greenhow.[65]

In a 2024 publication describing 'concussion-related concerns',[66] Greenhow highlights what are referred to as 'medical frictions' and 'polarised debates' between two groups of medical researchers, each holding distinct views on the cause of CTE. According to Greenhow, these differing perspectives have contributed to uncertainty among SGBs regarding whether RHT is a definitive cause of CTE. Greenhow further notes that the resulting tensions 'have divided the views of the Concussion in Sport Group (CiSG) affiliated with sporting organisations, and other research groups with no connections to sporting organisations'.[67]

Of these two groups, the first nominated by Greenhow comprised medicos associated with major sporting organisations, such as the National Football League (NFL) in the United States and the AFL in Australia. Greenhow notes that this 'affiliated group' of medical researchers has raised doubts as to whether CTE is caused by RHT and maintains that

> a 'cause-and-effect' relationship between CTE and sport-related concussion is yet to be established, identifying that there is 'much more to learn' about the potential relationship.[68]

The second group, which Greenhow refers to as the 'unaffiliated group', believes that 'CTE occurs *because* of repetitive mild traumatic brain injury'.[69] Why the 'unaffiliated group'? Because, as Greenhow states, this group 'has no connection to sporting organisations'.[70]

A critical yet implicit question arises: Do the 'affiliated' medical practitioners — or the SGBs with which they are associated — benefit from delaying, denying or questioning the link between 'their' contact sport and CTE?

Importantly, within the Greenhow schema, the term 'medicalisation' is explicitly applied to CTE with the assertion that the medicalisation of concussion has created 'friction' — 'especially about sport and longer-term neurological disease'.[71]

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65 Annette Greenhow is an Assistant Professor at Bond University Law Faculty. This is a truncated version of a longer section reduced for reasons of word limitations.

66 A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024). The publication is based on Greenhow's 'PhD research and findings in 2018 and updated to 2023': at xvii.

67 Ibid 106.

68 Ibid 112.

69 Ibid 113. Emphasis added.

70 Ibid 106.

71 Ibid.

Considering the proactive obligations of a duty-holder, if the 'conflict' between the two medical groups is so profound that reasonable individuals are not able to determine whether the risk of CTE warrants an SGB-issued warning, could an SGB justifiably withhold such a warning from athletes in its premier-level competition? Given the obligation of duty-holders to warn of risks of harm and the damaging symptoms of CTE, the answer is a categorical 'no'.

Even if one takes a very broad definition of 'medicalisation' as, say, a genuine uncertainty within the medical community that could create a situation where SGBs face legitimate difficulties in determining the appropriate course of action, it does not address the legal obligation of a duty-holder to warn of a serious risk of harm that is foreseeable rather than certain. Further, where the duty-holder relies on information flowing from a third party, it must take reasonable steps to ensure a system is in place to promote that that conveyance.

Almost all argument denying CTE as a 'real disease' is based on a proposition that proof must be provided to the 'scientific standard'. The legal concern, which is the subject of this thesis, is concerned with the legal standard of proof:

> The court's response to uncertainty arising from the absence of knowledge must be different from that of the medical practitioner or scientist. …
>
> The central question is whether it had been shown to me more probable than not that exposure to asbestos was a cause of [the victim's] death.[72]

The basis of argument to this thesis is very different to that of Greenhow's thesis, where the scientific standard appears more suited.[73] Greenhow's position *seems* to progress along the following lines: The 'medicalisation of concussion' caused two groups of medicos/scientists, the 'affiliated' and 'unaffiliated', to enter into 'conflict' or hold 'polarised views'. This denied SGBs the ability to comprehend or resolve the impasse, and necessitated government engagement mandating that 'both state and non-state actors'[74] take collective action and, in so doing, ensure that the 'precautionary principle' is recognised and applied.[75] The reference to the 'precautionary principle' is, states Greenhow, necessary because such 'standards of evidence should underpin decision-making in this complex and evolving landscape'.[76]

In these circumstances, the opposing views, the suggested 'conflict', may only be resolvable through reference to the scientific standard of proof. It is submitted that the notion of 'medicalisation of CTE' may well be argued as relevant to Greenhow's theory which deals in part with the 'precautionary principle', 'concussion related concerns' and the diagnosis of concussion, the proposition being that what was once treated on the sideline is not treated

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72 *Amaca Pty Ltd v Ellis* (2010) 240 CLR 111, [6].

73 A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024).

74 Ibid 105.

75 It is recommended that, to fully comprehend the Greenhow thesis, an individual should read at a minimum Chapters 3 and 4 of it before considering and judging the summary rendition provided here.

76 A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024) 105 [4.3].

medically. But the concept does not apply to the obligation of an SGB to warn its premier players of the risk of CTE. The reasons are discussed below.

2.6.1 To what extent can medicalisation be taken to have contributed to the 'friction' between the relevant groups of medicos?

In discussing the medicalisation of CTE, Greenhow mainly refers to publications of sociologist Dominic Malcolm. Malcolm defines 'medicalisation' as 'the process whereby, "more and more of everyday life has come under medical domain, influence and supervision".'[77]

According to Greenhow,

> Malcolm *explains* how an analysis of the medicalisation process of concussion in a sports context created 'friction' between sports medicine and neurology/neuroscience, especially about sport and longer-term neurological disease. [78]

The reference to Malcolm is not completely correct. What Malcolm in fact says, rather, is this:

> [I]n relation to concussion and CTE, for instance, we see *elements of friction* between the sports medicine community and that of neurology and neuroscience ...[79]

There is, of course, a difference in application between a 'friction' expressed in absolute terms and '*elements* of friction'. The word 'element' can be defined as 'one of the parts of a compound or complex whole … and often connotes irreducible simplicity', or as a substance 'that cannot be separated by ordinary chemical means into simpler substances', or as 'a distinct group within a larger group or community'.[80] The fact that 'medicalisation' is said to comprise an 'element' — perhaps a minor part — diminishes any argument that it could justify an SGB's failure to stay informed and issue appropriate warnings to its premier players.

Moreover, it is highly unlikely that a team medical practitioner, or an SGB for that matter, would have engaged with Malcolm's sociological analysis of CTE when deciding the approach to take in relaying information to players. Even if they had, the idea that a medico would

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[77] Dominic Malcolm, 'Concussion, Chronic Traumatic Encephalopathy, and the Medicalization of Sport' in M Ventrusca and M McDonald (eds), *Sociocultural Examinations of Sports Concussions* (Routledge, 2019) 4, referring to I Zola, 'Medicine as an Institution of Social Control' (1972) 20(4) *Sociological Review* 487.

[78] A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024) 83, [3.25]. Emphasis added. This paragraph is largely reproduced verbatim in the following chapter of the publication.

[79] Dominic Malcolm, 'Concussion, Chronic Traumatic Encephalopathy, and the Medicalization of Sport' in M Ventrusca and M McDonald (eds), *Sociocultural Examinations of Sports Concussions* (Routledge, 2019) 26. Emphasis added. The sentence continues: '(see, e.g., Sharp and Jenkins' (2015) call for the largely sport-related term "concussion", to be withdrawn from clinical use and replaced with a single classification system for traumatic brain injury (TBI))', referring to D Sharp and P Jenkins, 'Concussion is Confusing us All' (2015) 15 *Neurology* 172.

[80] 'Element', *Merriam-Webster* (Web Page) <https://www.merriam-webster.com/dictionary/element>.

dismiss the entire threat by reasoning 'since CTE is medicalised, I need not be concerned' or 'I need not warn the players', is entirely unrealistic.

At the very least, Greenhow arguably overstates the notion of friction or overlooks the specified meaning of Malcolm's statement. To the extent that Greenhow's concept of friction relies on Malcolm's assessment of the degree to which CTE has been medicalised, it must be reconsidered. Notably, Greenhow attributes the conflict between polarised views of 'affiliated' and 'non-affiliated' medical practitioners as a form of medicalisation itself. However, this interpretation may oversimplify the underlying tensions, potentially misconceiving the true basis to the divide.

2.6.2 Can 'medicalisation' apply to the disease of CTE — a real medical problem?

It should be immediately apparent from the definitions of 'medicalisation' recorded above that the term is misplaced with respect to CTE — a disease referenced in hundreds of medical journal articles published over the course of several decades of the twentieth century and into the twenty-first century. CTE is not a 'non-medical problem' but — and this cannot be overemphasised, a very real medical concern involving symptoms, sequelae and medical treatment, first noted by Dr Martland in a 1928 publication of *The Journal of the American Medical Association*.

Casper points out that the notion of CTE as a 'medicalised' condition (not a 'real' condition) was unsustainable following the publication of Martland's article:

> [P]aradoxically, having met the high threshold … [to be] termed 'biological specificity' in the making of disease, chronic traumatic encephalopathy resisted its opportunity to escape into medicalization.[81]

Martland's 1928 study indicated the early acceptance by the medical community, at least of the United States, of CTE as a real disease. The clinical presentation of typical symptoms leading to degenerative cognitive and motor processes, Casper noted, was paramount in the recognition of CTE as a disease caused by accumulated blows to the head. He continued:

> Other researchers soon published their own analyses in the 1930s, and still more followed in the 1940s and 1950s. By the 1950s it had become common for scientists to speak of traumatic encephalopathy or chronic traumatic encephalopathy. With the emerging field of neuropathology, more careful microscopic analysis of brain tissue produced evidence that violence resulted in pathological findings matching those seen in senile dementias.[82]

Casper opined that the publication of Martland's article 'stymied the medicalization of the disease' and that 'Martland's translation became an obstacle to medicalization'.[83] Such was the level of recognition of CTE-like harm that the resistance to medicalisation permitted 'the

[81] ST Casper, 'Punch-drunk Slugnuts: Violence and the Vernacular History of Disease' (2022) 113(2) *Isis: A Journal of the History of Science Society* 268.

[82] Ibid 281.

[83] Ibid 281, 284.

similarities to the disease experiences of others with recurrent exposure, particularly those with experiences of intimate partner violence' to be recognised.[84]

Supporting Casper's proposition is a 1929 editorial in the *Journal of the American Medical Association*, referring to Martland's study, which also referred to the full acceptance of CTE as a medical problem — a disease where the 'combination of anthropology and forensic medicine had resulted in "a new era in our knowledge" that produced "reliable details about the nature of the violence concerned in producing the pathological changes in the tissues"'.[85]

2.6.3 'Conflict' regarding the 'cause-and-effect' of CTE — is there an obligation to warn?

In a 2017 publication, Greenhow and Gowthorp refer to the argument about a 'conflict' between different medical/scientific groups affecting the assessment of CTE, stating that although the court-ordered settlement of the NFL players' concussion litigation[86] identified CTE 'as a medical condition',

> the nature, extent, and scope of the long-term risk is an area that has polarised views in the scientific research community.[87]

In support of this claim of 'polarisation', the authors cite the 2012 CISG Consensus Statement, which states that a 'cause and effect relationship' between 'CTE and concussions or exposure to contact sport had not been demonstrated', and that '[t]he CISG also observed that "the speculation that repeated concussion or sub-concussive impact causes CTE remains unproven".[88] In summary, Greenhow and Gowthorp state that

> there are, and continue to be, significant differences of opinion regarding the association between the cumulative effects of sports-related concussion and CTE.[89]

As this is said without objection, the authors are taken to have agreed with the position taken by the Consensus Statement.

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84 Ibid 288.

85 Editorial, 'Concussion of the Brain, or "Punch Drunk"' (26 January 1929) *Journal of the American Medical Association* 314, 314, 315, recorded ibid 281.

86 *In Re National Football League Players' Concussion Injury Litigation*, No 2:12-md-02323-AB, MDL No 2323; District Court of the Eastern District of Pennsylvania.

87 A Greenhow and L Gowthorp, 'Head Injuries and Concussion Issues' in N Schulenkorf and S Frawley (eds), *Critical Issues in Global Sport Management* (Routledge, 2017) 98.

88 Ibid, referring to P McCrory et al, 'Consensus Statement on Concussion in Sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012' (2013) 47(5) *British Journal of Sports Medicine* 250, 258.

89 A Greenhow and L Gowthorp, 'Head Injuries and Concussion Issues' in N Schulenkorf and S Frawley (eds), *Critical Issues in Global Sport Management* (Routledge, 2017) 258.

What is not quoted from the 2012 Consensus Statement — and what arguably alters the impact of Greenhow and Gowthorp's argument with respect to medicalisation as a polarising influence — is the following:

> It was agreed that CTE represents a distinct tauopathy with an unknown incidence in athletic populations.[90]

This is a positive recognition of CTE as a real disease — a 'distinct tauopathy'. The Statement then notes:

> It was further agreed that a cause-and-effect relationship has not *as yet* been demonstrated between CTE and concussions or exposure to contact sports [and that] at present, the interpretation of causation in the modern CTE case studies should proceed cautiously.[91]

As such, there is a 'real disease' known as CTE, though 'cause and effect' by playing contact sport is 'as yet' to be demonstrated — a term suggesting the possibility or probability that such a relationship may be established in the future.

Why, it may be asked, should the interpretation of the causation of CTE proceed 'cautiously'? The answer is because the CISG was not sure, or wished to avoid the pitfalls of making an absolute statement that was incorrect. Regardless, a serious disease perhaps caused through playing contact sport is sufficient, as argued in Chapter 4, to require an SGB to issue a risk warning to its premier players.[92]

As noted above, Greenhow and Gowthorp quote the 2012 Consensus Statement to substantiate their argument of 'polarised views in the scientific research community'.[93] However, in support of the CISG's claim that '[i]t was … agreed that a cause-and-effect relationship has not as yet been demonstrated between CTE and concussions or exposure to contact sports',[94] the same Consensus Statement references a 2005 article published in *Neurosurgery* by Guskiewicz[95] that, rather than denying a risk of CTE-like harm, confirms it:

> Our findings suggest that the onset of dementia-related syndromes may be initiated by repetitive cerebral concussions in professional football players. …

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[90] P McCrory et al, 'Consensus Statement on Concussion in Sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012' (2013) 47(5) *British Journal of Sports Medicine* 250, 255.

[91] Ibid. Emphasis added.

[92] Chapter 4: 'CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care'.

[93] A Greenhow and L Gowthorp, 'Head Injuries and Concussion Issues' in N Schulenkorf and S Frawley (eds), *Critical Issues in Global Sport Management* (Routledge, 2017) 98.

[94] P McCrory et al, 'Consensus Statement on Concussion in Sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012' (2013) 47(5) *British Journal of Sports Medicine* 250, 254.

[95] KM Guskiewicz et al, 'Association Between Recurrent Concussion and Late-life Cognitive Impairment in Retired Professional Football Players' (2005) 57 *Neurosurgery* 719.

> [Our] data suggest that a history of concussion, particularly recurrent concussion, may be a risk factor for the expression of late-life memory impairment, [mild cognitive impairment], and [Alzheimer's disease]. … [R]etired professional football players were found to have a progressive decline in mental health functioning and a higher rate of memory problems and cognitive decline associated with a history of concussion. …
>
> Retired professional football players with three or more concussions were twice as likely to be diagnosed with [mild cognitive impairment] as those with one or two previous concussions, and five times more likely than those with no previous concussions.[96]

Nonetheless, there is an absence of an association between 'concussion' and Alzheimer's disease in the following statement:

> Despite the earlier onset of [Alzheimer's disease], we failed to find an association between previous concussion and lifetime onset of [Alzheimer's disease].[97]

This statement refers to the fact that experiencing several concussions does not necessarily mean that the individual will suffer with CTE in their lifetime — noting here that Alzheimer's disease not uncommonly results from CTE. Similar to the effects of smoking, the assignment of an association is confounded where some people smoke and do not, unlike many others, suffer from tobacco-related smoking disorders.

Overall, one can contemplate whether the dissonance recorded in words taken from the 2012 Consensus Statement such as 'unknown incidence', 'not possible to determine … with any certainty', 'remains unproven', 'largely unaccounted for', 'not yet been demonstrated' and 'proceed cautiously' are reflected in words from Greenhow such as 'significant differences of opinion' and 'polarised views'.

Moreover, sociologist Malcolm says that 'fundamental questions [with regard to medicalisation] might include the consequences of using medicine as a tool of social control or medical professional expansion'.[98] Here, however, Greenhow's insistence upon methodological or scientific standards that cannot be provided because of ethical considerations in fact amounts to the use of 'medicine as a tool of social control.'

The term 'medicalisation' is not appropriately applied to CTE, a real medical condition with identifiable symptoms and a known pathology. The claim that the 'medicalisation' of CTE caused a conflict between different medical groups is vague and there is little evidence to support the contention. As such, the argument that 'methodological flaws' in research justify an SGB not warning of the risk of CTE has no legal standing.

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96 Ibid 719, 722, 723.

97 Ibid 722.

98 Dominic Malcolm, 'Concussion, Chronic Traumatic Encephalopathy, and the Medicalization of Sport' in M Ventrusca and M McDonald (eds), *Sociocultural Examinations of Sports Concussions* (Routledge, 2019) 24, quoting P McCrory et al, 'Consensus Statement on Concussion in Sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012' (2013) 47(5) *British Journal of Sports Medicine* 250, 258.

Malcolm's publication presents an interesting sociological treatise of a societal issue: What is the impact of the claimed medicalisation of concussion on society? An application to issues relating to the liability in negligence of an SGB in failing to warn its premier players of the risk of CTE is not objectively apparent.

2.6.4 A 'scientific application'

In summary, it seems, then, that Greenhow's position is along the following lines: The 'medicalisation of concussion' (which includes CTE) caused two groups of medicos/scientists, the 'affiliated' and 'unaffiliated', to enter into 'conflict' or hold 'polarised views'. This denied SGBs the ability to comprehend or resolve the impasse, and necessitated government engagement mandating that 'both state and non-state actors'[99] take collective action and, in so doing, ensure that the 'precautionary principle' is recognised and applied.[100] The reference to the 'precautionary principle' is, says Greenhow, necessary because such 'standards of evidence should underpin decision-making in this complex and evolving landscape'.[101]

Greenhow's thesis more readily concerns questions relevant to the 'scientific standard'. In these circumstances it may well argued that 'medicalisation' is relevant. It is difficult to see, however, that the term can apply to, or influence, the obligation of an SGB to take reasonable steps to be fully informed of the risk of CTE, which is argued here to include an ongoing and deep understanding of the historical record, and to warn its premier players of the risk of CTE; a real disease.

2.7 Conclusion

This appendix considered whether 'CTE denialism', in context a term referring to publications that intended or appeared to intend to deny that CTE was a real disease or was caused through RHT in the playing of contact sport. The relevance of 'denialism' to this thesis is the argument that these publications so discounted the threat of CTE to the point where it was not a foreseeable risk in playing contact sport. Exemplifying reference was made to the Consensus Statements of the CISG, and to neurologist Dr Paul McCrory (several publications of whom were retracted but would be argued to have been valid in the pre-2010 period). It was argued that while these sources did express reservations, they did not deny the reality of CTE and often supported its reality. Mention was made of a suggested 'medicalisation' of CTE, a proposition that could be argued to apply to symptomatic concussion, but could not apply to CTE, a disease characterised by symptoms, sequelae and ultimately death. The predominant resort of these commentators (and indeed other commentators insistent on adherence to the scientific standard of medical proof) was to 'methodological shortfalls' and, as argued, that

[99] Ibid 105.

[100] It is recommended that, to fully comprehend the Greenhow thesis, an individual should read at a minimum Chapters 3 and 4 of it before considering and judging the summary rendition provided here.

[101] A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024) 105 [4.3].

‘methodological flaws’ in research justify an SGB *not* warning of the risk of CTE as against a real risk of harm has no legal standing.

Questions regarding the objectivity of ‘denialists’ was raised by Dr Buckland on the basis that ‘lucrative sports’ may promote doubt about CTE because a threat to player health ‘threatens personal and commercial interests’. Legal commentator Greenhow referred to those who raised doubts regarding the ‘cause and effect’ of CTE as an ‘affiliated group’ in comparison to an ‘unaffiliated group that has ‘no connection to sporting organisations’.

In summary, there is no doubt that a large volume of material warning of the risk of CTE-like harm caused by RHT in the playing of contact sport had been amassing since Dr Martland’s 1928 publication in the *Journal of the American Medical Association*, dealing initially with boxing and then, from the 1970s, increasingly with commonly played Australia contact sports. The views of the CISG, McCrory do not negate the fact that RHT was recognised as a potential cause of CTE-like symptoms. Conflicting views among medical professionals does not justify an SGB’s failure to warn, especially when there is a known risk of serious harm.

What has been described as ‘denialism’ does not override the legal duty of SGBs to be informed of the risk of CTE and to warn their premier players. The legal standard focuses on foreseeability and the balance of probabilities, not scientific certainty.

Appendix 3:
A Brief Consideration of the Scope of Liability

3.1 Introduction

Satisfaction of legal causation requires a court to determine that it is appropriate that the scope of an SGB's negligence extends to harm in the form of CTE. This appendix is a supplement to Chapter 7: 'Factual Causation of Harm: Failure to Warn and the Athlete's Response Had a Warning Been Given'.

This appendix reviews the appropriateness that the scope of an SGB's liability should extend to a failure to warn of the risk of harm in the form of CTE. The appendix recognises that as the scope of liability does not require foresight of the specific harm caused by a defendant's breach of duty, CTE may be determined as foreseeable as a 'type' of cognitive harm known to occur in contact sport, such as symptomatic concussion or post-concussive trauma or on-field unconsciousness. It is argued, nonetheless, that as most athletes recover from these relatively benign forms of cognitive harm within a few days or weeks, as against the increasingly debilitation of CTE leading to death, 'type' must differentiate between the two forms of harm as a matter of foresight. CTE is more accurately characterised by type as 'a neurodegenerative disease', 'tauopathy' or more generally as 'head trauma leading to death' or 'cognitive harm from which the victim does not recover' is a more suitable in classifying CTE by type.

3.1.1 This appendix proceeds as follows

Section 3.2: The commonality of foreseeability in duty and scope of liability

This section briefly explains 'scope of liability' and its purpose in removing from consideration events that play little or no part in causing harm to the plaintiff, and in so doing accurately attributes legal responsibility.

Section 3.3: The content of the scope of liability

This section examines those matters that are considered relevant in determining a connection between the breach of duty and consequential damage, and evaluates these in respect of athlete affliction with CTE.

Section 3.4: An established class and the question of 'why?'

This section proposes that a failure of an SGB to warn of a material risk of harm falls within its duty of care and is an established class of case. The section argues further that the duty to warn of the risk of CTE falls within the scope of liability of an SGB.

Section 3.5: Genetic predisposition and the 'egg-shell skull' rule

This section addresses the proposition that an athlete's genetic predisposition to CTE, along with the use of anabolic steroids, could influence an assessment of an SGB's scope of liability.

Section 3.6: Conclusion

This section argues that it is appropriate that the scope of liability attaches to an SGB based primarily on the proposition that harm in the form of CTE was a foreseeable consequence of the SGB's breach in failing to warn its premier players of the risk of CTE.

3.1.2 Outline of 'scope of liability'

It is proposed that CTE is more accurately characterised as a unique disease the contraction of which must be shown as foreseeable with respect to an SGB's scope of liability. Foreseeability of this type of harm, or harm that 'ought to be foreseen', is argued to flow from the SGB's medical associates initiated through a system or proactive monitoring of extant and emerging risk of harm in contact sport.

This appendix also considers that suggestions that sufferers of CTE may possess a genetic predisposition are unsustainable under the 'egg-shell skull' rule. Proposals of a link to illicit steroid use by athletes are considered and argued as unlikely to apply in any degree to the Australian experience.

Scope of liability concerns the question of whether a defendant SGB is responsible for harm in the form of CTE consequential to its failure to warn its premier players of the risk of CTE in playing in its premier-level competition.

Liability for harm caused to a plaintiff under state civil liability legislation requires satisfaction of two limbs: that the defendant's negligence was a 'necessary condition' (a factual cause) of the claimed harm and, under a second limb, whether it is 'appropriate for the scope of the negligent person's liability to extend to the harm so caused (scope of liability)'. The purpose of the second limb is to articulate reasons as to why and 'whether the defendant ought to be held liable to pay damages for that harm.'[1]

Section 5D of the *Civil Liability Act 2000* (NSW) relevantly provides:[2]

(1) A determination that negligence caused particular harm comprises the following elements:

(a) that the negligence was a necessary condition of the occurrence of the harm (factual causation), and

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1 *Ruddock v Taylor* (2003) 58 NSWLR 269, [87] (Ipp JA).

2 *Civil Liability Act 2002* (NSW) s 5D (1), (4); *Civil Law (Wrongs) Act 2002* (ACT) s 45(1), (3); *Civil Liability Act 2003* (Qld) s 11(1), (4) (refers to 'breach of duty'); *Civil Liability Act 1936* (SA) s 34(1), (3); *Civil Liability Act 2002* (Tas) s 13(1), (4) (refers to 'breach of duty'); *Wrongs Act 1958* (Vic) s 51(1), (4); *Civil Liability Act 2002* (WA) s 5C(1), (4) (refers to 'fault').

(b) that it is appropriate for the scope of the negligent person's liability to extend to the harm so caused (scope of liability).

…

(4) For the purpose of determining the scope of liability, the court is to consider (amongst other relevant things) whether or not and why responsibility for the harm should be imposed on the negligent party

The *Ipp Report* referred to scope of liability as the 'ultimate question'; that is, 'should the defendant be held liable for any of the harmful consequences of the negligence and if so, for which?'[3] Stapleton described the question as 'whether, given the breach did contribute to the result, the features of that connection between breach and result are, in the context of the relevant legal rule, such that the defendant should be legally responsible for it'.[4]

The process in a failure to warn case, according to Stapleton, proceeds from establishing breach as a factual cause of harm after which 'we ask the scope question: does this consequence of the breach come within the appropriate scope of liability for breach of the relevant legal rule, a duty of care?', stating further that the harm in question 'must be of a type of harm that was a foreseeable consequence of the breach'.[5]

3.1.3 The relevance of the common law

Within the sweep of civil liability legislation, the common law remains relevant to the question of scope of liability. Where the case falls into an 'established class' the 'normative question posed', said the High Court, is 'properly answered by a court through the application of precedent. Section 5D guides but does not displace common law methodology. The common law method is that a policy choice once made is maintained unless confronted and overruled.'[6]

3.2 The commonality of foreseeability in duty and scope of liability

While foreseeability is a common feature in determining both duty of care and liability for causation of harm, the function of each differs. As noted by the High Court of Australia, foreseeability is forward looking to impose a rule of conduct, while causation of damage is backward looking to attribute responsibility. The policy considerations of each 'serve' different functions' such that policy may differ between the two.[7]

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3 *Review of the Law of Negligence* (Final Report, September 2002) [7.41] ('*Ipp Report*').

4 Jane Stapleton, 'Reflections on Common Sense Causation in Australia' in S Degeling and J Edelman (eds), *Torts in Commercial Law* (Thompson Reuters Australia, 2011) 354.

5 Ibid.

6 *Wallace v Kam* (2013) 250 CLR 375, [22].

7 Ibid [26].

3.2.1 The rationale of 'scope of liability'

The purpose of establishing a connection between breach (say, by failure to warn) and harm (say the contraction of CTE) is to remove from consideration causes that play little of no part in causing the harm but may be described as merely providing a setting in which the breach occurred. Hayne J explained that in respect to scope of liability that there may be a 'complex mixture of acts or omissions' that are causative of harm, but not every act or omission leads to a conclusion of legal responsibility. Consequently, '[i]t is necessary … to examine the role that is identified by reference to the purpose of the inquiry — the attribution of legal responsibility'.[8] In *Koehler v Cerebos* Callinan J said revealingly of a practice to be avoided: 'With enough imagination and pessimism it is possible to foresee that practically any misadventure, from mishap to catastrophe is just around the corner.'[9]

3.3 The content of the scope of liability

According to the *Ipp Report*:

> '[S]cope of liability' … concerns the normative issue of the appropriate scope of the negligent person's liability for the harm, once it has been established that the negligence was a factual cause of the harm. 'Scope of liability' covers issues, other than factual causation, referred to in terms such as 'legal cause', 'real and effective cause', 'commonsense causation', 'foreseeability' and 'remoteness of damage'.[10]

Stapleton, in considering a 'consequences approach' to determine liability for harm caused, proposed several 'features' or 'rules' that go to establishing a connection 'between the breach and the result': 'that the breach [would] have been necessary for the occurrence of the result; that the result [would] have been of a reasonably foreseeable type of result from the breach; that the result not be a coincidental consequence of the breach; that the result not have flowed from an unreasonable failure of the plaintiff to mitigate the position he found himself in due to the breach; and so on'.[11] The role of an appeal court, said Stapleton, was to 'choose' which rules should govern the scope of liability for the consequences of the breach.[12]

Allsop P in *Zanner v Zanner*[13] provided a list of 'all relevant considerations' that go to establishing the appropriate scope of the negligent person's liability — a list similar to Stapleton's. The case concerned a mother who permitted her 11-year-old son to manoeuvre her

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8 *Pledge v Roads and Traffic Authority* [2004] HCA 13, [10].

9 *Koehler v Cerebos Aust Pty Ltd* (2005) 214 ALR 355, [54].

10 *Review of the Law of Negligence* (Final Report, September 2002) Recommendation 29 ('*Ipp Report*').

11 Jane Stapleton, 'Reflections on Common Sense Causation in Australia' in S Degeling and J Edelman (eds), *Torts in Commercial Law* (Thompson Reuters Australia, 2011) 352.

12 Ibid 353.

13 [2010] NSWCA 343.

car into the family carport while she stood at its entrance. The boy's foot slipped from the brake onto the accelerator causing the car to surge forward and collide with his mother, injuring her.

While not suggesting a limit, the considerations of Allsop P, particularly that of foreseeability, are relevant to discerning the scope of liability in respect to an SGB failure to warn of the risk of CTE:

> Injury to the mother was entirely foreseeable should negligence occur. The scope of the risk of harm protected by the duty and created by the breach included injury to the mother. The injury was not coincidental to the breach. It was the direct and immediate consequence of the negligence. The son in the car ran over his mother. The content of the duty and the attenuated standard of care were directed to the exercise of care to avoid injury to the mother in the very manner that occurred. There was no intervening act of a third party or of an abnormal event.[14]

In the context of an SGB's failure to warn, the pattern of these 'considerations' can be structured along these lines:

- whether the SGB's obligation to warn of the risk of CTE fell within the ambit of its duty of care and the breach of this duty;
- whether harm in the form of CTE was incidental to the breach or a direct consequence of the breach by failure to warn;
- whether the content of the duty of care and the standard of care would have been directed to the avoidance of the injury that occurred; and
- whether there was an intervening act of a third party or abnormal event avoiding the SGB's liability.

3.3.1 Common sense and value judgments

The discernment of scope of liability concerns 'a normative weighing of all the relevant circumstances' such that the question is 'one to be determined by having regard to all the circumstances and then making a value judgment.'[15] Indeed, the use of the word 'appropriate' within the statute suggests the use of judgment. In *Zanner v Zanner*, Tobias JA commented that whether it is appropriate for the scope of a defendant's liability to extend to the harm caused 'is to be considered as a matter of common sense taking into account any relevant policy considerations that might assist in determining whether or not, and why, responsibility for the harm to the respondent should be imposed upon the first appellant'.[16] In *Wallace v Kam*, before the New South Wales Court of Appeal, Allsop P said that 'the enquiry and conclusion under s 5D(1)(b) involve a value judgment', stating further that the 'drawing of a conclusion that a

[14] Ibid [12].

[15] *Harvey v PD* [2004] NSWCA 97, 671 (Ipp JA).

[16] *Zanner v Zanner* [2010] NSWCA 343, [79].

consequence of posited liability would be "unjust", "absurd" or "unacceptable" would be relevant to the conclusion of appropriateness'.[17]

Clearly common sense and value judgments are not singular elements utilised to determine the scope of liability but are, rather, applied in evaluating the relevance of other factors, such as those listed by Allsop P and Stapleton above. In addition, exhortation to common sense and value judgment offer an explanation as to why subtle differences between similar factors relevant to the scope of liability render a judgment of either a positive or negative determination.[18]

3.3.2 Remoteness and reasonable foreseeability of harm

Of the factors propounded by Allsop P in *Zanner v Zanner*, foreseeability of harm is particularly useful in determining the scope of liability of an SGB in respect to CTE.[19] The test of remoteness (the obverse of foreseeability) concerns whether the harm suffered by a plaintiff was a foreseeable result of the defendant's negligence — foreseeability being often transposed to foreseeable risk; a risk that is not far-fetched or fanciful, or a 'possible risk'.[20] More precisely, a 'real risk' of harm is reasonably foreseeable where it 'would occur to the mind of a reasonable man … and which he would not brush aside as far-fetched'.[21] In context, is it foreseeable that the failure of an SGB to warn its premier players of the risk of CTE may cause the plaintiff athlete to suffer harm in the form of CTE?

A Foreseeability of harm as a 'type' or 'class'

A question of major concern for a plaintiff athlete is whether CTE is classifiable as a 'type' of cognitive harm or as a distinct disease. The law on foreseeability of harm by 'type' 'category' or 'class' was explained by Gummow J in *Rosenberg v Percival*:

> The precise and particular character of the injury or the precise sequence of events leading to the injury need not be foreseeable. It is sufficient if the kind or type of injury was foreseeable, even if the extent of the injury was greater than expected.[22]

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17 *Wallace v Kam* [2012] NSWCA 82, [4], [12].

18 For example, the differing determinations in the 'psychiatric harm' cases of *Rowe v McCartney* [1976] 2 NSWLR 72 and *Mount Isa Mines Ltd v Pusey* (1970) 125 CLR 383, discussed further below at **[C]**: 'Can CTE be incorporated into a "type"?'.

19 The obverse of 'foreseeability' is 'remoteness', the prevalence of one suggesting a dearth of the other.

20 *Rosenberg v Percival* (2001) 205 CLR 434, 455 [64]; *Overseas Tankship (UK) Ltd v Miller Steamship Co Pty Ltd (The Wagon Mound (No 2)* [1961] AC 388.

21 *Overseas Tankship (UK) Ltd v Miller Steamship Co Pty Ltd (The Wagon Mound (No 2)* [1967] 1 AC 617, 643 (Privy Council). In this case, the harm through fire was not judged as foreseeable.

22 *Rosenberg v Percival* (2001) 205 CLR 434, 455 [64]. As said by Villa, '[i]t is not necessary that the defendant ought to have foreseen the damage that actually happened, so long as the defendant ought to have foreseen damage of that general kind': D Villa, *Annotated Civil Liability Act 2002 (NSW)* (Lawbook Co, 2004).

In context, does CTE fall within a classification by type of, say, 'neurodegenerative disease', 'cognitive disfunction', 'concussive trauma', 'head trauma' or 'acquired cognitive injury'. Alternatively, is CTE foreseeable as a distinct disease only — that is, as CTE, whether described as *dementia pugilistica*, punch drunk syndrome, or by specific symptoms? For the plaintiff athlete the advantage lies in a categorisation by 'type' where a broad range of variants of severe cognitive harm are included within a foreseeable class of harm. However, as discussed in Chapter 9,[23] should this categorisation broaden to the point where the harm is described as, say, 'concussion', the athlete would be hard pressed to argue that the risk was not 'obvious' for the purpose of state civil liability legislation.

To illustrate this point, in *Hughes v Lord Advocate* the House of Lords held that while exploding gases were not a reasonably foreseeable cause of the plaintiff's burns as against burns from ignited paraffin, the 'distinction drawn between burning and explosion is too fine to warrant acceptance'.[24] In other words, harm by 'type' — in this case by burning — was sufficient to satisfy the question of scope of liability through foreseeability of harm by burning irrespective of its source.

That the precise form of harm need not be foreseen by a defendant raises the question of how unique, or what degree of variability places a particular form of harm beyond foreseeability 'by type'. For example, are all forms of harm associated with playing contact sport foreseeable such that CTE, a form of harm, is also foreseeable? What of harm of a particular kind or to a particular area of the body, such 'head trauma' or 'concussion'? What of a broader classification like 'acquired brain injury'? Or alternatively must the risk of CTE be foreseen precisely? These considerations are discussed below.

B The advantage of foreseeability by 'type' of harm for plaintiff athletes

An athlete plaintiff is advantaged should CTE be adjudged as falling within a broad type of harm, say, 'cognitive disfunction' or 'head trauma', forms of classifying harm that are clearly a foreseeable consequence of playing contact sport. Harm of this 'type' could include 'symptomatic concussion' or 'unconsciousness through knockout' and, as the argument would run, CTE. To illustrate through a syllogism: CTE causes cognitive disfunction. Cognitive disfunction is a foreseeable result of playing contact sport. Since CTE is a form of cognitive disfunction, the risk of CTE is a foreseeable consequence of an SGB's failure to warn. In other words, just as a distinction between burning by explosion and burning by paraffin fire is too fine to warrant a distinction, the difference between frank concussion or cognitive disfunction and CTE is 'too fine to warrant acceptance'.[25] Or, to paraphrase Gummow J, is it sufficient where the 'kind or type of injury' categorised as 'cognitive disfunction' is foreseeable, even if

[23] Chapter 9: 'CTE and the Characterisation of "Risk of Harm": Dangerous Recreational Activity, Assumption of Risk and Other Defences'.

[24] *Hughes v Lord Advocate* [1963] 1 All ER 705, 710 (Lord Jenkins).

[25] Ibid.

the 'extent of the injury was greater than expected'?[26] Under such an argument, 'the precise and particular character of the injury or the precise sequence of events leading to the injury [in the form of CTE] need not be foreseeable'.[27]

Consideration must be given to the characterisation of risk as regards 'obvious risk', and while concussion is an obvious risk in playing contact sport, this chapter argues that the material risk of CTE, or a tauopathy or neurodegenerative disease is not obvious to a reasonable premier contact sport athlete.

C Can CTE be incorporated into a 'type'?

While foreseeability by 'type' is a well-recognised methodology in determining the scope of liability, the application is far from universal in all cases. Consider the cautionary words of Windeyer J with respect to determining foreseeability by 'harm of like kind': '[T]his comfortable latitudinarian doctrine has however the obvious difficulty that it leaves the criterion for classification of kinds or types of harm undefined and at large.'[28] Harm classified as 'cognitive harm' or an 'acquired brain injury' is not a term of definition and therefore is too broad to supply the criterion necessary to identify CTE by type of disease..

When scrutinised, similar forms of harm may not in fact be identical nor identically foreseeable. For the athlete plaintiff, there is little facility in the sole pursuit of a claim based on an assumption that 'cognitive damage' or 'acquired cognitive harm' in the form of, say, symptomatic concussion necessarily incorporates other forms of cognitive damage, such as CTE. In short, a subset of a type of foreseeable harm may not be foreseeable for the purposes of scope of liability — in effect, a 'subtype'.

Where harm ostensibly belongs to a category, type, or class of harm, it is a question of fact as to whether a particular variation is substantial enough to preclude membership in that type. Whether a specific form of harm is to be included or excluded from a classification of harm involves, then, consideration of factual differences, at times subtle, and the utilisation by a court of 'common sense' and a 'value judgment'.[29]

Consider the following examples. In *Tremain v Pike*[30] a farm employee contracted Weil's disease, an infection carried by rats. According to expert evidence, the illness was caused by rat urine contacting broken or sodden skin rather than the more predictable transmission sources of bites and food contamination. There was no evidence that the farmer knew or ought to have known of the risk of infection by Weil's disease. The plaintiff framed his argument of foreseeable harm in broad terms as essentially any rat-borne disease: 'The plaintiff would be

[26] See *Rosenberg v Percival* (2001) 205 CLR 434, 455 [64] (Gummow J).

[27] Ibid.

[28] *Mount Isa Mines Ltd v Pusey* (1970) 125 CLR 383, 414.

[29] *Wallace v Kam* [2012] NSWCA 82, [4].

[30] [1969] 3 All ER 1303.

exposed to the risk of disease if the rats were not controlled.' However, as infection with Weil's disease was a 'remote possibility' only, Payne J found that the plaintiff's harm 'was, therefore, unforeseeable and too remote to be recoverable'.[31]

In *Rowe v McCartney*[32] the plaintiff suffered depressive neurosis when she permitted a friend to drive her car in adverse weather conditions in which he crashed it into a telegraph pole. The friend was rendered quadriplegic. The Court of Appeal by majority found that psychiatric damage of this type was not foreseeable. Rather, it was the result of the 'plaintiff's abnormal reaction' and the 'irrational reasoning in relation to, but external to, the negligent act'. In summary, the psychiatric injury 'was too remote from the defendant's breach of duty'.[33]

Irrespective of whether or not it falls within a particular type of harm, the specific harm in question may nonetheless be adjudged as not foreseeable. In *Koehler v Cerebos*[34] a worker whose job required her to lift boxes complained that the aches, pain and difficulty in moving were due to the physical demands of her job and 'overwork'. In fact, the causes were a 'psycho-physical disorder' and a 'major depressive illness' that were, according to her psychiatrist, 'entirely attributable to her conditions of employment'.[35] Nonetheless, psychiatric harm or nervous shock was not foreseeable in the circumstances. There were no outward indications that the plaintiff was under a psychological threat. The plaintiff made no express complaint that she was at risk of psychological harm. While the court said that 'signs' of an employee being at risk would indicate foreseeability, none were displayed; nor did the plaintiff suggest 'vulnerability to psychiatric injury',[36] or take any sick leave suggestive of psychological distress. Callinan J noted that foreseeability may be assessed on the basis 'of the impression created by, and the other overt or foreseeable sensitivities of the actual person affected', including changes to personality or symptoms'.[37]

In *Rowe*, differences in mode of transmission, a different form of disease to that commonly associated with rats and the rarity of the disease in humans were noted, and there was no expectation that farmers would know of Weil's disease. In *Koehler v Cerebos*, the psychological reaction was abnormal and irrational, so as to place it beyond the foreseeable. It is 'difference' of a material kind that precludes a particular form of harm from falling within a classification by type where its members share similar traits of a fundamental type.

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31 Ibid 1307.

32 [1976] 2 NSWLR 72.

33 Ibid.

34 [2005] HCA 15.

35 Ibid [14].

36 Ibid [35].

37 Ibid [55]–[56]

As explained further below, the argument that an SGB's scope of liability extends only to the consequential harm of CTE based on foreseeability as a 'type' of cognitive harm is at best doubtful. The factual differences between, say, symptomatic concussion and CTE are sufficiently pronounced as to preclude any assumption of amalgamation by type. Therefore, foreseeability of consequential harm in the form of CTE is argued below to flow through an SGB's medical advisers.

D Why foreseeability of CTE as a 'type' of cognitive harm/ concussion is unlikely

Several forms of cognitive harm associated with contact sport are concussion, loss of consciousness, post-traumatic amnesia, post-concussive syndrome, among others. At its most basic, a hallmark of these conditions is a relatively short recovery period in comparison to CTE, which is characterised by increasing debilitation and death.

While foresight need only be 'harm of a like kind',[38] differences between simple forms of cognitive distress associated with contact sports and that of CTE are at such a degree of variance that each is unlikely to be judged as belonging to a common type or kind.

One need only reference the sequelae and the increasingly debilitating effects of CTE expressed throughout this thesis to realise that foreseeability by 'type' as a common category based on cognitive disfunction or similar descriptor is unlikely to be sustainable. There is no warrant to revisit chapters that describe the differences between CTE and, say, frank concussion. It is sufficient to note the histological uniqueness of CTE marked by deposits of phosphorated tau, the behavioural manifestations common to affliction with CTE and the range of long-term sequelae, including confusion, impaired judgment, impulse control problems, aggression, depression, suicidality, parkinsonism, and eventually progressive dementia and death.[39]

In summary, a type of harm to which CTE could properly belong is proposed as one best classified along the lines of 'head trauma leading to death' or 'cognitive disfunction from which a victim does not recover' or of similar description.

E CTE as a foreseeable risk of harm

Establishing scope of liability through foresight of harm consequential to breach requires a defendant to have had reasonable access to information revealing a risk of harm to the plaintiff.[40] The risk of CTE was argued in several chapters of this thesis as revealed, and thereby foreseeable through an SGB's medical associates and through observation by an SGB of players who display the symptoms or sequelae of CTE-like harm. Again, there is no need to

[38] *Mount Isa Mines Ltd v Pusey* (1970) 125 CLR 383, 402.

[39] 'Frequently Asked Questions about CTE', *Boston University, Center for the Study of Traumatic Encephalopathy* (Web Page) <https://www.bu.edu/cte/about/frequently-asked-questions/>.

[40] *Rosenberg v Percival* (2001) 205 CLR 434.

revisit the detail of these chapters other than to say that provided an SGB's medical associates had reasonable access to medical journals that reported on the risk of CTE in contact sport prior to *circa* 2010, foreseeability of harm consequential to breach by failure to warn is likely to be satisfied.

F Notification and foreseeability

In addition to information available from other sources, two studies suggest that the governing bodies of rugby league and Australian Football League (AFL) possessed direct foresight of the risk of CTE well before *circa* 2010.[41] In 1994, sports medico Dr Nathan Gibbs published an article titled 'Common Rugby League Injuries' in *Sports Medicine*, referencing 'a belief' based on 'evidence from boxing' that *dementia pugilistica* could be caused by playing rugby league.[42]

In 1993, a report by AFL medical officers to the National Health and Medical Research Council (NHMRC) on head and neck injuries in football expressed concerns regarding the long-term effects of concussion.[43] The AFL also informed that since 1985 it had been researching the risk of long-term cognitive harm 'as a result of impacts to the head during games or practice, and not just those hits resulting in concussion'.[44] It is inconceivable that AFL medical officers would put their views to the NHMRC without the knowledge and approval of the AFL. Had they done so, for the sake of argument, it is not conceivable that information of the risk of long-term harm in playing AFL would not have been brought to the attention of the governing body of AFL. In similar vein, the publication of Dr Gibbs' article would undoubtedly have alerted the governing body of rugby league of the risk of CTE, and would in all probability have come to the notice — directly or by word of mouth — of other governing bodies of contact sport.

3.3.3 Other considerations

Other 'relevant considerations' noted in *Zanner v Zanner*[45] and in commentary and other judgments revealing the content appropriate to determining the scope of liability are touched upon briefly below.

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41 For further discussion, Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times'.

42 Nathan Gibbs, 'Common Rugby League Injuries' (1994) 18(6) *Sports Medicine* 438, 438.

43 *AFL Information Paper and the AFL Medical Officers Report to the National Health and Medical Research Council on Head and Neck Injuries in Football* (1993).

44 See AFL, 'Responsible Approach to Concussion in the AFL', *Information Paper*, May 2012.

45 [2010] NSWCA 343: see **[3.3]** above.

A The scope of the risk of harm protected by the duty and created by the breach was foreseeable

In the words of the High Court, 'the scope of liability for the consequences of negligence is often coextensive with the content of the duty of the negligent party that has been breached'.[46] The duty of an SGB was to protect the health and well-being of its premier players. CTE was within the scope of the risk of harm and was created by the breach.

B The injury was not coincidental to the breach but the direct and immediate consequence of the negligence

Injury in the form of CTE was not coincidental to the breach by failure to warn and exposure to RHT, but was the direct and immediate consequence of it.

C There was no intervening act or abnormal event

There were no intervening acts that led to harm in the form of CTE. Concerns as to head trauma experienced in other contact sport do not represent an intervening act, but are argued to be a contributing cause. Any susceptibility due to genetic predisposition is not an intervening act, but falls under the proposition that a defendant must take the victim as found.[47]

D Harm was from the same cause

Can it be said that symptomatic concussion and CTE are merely degrees of the same harm? In *Uniting Church in Australia Property Trust (NSW) v Miller*, where a swimmer suffered spinal damage when she slipped on entering the shallow end of a pool, Leeming JA stated:

> Plainly enough, the class of harm in the present case includes all injuries — minor, major, catastrophic or even fatal — suffered from a dive which results in the swimmer colliding with the bottom of the pool.[48]

The components of this class of harm, however, are all known and foreseeable consequences of falling or diving into shallow water. In contrast, although sharing head trauma as a deriving source, CTE and transient disturbances such as symptomatic concussion are, as noted, different diseases.

3.4 An established class and the question of 'why?'

A principal argument of this thesis is that SGBs owe their premier players a duty of care, the scope of which extends to an obligation to warn of material threats to player health and well-being, where these threats would not be obvious to the reasonable player.

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[46] *Wallace v Kam* (2013) 250 CLR 375, [26].

[47] See the discussion at **[3.5]** below.

[48] *Uniting Church in Australia Property Trust (NSW) v Miller; Miller v Lithgow City Council* (2015) NSWCA 320, [110].

The breach of duty, as argued, is a failure of an SGB to warn their players of the risk of CTE-like harm. Breach of duty by failure to warn is an established class of case.[49] The High Court of Australia in *Wallace v Kam* said that where a case is within an established class, the normative question under s 5D(1)(b) of the *Civil Liability Act 2002* (NSW) (and its equivalents) is properly answered through the application of precedent.[50] In simple terms, where a defendant is shown to have an obligation to warn, and harm results from the failure to warn, and precedent establishes a failure to warn as an established class then, *ceteris paribus*, the defendant is liable for the harm caused. As argued in this thesis, a duty of care owed by an SGB to its premier players is 'a novel case'.

Once a duty of care residing in an SGB to its premier players is established as a novel case, it flows naturally that the obligation of an SGB to warn of material risks of material harm is a consequence of that duty. The High Court instructed that a court must explain 'in terms of legal policy' whether or not and why 'responsibility for the harm should be imposed on the negligent party'.[51] The methodology for making what was described as an 'evaluative judgment' requires 'reference to the purposes and policy of the relevant part of the law'.[52] The High Court provided two contextually relevant guides to so judge:[53]

1. '[T]he scope of liability in negligence normally does not extend beyond liability for the occurrence of such harm the risk of which it was the duty of the negligent party to exercise reasonable care and skill to avoid.'[54]

2. '[T]he policy of the law in imposing the duty on the negligent party will ordinarily be furthered by holding the negligent party liable for all harm that occurs in fact if that harm would not have occurred but for breach of that duty …'[55]

With respect to legal policy, an SGB that fails in its duty to warn a player of the risk of CTE is liable for the consequential harm of CTE. Had the player been warned by the SGB of the risk of CTE, the player would not have contracted CTE.[56] As such, the policy and purposes of the law is furthered in a finding that harm in the form of CTE was within an SGB's scope of liability.

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49 See, eg, *Nagle v Rottnest Island Authority* (1993) 177 CLR 423; *Rosenberg v Percival* (2001) 205 CLR 434.

50 *Wallace v Kam* (2013) 250 CLR 375, [26].

51 Ibid [23]. The position taken is that while establishing that a duty of care resides in an SGB is a novel case, once established, the scope of liability with respect to an obligation to warn falls within an established class.

52 Ibid.

53 Ibid [24], [26].

54 Ibid [24].

55 Ibid [26].

56 Assuming the warning would have been responded to.

There is, however, a caveat to the application of these guides. The High Court indicated that the consequences of negligence may not, in some cases, be coextensive with the content of the duty of care. In these circumstances, 'policy' may direct that liability does not attach to the defendant, despite a breach of duty. In respect to an SGB's failure to warn, lability will not attach should it be deemed that a plaintiff player would have ignored a warning had it been given.[57]

3.5 Genetic predisposition and the 'egg-shell skull' rule

Legal commentator Elise Michael suggested that the extraneous factors of 'genetics' and 'steroid use' may be contributing factors in the causation of CTE in NFL players.[58]

3.5.1 On genetic risk and CTE

The major genetic link to the contraction of CTE appears to be the presence of the Apolipoprotein E gene, the allele APOE4 of which is implicated in Alzheimer's disease and traumatic brain injury.[59] Dr Omalu et al's early research noted that '[i]ndividuals who inherit one or two copies of APOE4 have an earlier age of onset for late-onset and sporadic [Alzheimer's disease] and increased long-term sequelae of brain trauma'. However, they added:

> The presence of the APOE4 allele does not confer absolute predisposition to CTE and/or other sporadic or posttraumatic AD pathological changes, and absence of the APOE4 allele does not confer absolute protection from these diseases … [In conclusion,] CTE can occur in the absence of APOE4.[60]

McKee et al, drawing on several studies, also noted possible genetic influences associated with head trauma.[61] Of 'juvenile head trauma syndrome', McKee et al stated that several individuals 'have been reported to have a mutation in the calcium channel subunit gene (CACNA1A) associated with familial hemiplegic migraine.'[62]

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[57] 'Player response' is discussed extensively in Chapter 7: 'Factual Causation of Harm: Failure to Warn and the Athlete's Response Had a Warning Been Given'.

[58] Elise Michael, 'School of Hard Knocks — The Impact of the NFL Concussion Litigation' [2015] 33 *Cardozo Arts and Entertainment Journal*, 289, 306. Expedition permits only a cursory treatment.

[59] HR Winn, 'Neurochemical Pathomechanisms in Traumatic Brain Injury' (2017) 341 *Youmans and Winn Neurological Surgery* 2786.e5. See also 'Apolipoprotein E — An Overview', *ScienceDirect* (Web Page) <https://www.sciencedirect.com/topics/neuroscience/apolipoprotein-e>.

[60] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[61] Ann McKee, 'The Neuropathology of Sport' (2014) 127 *Acta Neuropatholiga* 29.

[62] Ibid 32.

That an individual carries a risk factor for CTE (which has not been established) is of little legal consequence given that all athletes exposed to RHT are at risk of CTE.

Kirby J remarked in *Shorey v PT Ltd*, a case that concerned a fall in a shopping centre resulting in a psychiatric disorder: 'The principle of law is that the negligent defendant must take its victim as it finds her and must pay damages accordingly. … It does not render the damage "unforeseeable".'[63] Similarly:

> It is trite law that a tortfeasor must take the victim of the tort as the victim is found to be. Hence, if as a result of an employer's negligence an employee is caused to inhale asbestos fibres and, due to the employee's predisposition to mesothelioma, the inhalation of fibres results in mesothelioma, the employer will be held liable for the damage thereby inflicted.'[64]

3.5.2 On steroid use and CTE

Michael's suggested use of steroids as an extraneous factor in the development of CTE[65] is seemingly based on the not uncommon use of anabolic steroids in American Football, particularly in the National Football League (NFL). Informatively, Horn found that 9.1% of NFL players 'self-reported' the use of anabolic steroids during their playing career.[66] Several medical journals have also catalogued the use of anabolic steroids across several sports,[67] 'particularly among athletes seeking a competitive advantage.'[68] Mills, however, disclosed that 'to date no experimental paradigm has been carried out to attempt to discern the mammalian brain of a maintenance administration of testosterone combined with a [traumatic brain injury]'.[69]

As that a controlled clinical trial, given the ethical difficulties in administering anabolic steroids is unlikely, Mills experimented with rats injected with anabolic steroids for 12 weeks and then subjected to repeated 'impact acceleration injury' to the head. Control groups did not

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63 *Shorey v PT Ltd* (2003) 197 ALR 410, [44].

64 *Alcan Gove Pty Ltd v Zabic* (2015) 257 CLR 1, [44]

65 Elise Michael, 'School of Hard Knocks — The Impact of the NFL Concussion Litigation' (2015) 33 *Cardozo Arts and Entertainment Journal* 289, 306.

66 S Horn et al, 'Self-reported Anabolic-Androgenic Steroids Use and Musculoskeletal Injuries: Findings from the Center for the Study of Retired Athletes Health Survey of Retired NFL Players' (2009) 88(3) *American Journal of Physical Medicine & Rehabilitation* 192.

67 See, eg, S Bhasin et al, 'The Effects of Supraphysiologic Doses of Testosterone on Muscle Size and Strength in Normal Men' (1996) 335(1) *New England Journal of Medicine* 1; P Vandenberg et al, 'Steroid Use among Adolescents: Longitudinal Findings from Project EAT (2007) 119(3) *Pediatrics* 476; DM Scott et al, 'Anabolic Steroid Use among Adolescents in Nebraska Schools' (1996) 53(17) 53(17) *American Journal of Health-System Pharmacy* 2068; cited in JD Mills et al, 'Anabolic Steroids and Head Injury' (2012) 70 *Neurosurgery* 205.

68 JD Mills et al, 'Anabolic Steroids and Head Injury' (2012) 70 *Neurosurgery* 205, 208, referring to research by F Talih et al, 'Anabolic Steroid Abuse: Psychiatric and Physical Costs' (2007) 74(5) *Cleveland Clinical Journal of Medicine* 341, 346, 349–52.

69 JD Mills et al, 'Anabolic Steroids and Head Injury' (2012) 70 *Neurosurgery* 205, 208, 209.

receive steroids. The rats were euthanised and brain tissue examined. The experimenters reported: 'Using this model and within its limitations, we see no adverse effect or causative role of anabolic steroid administration on the brain following [mild traumatic brain injury] with the use of APP counts as a marker for anatomic injury.'[70] Similar results were recorded in a meta study of 153 CTE cases reported in medical journals, revealing 'anabolic steroids' use in only four CTE 'diagnosed subjects'.[71] Some more recent studies, moreover, make no mention of steroids as a causative factor in CTE, nor are steroids causative of CTE in reported domestic abuse cases nor in head banging syndrome.[72]

Any connection would seem to be based on an association rather than a clinical link between CTE and steroids. Also relevant is the prevalence of CTE in other groups exposed to RHT, including artillery personnel and those with 'head bang syndrome' who, it would seem, have less reason that a professional athlete to engage in steroid use.

3.6 Conclusion

This appendix argued that an SGB's scope of liability extends to the contraction of CTE consequential to a failure to warn players who participated in its premier competition prior to *circa* 2010 of the risk of CTE-like harm and where those players would have adopted ameliorating measures, such as choosing not to play, had they been warned.

That it is appropriate that the scope of liability attaches to an SGB is based primarily on the proposition that harm in the form of CTE was a foreseeable consequence of the SGB's breach. Foreseeability was noted by Allsop J in *Zanner v Zanner*[73] and by Stapleton as a 'consideration' in determining the scope of liability.[74] In context: it was foreseeable that if an SGB did not warn a premier player of the risk of CTE, the player may contract CTE through participation in a sporting activity that he or she may, if warned, have chosen to avoid.

It was suggested that CTE may be classified 'by type' as a form of cognitive damage, similar to other well-recognised forms of foreseeable harm such as frank concussion, acquired brain injury or unconsciousness. Such a classification is advantageous to a plaintiff athlete given that cognitive damage as a type of harm is clearly a foreseeable consequence of contact

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[70] Ibid 209. One group of rats received steroids post exposure to head impacts, but not before.

[71] JC Maroon et al, 'Chronic Traumatic Encephalopathy in Contact Sports: A Systematic Review of All Reported Pathological Cases' (2015) 10(2) *PLoS One* 6.

[72] See, eg, KF Bieniek, 'The Second NINDS/NIBIB Consensus Meeting to Define Neuropathological Criteria for the Diagnosis of Chronic Traumatic Encephalopathy' (2021) 80(3) *Journal of Neuropathology & Experimental Neurology* 210; T Danielsen et al, 'Chronic Traumatic Encephalopathy (CTE)-Type Neuropathology in a Young Victim of Domestic Abuse' (2021) 80(6) *Journal of Neuropathology & Experimental Neurology* 21; PR Hof et al, 'Neuropathological Observations in a Case of Autism Presenting with Self-Injury Behavior' (1991) 82(4) *Acta Neuropatholiga* 321.

[73] [2010] NSWCA 343, [12]

[74] Jane Stapleton, 'Reflections on Common Sense Causation in Australia' in S Degeling and J Edelman (eds), *Torts in Commercial Law* (Thompson Reuters Australia, 2011) 352.

sport and CTE, as a form of cognitive damage, is to be included within that type. In fact, it was proposed that a potential error of great moment would be an assumption by a plaintiff athlete that because cognitive harm is a recognised 'type' of harm associated with the playing of contact sport, CTE is also foreseeable consequence of an SGB's breach by failure to inform.

While foreseeability of CTE 'by type' is recognised as a possibility, it was argued more forcefully that the differences between CTE and other forms of cognitive harm are so substantial as to preclude CTE from inclusion within such a broad classification of type. Redlich JA in *Metrolink Victoria v Inglis* considered the application of scope of duty where the harm that eventuated was unusual, stating: 'It seems that those cases which descend to a further level of detail such as *Doughty* and *Rowe v McCartney*, are those which involve an unusual injury or an injury which arises from a particularly unusual sequence of events.'[75] In support of this proposition, it was argued that CTE is a disease of increasing debilitation leading ultimately to death, while those affected by conditions like concussion or on-field unconsciousness recover within a few days or weeks. Descriptions by type, such as 'acquired brain injury', are perhaps broad enough to cover the harm of symptomatic concussion and CTE, yet again the sequelae and life expectancy of each are starkly at variance. Differences in symptoms and histology have been referred to in detail throughout this thesis. It is proposed that CTE the distinct clinical features of CTE in comparison to other forms of cognitive harm, does not fall within a classification by type referred to as cognitive harm or other general description.

In summary, this appendix argued that the scope of an SGB's liability in failing to warn its premier-level athletes of the risk of CTE extends to harm in the form of CTE. A major consideration in determining scope of liability is that of foreseeability of harm. Harm in the form of CTE is a foreseeable consequence of failing to warn an SGB's premier players of the risk of long-term cognitive harm in the form of CTE. In applying Stapleton's formulation: the failure to warn was a breach of duty. This breach of duty caused the plaintiff athlete to participate in the SGB's contact sport. This participation caused the plaintiff harm in the form of CTE. With respect to the 'scope question', this form of harm is a 'foreseeable consequence of the breach'.[76]

[75] *Metrolink Victoria Pty Ltd v Inglis* [2009] VSCA 227, [95]–[96], referring to *Doughty v Turner Manufacturing* [1964] 1 QB 518; *Rowe v McCartney* [1976] 2 NSWLR 72.

[76] Jane Stapleton, 'Reflections on Common Sense Causation in Australia' in S Degeling and J Edelman (eds), *Torts in Commercial Law* (Thompson Reuters Australia, 2011) 354.

Appendix 4:
Factual Means of Assessing Generic Capacity to Cause CTE

4.1 Introduction

Chapter 8 of this thesis — 'The Question of "Generic Capacity": Does the SGB's Contact Sport Cause CTE?' — concerned the law applicable to proving that the playing of contact sport had the generic capacity of causing CTE. The analysis in Chapter 8 was necessary to show that the accepted methodology of proving 'generic capacity' in a case where latency in the development of a disease following exposure to a source of harm is through the application of the 'but for' test of causation. It must be stressed that the 'but for' test is the recognised means of proving causation to the legal standard of assessment on the balance of probabilities rather than the more absolute scientific standard which may be inapt for purposes of judgment in law.[1]

This appendix is supplementary to Chapter 8 and aims to provide discussion of the factual means by which a plaintiff athlete may prove on the balance of probabilities that the contact sport he or she played had the capacity to cause CTE. Again, proof that a sport administered by an SGB has the capacity or potential to cause CTE does not prove that the symptoms displayed by the athlete are necessarily those of CTE.[2]

This appendix is concerned with research argued as proving to the legal standard that RHT in playing contact sport is a cause of CTE. The related question of whether there was sufficient information regarding the availability and access to information on the risk of CTE is considered in Chapter 4: 'CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care' and Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times'. It is important that the two questions not be confused. Knowledge of CTE dating back decades is relevant to the question of whether information on the risk of CTE was available and accessible to SGBs in the pre-2010 era and ought to have been known. Proof that the generic capacity of RHT in playing contact sport is a cause CTE, or whether CTE is a real disease, must be proven on the balance of probabilities and draws upon scientific information available at any date, including post-2010.

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[1] Where the scientific standard actually proves causation the purposes of a plaintiff are served, however, it is a higher standard than required for legal proof and in many cases make legal proof of causation impossible.

[2] That question — proof of individual agency — is discussed in Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"'.

The common law 'but for' test used in cases such as *Amaca Pty Ltd v Booth*[3] is equivalent to the statutory 'necessary condition' test. In *Adeels Palace v Moubarak*, the High Court stated that 'it is necessary to observe that the first of the two elements identified in s 5D(1) [of the *Civil Liability Act 2002* (NSW)] (factual causation) is determined by the "but for" test: but for the negligent act or omission, would the harm have occurred?'[4] Here, of course, the consideration is the generic capacity of RHT in the playing of contact sport to cause CTE, and how this capacity in determined as a matter of law by the 'but for' test.

4.1.1 Two interrelated questions

Given the nature of the occurrence of CTE and the long latency period prior to the onset of symptoms, proof of legal causation of CTE involves two interrelated questions:

1. Is RHT in playing the type of contact sport for which the governing body provides a competition a probable cause of CTE?

2. Did playing in the SGB's competition cause the head trauma that led to the plaintiff's CTE?

While proof that conduct *can* cause injury does not prove that it *did* cause injury,[5] both questions are logically interlinked and, in some cases, will be identical in outcome — if playing a particular contact sport is a cause of CTE and the governing body provides a competition in that contact sport, then, *ceteris paribus*, playing in the governing body's sporting competition is probably a cause of the plaintiff's CTE.[6] Nonetheless, the plaintiff must prove this logical assumption.

This appendix now turns to consider the scientific and medical information regarding the risk of harm in the form of CTE in the playing of contact sport with the object of determining whether this risk is affirmed to the legal standard.

4.1.2 This appendix proceeds as follows

Section 4.2: The general body of medical knowledge

This section explains how proof of causation of CTE by RHT in playing contact sport can be provided by a preponderance of medical knowledge, including inferences drawn from hundreds of research articles, a connection to a known risk, cases of similar application, and the evidence of experts.

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3 (2011) 246 CLR 36.

4 *Adeels Palace Pty Ltd v Moubarak* (2009) 239 CLR 420, [51].

5 *Amaca Pty Ltd v Ellis* (2010) 240 CLR 111, [10].

6 Of course, as explained in Appendix 5, the amount of participation in a contact is a determining factor.

Section 4.3 The Bradford Hill criteria: Determining the cause of CTE

This section examines the use of the judicially well-recognised Bradford Hill criteria as a means of proving generic causation.

Section 4.4: Nowinski's application of the Bradford Hill criteria to CTE

This section reviews Dr Christopher Nowinski's application[7] of all nine Bradford Hill criteria in forming the reasoned opinion that RHT in playing contact sport is a cause of CTE.

The section also includes a short treatise on the apparent reliability of the Nowinski study as a tool in showing the generic cause of CTE.

Section 4.5: Other means of proving generic capacity

This short section briefly notes that the 2023 Australian Senate Inquiry titled *Concussions and Repeated Head Trauma in Contact Sports*[8] provides some limited factual material to prove the generic capacity of contact sport to cause CTE, and in addition points out that some submissions to the inquiry denied a provable link.

Section 4.6: Conclusion

This section reviews the content of this appendix, stating that the test of generic capacity is determined under the common law 'but for' test, which is equivalent to the statutory test of causation as a 'necessary condition' or 'factual causation'. It concludes that generic causation is provable on the balance of probabilities through various scientific/medical tests, and particularly through use of Bradford Hill criteria.

4.2 The general body of medical knowledge

Some questions of medical causation can be answered from 'the general body of medical knowledge', said Sir Austin Bradford Hill; for example, where 'a particular chemical is known to be toxic to man and therefore suspect on the factory floor. … [Or] sometimes, alternatively, we may be able to consider what might a particular environment do to man, and then see whether such consequences are indeed to be found.'[9]

With several hundred studies linking CTE with RHT in the playing of contact sport published since Dr Harrison Martland's 'Punch Drunk' article in 1928,[10] proof of causation of

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[7] Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1.

[8] The Senate, Community Affairs References Committee, *Concussions and Repeated Head Trauma in Contact Sports*, September 2023.

[9] Austin Bradford Hill, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965) 295, 295.

[10] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

CTE may, as argued, be inferred from the general body of medical knowledge. Many of the publications referenced throughout this thesis strongly indicate an affirmative response to the question: 'Is RHT in playing the type of contact sport for which the governing body provides a competition a probable cause of CTE?' Major and widely noted research since Martland's include Dr Bennet Omalu et al's revelations of CTE in an NFL player,[11] Dr Ann McKee et al's 2009 report of CTE across a range of contact sports including rugby and Australian football,[12] and various publications concerned specifically with bodily contact in rugby and football.[13]

4.2.1 Causal connection and a known risk

Where a plaintiff is exposed to a known risk of a disease and the disease materialises, the trier of fact may infer causation. In context, if head trauma in playing contact sport is a known risk in contracting CTE, and a plaintiff athlete played contact sport and contracts CTE, can causation be inferred?

4.2.2 Cases of similar application

In terms of application, proof of causation of CTE has some parallel with the mesothelioma cases. Asbestos as a risk factor in the causation of mesothelioma is well established. As Heydon J explained in *Amaca Pty Ltd v Booth*,

> a person who is in a room containing asbestos dust is at risk of inhaling asbestos fibres. If the risk materialises and the fibre is inhaled, he will be at risk of some fibres lodging in his lung … [and] he is at risk of contracting mesothelioma. The concept of 'risk' looks at the matter prospectively; if the risk materialises, a causal connection may be inferred.[14]

French CJ stated similarly in the same case that although a mere 'risk' is not sufficient to justify a finding of causation in law,

> if the association between two events is shown to have a causal explanation, then the conclusion may be open, if the second event should occur, that the first event has been at least a contributing cause of that occurrence.
>
> … Where the existence of a causal connection is accepted it can support an *inference*, in the particular case, when injury has eventuated, that the defendant's conduct was a cause of the injury.[15]

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[11] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[12] Ann McKee et al, 'Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy after Repetitive Head Injury' (2009) 68(7) *Journal of Neuropathology & Experimental Neurology* 709.

[13] For example, AJ Gardner et al, 'A Systematic Review and Meta-analysis of Concussion in Rugby Union' (2014) 44 *Sports Medicine* 1717; DT Kirkendall et al, 'Heading and Head Injuries in Soccer' (2001) 31(5) *Sports Medicine* 369.

[14] *Amaca Pty Ltd v Booth* (2011) 246 CLR 36, [121].

[15] Ibid [43], [49]. Emphasis added. 'Inference' was discussed at length in Chapter 6 at **[6.5]**.

French CJ then quoted a passage of Lord Reid's in *Gardiner v Motherwell Machinery & Scrap Co Ltd*[16] that is relevant to proving causation of CTE:

> [W]hen a man who has not previously suffered from a disease contracts that disease after being subjected to conditions likely to cause it, and when he shows that it starts in a way typical of disease caused by such conditions, he establishes a prima facie presumption that his disease was caused by those conditions.[17]

McHugh J in *Chappel v Hart*, which concerned a failure of a surgeon to warn a patient of the risk of vocal cord damage through infection, did not refer to 'inference', but held that '[i]f a wrongful act or omission results in an increased risk of injury … and that risk eventuates, the defendant's conduct had materially contributed to the injury … whether or not other factors have also contributed'.[18]

An individual who experiences RHT in contact sport is exposed to a known risk — that of CTE. Where the risk of CTE in playing contact sport materialises, then arguably, in the words of Heydon J, 'a causal connection may be inferred', or in the words of French CJ, it may be inferred that 'the first event has been at least a contributing cause', or according to McHugh J, where the 'risk eventuates', the defendant contributed to the harm.

4.2.3 Inference and expert evidence

A causal connection can be inferred by a relevant expert.[19] In *Amaca Pty Ltd v Booth*, French CJ referred to the testimony of an expert witness called by the plaintiff:

> As demonstrated by medical evidence in this case and in particular by Professor Henderson's evidence, a causal connection may be inferred by somebody expert in the relevant field considering the nature and incidents of the correlation.[20]

As said, a causal connection in *Amaca v Booth* was established by Prof Henderson, who, in doing so, utilised the Bradford Hill criteria — a set of medically based indicators designed to reveal the strength of association between a believed cause and the actual cause of a disease. Prof Henderson opined that a 'causal-contributory relationship' exists on the balance of probabilities between 'exposure to dust derived from brake linings that contain chrysotile asbestos' and the contraction of mesothelioma.[21]

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16 [1961] 3 All ER 831, 832.

17 Ibid [50].

18 *Chappel v Hart* (1998) 195 CLR 232, [68].

19 'Inference' is discussed at length in Chapter 6 at **[6.5]**.

20 *Amaca Pty Ltd v Booth* (2011) 246 CLR 36, [49].

21 Ibid [45].

A causal connection, if accepted, 'can support an inference, in the particular case, when injury has eventuated, that the defendant's conduct was a cause of the injury'.[22] The inference drawn by Prof Henderson was sufficient, in the view of French CJ, to establish 'factual causation'; that is, 'but for' the negligence of the defendant, the plaintiff would not have sustained harm.[23]

The conditions that cause CTE are well researched and documented, as are its symptoms and sequelae. Nonetheless, doubts have been expressed as to whether CTE is a 'real' disease or whether, if real, it is caused by RHT in the playing of sport and are discussed and considered as to any impact of the assessment of the causation of CTE more fully in Chapter 2 and Appendix 1 and 2.[24] Irrespective, most concerns are expressed in terms of what are referred to as 'methodological flaws', mainly biased sampling at autopsy, a shortcoming noted by those scientists and medicos who accept CTE as a real disease but believe that such imperfections do not alter the more pressing imperatives: 'Waiting for results and conclusions from studies that require many decades is unethical in light of the significant probability of harm to at least some nonzero proportion of any collision sport cohort.'[25]

What is important with respect to this discussion of the general body of medical knowledge is that an inference is drawn from the fact of medical research from multiple sources by experts in the field.[26] From this research, an inference of causation due to RHT is, as argued, likely to be established and from which the claim can rightly be put that an SGB 'ought to have' possessed knowledge of the risk and warned its players.

4.3 The Bradford Hill criteria: Determining the cause of CTE

Attention now turns to the well-recognised Bradford Hill criteria to infer causation of CTE as a 'necessary condition'. It should be noted, as a matter of practicality, that an assessment of causation based on the Bradford Hill criteria may well utilise many of the hundreds of scientific reports within the general body of medical knowledge — many referred to in this thesis.

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[22] Ibid [49].

[23] Ibid [47]–[48].

[24] Chapter 2: 'The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments'; Appendix 2: 'CTE and "Negativing Influences": Do They Determine Liability?'

[25] ST Casper et al, 'Toward Complete, Candid, and Unbiased International Consensus Statements on Concussion in Sport' (2021) 49(3) *Journal of Law, Medicine & Ethics* 372, 376. This topic is discussed extensively in Chapter 5: 'Accessible Knowledge of CTE in Australia at Relevant Times'. For further discussion, see **[4.4.3]** below: 'Is the Nowinski study factually and legally acceptable?'

[26] Austin Bradford Hill, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965) 295.

In 1965, medical statistician Sir Austin Bradford Hill posed several questions to the Royal Society of Medicine on the determination of the medical cause of a disease:

> How … do we detect … relationships between sickness, injury and conditions of work? How do we determine what are physical, chemical and psychological hazards of occupation, and in particular those that are rare and not easily recognized? …[27]
>
> [M]ore often than not we have no such guidance, no such means of proceeding; more often than not we are dependent upon our *observation* and *enumeration* of defined events for which we then seek antecedents.[28]

To give structure to 'observation' and 'enumeration', Bradford Hill proposed nine criteria by which to evaluate 'human epidemiological evidence' to determine the strength of a causal relationship between an apparent causative event and the onset of a disease believed to be caused by that event. The criteria are: strength of association, consistency, specificity, temporality, biological gradient, plausibility, coherence, experiment and analogy. These are reviewed in greater detail below.

The Bradford Hill criteria, says Nowinski, are designed 'specifically to address causation involving environmental exposures' by providing 'a framework to determine if one can justifiably move from an observed association to a verdict of causation'.[29] In the present context, an 'environmental exposure' is RHT experienced in the playing of contact sport. Importantly, with respect to newly discovered (or 'rediscovered') medical conditions such as CTE, absolute proof is not necessary to establish positive causation in medical terms — in Nowinski's words, 'before deducing "causation" and taking action we shall not invariably have to sit around awaiting the results of that research. The whole chain may have to be unravelled or a few links may suffice.'[30]

The Bradford Hill methodology has been applied by courts of appeal across several decades thereby attesting to its usefulness as a legal tool in discerning generic causation.

French CJ in *Amaca Pty Ltd v Booth*[31] spoke at length on the utility of the Bradford Hill criteria in determining the causation of mesothelioma. His Honour observed that a positive finding based on the Bradford Hill criteria does not establish 'but for' causation, but it does 'provide a guide to the kind of considerations that lead to an inference of causal connection'.[32] French CJ also pointed out that Bradford Hill's nine criteria 'were not presented in his paper

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[27] Ibid 295.

[28] Ibid 295. Emphasis added.

[29] Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1, 3.

[30] Austin Bradford Hill, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965) 295, 295.

[31] (2011) 246 CLR 36.

[32] Ibid [49].

as necessary conditions of a cause and effect relationship', but rather 'have the character of circumstantial evidence of such a relationship'.[33]

In *Seltsam Pty Ltd v McGuiness*, Spigelman CJ, Stein JA and Davies A-JA referenced the Bradford Hill criteria in determining whether asbestos was a legal cause of the plaintiff's renal cell carcinoma.[34] French J in *Reay v British Nuclear Fuels* referred to the criteria as the factors against which epidemiological results should be examined.[35]

Discussion above concerned the formation of a causal connection from which an inference of causation is established. There is no need to repeat that analysis here; rather, focus is directed to the application of the Bradford Hill criteria with respect to an inference of causation of CTE as a matter of law.

4.4 Nowinski's application of the Bradford Hill criteria to CTE

In his original paper, Bradford Hill sought a method to establish a 'verdict of causation'.[36] His criteria were applied by Nowinski, a neuroscientist, directed to the question of an 'observed association' between head trauma and the onset of CTE. Utilising medical literature on RHT, Nowinski used the Bradford Hill criteria '[t]o better understand the strength of evidence underlying the possible causal relationship between [RHT] and CTE'.[37] Each criterion, in the context of CTE and Nowinski's findings, is considered in brief below.

4.4.1 The criteria

A Criterion 1: Strength of association

The researchers applied an 'odds ratio' or 'relative risk' statistical assessment to determine the 'strength of association', or relationship, between RHT and CTE. In general terms, a ratio >1.0 is 'very unlikely to be caused by random chance' and, the larger the ratio, 'the less likely [it is] that the significant association is an artifact of some non-random flaw such as confounding, bias, or misclassification'.[38] Six medical journal articles that had been identified as 'well-conducted case-control studies where … a reasonable attempt to identify [RHT] history and

[33] Ibid [44].

[34] *Seltsam Pty Ltd v McGuiness* [2000] NSWCA 29. See further comments below.

[35] *Reay v British Nuclear Fuels* (1994) 5 Med LR 1.

[36] Austin Bradford Hill, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965) 295, 295.

[37] Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1, 1. The Australian researchers included Michael E Buckland, Elisa L Hill-Yardin and Alan J Pearce.

[38] Ibid 3.

had more than 50 subjects to be sufficiently powered for statistical significance', were reviewed by Nowiski in the assessment of the medical causation of CTE.[39]

The results for 'strength of association' were as follows. Nowinski stated:

> [W]e found that all six studies reported large and significant ORs [odds ratios] of CTE cases in subjects with substantial exposure to [RHT] compared to controls. … The clear and unremitting message from these studies is that the associations between [RHT] (and reliable proxies for it) and CTE cases are strong: consistently above the 2.0 benchmark, with some above ratios of 10:1 and even 100:1.[40]

Furthermore, when criteria developed by the Oxford Centre for Evidence-Based Medicine — the 'Oxford Levels of Evidence' — were applied to the same studies, Nowinski found that the findings 'would also be considered as "strong evidence" for a causal association by any reasonable interpretation of the Oxford Levels'.[41]

B Criterion 2: Consistency

The criterion of 'consistency' refers to an association between RHT and CTE pathology 'across multiple studies conducted by independent groups investigating different patient populations using different approaches'.[42]

Nowinski referred to research at the Boston University Brain Bank, which has an 'extensive collection of brains' and reported that

> CTE pathology has been found in former athletes at amateur and professional levels from American football, soccer, rugby, ice hockey, lacrosse, mixed martial arts, wrestling and boxing, as well as military veterans, victims of domestic violence, an individual who suffered multiple falls and an individual who suffered from head-banging behavior.[43]

Nowinski also referred to other studies, where CTE was detected in players of soccer, rugby and Australian football.

In addition, the 'consistency' measure included 'independent studies' by brain banks in several countries, where research had revealed an association between head trauma and CTE, including Scotland, England, Canada, Ireland, France, Australia, Brazil, the United States and South Korea.

Nowinski concluded:

> Together, these brain bank studies highlight that exposure to [RHT] from various sources is a conserved factor between individuals with CTE pathology, regardless of whether other comorbid

[39] Ibid 4.

[40] Ibid.

[41] Ibid 5.

[42] Ibid.

[43] Ibid

> pathologies are present. Overall, current literature from independent researchers and brain banks around the world provides strong support for [Bradford] Hill's consistency criterion.[44]

C Criterion 3: Specificity

'Specificity' refers to a disease occurring 'within a specific population and/or at a specific anatomical site with no reasonable explanation other than involvement of the factor in question', such that 'the greater the specificity of association between factor and outcome, the greater the probability of a causal relationship'.[45] Bradford Hill notes that 'if specificity exists we may be able to draw conclusions without hesitation'.[46]

Nowinski's study found that

> [s]pecificity can be observed in a number of ways, but perhaps most significantly by the fact that prior exposure to [RHT] is the *only* known unifying factor among CTE cases reported in the literature to date … which indicates a specific relationship between the two. …
>
> Taking all of the evidence together, we can conclude that specificity certainly exists for the association between [RHT] and CTE. … [T]his should lead us to consider that the relationship between [RHT] and CTE is indeed a causal one.[47]

D Criterion 4: Temporality

This characteristic was described by Bradford Hill as 'the temporal relationship of the association — which is the cart and which the horse?'[48] Logically, said Nowinski, 'exposure necessarily must precede its purported outcome'.[49] He continued:

> The [RHT] exposure experienced by nearly all individuals diagnosed with CTE occurred before the observed pathology, typically years if not decades earlier. As such, the pathology of CTE is considered a long-term consequence of [RHT] exposure. …
>
> Together, these data provide overwhelming evidence supporting the temporal relationship between [RHT] and CTE.[50]

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[44] Ibid 6.

[45] Ibid.

[46] Austin Bradford Hill, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965) 295, 297.

[47] Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1, 7.

[48] Austin Bradford Hill, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965) 295, 297

[49] Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1, 7.

[50] Ibid 7–8.

E Criterion 5: Biological gradient

This criterion is revealed as a 'dose response curve'. The greater the degree of exposure to the purported cause, the more likely the individual is to contract the disease in question. Affirming a positive response necessarily depends on the availability of information regarding an individual athlete's playing history. Nowinski considered studies published over several decades, noting that Martland's 1928 research 'included the recognition of a dose-response between [RHT]and possible CTE symptoms'.[51]

Nowinski referred to a study of the brains of American footballers by Boston University, Center for the Study of Traumatic Encephalopathy, and observed that

> players who played 14.5 years or more were 10 times more likely to have CTE than someone who played fewer than 4.5 years. … Even under conditions of simulated extreme brain bank selection bias, the estimated magnitude of the relationship between duration played and CTE status remained consistent.[52]

In addition, Nowinski noted that 'each year of additional football play was associated with an increased OR of 1.30, effectively doubling the odds of disease every 2.6 years'.[53] Nowinski concluded that 'from its first description nearly 100 years ago to the present, CTE has been recognized as having a strong biological gradient, one of [Bradford] Hill's most salient criteria, in boxing, American football, and ice hockey'.[54]

F Criterion 6: Plausibility

This characteristic refers to causation that is 'biologically plausible' but, as Bradford Hill said, 'this is a feature I am convinced cannot be demanded' as the association 'we observe may be one new to science or medicine and we must not dismiss it too light-heartedly as just too odd'.[55] Or, as Nowinski explained, this criterion 'requires that the association between [RHT] and CTE pathology can be explained in the presence of existing biological or social models'.[56] Nonetheless, referring to human, animal and computational research, Nowinski stated that

> these independent studies provide evidence of a credible mechanistic hypothesis for the location of the pathognomonic lesion, and the association between [RHT] and CTE alongside the paucity of CTE

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[51] Ibid 8.

[52] Ibid. Apparent 'selection bias' occurs because those suffering cognitive symptoms are more likely to have their brain donated for autopsy examination.

[53] Ibid.

[54] Ibid 9.

[55] Austin Bradford Hill, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965) 295, 298.

[56] Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1, 9.

cases in individuals not exposed to [RHT] all support that [RHT] exposure is a plausible cause of CTE.[57]

G Criterion 7: Coherence

According to Bradford Hill, 'the cause-and-effect interpretation of our data should not seriously conflict with the generally known facts of the natural history and biology of the disease'.[58] In this context, the criterion of 'coherence' requires 'that the association between [RHT] and CTE pathology does not conflict with what we know about the development of CTE pathology or [RHT]'.[59] Nowinski found 'coherence' evidenced in the 'location of the pathognomonic lesion and the absence of CTE cases in individuals without exposure to [RHT] or with only a single TBI [traumatic brain injury] exposure', and the marked rarity of CTE in females as against males, given that historically 'more males have athletic exposure to [RHT], a greater duration of athletic exposures, and more years since [RHT] exposure'.[60] Alternative hypotheses, such as opiate use, were found 'not [to] hold up to scrutiny'.[61] Nowinski concluded that '[w]ith what is known in the literature about computer modelling of brain trauma, post-mortem confirmed cases without a history of [RHT] exposure, sex differences, opiate use, and CTE genetics, [RHT] remains the only candidate risk factor for CTE causation'.[62]

H Criterion 8: Experiment

Bradford Hill offered that '[o]ccasionally it is possible to appeal to experimental, or semi-experimental, evidence'.[63] This may include an observed outcome — positive or negative — following the removal of a proposed factor of causation. Two problems present regarding head trauma as a cause of CTE: individuals cannot ethically be exposed to experimental RHT, and it is 'too soon to measure the potential impact of recent efforts made to reduce [RHT]'.[64] Nonetheless, Nowinski states that 'pre-clinical models' have proven to be effective indicators: '[T]he findings from pre-clinical [RHT] studies replicating elements of observed human

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57 Ibid.

58 Austin Bradford Hill, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965) 295, 298.

59 Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1, 9.

60 Ibid 10.

61 Ibid.

62 Ibid.

63 Austin Bradford Hill, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965) 295, 298.

64 Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1, 10.

pathophysiological, imaging, and epidemiological studies suggests a causal relationship [between RHT and CTE].'[65]

I Criterion 9: Analogy

Bradford Hill stated that in 'some circumstances, it would be fair to judge by analogy'.[66] That is, explained Nowinski, 'scientists can use prior knowledge and patterns to infer similar causal associations … [and while this category may not be necessary] it does engender a sense of credibility through resembling other accepted scientific truths'.[67]

Nowinski found that 'evidence supporting TBI as a causal risk factor for dementia is analogous to the relationship between [RHT] and CTE', and that 'while the differences between TBI and [RHT]models are outlined in the plausibility and experiment sections, pre-clinical studies suggest repeated and multiple [RHTs] result in more pathologies than a single impact'.[68] Nowinski also stated that

> [t]he discovery of the link between smoking and environmentally caused lung cancer is analogous to the accruing body of evidence linking [RHT] to CTE. Both links were controversial, specifically because [of] the extreme latency between exposure and symptomatic disease. In fact, the Bradford Hill criteria was partly built on [Bradford] Hill's experience demonstrating a causal relationship between smoking and lung cancer.[69]

4.4.2 Nowinski's conclusions

As French CJ said in *Amaca Pty Ltd v Booth*,[70] the Bradford Hill criteria are not applied as 'necessary conditions of a cause and effect relationship', but they have 'the character of circumstantial evidence of such a relationship'. A direct causal relationship cannot be established through the criteria. Consequently, a determination of causality or diagnosis requires, in the words of French CJ, the exercise of 'judgment'. His Honour referenced the application of the Bradford Hill criteria in the *American Restatement of the Law (3d) of Torts*:

> Whether an inference of causation based on an association is appropriate is a matter of informed judgment, not scientific methodology, as is a judgment whether a study that finds no association is exonerative or inconclusive. No algorithm exi-sts for applying the [Bradford] Hill guidelines to determine whether an association truly reflects a causal relationship or is spurious.

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65 Ibid 11.

66 Austin Bradford Hill, 'The Environment and Disease: Association or Causation?' (President's Address, Proceedings of the Royal Society of Medicine, 14 January 1965) 295, 299.

67 Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1, 11.

68 Ibid.

69 Ibid.

70 (2011) 246 CLR 36, [44].

Because the inferential process involves assessing multiple unranked factors, some of which may be more or less appropriate with regard to a specific causal assessment, judgment is required.[71]

Exercising medical judgment, Nowinski offered several conclusions as to the cause of CTE:

- '[A]n exploration of the literature through each of [Bradford] Hill's nine viewpoints suggests an extremely high likelihood of a causal relationship [between RHT and CTE]. That conclusion is bolstered by the complete absence of evidence for plausible alternative hypotheses.'
- '[T]here does not appear to be any other common variable or possible mechanism that would explain why so many contact sport athletes, in diverse sports, and in multiple countries, are being diagnosed with CTE, while individuals without [RHT] exposure are not.'
- 'The extraordinary statistical relationship between [RHT] and CTE, both in strength of association as well as dose-responsiveness, supports causality.'
- '[T]he largest neuropathologic studies ever published of American football and ice hockey players have revealed an increased OR for developing CTE exceeding 1.2 for *each* additional year of play, on par with studies of years of smoking and lung cancer.'
- '[N]umerous independent research groups, using data from multiple independent brain banks with varying selection criteria, have consistently diagnosed CTE nearly exclusively in individuals with documented exposure to [RHT].'[72]

After noting that 'evidence on the link between [RHT] and CTE is imperfect, and like all similar research, it will remain imperfect in perpetuity', Nowinski affirmed a strong positive relationship between RHT and CTE:

> After reviewing the medical literature on [RHT] and CTE through the Bradford Hill criteria, we have the highest confidence in the conclusion that [RHT] *causes* CTE. We encourage the medical, scientific and public health communities to now act under the premise of a causal relationship and take immediate action to prevent CTE, minimize risk, and develop therapeutics to slow or stop disease progression.[73]

While the criteria are nine in number, a 'probable' correlation does not require each to be operative. Indeed, French CJ noted in *Amaca Pty Ltd v Booth* that expert witness Prof Henderson applied six only: 'capacity to induce', 'dose-response', 'experiment',

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[71] Ibid [45].

[72] Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1, 12. Original emphasis.

[73] Ibid 14. Original emphasis.

'biological plausibility', 'temporality' and 'analogy'.[74] It is informing that Nowinski found a positive causal relationship between *all nine* criteria and CTE.

4.4.3 Is the Nowinski study factually and legally acceptable?

As seemingly the only scientist to have applied the Bradford Hill criteria establishing RHT as cause of CTE, some emphasis is placed in this appendix upon the expressed 'confidence' of Dr Nowinski in declaring RHT in the playing of contact sport as causative, raising the question of whether the assessment of Nowinski and his team is sufficient for the purposes of inferring legal causation of CTE. To give some perspective to this question it should be said initially that, as explained in Chapter 6, legal causation is proven not on the scientific standard but according to the legal standard 'on the balance of probabilities'.

Acceptance of the criteria as applied by Nowinski is not, however, universal. Fortington, an injury epidemiologist, believes some of the criteria in Nowinski's study when considered as an epidemiological tool underestimate the possible contribution to CTE of other factors:

> Considerable uncertainty among experts remains about possible environmental causes of canonical neurodegenerative diseases (e.g. Alzheimer's disease, Parkinson's disease, frontotemporal dementia, amyotrophic lateral sclerosis) and in fact the causes of most neurodegenerative diseases are yet to be established despite significant research efforts.[75]

Concerns are also expressed as to differentiating between concussive and sub-concussive events where 'impact events are described in terms of the outcomes from them, rather than in terms of the bio mechanical characteristics of the impacts themselves'. Imperfections in the collection of data — such as utilising years of a contact sport played as a proxy for the quantity of head trauma experienced — were also noted. In conclusion, Fortington opines that Nowinski's study does not support a finding of definitive causation. Her view is that until 'methodological work to establish clearly defined and quantifiable variables, [and] well-designed cohort studies, with attention on a wide range of candidate risk and protective factors' are conducted, with 'rigorous scientific evaluation of the evidence', 'the scientific community, and all those who distribute research findings, must be cautious of making or accepting causal claims in this field'.

Fortington's favouring of an epidemiological assessment reflects the scientific standard rather than the legal standard. To illustrate, Fortington said: 'In order to say that A leads to B we first need to understand what A and B are. Scientific consensus on [RHTs] and CTE-NC is

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[74] *Amaca Pty Ltd v Booth* (2011) 246 CLR 36, [44].

[75] LV Fortington et al, 'Epidemiological Principles in Claims of Causality: An Enquiry into Repetitive Head Impacts (RHI) and Chronic Traumatic Encephalopathy (CTE)' (2025) 55(2) *Sports Medicine* 255, 258.

lacking — we don't have a clear understanding of what the exposure and outcomes are, and we don't have suitable ways to accurately record them.'[76]

A co-author, New Zealand Rugby's Chief Scientist Dr Ken Quarrie, commented that '[u]ntil the findings of several high-quality studies are published, the scientific community, and all those who distribute research findings, must be more critical of causal claims in this field'.[77] In summary, Fortington's study applies a 'modern epidemiological approach' to find that Nowinski's assertions of 'causality are premature'.

However, as indicated several times previously, epidemiological evidence is but one avenue of consideration and not determinative. As summed up by Spigelman CJ in *Seltsam Pty Ltd v McGuiness*:

> The courts must determine the existence of a causal relationship on the balance of probabilities. However, as is the case with all circumstantial evidence, an inference as to the probabilities may be drawn from a number of pieces of particular evidence, each piece of which does not itself rise above the level of possibility. Epidemiological studies and expert opinions based on such studies are able to form 'strands in a cable' of a circumstantial case.[78]

Of the difference between the scientific standard and the legal standard, Spigelman CJ in the same case commented:

> When assessing expert evidence on causation, the legal concept of causation requires the court to approach the matter in a distinctively different manner from that which may be appropriate in either philosophy or science, including the science of epidemiology.[79]

Herron CJ remarked in *EMI (Australia) Ltd v BES*:

> It may be, and probably is, the case that medical science will find a possibility not good enough on which to base a scientific deduction, but courts are always concerned to reach a decision on probability and it is no answer, it seems to me that no medical witness states with certainty the very issue which the judge himself has to try.[80]

Fortington's concern, as she states, is directed to establishing a 'scientific consensus'. However, the standard of proof relevant to legal causation is not to the strict scientific standard, but to the legal standard of 'likely'. As Kitto J said,

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[76] 'New Publication Queries Evidence from Studies of Repetitive Head Impacts and CTE', *Edith Cowan University* (Web Page) <https://www.ecu.edu.au/newsroom/articles/research/new-publication-queries-evidence-from-studies-of-repetitive-head-impacts-and-cte>.

[77] Ibid. See also GL Iverson et al, 'Examining Later-in-Life Health Risks Associated with Sport-Related Concussion and Repetitive Head Impacts: A Systematic Review of Case-Control and Cohort Studies' (2003) 57 *British Journal of Sports Medicine* 810.

[78] [2000] NSWCA 29, [98].

[79] Ibid [142].

[80] [1970] 2 NSWR 238, 242.

> I agree that no ground for an inference is to be found in general considerations as to the likelihood of negligent conduct occurring in the not pass from the realm of conjecture into the realm of inference until some fact is found which positively suggests, that is to say provides a reason, special to the case under consideration, for thinking it likely that in that actual case a specific event happened or a specific state of affairs existed.[81]

Many of the criteria presented by Nowinski are objective facts, which carry considerable weight in forming an inference of causation, present a strong argument of a claim beyond the 'realm of conjecture' referred to by Kitto J.[82] As Lord Wright opined:

> Inference must be carefully distinguished from conjecture or speculation. There can be no inference unless there are objective facts from which to infer the other facts which it is sought to establish. In some case the other facts can be inferred with as much practical certainty as if they had been actually observed. In other cases the inference does not go beyond reasonable probability. But if there are no positive proved facts from which the inference can be made, the method of inference fails and what is left is mere speculation or conjecture.[83]

To take but two Bradford Hill criteria discussed by Nowinski, those of 'consistency' and 'specificity', both criteria are objectively certain. With respect to 'consistency', CTE is found across a wide range of contact sports and is confirmed through its presence in other activities that involve RHT and across multiple nations where contact sport is played. 'Specificity' is objectively clear because of the fact that RHT is the only known unifying factor among all cases of CTE.

The concerns expressed within Fortington's article are obviously to be considered; however, in terms of a legal obligation of an SGB to inform itself of the risk of CTE and to warn of that risk, these concerns must be categorised similarly to the duty recognised in *H v Royal Alexandra Hospital*[84] requiring the hospital to warn of the risk of blood products being contaminated with AIDS *prior* to the fact being established with scientific certainty.[85]

In the Nowinski study, the standard of proof is on the balance of probabilities and based on inferences formulated from expert evidence, including any affirmation from other researchers. Nowinski's is but one study contributing to an inference of causation of CTE by RHT in the playing of contact sport. Multiple studies referenced throughout this thesis testify to the same conclusion. As said by Spigelman CJ:

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81 *Jones v Dunkel* (1959) 101 CLR 298, 305

82 'Inference' is discussed in greater detail in Chapter 6: 'Proof *In Vivo* of Actual Harm in the Form of CTE: The Question of "Individual Agency"'.

83 *Caswell v Powell Duffryn Associated Collieries Ltd* [1940] AC 152, 169–70.

84 [1990] 1 Med LR 297.

85 See Chapter 4: 'CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care'.

> [A]s is the case with all circumstantial evidence, an inference as to the probabilities may be drawn from a number of pieces of particular evidence, each piece of which does not itself rise above the level of possibility.[86]

Worth noting is that Nowinski's study has been quoted and referenced, and its conclusions endorsed in several scientific publications. For example, medicos Kim and Beran state, in referring to the Nowinski article, that '[t]he general consensus of the medical and scientific community is unanimous in that there is a cause-effect relationship [between RHT and CTE]'.[87] McKee et al noted with reference to the study that '[t]he Bradford-Hill criteria are the most frequently cited framework for causal inference in epidemiologic studies and include nine aspects of association by which to gauge epidemiological evidence for causation'.[88]

The use of the Bradford Hill criteria appears to find some support with legal commentator Greenhow, who states: 'In 2022, a group of 14 researchers and physicians collaborated on an alternative methodological approach — the Bradford Hill Criteria for Causation. The differences in methodological approaches across the CiSG and the proponents of alternative approaches has divided opinions and will likely continue in the future. In the meantime, it is worth asking the question: what if the CiSG methodological approach is wrong and the alternative approach is adopted.'[89] Why the Bradford Hill methodology would divide opinions is not stated by Greenhow, although it would seem that the medicos associated with major SGBs of contact sport say that a methodology establishing an epidemiological cause to CTE in playing contact sport, or conducting a 'randomised controlled trial (RTC)' is necessary before CTE can be accepted as a real disease.

However, as said by McNamee: 'Yet RCTs are not always feasible due to a variety of reasons: ethical (eg, deliberately inflicting concussions in humans even with their consent is unlikely to pass any kind of ethical review board) and practical (eg, how to keep participants blinded with respect to their assigned group?).'[90]

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[86] *Seltsam Pty Ltd v McGuiness* [2000] NSWCA 29, [98].

[87] PS Kim and RG Beran, 'Legal Medicine Implications of a Multidisciplinary Approach to Managing Traumatic Encephalopathy Syndrome in Australia' (2023) 14 *Frontiers in Neurology* 1179319, 1.

[88] Ann McKee et al, 'Chronic Traumatic Encephalopathy (CTE): Criteria for Neuropathological Diagnosis and Relationship to Repetitive Head Impacts' (2023) 145 *Acta Neuropathologica* 371, 394. See also L Filip and S Ilut, 'Chronic Traumatic Encephalopathy and Repetitive Head Impacts' , *Academy for Multidisciplinary Neurotraumatology* (Web Page) <https://brain-amn.org/repetitive-head-impacts/>; Royal College of Pathologists of Australasia, *Position Statement: Chronic Traumatic Encephalopathy (CTE)* (October 2023); R Byard, 'Chronic Traumatic Encephalopathy (CTE) — Features and Forensic Considerations' (2023) 19 *Forensic Science, Medicine and Pathology* 620; J Manning, 'Breakthrough Study Reveals Repetitive Head Impacts Are a Definitive Cause of CTE: Sports Organizations Must Acknowledge that Head Impacts Cause CTE to Protect Children', *Concussion Legacy Foundation* (Web Page, 26 July 2022); E Anderson et al, 'Sport Structured Brain Trauma is Child Abuse' (2023) *Sport, Ethics and Philosophy* 1.

[89] A Greenhow, *The Brain Matters: Regulation of Concussion in Sports-related Injuries* (LexisNexis, 2024) 115. Nowinski's article and the original article by Bradford Hill were not, curiously, referenced in Greenhow's footnotes.

[90] Mike McNamee et al, 'Sport-related Concussion Research Agenda Beyond Medical Science: Culture, Ethics, Science, Policy' (2025) 51 *Journal of Medical Ethics* 68, 70 (published online March 2023).

In addition, it should be emphasised that the Bradford Hill criteria are recognised as a means by which to judge the efficacy of epidemiological findings. In *Seltsam Pty Ltd v McGuiness* Spigelman CJ commented that '[t]here is widespread acceptance amongst epidemiologists of the principles or postulates which are applied to assess the evidence of a statistical correlation or association'.[91] His Honour went on to state:

> These postulates or criteria are uncomplicated statements of commonsense propositions. … The postulates or criteria are all matters which a court can take into account in determining whether or not it should infer, on the balance of probabilities, that a particular exposure caused injury in the specific case before the court.[92]

Ultimately, as stressed in this thesis, the matter returns to the question of 'legal proof':

> When assessing expert evidence on causation, the legal concept of causation requires the court to approach the matter in a distinctively different manner from that which may be appropriate in either philosophy or science, including the science of epidemiology.[93]

His Honour concluded by stating that '[a]n inference of causation for purposes of the tort of negligence' may be drawn by a court, where a 'a scientist, including an epidemiologist' would not draw such an inference'.[94]

4.5 Other means of proving generic capacity

In 2023, members of the Australian Senate published a report titled *Concussions and Repeated Head Trauma in Contact Sports*.[95] Many submissions on CTE-like harm recorded in the report are likely to be of use to a plaintiff athlete in attempting to prove the generic capacity of contact sport to cause CTE. Being published in 2023, clearly after the estimated date of *circa* 2010 by which CTE would likely be declared an 'obvious risk' of playing contact sport, the report itself is of no use in that regard to a defendant SGB.

However, within the report are several submissions that question the methodology by which reports of a link between RHT in playing contact sport and CTE are said to be substantiated. These submissions by and large look for a higher level of assessment to the scientific standard. For example, the Australian Sports Commission stated that 'there is currently a lack of high-quality evidence indicating the degree of association between RHT[repeated head trauma] and concussion with CTE-NC [Chronic Traumatic Encephalopathy Neuropathological Change]'.[96] Many of the submissions refer to a failure to include factors such as 'alcohol abuse, recreational

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91 *Seltsam Pty Ltd v McGuiness* [2000] NSWCA 29, [139].

92 Ibid [140]–[141].

93 Ibid [142].

94 Ibid [143].

95 The Senate, Community Affairs References Committee, *Concussions and Repeated Head Trauma in Contact Sports*, September 2023.

96 Ibid [3.38], quoting Australian Sports Commission, Submission No 10, 3.

drug use, education exposure, past history of psychiatric illness and genetic factors [that] interact with repeated head trauma [and] will only be discovered by appropriately structured, prospective, longitudinal studies that include a control group of individuals who have not been exposed to repeated head trauma'.[97] As noted in this appendix, such shortcomings are accepted by those proffering the ready acceptance of CTE as a disease, but who claim they do not alter the essential nature of the occurrence of CTE — RHT in the playing of contact sport.

Nonetheless, from a plaintiff athlete's perspective, the 'Committee view' offers the following affirmation:

> There is clear evidence of a causal link between repeated head trauma and concussions and subsequent neurodegenerative diseases such as CTE. While important research questions remain regarding the degree of causation and the nature of long-term impacts, these questions should not be used to undermine the fundamental nature of that link.[98]

The Senate report and its relevancy has been discussed only briefly. Again, it should be noted that the submissions heard by the Senate Committee were not infrequently opinion-based (though perhaps, but not stated, based on extensive research), and were not given under oath, nor subject to cross-examination.

4.6 Conclusion

This appendix informed that a plaintiff athlete must prove any fact relevant to the question of causation: 'The issue … is whether one substance that *can* cause injury *did* cause injury.'[99] Several recognised means by which a plaintiff may prove to the legal standard on the balance of probabilities that RHT in the playing of contact sport is a cause of CTE. While a particular plaintiff athlete may be able to show that the specific contact sport he or she participated in possessed the generic capacity to cause CTE, it is likely — given a commonality across the major contact sports — that any research revealing causation by participation in any contact sport will form a 'strand in the cable'[100] to determine an inference of exposure to RHT as a cause of CTE.

In the absence of direct evidence, causation of harm is proven by drawing an inference from facts and circumstantial evidence. Several methods are recognised in law as providing a factual source from which to draw an inference that CTE is caused in the playing of contact sports. The 'general body of medical knowledge' recorded in hundreds of medical research

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97 Ibid [3.42], quoting Dr David Hughes, Chief Medical Officer, Australian Institute of Sport, *Committee Hansard*, 1 March 2023, 39.

98 The Senate, Community Affairs References Committee, *Concussions and Repeated Head Trauma in Contact Sports*, September 2023, [3.124].

99 *Amaca Pty Ltd v Ellis* (2010) 240 CLR 111, [10]. Emphasis added.

100 *Seltsam Pty Ltd v McGuiness* [2000] NSWCA 29, [151].

publications since that of Dr Martland in 1928[101] (and indeed, retrospectively, well into the nineteenth century) have recognised a causative link between RHT and CTE — whether described as *dementia pugilistica* or punch drunk, or through its long-term widely recognised symptoms. An inference may be drawn from several disparate sources[102] — a fact recognised in research studies that tie together multiple symptoms and sequelae of CTE commonly found in CTE sufferers, including physical, mental and behavioural deterioration and death.

A recognised 'causal connection' occurs where one event, such as RHT, is shown to cause a second event, such as CTE; then, should the second event occur following the first event, causation will be accepted.[103] The connection between RHT and CTE affirmed at autopsy suggests a 'first to second event' relationship. Expert evidence is also an accepted means of inferring causation of harm, to which hundreds of science research journal articles testify.

The Bradford Hill methodology, widely recognised by Courts of Appeal in Australia, offers perhaps the most advanced means of proving causation of CTE by RHT to the legal standard. The utilisation of the Bradford Hill criteria by Dr Nowinski indicated a causative connection between RHT in the playing of contact sport and CTE across all nine criteria — an unusual occurrence in most relationships. Nowinski's application of the Bradford Hill criteria was directed specifically to the question of the causation of CTE.[104]

By the utilisation of the Bradford Hill criteria in several High Court judgments and the sound use of its criteria across several methodologies, most prominently that of traumatic encephalopathy syndrome (TES), the argument that RHT in the playing of contact sport has the generic capacity to cause CTE.

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[101] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

[102] *Chamberlain v The Queen* (1984) 153 CLR 521, 536.

[103] *Amaca Pty Ltd v Booth* (2011) 246 CLR 36, [121].

[104] Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1.

Appendix 5: Multiple Causes of CTE and 'Material Contribution' to Harm

5.1 Introduction

All premier-level athletes invariably played contact sport in a junior adolescent competition. This fact means that if challenged by the defendant on causation of actual harm, the plaintiff athlete, who carries the burden of proving any fact relevant to the issue of causation,[1] must be able to prove on the balance of probabilities that the CTE with which he or she claims to suffer was not caused solely by play as a junior. Without provision of such proof, a defendant SGB has a strong argument to absolve itself from charges of causation of CTE by claiming that head trauma *prior* to an athlete's professional career with the SGB or when playing other contact sports was the cause the plaintiff athlete's CTE. The question is resolved by proof that head trauma, whether concussive or sub-concussive, in each year of play — accumulating over the junior and senior years in all contact sports played — is unavoidable. In other words, multiple sources of RHT 'materially contributed' to the causation of CTE. The same response applies to a defendant's claim that the plaintiff's CTE was caused by RHT caused while he or she was playing in a competition organised by a different SGB.

The legal descriptor 'material contribution' refers to harm caused to a plaintiff by more than one tortfeasor. To make the point in context: from the first time an athlete plays contact sport, they are vulnerable to concussive and sub-concussive head trauma. This does not mean that all contact sport athletes will suffer from CTE and, in fact, in the absence of demonstrable cognitive damage no legal issue arises. It does mean, though, that in all but the most unusual of circumstances, an athlete who develops symptoms of CTE began to accumulate the RHT that causes CTE in their first season of playing contact sport and in every game subsequent.

Reflecting the relevance of a claim that the plaintiff's CTE was not caused by head trauma in the defendant SGB's competition Elise Michael, a legal commentator writing on the 2015 US National Football League (NFL) concussion settlement, said that had the matter proceeded to trial, the players would have been hard pressed to establish a causal nexus between 'concussion and football' as 'scientists cannot show a direct relationship between professional

[1] *Civil Law (Wrongs) Act 2002* (ACT) s 46; *Civil Liability Act 2002* (NSW) s 5E; *Civil Liability Act 2003* (Qld) s 12; *Civil Liability Act 1936* (SA) s 35; *Civil Liability Act 2002* (Tas) s 14; *Wrongs Act 1958* (Vic) s 52; *Civil Liability Act 2002* (WA) s 5D.

football and the long-term cognitive impairment because there may be extraneous factors, such … concussions from youth leagues or high school or college'.[2]

Causation of harm is a question of fact. Without the acquisition and presentation of factual evidence sufficient to prove that harm was caused, or materially contributed to in the defendant SGB's competition, the plaintiff athlete is unable to prove his or her case. As such, this appendix engages with extensive scientific research for the purpose of establishing that head trauma is an inherent risk of any contact sport and, moreover, is unavoidable irrespective of the level of a player's participation.

Also considered in this appendix a possible application of material contribution as an 'exceptional case' under relevant provisions of state civil liability legislation.

5.1.1 This appendix proceeds as follows

Section 5.2: The concept and operation of 'material contribution' in law

The section explains the concept of 'material contribution' to harm — in essence, where more than one source of harm is a cause of the plaintiff's injury. The section illustrates its operation through the facts and determinations of several cases, and explains that each material contribution to harm must be proven as a 'necessary condition'.

Section 5.3: 'Material contribution' and the joinder of other sporting organisations

This section reviews broadly the various other contact sport competitions that could have contributed to the RHT that caused the athlete plaintiff's CTE and thereby be joined as co-defendants. The section points out the financial advantage of a defendant SGB joining these entities as contributing tortfeasors to payment of damages in compensation. The section also points out, however, that liability is joint and several, meaning that the defendant SGB may be liable for the entire payment should the other tortfeasors be impecunious.

Section 5.4: The fact of multiple tortfeasors and cumulative head trauma

Without factual evidence that counters the argument that all CTE inducing RHT occurred in the junior years of play, the SGB has the defensive claim that either none occurred while the plaintiff played in its competition or, if it did occur, the critical threshold had already been

[2] Elise Michael, 'School of Hard Knocks — The Impact of the NFL Concussion Litigation' [2015] 33 *Cardozo Arts and Entertainment Journal* 289, 306. A possible association between CTE and steroid use is now rarely mentioned as an issue, and, as noted by Christopher Nowinski, 'Applying the Bradford Hill Criteria for Causation of Repetitive Head Trauma Impacts and Chronic Traumatic Encephalopathy' (2022) 13 *Frontiers in Neurology* 1, has been largely dismissed as a causative factor. Note also that 'CTE can only be diagnosed by post-mortem neuropathologic examination of the brain tissue': K Pierre et al, 'Chronic Traumatic Encephalopathy: Update on Current Clinical Diagnosis And Management' (2021) 9 *Biomedicines* 415, 417. See the discussion in Appendix 1 at **[1.2]**: 'The US NFL and NHL litigations'.

reached, such that the SGB's failure to warn made no difference to the plaintiff's ultimate development of CTE — in effect, that the SGB did not contribute to a result that had already been determined.

This section presents fact-based evidence to counter an SGB's argument that it made no material contribution to the plaintiff's CTE or, alternatively, if RHT was experienced by the plaintiff athlete while playing in the SGB's competition, this made no difference in science or law because the threshold at which tau is released from the axons of the brain had already been reached in the years the plaintiff played junior contact sport. In essence, the argument is made through the presentation of scientific studies that RHT is unavoidable at any level at which contact sport is played; and, further, that scientific evidence does not indicate that the threshold for the release of tau is reached in the junior years — rather, the threshold is reached at some point through the accumulation of head trauma in all games from junior to premier to retirement.

Section 5.5: Evaluation of 'material contribution'

This brief section summarises and evaluates the relevance of 'material contribution' to proving the plaintiff athlete's case that the SGB caused the symptoms of CTE with which he or she suffers. The section argues that several of the studies presented in this appendix and throughout the thesis indicate that such an outcome with respect to a premier-level SGB is a near impossibility, other than where a player had an extremely limited premier career. These studies, it is argued, show that the contribution of an SGB to the RHT experienced by a plaintiff athlete exceeds the *de minimis* level and is thereby causative in law.

Section 5.6: Multiple tortfeasors and the 'exceptional case'

This section reviews the statutory and common law application of the 'exceptional case' and the advantage it may offer to both a defendant and tortfeasor. It also considers whether the exceptional case provisions of civil liability legislation apply, such that causation need not be proven on the balance of probabilities but, rather, a to a lesser standard. The rationale is drawn from judgments affirming that where the degree of material contribution cannot be ascertained, the exceptional case provisions may apply where countenanced by 'established principles'.

The section also considers a possible SGB argument that sufficient head trauma was experienced by the plaintiff athlete in junior competition; thus the SGB could not be responsible for a disease that had already occurred. Alternatively, and more logically, is the argument that the plaintiff participated in several senior competitions for different SGBs, perhaps switching between each on several occasions, such that each, alone, is likely to have caused the threshold to be reached at which tau is released from the axons of the brain — a situation of 'multiple sufficient causes'.

Section 5.7: Conclusion

This concludes that a defendant SGB will almost certainly — based on the factual scientific evidence and inferences formed from that evidence — be considered in law a 'material contributor' to a plaintiff athlete's contraction of CTE.

5.2 The concept and operation of 'material contribution' in law

A 'material contribution' to the causation of the plaintiff's harm occurs where a defendant's negligence contributes, along with the negligence of another or several defendants, to the harm suffered by the plaintiff. Liability is not dependent on proof of one defendant's negligence.[3]

'Material contribution' (excluding the 'exceptional case') must be proven by a plaintiff as a 'necessary condition'.[4] In *Merck Sharp & Dohme (Australia) Pty Ltd v Peterson*,[5] the plaintiff claimed that the anti-inflammatory drug Vioxx caused a heart attack. Several other risk factors were listed by the court, including obesity and hypertension. Allsop P found causation had not been proven, stating that

> [t]he Court must proceed on the footing that for [the plaintiff] to show that the consumption of Vioxx materially contributed to his MI [myocardial infarction] … he is obliged to show that his consumption of Vioxx was a *necessary condition* for the occurrence of the heart attack. … To say that the consumption of Vioxx was, for example, 'in the mix' of possible causes is not enough in this regard.[6]

Material contribution was the basis of the House of Lords determination of liability in *Bonnington Castings Ltd v Wardlow*.[7] The relevant facts centred on the accumulation of silica particles from two different sources that each contributed to the plaintiff's harm, an arguable parallel to CTE caused by the accumulation of RHT by the material contribution of playing contact sport in more than one competition.

In *Bonnington*, the plaintiff was employed by the defendant on the factory floor over an eight-year period, during which he contracted pneumoconiosis from inhaling minute particles of silica. Two sources of silica leakage were identified: (1) from 'swing grinders' with blocked extractors, for which the employer would be liable in negligence; and (2) from pneumatic hammers, for which the employer would not be liable: 'As there was no known means of collecting or neutralising this dust, and as it is not alleged that these machines ought not to have been used, there was no breach of duty on the part of the [defendant] in allowing this dust to

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3 *Strong v Woolworths* (2012 246 CLR 182, [22].

4 See the following quote of Allsop P.

5 (2011) 284 ALR 1.

6 Ibid [104]. Emphasis added.

7 [1956] AC 613.

escape into the air.'[8] It should be noted that the harm to the plaintiff was not categorised as due to 'multiple sufficient causes' (where any cause is sufficient in itself to cause the total harm); rather, in the words of Lord Keith of Avonholm,

> the particles inhaled are acting cumulatively, and I think the natural inference is that had it not been for the cumulative effect the [plaintiff] would not have developed pneumoconiosis when he did and might not have developed it at all.[9]

Lord Reid recognised similarly that 'pneumoconiosis is caused by a gradual accumulation in the lungs of minute particles of silica inhaled over a period of years'; consequently, the disease 'is caused by the whole of the noxious material inhaled and, if that material comes from two sources, it cannot be wholly attributed to material from one source or the other'.[10]

In continuing, his Lordship revealed the logic to the concept of 'material contribution':

> It appears to me that the source of [the plaintiff's] disease was the dust from both sources, and the real question is whether the dust from the swing grinders *materially contributed* to the disease. What is a material contribution must be a question of degree. A contribution which comes within the exception *de minimis non curat lex* is not material, but I think that any contribution which does not fall within that exception must be material. I do not see how there can be something too large to come within the *de minimis* principle but yet too small to be material.[11]

Notably, his Lordship considered that the contribution of the swing grinders to causing a 'guilty atmosphere'[12] exceeded the *de minimis* and was thereby material:

> It may be that, of the noxious dust in the general atmosphere of the shop, more came from the pneumatic hammers than from the swing grinders, but I think it is sufficiently proved that the dust from the grinders made a substantial contribution.[13]

Several cases in Australia have considered 'material contribution' through multiple causative factors and are examined below.

In *Amaca Pty Ltd v Ellis*[14] Mr Cotton contracted and died of lung cancer, the claim in negligence against his employer being initiated by his executor, Ellis. The question of concern for the High Court was 'whether it had been shown to be more probable than not that exposure to asbestos was a cause of Mr Cotton's death'.[15] Cotton had smoked on average somewhere between 15 and 20 cigarettes a day for over 26 years. Epidemiological evidence assigned a

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8 Ibid 615.

9 Ibid 626.

10 Ibid 621.

11 Ibid. Emphasis added.

12 Ibid 617.

13 Ibid 622.

14 *Amaca Pty Ltd v Ellis* (2010) 240 CLR 111.

15 Ibid [68].

probability of no more than 23 per cent that his cancer was caused by exposure to asbestos whether he was a smoker or not.

The High Court found:

> It was not shown to be more probable than not that asbestos was a cause of (a necessary condition for) his cancer. It was not shown that exposure to asbestos made a material contribution to his cancer. Material contribution was not shown because a connection between Mr Cotton's inhaling asbestos and his developing cancer was not demonstrated.[16]

The court explained the difference between Cotton's case and that of the plaintiff in *Bonnington*:

> The issue in *Bonnington Castings* was whether one source of an injurious substance contributed to a gradual accumulation of dust that resulted in disease. The issue here is whether one substance that *can* cause injury *did* cause injury.[17]

A difficulty the plaintiff faced in *Amaca v Ellis* was that the substances purported to have caused Cotton's cancer were different, one being asbestos and the other cigarette smoke. Had the substances been the same, each, given the *Bonnington* decision, may have been ruled as a material contributor to Cotton's harm. This point was affirmed by the High Court in *Amaca Pty Ltd v Booth*: '[T]he evidence in the present case … was that, unlike the situation regarding [Mr Cotton's] lung cancer [in *Ellis*], exposure to asbestos is effectively the only known cause of mesothelioma.'[18]

In *Amaca v Booth* the High Court considered the application of material contribution where two companies were each charged with causing the plaintiff's mesothelioma. Both defendants, as noted above, exposed the plaintiff to the same agent of harm: asbestos. The plaintiff was a motor mechanic who worked on brake linings containing asbestos that were manufactured by the defendants — he worked for nine years with Amaca Pty Ltd and for 19 years with Amaba Pty Ltd. Two other minor sources of asbestos exposure were considered *de minimis*.

The High Court majority found that each company had materially contributed to the plaintiff's harm. French CJ stated: 'The cumulative effect mechanism, accepted by [the judge at first instance], implicated the products of both Amaca and Amaba in the development of [the plaintiff's] disease.'[19]

It is clear, of course, that the agent of harm in the contraction of CTE is repetitive, or 'cumulative', head trauma experienced during in playing in more than one contact sports competition. In the context of a plaintiff athlete suffering CTE-like harm, the process of

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16 Ibid [65].

17 Ibid [68]. Original emphasis.

18 *Amaca Pty Ltd v Booth* (2011) 246 CLR 36, [71].

19 Ibid [51].

accumulation began as a junior and continued into senior professional competitions, concluding only with the last game of the athlete's career.[20]

5.3 'Material contribution' and the joinder of other sporting organisations

Possessing potential benefits for a defendant governing body is the joining as co-defendants of other sporting organisations as 'material contributors' to the athlete's harm in the form of CTE. Each entity that is found negligent is jointly and severally liable. Several possible combinations emerge:

1. the teams the athlete played for as a junior;

2. the governing bodies of different codes of sport that the athlete played, where head trauma is a trait common to each; for example, rugby, rugby league or Australian Football League (AFL);

3. other governing bodies administering the same sport; for example, if the athlete played in the National Rugby League (NRL) competition in Australia and the English Super League; or in an Australian state team in the Super Rugby tournament as well as, say, a New Zealand or South African rugby team or another state rugby team.

The discussion below refers in the main to 'junior sport', given its commonality to all premier players. Nonetheless, the approach is also relevant to plaintiff athletes who played for senior sporting organisations other than a defendant SGB.

5.3.1 Possible combinations

Several combinations as between junior or senior contact sport and the causation of actual harm in the form of CTE are proposed:

1. The only head trauma the athlete incurred was as a junior player.

2. The only head trauma the athlete incurred was as a senior player.

3. The athlete incurred head trauma in playing both as a junior and senior player.

Proposition one, which is largely theoretical if not fanciful, would absolve a major sporting organisation of responsibility in causing CTE-like harm to the athlete concerned. Propositions two and three suggest liability.

[20] The scientific and factual basis to material contribution through play in multiple contact sport competitions is discussed below at **[5.4]**.

5.4 The fact of multiple tortfeasors and cumulative head trauma

Few athletes who stop playing contact sport at, say, age 18 or 19, come later in life to suffer from CTE, suggesting, on one argument, that all or at least most cognitive harm is incurred in senior sport alone. A contrary argument suggests that a retired senior player who suffers CTE but incurred no concussive blows as a senior must, thereby, have incurred all CTE-causative head trauma as a junior player. Opposed to these propositions is the practical impossibility of avoiding even mild head trauma in contact sport, at any level, at any age. Here, the recognition that CTE is caused not only by repetitive concussive blows to the head, but also asymptomatic sub-concussive blows is material.[21] That is, the avoidance of symptomatic concussion does not indicate an absence of CTE-causing head trauma. As the argument goes, irrespective of having avoided symptomatic concussions as either a junior or a senior player, the sub-concussive blows accumulated as a junior or senior athlete are recognised causes contributing to CTE in later life.

5.4.1 The scientific fact of head trauma in junior and senior contact sport

This section concerns the argument, supported by factual evidence, that concussive and sub-concussive trauma incurred in playing both junior and senior contact sport in Australia materially contributed to the causation of CTE. The purpose is not to measure the precise quantity and severity of concussions in junior and senior contact sport, but rather to show that head trauma is common to both and, should it be necessary, to counter a governing organisation's argument that the plaintiff athlete sustained all head trauma in junior sport, not when playing in its senior premier sport.

Two matters relevant to the question of causation with respect to a premier-level athlete's negligence claim against a governing sporting organisation are:

1. Did the athlete suffer some concussion or asymptomatic concussion as a junior?

2. Did the contribution to harm the athlete experienced as a premier player rise above the trivial, or *de minimis*?

To expand on the second point: as causation applies only to harm that exceeds the *de minimis*, can a governing organisation claim that its contribution to a plaintiff's harm is trivial given, perhaps, that junior participants are known to be more vulnerable to harm through head trauma? On a simple level, the science of physics suggests otherwise: force is a product of mass

[21] 'Frequently Asked Questions about CTE', *Boston University Center for the Study of Traumatic Encephalopathy* (Web Page) <https://www.bu.edu/cte/about/frequently-asked-questions/>.

multiplied by acceleration, such that greater risk of experiencing head trauma is found in the heavier, faster premier grades of contact sport.[22]

Relevant also to the question of causation is that the progressive histological reaction to RHT over many years prior to the common onset of symptoms of CTE does not occur until early to late middle age. Therefore, the fact that symptoms appear more proximate to premier-level competition does not, given the decades over which CTE usually develops, necessarily lead to the conclusion that a senior governing body alone is causative.

Discussed below are several research papers concerned with head trauma in junior and senior contact sport. These papers, in the main, appraise the incidence and circumstances of symptomatic concussion. Several papers consider asymptomatic concussion as a contributing factor in CTE. The many bruises, facial cuts and abrasions seen around the head and neck of players of contact sport — both junior and senior — offer some insight into the incidence of asymptomatic concussion.

These research papers are purposed to show that head trauma is a condition of both junior and senior contact sport. The articles referenced also reveal differences in the forces of contact as between junior and senior competitions — a further point, should it be necessary, suggesting that a denial of head trauma in premier-level competition is unsustainable.

In a study of youth concussion in Canadian junior ice hockey, as observed by physicians in attendance at games, Echlin recorded: 'Twenty-one concussions were physician observed or self-reported and subsequently physician diagnosed in 52 physician-observed regular season games, yielding an incidence of 21.52 concussions per 1000 athlete exposures.'[23]

A meta-study of rugby and rugby league players under 20 years of age conducted by Kirkwood[24] examined 1,576 cases of concussion reported in medical journals published in Australia, South Africa, New Zealand, Scotland, the United States, Canada and Ireland. Using a 'concussion incidence per 1,000 player hours' methodology, the study found, as might be expected, evidence of 'a significant risk of concussion in children and adolescents playing rugby union and rugby league'.[25] Across the sample, the incidence of concussion in youth rugby 'ranged from 0.2 to 6.9 concussions per 1000 player-hours, equivalent to a probability for a player over a season of sustaining a concussion of between 0.3% and 11.4%', while that of rugby league taken from just two studies was between '4.6 and 14.7 concussions per 1000 player-hours equivalent to a 7.7% and 22.7% probability of concussion over a season …

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[22] Nonetheless, factors associated with the developing brain in junior contact sport may pose an increased risk of concussion.

[23] PS Echlin et al, 'A Prospective Study of Physician-observed Concussions during Junior Ice Hockey: Implications for Incidence Rates' (2010) 29(5) *Neurosurgical Focus* E4, 4.

[24] G Kirkwood et al, 'Concussion in Youth Rugby Union and Rugby League: A Systematic Review' (2005) 49 *British Journal of Sports Medicine* 506. The advantage of a meta-study, of course, is the collection of findings into a single study in which patterns of causation may be observed.

[25] Ibid 508.

or one or two players sustaining a concussion every season in every school or club rugby team of 15 players'.[26] Two studies of players reporting incidents of concussion relayed that '48.1% of 133 interviewees in Ireland and 14.1% of 3330 respondents in a South African survey report[ed] at least one prior concussion'.[27]

An earlier meta-study by Marshall and Spencer critically examined medical reports of concussed players from the Highland High School rugby team in Utah over the seasons 1998 to 2000. During that period, 'seventeen concussions were recorded' — 12 concussions occurred in games, giving 'a rate of 11.1 per 1000 player-games, and 5 occurred in practices [at] 1.5 per 1000 player-practices'. Overall, 'the concussion rate was 11.3 per 100 player seasons'.[28] The incidence is within the range found in Kirkwood's research.

With respect to asymptomatic concussion, a study by Bailes found that the 'evidence now suggests that head impacts commonly occur during contact or collision sports in which symptoms may not develop and there are no outward or visible signs of neurological dysfunction', suggesting it is 'the accumulation of multiple blows to the head, rather than a single event, that can produce neurological impairments'.[29] Rasmussen reviewed over 100 studies on concussion in youth sports and made the point with respect to asymptomatic concussion that 'present surveillance methods are, by definition, incapable of ever capturing [asymptomatic] impacts. This is because concussion, for the present, is clinically defined and assessed based on self-reported symptoms and cognitive-behavioral testing.'[30]

At times, studies conflict. For example, 'a cohort study of Wisconsin men who played football in the late 1950s found no relationship between football participation and the onset of cognitive impairment or depression in later life'.[31] In contrast, Rasmussen found that

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[26] Ibid 508–9.

[27] Ibid 509. Of collateral interest, Kirkwood (at 508) referenced a study by Collins on rugby union that found little difference in incidence between sexes: '83 concussions among the boys (16.1% of all injuries) and 11 concussions among the girls (14.3% of all injuries)' (CL Collins et al, 'Injuries Sustained by High School Rugby Players in the United States, 2005–2006' (2008) 162 *Archives of Pediatrics & Adolescent Medicine* 49).

[28] SW Marshall and RJ Spencer, 'Concussion in Rugby: The Hidden Epidemic' (2001) 36(3) *Journal of Athletic Training* 334, 335. 'Player-seasons' is the number of athletes per team multiplied by the number of seasons for which the team was followed': ibid.

[29] Julian Bailes, 'Role of Subconcussion in Repetitive Mild Traumatic Brain Injury: A Review' (2013) 119 *Journal of Neurosurgery* 1235, 1235, 1242, reported in C Rasmussen et al, 'How Dangerous Are Youth Sports for the Brain? A Review of the Evidence' (2018) 7 *Berkeley Journal of Entertainment & Sports Law* 67, 112. Of interest, Rasmussen commented (at 112–13): 'To the extent that accumulated sub-concussive impacts are a significant, or perhaps the most important, contributor to impaired mental functioning, the focus placed on "concussion incidence" itself may be misplaced.'

[30] C Rasmussen et al, 'How Dangerous Are Youth Sports for the Brain? A Review of the Evidence' (2018) 7 *Berkeley Journal of Entertainment & Sports Law* 67, 111.

[31] K Sameer et al, 'Cognition and Mental Health Later in Life' (2017) 74 *JAMA Neurology* 909, 916–17, reported in C Rasmussen et al, 'How Dangerous Are Youth Sports for the Brain? A Review of the Evidence' (2018) 7 *Berkeley Journal of Entertainment & Sports Law* 67, 114.

> athletes who began playing football before 12-years-old performed worse on cognitive tests, and had more symptoms of depression and apathy, independent of age, education and duration of play. Beginning football before 12-years-old more than doubled the risk of 'clinically meaningful impairments in reported behavioral regulation, apathy and executive function' and more than tripled the risk of 'clinically elevated depression scores'.[32]

Rasmussen concluded:

> On one hand, the data suggests that the vast majority of youth athletes will not sustain a concussion. Moreover, a majority of those who do experience a concussion will see their post-concussion symptoms dissipate within three weeks. At the same time, the data clearly shows that there are serious, non-zero risks of brain injury from playing contact sports before and during high school. These risks are elevated in collision sports, and current data collection methods likely underestimate actual incidence rates.[33]

While obviously junior players experience injuries in contact sport, as the size, speed and strength of players increase with age, training and professionalism, the force of blows to the head, face and neck also increase, and with it, the risk of head trauma.[34] McIntosh's study of youth rugby found 'a slightly higher proportion of U13s injured while being tackled compared with tackling (33% and 25%, respectively), and this is reversed with U15s (13% and 40%, respectively)'. McIntosh suggested that the reversal was 'related to skill, player size and game speed developments'.[35] Clearly the body mass of players influenced the outcome:

> Mean body mass increased by 15 kg from the U13 age group to the U15 age group, a further 9 kg in the U18 age group, and a further 10 kg in the U20 group. Stature increased from 161 cm in the U13 age group to 181 cm in the U20 age group, with an average increase of 14 cm from age groups U13 to U15.[36]

Size and speed as risk factors in New Zealand rugby were studied by Quarrie.[37] The research, which was conducted over a full season involving 258 male players with an average age of 20.6 years, found that as an athlete's size and speed increases with age, so does the incidence of harm: 'Players with a body mass of greater than 81 kg sustained a higher IR [injury rate] than players whose body mass was less than 74 kg. Players with a BMI of greater than 26.5 sustained more injuries than players with a BMI of less than 23'.[38] Similarly, 'players

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[32] C Rasmussen et al, 'How Dangerous Are Youth Sports for the Brain? A Review of the Evidence' (2018) 7 *Berkeley Journal of Entertainment & Sports Law* 67, 114, quoting Michael Alosco, 'Age of First Exposure to American Football and Long-term Neuropsychiatric and Cognitive Outcomes' (2017) 7 *Translational Psychiatry* 1, 1, 3.

[33] C Rasmussen et al, 'How Dangerous Are Youth Sports for the Brain? A Review of the Evidence' (2018) 7 *Journal of Entertainment & Sports Law* 67, 122–3.

[34] AS McIntosh et al, 'Head, Face and Neck Injury in Youth Rugby: Incidence and Risk Factors' (2010) 44 *British Journal of Sports Medicine* 188.

[35] Ibid 191.

[36] Ibid.

[37] KL Quarrie, 'The New Zealand Rugby Injury and Performance Project. VI. A Prospective Cohort Study of Risk Factors for Injury in Rugby Union Football' (2001) 35 *British Journal of Sports Medicine* 157.

[38] Ibid 161–2.

whose body mass fell in the middle quintile (81–87 kg) missed a greater proportion of their season (17%) than players with the lowest body mass'.[39] Quarrie reported that the association between body mass and injury was consistent with a previous study, which found that 'injuries had an age adjusted mean BMI of 25.4 compared with an age adjusted mean BMI of 24.6 for players who were not injured'.[40]

Similar results were recorded for speed: 'Players in the fastest group (4.06 seconds) reported a higher IR than those in the slowest (reference) group (> 4.06 seconds).' In relation to height, '[p]layers whose height was in the middle quintile (179–181 cm) missed a greater proportion of their season (16%) than the shortest players'. With respect to strength, '[p]layers who performed between 20 and 33 push ups missed a greater proportion of their playing season than those who completed fewer than 19 push ups'.[41] The risk of injury also increased with age: 'Players in the under 19/18 grades had a lower IR than players at all higher grades, and players who were injured at the preseason assessment had a higher IR than players who entered the season injury free.'[42]

Speed and size were implicated in concussion and CTE in the NFL by medico Dr John Orchard:

> Because of unlimited substitutions, heavy protective equipment and … enormous numbers of stoppages and of players on the interchange bench, American football has done away with any meaningful need for endurance, allowing players to completely focus on strength and speed. … [T]he rate of high-speed collisions has meant that the NFL has entered what is being dubbed the 'concussion crisis' era.[43]

An earlier study by Orchard compared injuries in elite senior and junior (under-18) AFL players. The six most common injuries across AFL were 'hamstring strains, ankle sprains, thigh haematomas ("cork thighs")' and, tellingly, 'head and facial lacerations, and concussion', followed by groin strains. When isolated for under-18 players the 'significantly' more common injuries were 'concussion and foot stress fractures'.[44] Overall, the study found that 'there was

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[39] Ibid 162.

[40] Ibid 164, citing AJ Lee, JL Myers and WM Garraway, 'Influence of Players' Physique on Rugby Football Injuries' (1997) 31 *British Journal of Sports Medicine* 135.

[41] KL Quarrie, 'The New Zealand Rugby Injury and Performance Project. VI. A Prospective Cohort Study of Risk Factors for Injury in Rugby Union Football' (2001) 35 *British Journal of Sports Medicine* 157, 161–2.

[42] Ibid 163. Perhaps relevant (at 163) is that '[p]layers who engaged in strenuous physical activity for 39 hours or more a week missed a greater proportion of the season than did players who were active for five hours or less a week'.

[43] J Orchard, 'Match of the Decade: Risk Management of Concussion versus High-speed Collisions in the Football Codes' (2015) 203(7) *Medical Journal of Australia* 1, 1.

[44] J Orchard, 'Comparison of Injuries in Elite Senior and Junior Australian Football (1998) 2 *Journal of Science and Medicine in Sport* 82, 86.

a higher reported injury incidence, but lower prevalence, indicating the injuries were generally less severe in the junior competition'.[45]

The frequency with which a player 'impacts' other players, either in defence or attack, also influences the likelihood of head trauma. While most players in Quarrie's study displayed 'fairly uniform injury rates' across all positions, the midfield backs, due to the frequency with which they are 'used as "battering rams" to run the ball directly at the opposing players … and the high speeds they attain when they enter tackles', missed a 'significantly greater proportion of their season' due to injury compared to the front row reference group.[46] To emphasise the point: '[M]idfield backs missed, on average, 21% of their playing season through injury,' while 'the inside backs, who are generally involved in fewer high speed impacts, missed only 7% of their season.' Injuries to midfield backs 'were, on average, more severe and kept them out of play for longer periods'.[47]

Quarrie's study did not isolate head trauma from other forms of bodily harm; it merely considered all injuries sufficient to cause an athlete to miss a match. Nonetheless, size and speed as factors of harm in general, with almost complete certainty, increase the risk of concussive and sub-concussive head trauma — after all, the head and neck are as exposed to collateral or incidental on-field collision as other parts of the body. Quarrie also noted the risk of harm in 'contact training', such as tackling practice: 'Anecdotally, players and coaches often refer to "match fitness" ... Part of this match fitness may be physical conditioning to the impacts with other players and the playing surface that players are exposed to during games.'[48]

Bailes and Cantu found: 'There are studies that clearly suggest that the damaging effects of the shearing injury to nerve fibres and neurons is proportional to the degree of acceleration to the head, and that these changes are cumulative.'[49] *Ceteris paribus*, the influence of size and speed on the degree of cognitive harm as between junior and senior contact sport is explained through Newtonian physics: Force equals mass times acceleration.[50] As a player's size and speed increase, their capacity to impact an opponent with force increases commensurately. That

[45] Ibid 87. Notably, while junior AFL players suffered more injuries, the prevalence, or severity, 'over three years and in total was significantly higher in the AFL than U/18 competition ($p < 05$)': at 87. The explanation for juniors suffering more injuries, but injuries being less prevalent, is likely the definition of injury as 'unavailability for selection in a match or training session', or '[received] specific medical treatment', which may be reflected in quicker recovery times and less severe injury in juniors.

[46] KL Quarrie, 'The New Zealand Rugby Injury and Performance Project. VI. A Prospective Cohort Study of Risk Factors for Injury in Rugby Union Football' (2001) 35 *British Journal of Sports Medicine*157, 164.

[47] Ibid.

[48] Ibid 165.

[49] JE Bailes and RC Cantu, 'Head Injury in Athletes' (2001) 48(1) *Neurosurgery* 26, 35.

[50] Isaac Newton's Second Law of Motion: F = ma.

is, whether making a tackle or being tackled, the force expended on the human body is greater for the speed and mass of each player.

A study by Mez et al of players of American football, found a strong correlation between physical size, the grade of play and the severity of CTE:

> The severity of CTE pathology was distributed across the highest level of play, with all former high school players having mild pathology (3 [100%]) and the majority of former college (27 [56%]), semi-professional (5 [56%]), Canadian Football League (6 [86%]), and NFL (95 [86%]) players having severe pathology. The mean duration of play for participants with mild CTE pathology was 13 years (SD, 4.2 years) and for participants with severe CTE pathology was 15.8 years (SD, 5.3 years).[51]

Medical studies prior to, and after, Martland's *Journal of the American Medical Association* article in 1928, 'Punch Drunk',[52] reported on CTE-like harm affecting players of contact and collision sport.[53] Although the histological link was not revealed until 2005, by Omalu et al,[54] the physical cause of CTE — RHT — was known decades earlier. There is, then, a clearly established 'causal connection' between the number and severity of blows to the head over time in both junior and senior contact sport and the disease of CTE. The studies cited above were conducted by scientists who can reasonably be regarded as experts in their field, the evidence of whom is relevant to the question of proof of causation. As the High Court remarked in *Amaca Pty Ltd v Booth*: '[A]s demonstrated by medical evidence in this case … a causal connection may be inferred by somebody expert in the relevant field considering the nature and incidents of the correlation.'[55]

5.5 Evaluation of 'material contribution'

Liability for failure to warn requires proof of two causative outcomes, each with differing levels of satisfaction.[56] The first is that a duty-holder failed to warn of a 'material risk' — irrespective of how small the material risk. Consequently, the material risk that eventuated may be minute. For example, in *Rogers v Whitaker*[57] an ophthalmic surgeon was held to be negligent in not warning of a 1-in-14,000 chance of a patient suffering sympathetic ophthalmia as a possible

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[51] J Mez et al, 'Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football' (2017) 318(4) *Journal of the American Medical Association* 360, 362.

[52] Harrison Martland, 'Punch Drunk' (1928) 91(15) *Journal of the American Medical Association* 1103.

[53] See the brief description above and detailed discussion in Chapter 2: 'The Science of Concussion and CTE in Sport: Essential to Understanding the Legal Arguments'.

[54] Bennet Omalu et al, 'Chronic Traumatic Encephalopathy in a National Football League Player' (2005) 57(1) *Neurosurgery* 128.

[55] (2011) 246 CLR 36, [49].

[56] See Chapter 4: 'CTE and the Obligation to be Informed of the Scientific Knowledge of the Day and to Warn of Material Risks: Breach of the Standard of Care'.

[57] (1992) 175 CLR 479.

result of the surgical procedure. The second causative outcome is that the failure to warn by a governing body caused actual harm to the plaintiff.

Although a plaintiff athlete must show that actual cognitive harm was caused in playing the governing organisation's sporting competition, with respect to 'material contribution' this need not be *all* harm, provided the organisation caused *some* harm. The degree of harm caused by each contributor must, however, rise above the *de minimis*; that is, it must be not so trivial or minor or too small to merit consideration. This position was made clear by Lord Reid in *Bonnington Castings Ltd v Wardlaw*: 'A contribution [to harm] which comes within the exception *de minimis non curat lex* is not material.'[58]

The research cited in **[5.4.1]** above indicates that as athletes increase in size and strength, the incidence and force of head trauma in contact sport also increases. This does not mean that head trauma experienced as a junior or adolescent is insignificant or non-existent — in fact, several studies indicate that junior athletes are at a greater risk of cognitive damage from head trauma[59] — but those withdrawing from contact sport as juniors, even after experiencing concussive and sub-concussive trauma, rarely suffer long-term cognitive damage. However, as an athlete's career extends into the senior ranks of play, trauma to the head accumulates.

Consider a possible application: An athlete competes in contact sport as a junior. During this period, he or she begins to accumulate RHT. Should the athlete retire at age 18, he or she is not likely to suffer CTE at a later age. Should the athlete continue to play contact sport, he or she will accumulate further head trauma — perhaps to the point where, as Lipton stated, the 'body's repair mechanism' is 'unable to keep pace'.[60]

Several chapters and appendices of this thesis more than adequately record the severe symptomatic and psychological harm experienced by a CTE sufferer — a level of harm that clearly exceeds the *de minimis* in both junior and senior grades of play.

Although an SGB may argue material contribution or the application of the exceptional case (see below), other than where it can show that play in its competition caused no head trauma above the *de minimis* level, a successful determination by the court is likely to afford little benefit. While junior play over several years almost invariably contributed to the head knocks that cause CTE, it is largely immaterial in reducing the financial liability of a major sporting organisation. With respect to payment of damages, one point is clear: there is little utility in a plaintiff athlete who is currently suffering CTE-like symptoms seeking compensation from junior clubs for which they played in their adolescence. Such clubs usually

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58 [1956] AC 613, 621.

59 See, eg, Kathleen Bachynski, 'Tolerable Risks? Physicians and Youth Tackle Football' (2016) 374(5) *New England Journal of Medicine* 405; C Rasmussen et al, 'How Dangerous Are Youth Sports for the Brain? A Review of the Evidence' (2018) 7 *Berkeley Journal of Entertainment & Sports Law* 67.

60 ML Lipton et al, 'Soccer Heading is Associated with White Matter Microstructural and Cognitive Abnormalities' (2013) 268(3) *Neuroradiology* 850, 854.

possess few financial resources and were at the time often run as unincorporated organisations by voluntary coaches or officials; and the teams have perhaps long been disbanded. Nor can it be expected that a junior sporting organisation operated part-time by parents and community-minded citizens possessed the legal duties expected of a major sporting organisation, or that access to relevant medical advice and information was readily available to such a junior organisation. Of course, an athlete who played for several financially viable senior organisations is advantaged in prosecuting a case against all.

In these circumstances, any financially viable organisation could well be the sole contributor to the payment of damages; although of course a sole defendant may also join other SGB's believed to have materially contributed to the plaintiff's harm as co-defendants. As Lord Rodger remarked on damages fixed to a single tortfeasor from among several other tortfeasors in a mesothelioma case:

> Defendants whose breaches of duty materially increase the risk that the victim will develop mesothelioma are liable jointly and severally for the damage which the victim suffers if he does in fact develop mesothelioma.[61]

5.6 Multiple tortfeasors and the 'exceptional case'

The 'exceptional case' is concerned with limitations inherent in the application of the 'but for' test of causation in law. To remedy this shortfall, state civil liability legislation introduced 'exceptional case' provisions built on a common law foundation. Where an SGB argues that an athlete's participation in junior contact sport and in its premier competition were each, singularly, sufficient to have caused the CTE suffered by a plaintiff athlete the 'exceptional case' provisions offer a possible means to nonetheless prove legal causation of harm without reliance on the 'but for' test. In other words, the provision addresses what is referred to as 'multiple sufficient causes' or 'causative overdetermination' which, as explained below renders the 'but for' (necessary condition) test of causation illogical and inapplicable. In addition, the provision is said, as explained below, to apply to material contribution to harm where the contribution of each cannot be ascertained.[62]

5.6.1 The statutory approach to causation as an 'exceptional case'

Section 5D(2) of the *Civil Liability Act 2002* (NSW) provides:

> In determining in an exceptional case, in accordance with established principles, whether negligence that cannot be established as a necessary condition of the occurrence of harm should be accepted as

[61] *Sienkiewicz v Grief* [2011] 2 AC 229, [141]–[142].

[62] *Review of the Law of Negligence* (Final Report, September 2002) [7.28] ('*Ipp Report*').

> establishing factual causation, the court is to consider (amongst other relevant things) whether or not and why responsibility for the harm should be imposed on the negligent party.[63]

It can be noted that the *Civil Liability Act* provides no guidance as to the application of the 'exceptional case' provisions other than 'in accordance with established principles'. As the High Court remarked:

> Whether, or when, s 5D(2) is engaged must depend, then, upon whether and to what extent 'established principles' countenance departure from the 'but for' test of causation.[64]

The High Court majority in *Strong v Woolworths* explained that s 5(D)(2) 'permits a finding of causation in exceptional cases, notwithstanding that the defendant's negligence cannot be established as a "necessary condition" of the occurrence of the harm'.[65] The exceptional case provisions, noted the Victorian Court of Appeal in *Powney v Kerang and District Health*, were 'designed to accommodate cases quite out of the ordinary — particularly those involving exposures to a particular agent on multiple occasions, all of which contributed to a disease process but factual causation could not be attributed to a specific exposure'.[66] The Court of Appeal said that the case under appeal was not exceptional because 'there is one alleged tortious act and no question of multiple causes or unknown aetiology of the alleged damage'.[67] The court's supporting reference is to the High Court's ruling in *Amaca Pty Ltd v Booth*.[68] For the exceptional case provisions to apply it is 'necessary for the court to be satisfied that the appellant was unable to establish factual causation under [s 5D(1)(a)] prior to [s 5D(2)] being engaged'.[69]

An 'exceptional case', the *Ipp Report* explained, applies to 'two types of situations in which an evidentiary gap may exist'.[70] These 'types', or as they were later termed, 'categories', are exemplified in two House of Lords decisions: *Bonnington Castings v Wardlaw*[71] and *Fairchild*

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[63] The *Civil Liability Act 2002* (NSW) s 5D(2) is largely identical in wording to the 'exceptional case' provisions in other Australian jurisdictions. See *Civil Law (Wrongs) Act 2002* (ACT) s 45(2); *Civil Liability Act 2002* (Qld) s 11(2); *Civil Liability Act 2002* (Tas) s 13(2); *Wrongs Act 1958* (Vic) s 51(2); *Civil Liability Act 2002* (WA) s 5C(2). The question of 'whether or not and why responsibility for harm should be imposed on the negligent party' is outside the scope of this thesis other than collaterally.

[64] *Adeels Palace Pty Ltd v Moubarak (*2009) 239 CLR 420, [81].

[65] (2012 246 CLR 182, [26].

[66] (2014) 43 VR 506, [96]. 'Exceptional case' is termed 'appropriate case' in Victoria.

[67] Ibid [99].

[68] (2011) 246 CLR 36, [52].

[69] *Powney v Kerang and District Health* (2014) 43 VR 506, [95]. The provisions of ss 51 and 52 of the *Wrongs Act 1958* (Vic) are almost identical to those of s 5D(1) and (2) of the *Civil Liability Act 2002* (NSW).

[70] *Review of the Law of Negligence* (Final Report, September 2002) [7.27] ('*Ipp Report*').

[71] *Bonnington Castings v Wardlaw* [1956] AC 613. Emphasis added; discussed below at **[5.64]**.

v Glenhaven Funeral Services Ltd.[72] The 'second category', known as 'inadequate scientific or medical knowledge', is not presently of concern. The first category 'cumulative operation of factors', and the concomitant application of a 'material contribution' to harm is the subject of this section.

Section 5D (2) also requires that a court consider with respect to an exceptional case the 'normative question' of 'whether or not and why responsibility for the harm should be imposed on the negligent party'. The *Ipp Report* suggested that this inclusion 'would be useful to make explicit the normative character of the issue' that 'depends ultimately on a value judgment about how the costs of injuries and death should be allocated'.[73]

5.6.2 The advantage of an 'exceptional case'

The advantage of the 'exceptional case' that it is an exception to the statutory requirement of proving causation as a 'necessary condition'. It applies where circumstances render the 'but for' test illogical in operation. French CJ in *Amaca Pty Ltd v Booth* said that the effect of the provision was to extend legal causation 'beyond the limits of factual causation'. His Honour continued: 'In the result, a new head of tortious liability appears to have been created.'[74] Prof Barbara McDonald notes that where the application of one provision against the other is 'unclear', then 'in exceptional cases involving an evidentiary gap the court may relax the causation requirements including the onus of proof. This … will be a matter of interpretation for the courts.'[75] Although some years on from the publication of Prof McDonald's article, the precise application of the exceptional case provisions is yet to be settled.

5.6.3 The application of the exceptional case

The exceptional case provisions commonly applied to cases where there were 'multiple sufficient causes' or, as it is also known, 'causative overdetermination'. The term 'multiple sufficient causes' refers to harm caused from two or more different and independent events where each event, alone, is sufficient to have caused the total damage suffered by the plaintiff. On a strict application of the 'but for' test, the harm suffered would have occurred irrespective of each defendant's negligence. Several examples are used by commentators to illustrate the concept, most commonly, the 'double hunters' case. As Stapleton explained it:

> [T]wo hunters carelessly shoot into a wood, both bullets hitting a beater who dies, the medical evidence being clear that either shot would have been sufficient to kill the beater: in philosopher's

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[72] *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32. This application is not discussed here due to reasons of length, although arguably available to the exceptional case in respect to inadequate 'scientific and medical knowledge' of CTE.

[73] *Review of the Law of Negligence* (Final Report, September 2002) [7.33] ('*Ipp Report*'). Other than in passing, discussion of s 5D(2) lies outside the scope of this thesis.

[74] (2011) 246 CLR 36, [52]. Although French CJ was referring specifically to the second category, the description is applicable to both categories.

[75] B McDonald, 'Legislative Intervention in the Law Of Negligence: The Common Law, Statutory Interpretation and Tort Reform in Australia' [2005] 27 *Sydney Law Review* 443, 477.

> jargon, the death was 'overdetermined'. Scientists see no difficulty in this case because it is quite clear how the death came about. …
>
> But lawyers regard the case as very problematic: some arguing that each hunter would be able to deny he was a 'cause' because 'but for' his act the death would still have occurred.[76]

The 'double hunters' scenario would apply in CTE cases where more than one source of head trauma leading to CTE is identified as a cause of harm, and each source is sufficient in itself to cause the plaintiff athlete's CTE (or its symptoms). The two most likely sources of sufficient head trauma leading to CTE are play as a junior and play in a premier-level competition. In such circumstances a senior competition organiser, such as the National Rugby NRL, may argue 'no liability' under the 'but for' test because, irrespective of its conduct, the plaintiff athlete would still have suffered RHT sufficient to cause CTE due to participation in junior competitions, or indeed in other senior sporting competitions. Alternatively, and more logically, where a player switched between the competitions of more than one SGB, especially where the player returned to play for the original SGB.

A logical difficulty with the application of the argument above, such as defendant SGB to inculcate a junior competition should be pointed out. If sufficient harm was caused by play in a junior competition, any head trauma experienced in the premier competition would be superfluous and play in the SGB's competitions could not be taken to have caused the plaintiff's CTE. This circumstance is equivalent to the 'double hunters' example but where one hunter shot and killed the victim seconds before the second hunter shot the now dead victim (and where it could be determined who shot first).

Alternatively, where a plaintiff athlete had switched between competitions, or played in several different senior competitions, and the head trauma experienced in each competition was sufficient to induce CTE but it was not possible to know in which competition that threshold was reached (noting that there is no set number of games to reach that threshold) the 'but for' test could *not* apply and the 'multiple sufficient causes' exception may apply. This configuration of facts is used because if it can be known the point when the threshold was reached, any competition played in after that point could not be causative of a condition that was already caused. Further, where each competition is not sufficient to cause CTE, the cause must be the material contribution made by play in each competition.

5.6.4 Can the 'exceptional case' apply to multiple contributors to CTE?

In logic, where one cause is sufficient to cause the plaintiff's total harm, that harm would have occurred irrespective of another tortfeasor's negligence. When the question is asked, 'Would the defendant have suffered harm "but for" the second tortfeasor's negligence', the answer is 'yes', hence supposedly releasing the second tortfeasor from liability.

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[76] Jane Stapleton, 'Scientific and Legal Approaches to Causation' in Danuta Mendelson and Ian Freckelton (eds), *Causation in Law and Medicine* (Taylor & Francis Group, Milton Park, 2002) 19. A 'beater' flushes birds out of the bush to direct them towards shooters.

A The classic exceptional case

The application of 'multiple sufficient causes' as exemplified in the 'double hunters' case can be described as the classic exceptional case. In conformity, Dean J stated that

> the 'but for' test as even a negative or exclusionary test of causation for the purposes of the law of negligence would lead to the absurd and unjust position that there was no 'cause' of an injury in any case where there were present two independent and sufficient causes of the accident in which the injury was sustained.[77]

Nonetheless, the exceptional case has been said to apply to material contribution where the level of contribution of each defendant cannot be ascertained as a necessary condition. While the 'classic' exceptional case concerns 'multiple sufficient causes', both the *Ipp Report* and the High Court indicate a broader application — an application relevant to proof of the causation of CTE by material contribution. The *Ipp Report* refers to the 'cumulative operation of two or more factors, but which is indivisible in the sense that it is not possible to determine the relative contribution of the various factors to the total harm suffered'.[78] If it is not possible to determine the relative contribution of each factor to the plaintiff's harm, why would it be assumed that each factor is or would be sufficient in itself? This description, on the wording of the *Ipp Report*, could apply to 'multiple contribution' to harm falling short of causative overdetermination. An exemplar provided in the *Ipp Report* is the case of *Bonnington Castings v Wardlaw*.[79]

The plaintiff, Wardlow, was employed by the defendant on the factory floor over an eight-year period, during which he contracted pneumoconiosis from inhaling minute particles of silica. Two sources of silica leakage were identified: (1) from 'swing grinders' with blocked extractors, for which the employer would be liable in negligence; and (2) from pneumatic hammers, for which the employer would not be liable: 'As there was no known means of collecting or neutralising this dust, and as it is not alleged that these machines ought not to have been used, there was no breach of duty on the part of the [defendant] in allowing this dust to escape into the air.'[80] It should be noted that the harm to the plaintiff was not categorised as due to 'multiple sufficient causes' (where any cause is sufficient in itself to cause the total harm); rather, in the words of Lord Keith of Avonholm,

> the particles inhaled are acting cumulatively, and I think the natural inference is that had it not been for the cumulative effect the [plaintiff] would not have developed pneumoconiosis when he did and might not have developed it at all.[81]

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77 *March v E & MH Stramare Pty Ltd* (1991) 171 CLR 506, 523.

78 *Review of the Law of Negligence* (Final Report, September 2002) [7.28].

79 [1956] AC 613.

80 Ibid 615.

81 Ibid 626.

In fact the judgments did not refer to 'multiple sufficient causes' and indeed implied that one cause was inadequate in itself:

> It may be that, of the noxious dust in the general atmosphere of the shop, *more* came from the pneumatic hammers than from the swing grinders, but I think it is sufficiently proved that the dust from the grinders made a substantial contribution.[82]

Lord Reid recognised similarly that 'pneumoconiosis is caused by a gradual accumulation in the lungs of minute particles of silica inhaled over a period of years'; consequently, the disease 'is caused by the whole of the noxious material inhaled and, if that material comes from two sources, it cannot be wholly attributed to material from one source or the other'.[83]

In continuing, his Lordship revealed the logic to the concept of 'material contribution':

> It appears to me that the source of [the plaintiff's] disease was the dust from both sources, and the real question is whether the dust from the swing grinders *materially contributed* to the disease. What is a material contribution must be a question of degree. A contribution which comes within the exception *de minimis non curat lex* is not material, but I think that any contribution which does not fall within that exception must be material. I do not see how there can be something too large to come within the *de minimis* principle but yet too small to be material.[84]

Notably, his Lordship considered that the contribution of the swing grinders to causing a 'guilty atmosphere'[85] exceeded the *de minimis* and was thereby material:

Unlike the 'double hunters' illustration, where each negligent act was sufficient in itself to produce the total harm suffered by the plaintiff, the *Ipp Report* refers to the cumulative operation of various factors. The 'total harm' caused to the plaintiff is the sum of each defendant's contribution to that harm and *not* harm that was 'sufficient in itself'.[86]

The wording of the relevant statements indicates that the exceptional case may apply where: the 'but for' test cannot be satisfied; the contribution to each defendant is 'sufficient in itself'; or where the relative contribution of each is unascertainable. A limiting factor in that application must accord with established principles. Can it be said that the determination in *Bonnington* established a principle that accepts material contribution as a valid application of exceptional case? Consider in this regard the statement of the High Court in *Strong v Woolworths*:

> Section 5D (2) makes special provision for cases in which factual causation *cannot be established* on a 'but for' analysis. The provision permits a finding of causation in exceptional cases,

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82 Ibid 622. Emphasis added.

83 Ibid 621.

84 Ibid. Emphasis added.

85 Ibid 617.

86 *Review of the Law of Negligence* (Final Report, September 2002 [7.28]. Emphasis added.

notwithstanding that the defendant's negligence cannot be established as a necessary condition of the occurrence of the harm.

Negligent conduct that materially contributes to the plaintiff's harm but which cannot be shown to have been a necessary condition of its occurrence may, in accordance with established principles,[38] be accepted as establishing factual causation, subject to the normative considerations to which s 5D(2) requires that attention be directed.[87]

Given the discussion above at **[5.4.1]** 'regarding the probability that all contact sport competitions a premier-level athlete played in contributed to the development of CTE, the exceptional case application would seem unnecessary. Irrespective, access to the exceptional case provision supplies an avenue of response to the SGB defensive argument, even if theoretical, that all harm was caused in junior play, or play in a different competition and it could not be responsible. Or, indeed, in addressing the reality that ascertaining an even approximate estimate of the degree of harm between each level grade of play or each competition 'cannot be established'.

The High Court did, however, suggest a caveat regarding the *Bonnington* approach. In essence, whether the test of factual causation could not be met must depend upon the state of scientific or medical knowledge, citing as an example *Amaca Pty Ltd v Booth*,[88] where sufficient scientific evidence was available to find the defendant liable without resort to the exceptional case approach.[89]

In these situations, while the plaintiff athlete continues to bear the onus of proving any fact relevant to the issue of causation, the 'exceptional case' provisions permit causation of harm to be proven to a lower standard than that required as a 'necessary condition'. That is, while not defined by percentage, the standard is less than that of probability. Such an application is clearly advantageous to a plaintiff athlete.

However, it is likely under the 'classic application', which focuses on multiple sufficient causes, that a 'material contribution' approach to 'material contribution' of CTE as an exceptional case may be rejected on the basis that neither the junior nor senior competitions had, alone, been a 'sufficient' cause of a player's contraction of CTE but, rather, each was a contributing factor provable on factual evidence as 'necessary condition'. To reiterate a statement above, it is 'necessary for the court to be satisfied that the appellant was unable to establish factual causation under [s 5D(1)(a)]'.[90]

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87 *Strong v Woolworths* (2012) 246 CLR 182, [26]–[27] (French CJ, Gummow, Heydon, Crennan and Bell JJ). Emphasis added. A footnote within the passage noting 'established principles' refers to *March v E & MH Stramare Pty Ltd* (1991) 171 CLR 506, 514 (Mason CJ).

88 (2011) 246 CLR 36.

89 *Strong v Woolworths* (2012) 246 CLR 182, [27]. See the extensive discussion of *Amaca Pty Ltd v Booth* (2011) 246 CLR 36 in Chapter 8: 'The Question of "Generic Capacity": Does the SGB's Contact Sport Cause CTE?'

90 *Powney v Kerang and District Health* (2014) 43 VR 506, [95]. The provisions of ss 51 and 52 of the *Wrongs Act 1958* (Vic) are almost identical to those of s 5D(1) and (2) of the *Civil Liability Act 2002* (NSW).

Having said that, for purposes of completeness Prof Sanderson believes that 'a further type of exceptional case' apparently based on the normative considerations 'spelt out in the Civil Liability Acts'[91] may apply where 'uncertainty of factual causation can be resolved by considering whether and why responsibility for harm should be imposed on the SSB manufacturers.'[92] The harm Sanderson speaks of is disease caused by the consumption of sugar in beverages where different suppliers contributed to the harm. The exceptional case is argued to apply through material contribution given that sugar-sweetened beverages are generally a component of an unhealthy dietary patten. Justifying this application of material contribution is wording in the High Court judgment in *Adeels Palace v Moubarak*:

> Section 5D(2) makes provision for what it describes as 'an exceptional case'. But the Act does not expressly give content to the phrase 'an exceptional case'. All that is plain is that it is a case where negligence cannot be established as a necessary condition of the harm; the 'but for' test of causation is not met. In such a case the court is commanded 'to consider (amongst other relevant things) whether or not and why responsibility for the harm should be imposed on the negligent party.[93]

It seems in this sparsely explained application that 'material contribution' is an 'established principle' such that all that is then required is whether the court will 'countenance departure from the "but for" test of causation'.[94]

5.7 Conclusion

To begin with a syllogism:

> Where it is accepted that CTE is caused by repetitive head trauma in the playing of contact sport and is a product of accumulation of head trauma, it must also be accepted that both junior and senior sport contact sport contributed to that harm.

The previous appendix considered the important element of proof that playing the SGB's type of contact sport had the generic capacity to cause CTE. Without such proof, the plaintiff athlete is unable to meet the statutory obligation of 'proving any fact relevant to causation'.

This appendix considered the possible response of a plaintiff athlete where, following a determination of the generic capacity of the plaintiff's contact sport to cause CTE, a defendant SGB maintains that the CTE with which plaintiff suffers was caused not through play in its competition, but as a junior, or in a senior competition different from its own. Again, either contention is a question of fact relevant to causation that the plaintiff must be able to prove or disprove.

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[91] Jay Sanderson, D Moritz and Z Smithers, 'Sugar-sweetened Beverages and Negligence: Who Is To Blame?' (2020) 26 *Torts Law Journal* 128, 154.

[92] Ibid 155.

[93] *Adeels Palace v Moubarak* (2009) 239 CLR 420, 443 [54] (French CJ, Gummow, Hayne, Heydon and Crennan JJ).

[94] Jay Sanderson, D Moritz and Z Smithers, 'Sugar-sweetened Beverages and Negligence: Who Is To Blame?' (2020) 26 *Torts Law Journal* 128, 154.

The appendix argued that a plaintiff athlete may counter the SGB's defence by proving that while it accepted that head trauma was experienced in playing junior contact sport or in a different competition, the defendant SGB nonetheless made a 'material contribution' to the RHT experienced by the plaintiff and hence was a cause of the plaintiff's acquired CTE.

In law, provided a defendant made material contribution, which is causation of harm beyond the *de minimis*, the defendant will be jointly and severally liable for the harm that the defendant experiences. Assuming other elements of negligence have been proven, an SGB that will be liable for the causation of CTE where the defendant played sufficient games in its competition that head trauma exceeded the *de minimis* level.

The challenge the plaintiff athlete faces is proving, as required under statute, that play in the SGB's competition was the source of the head trauma that led to CTE rather than head trauma experienced in other contact sport competitions as a junior, at school or in the senior competition of a different SGB.

The fact that a defendant SGB materially contributed to the plaintiff's CTE is argued in this appendix to be provided though scientific evidence that reveals that RHT is a consequence of all contact sports. Several journal publications were referred to in this appendix to provide a source of factual proof that head trauma is an unavoidable product of contact sport irrespective of the age or the grade at which it is played. Research from scientific and medical journals also revealed that as the size and weight of competitors increase with age the force of blows that lead to head trauma increase. From the research published in these scientific papers and discussed in other chapters — particularly Chapter 2 and Appendix 1 — informing as to how CTE is contracted and the influence of both symptomatic and asymptomatic concussion in causing CTE and how it progresses once a threshold of trauma is experienced, this appendix submits that it may be inferred that a defendant SGB is in fact a material contributor to a plaintiff athlete's CTE.[95]

This appendix also considered the possibility that the 'exceptional case' provisions of civil liability legislation may be argued as applicable to the question of a plaintiff's exposure to head trauma through several distinct sources. Under what was referred to as the 'traditional application' of the exceptional case, all competitions in which the plaintiff played each were a source of sufficient head trauma that the threshold level sufficient to cause CTE was reached. Also considered where judgments and legal commentary indicating that the exception case would apply where the material contribution of an SGB and that of other competition in which the plaintiff athlete played 'could not be ascertained.' Against this proposition was the argument that material contribution alone is sufficient to establish 'but for' causation as a 'necessary condition' under civil liability legislation.

[95] For discussion of fact and inference, see Chapter 6: 'Proof *In Vivo* of Personal Damage in the Form of CTE: The Question of "Individual Agency"'; Chapter 7: 'Factual Causation of Harm: Failure to Warn and the Athlete's Response Had a Warning Been Given'.