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Case No: KB-2022-004870

IN THE HIGH COURT OF JUSTICE
KING'S BENCH DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 18th December 2025

Before :

Mrs Justice Lambert DBE

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Between :

Sylvan Ebanks-Blake	**Claimant**
- and -	
Professor James Calder	**Defendant**

- -
- -

Simeon Maskrey KC (instructed by **Stewarts**) for the **Claimant**
Martin Forde KC (instructed by **Capsticks**) for the **Defendant**

Hearing Dates: 15 – 22 July, 30 July 2025

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Approved Judgment

This judgment was handed down remotely at 14:00 on 18th December 2025 by circulation to the parties or their representatives by e-mail and by release to the National Archives.

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MRS JUSTICE LAMBERT DBE

Mrs Justice Lambert DBE:

1. The claimant was born on 29 March 1986. In April 2013 (aged 27) he was a professional football player contracted with Wolverhampton Wanderers Football Club ("Wolves") which was then in the Premier League. During a match on 1 April 2013 his lower left fibula was fractured in a tackle. He was referred to the defendant, a consultant orthopaedic surgeon, who reduced and fixed the fracture to the fibula and additionally performed an ankle arthroscopy with debridement of scar tissue and loose cartilage in the ankle together with microfracture to the claimant's left talus bone.

2. The claimant brings this action for damages for personal injury arising from the alleged negligence of the defendant in performing ankle surgery. It is the claimant's case that the defendant's surgery ought reasonably to have been limited to reduction and fixation of the fibula and the ankle should have been left alone.

3. This is a split trial and I am deciding the issues of breach of duty and causation only. The claimant was represented by Simeon Maskrey KC and the defendant by Martin Forde KC. I repeat my thanks to them both for their assistance in this case which was very well prepared and presented with a clear focus on the key issues.

Background Facts

4. I set out below the relevant uncontentious factual background.

5. The claimant's career started in 2002 when he was 15 years old and talent scouted by Manchester United Football Club to play for the Youth Academy. He made his first team debut in 2004. On 10 February 2005 while playing for the Manchester United Academy he suffered a left fibula fracture. He described the injury as having been caused by an "awkward on-pitch tackle" with the defender's weight having fallen on to his left leg, fracturing the fibula and causing ligament damage. He underwent open reduction and internal fixation surgery on 11 February 2005. The procedure was undertaken by a consultant orthopaedic and trauma surgeon based in Manchester. The claimant was unable to play for the rest of the season but returned to training on 2 July 2005 and was back playing again at the beginning of the 2005/2006 season. He signed for Wolves in 2008 finishing the season as that club's top goal scorer. He continued to play well for the club after they were promoted to the Premier League for the three seasons between 2009-2012.

6. On 1 April 2013, whilst playing for Wolves in a fixture against Birmingham City, he was involved in another on-pitch collision with a defender. The claimant describes in his witness statement how the other player's full body weight landed on the lower part of his left leg causing a similar feeling to that which he had experienced during the incident in 2005. Although he understood that the injury was serious he believed that he had only suffered ligamentous damage and was able to drive home from the match. On 2 April 2013 he underwent an MRI scan and plain x rays which demonstrated that he had in fact sustained a left fibular fracture.

7. On or around 2 April 2013 the claimant was referred by the club doctor, Dr Matt Perry, to the defendant, a consultant orthopaedic surgeon. The defendant is, and was then, an experienced orthopaedic surgeon specialising in trauma surgery and, in particular, trauma surgery involving elite athletes. The defendant was on holiday at the time of the referral

but he reviewed the MR images and discussed the case with Dr Perry before seeing the claimant at the Cromwell Hospital on 8 April 2013.

8. On 8 April, the defendant met the claimant. He recorded in his letter to Dr Perry of the same date that the claimant had "*played very well indeed*" that season for Wolves where, he understood, the claimant had been the top goal scorer. He remarked that:

> *"he has not had any significant symptoms in his ankle since the previous fracture other than some stiffness and he certainly has always had a reduced knee to wall test on this side but has been asymptomatic; he says this has never bothered him"*

The defendant continued:

> "*clinically today the ankle itself appears a little swollen but there is no tenderness across the medial aspect of the ankle in the region of the deltoid.. and there is no significant tenderness around the lateral ligament complex – in particular there is no tenderness around the AITFL."*

9. The anterior inferior tibiofibular ligament or "AITFL" is a ligament which connects the distal tibia and fibula bones and one of the three structures which collectively form the "syndesmosis" or fibrous joint connecting the distal tibia and fibula. The other structures forming the syndesmosis are the posterior inferior tibiofibular ligament or "PITFL" and the interosseous membrane.

10. It is common ground that the defendant did not have a report from a consultant radiologist on the imaging which had been obtained. However, he reviewed the films himself, noting that the plain radiograph demonstrated a high fibular fracture with a butterfly component and that the fracture pattern appeared "*compatible with a direct blow rather than any torsional/rotational movement through the ankle joint itself*." As for the MRI scan, he noted that it demonstrated:

> *"more worrying features with regards to the articular surface and particularly in the region of the talar dome centrally. Although there is no significant bone oedema underlying this presumed chondral lesion of the talus, it would certainly appear unstable and there is also a small amount of bone oedema in an osteochondral lesion which is apparent on the posterior tibia centrally."*

11. He went on to observe that there were abnormalities within the AITFL and the PITFL but that he thought that these were old.

12. The defendant's management plan included open reduction and internal fixation of the fractured fibula and an examination under anaesthesia to check the integrity of the syndesmosis, although he recorded his thinking that the "*syndesmosis itself was normal*." His letter to Dr Perry outlining his plan continued: "*in view of the chondral lesion of the talus (and this may be substantial) I would advocate an ankle arthroscopy to assess this*

and potentially debride it if it is unstable". By "chondral lesion of the talus" the defendant was referring to a lesion (or defect) of the cartilage overlying the talus bone which sits between the heel bone and the two bones of the lower leg, the tibia and fibula. As to the advice which he gave the claimant, his letter stated that "*I have explained to Sylvan that this is not a simple break and although he thinks he will be back in three months, if there is an osteochondral lesion I have explained it is more likely to be six to eight months*.." He recorded that the claimant "*was keen to proceed with surgery*" and that he planned to perform the surgery later on 8 April.

13. The claimant signed a consent form for a "left ankle EUA + Arthroscopy + ORIF +/- ligament reconstruction." The "*intended benefits*" of the surgery were to "*diagnose, treat and optimise return to sport*" and the "*significantly or frequently occurring risks"* were "*infection, nerve injury, tendon/ vessel injury, non -union, need for metalwork removal, re-fracture arthritis, deep vein thrombosis.*"

14. The claimant was placed on the surgical list and went into theatre at around 11 am. The defendant's typed operative record records that:
    a. an examination under anaesthesia demonstrated a stable ankle with very limited movement, probably only around 5 – 10 degrees of dorsiflexion of the ankle joint;
    b. haemarthrosis (or blood in the joint) was washed out from the ankle joint;
    c. a "*large amount of scar tissue was present from the previous injury*" which was debrided "*in order to gain access/good views of the ankle joint*." The defendant noted the presence of a "*very large unstable chondral lesion affecting the central portion of the talar dome measuring 1.8 cm in the medial/lateral direction and 1 cm in the AP direction. This lesion was debrided back to stable edges… A microfracture technique was then performed to the underlying subchondral bone.*"
    d. As for the fibula fracture, this was observed to be a "*multi-fragmentary fracture compatible with a direct blow rather than any torsional element to the fracture*." Good fixation was achieved.
    e. The syndesmosis "*appeared stable*" and this was confirmed by the performance of the "Hook test."

15. In his letter to Dr Perry setting out his surgical findings, the defendant added that the osteochondral lesion of the talar dome had been significant and "very large" measuring 1.8cm x 1 cm. He confirmed that he had performed a microfracture technique to the underlying subchondral bone in order to encourage fibrocartilage formation. He advised non-weight bearing for 4 weeks and partial weight bearing for 6 weeks.

16. The radiology report of Dr James, consultant musculoskeletal radiologist, post-dated the surgery (stamped as received on 14 April 2013) and it is common ground that the contents of the report formed no part in the development of the defendant's management plan. Dr James recorded the fibula fracture. He also recorded his finding that there was disruption of the AITFL and that the PITFL appeared abnormal, potentially having been stripped from its posterior tibial attachment. He noted widening of the medial joint line suggesting disruption of the syndesmosis with the deltoid ligament appearing to be markedly irregular. He postulated however that this might be a more chronic finding. He noted the presence of established arthropathy of the ankle joint with oedema present in the talar dome. He found on the talar side approximately 1.5 cm area of chondral fissuring, irregularity and delamination in the talar dome. Osteophytes were present. I pause here to note that the radiology experts in this case agree that there is nothing in the James report which suggests

the presence of an acute ankle injury, that is, an ankle injury sustained during the tackle on 1 April.

17. The claimant was discharged on 9 April 2013. He remained under the care of the medical team at Wolves. He underwent a period of residential rehabilitation at St George's Park. His progress was monitored and supervised by physiotherapists.

18. The defendant saw the claimant for follow up on one occasion only, on 19 June 2013. Following the consultation, the defendant wrote to Dr Perry saying that "*overall, he appears to be making satisfactory progress and has come out of the boot now. He is comfortable walking and is regaining ankle dorsiflexion although this is still a little down as is probably to be expected at this stage. ..The thickening that is currently present around the ankle is to be expected to this stage following this significant injury but it does not appear to be a significant joint effusion*."

19. The claimant underwent a further MR scan on 18 December 2013. It was reported by Dr Steve Garber (consultant radiologist) who said that the "*osteochondral lesion in the lateral talar dome is still visible. It measures approximately 1 cm in transverse diameter but there is quite extensive oedema seen extending from this into the body of the talus….There is no significant ligamentous damage and the anterior talofibular ligament is intact*." He noted his conclusion thus: "*There is residual osteochondral lesion in the lateral talar dome with some oedema in the body of the talus*."

The Issues

20. This claim revolves around the defendant's decision, on 8 April 2013, to perform ankle surgery.

The claimant's case

21. It is the claimant's case that the defendant's decision to undertake an arthroscopy of the claimant's left ankle (with consequential debridement of arthrofibrosis and of loose or loosened cartilage over the dome of the talus bone) and perform a microfracture technique on the exposed talus bone was negligent for the following main reasons.
    a. The claimant suffered a significant injury to the syndesmosis during the 2005 tackle. This was inadequately treated and led to the formation of scar tissue (arthrofibrosis) and degenerative changes in the ankle. However, the claimant's ankle joint had been, notwithstanding the 2005 injury, pain free. He had been able to accommodate ankle stiffness by adapting his playing style and had been playing very successfully to an elite standard.
    b. The tackle on 1 April 2013 had caused no sufficiently significant acute injury to the ankle ligaments forming the syndesmosis nor to the cartilage overlying the talar bone to justify an arthroscopy procedure which, in this case, was inherently destructive. The arthroscopy entailed the removal of arthrofibrosis in order to visualise and access the ankle joint and involved the debriding and removing of degenerate cartilage overlying the talar dome. The arthrofibrosis and cartilage had however been serving a useful function, acting as a cushion for the ankle joint and its removal altered the biomechanics of the joint and caused pain, instability and an acceleration of degenerative changes. The dissection and removal of cartilage overlying the talar dome was followed by the use of the microfracture technique on a lesion or area of exposed bone of considerable dimensions and well beyond the size associated with a favourable outcome. The process damaged the articular surface and created

inflammation without any certainty that it would induce the formation of replacement cartilage.

c. None of these procedures should have been performed on a pain-free and functioning ankle. They caused or contributed to an acceleration in the degenerative process already affecting the joint. They led to the claimant suffering a "bone on bone" type of pain which was first demonstrated when the claimant began weight bearing and which affected his ability to play football at a high level. The only procedure which the claimant ought to have undergone was reduction and fixation of the fracture to the left fibula. The ankle joint itself should have been treated conservatively.

d. The claimant accepts that at some point in the future the ankle joint would have become symptomatic as a consequence of ongoing degenerative changes but it is asserted that those changes would not have prevented the claimant from being able to play football to a Premier and/or Championship league level for a period of 3 to 5 years.

22. The claimant raises a secondary case on informed consent which the parties agree I need address only if I am against the claimant on his case arising from the surgical management of the ankle. I say no more about this topic and return to it at the end of this judgment.

The Defendant's Case

23. The defendant's case is that during the tackle in April 2013 the claimant sustained significant acute injuries affecting two structures within the ankle: the syndesmosis and the cartilage on the talar dome. Both of those injuries mandated intervention and it would have been negligent not to have undertaken the arthroscopy and other procedures on 8 April.

24. Arthroscopy is the "gold standard" intervention for a suspected unstable syndesmosis, particularly for an elite athlete. There is literature to that effect. Given that the claimant had suffered a significant ligamentous injury in 2005 and that that injury had been inadequately treated, it was important that any suspected ligamentous damage was ascertained and addressed by the defendant.

25. Arthroscopy is also the "gold standard" intervention for the investigation of an acute and/or unstable talar lesion. The cartilage had sheared off the talus bone as a result of the forces applied during the tackle on 1 April 2013 and the loose or loosened chondral tissue had to be trimmed and removed from over the talar bone. If left, it would fragment. The fragments would then cause wear and tear to adjacent structures and would impinge on the movement of the bones of the ankle joint causing the ankle to lock and/or causing pain and an acceleration of pre-existing ankle arthritis. There were also fresh loose fragments of cartilage within the ankle joint, caused by the tackle, which had to be removed for the same reason.

26. The surgical procedure involved the removal of arthrofibrosis in front of, but not inside, the joint. It was not providing any cushioning and its removal would not affect the biomechanics of the joint. The defendant's surgical expert is strongly of the view that scar tissue should be removed. It is a noxious, cytokine-producing substance which if not removed gets bigger and accelerates arthritic changes.

27. Microfracture was, following the trimming of the loose cartilage, a reasonable process to apply to the talar bone. Despite its name it does not involve the "fracture" of any structure

but the making of a series of tiny holes in the exposed bone with a fine probe in order to provoke inflammation and so the formation of new cartilage. It was likely to be successful but even if it was not, then there was no down side. It either worked or not. It would not make the situation worse.

28. The defendant denies that the ankle surgery curtailed the claimant's football career. The defendant says that, if anything, the surgical treatment prolonged the claimant's career. He did not retire from football until 2019 and so enjoyed a further 6 years of playing, only retiring when he suffered another injury to his left fibula. The claimant's gradual career demise (in terms of his level of playing) was a combination of the 2013 acute injuries to the ankle in conjunction with the chronic degenerative changes following the 2005 injuries.

29. For completeness I record that although the defendant's pleaded case asserted that the claimant suffered from a vitamin D deficiency and that this caused or contributed to the claimant's outcome, this point was not pursued by Mr Forde at trial.

The Main Issues

30. The parties agreed a list of questions for my determination. The breach of duty questions focus upon the defendant's decision to perform an arthroscopy (and associated procedures) on 8 April 2013. Consideration of that decision involves an examination of, amongst other things:
    a. the defendant's rationale for performing the arthroscopy;
    b. what, if any, acute injuries were demonstrated clinically and/or radiologically in April 2013;
    c. the significance of any acute injuries;
    d. the likely risks and benefits of debridement; the likely risks and benefits of microfracture involving lesions of the size affecting the claimant.

31. The causative impact of the alleged negligence involves consideration of, amongst other things:
    a. the extent of the surgery performed on 8 April;
    b. whether and when the claimant suffered pain following the surgery in April 2013;
    c. the location and nature of changes apparent on MR imaging in December 2013;
    d. the natural history of the chronic and acute changes affecting the claimant's ankle;
    e. the defendant's likely course had conservative treatment been pursued.

Legal Framework

32. Negligence in a clinical context is judged by reference to the *Bolam* test (referring to *Bolam v Friern Hospital Management Committee* [1957] 1 WLR 582). A doctor is not negligent if "*he has acted in accordance with a practice accepted as proper by a responsible body of medical men skilled in that particular art"*. Thus, it follows that "*a man is not negligent, if he is acting in accordance with such a practice, merely because there is a body of opinion who would take a contrary view"* (per McNair J). The *Bolam* test is qualified by *Bolitho v City and Hackney Health Authority* [1988] AC 232 in the following way: "*if, in a rare case, it can be demonstrated that the professional opinion is not capable of withstanding logical analysis, the judge is entitled to hold that the body of opinion is not reasonable or responsible."* (per Lord Brown-Wilkinson at 243 C)). Ultimately therefore it is for the court and not for medical opinion to decide the standard of care required of a professional in the circumstances of a particular case.

The Claimant's Evidence

33. The claimant said that the ankle joint was initially stiff. He underwent treatment with Dr Perry and gradually his range of movement increased. However he struggled to put much weight through the ankle without experiencing a sharp pain and a "bone on bone" sensation within the ankle. This affected his mobility. He returned to training in November 2013 but continued to find his ankle painful. It was treated by Dr Perry with lidocaine and steroid injections.

34. During his time out of the 2012/2013 season Wolves had been relegated from the Premier League to the Championship League. The claimant signed for Ipswich Town Football Club in January 2014 on a fixed term contract and returned to full-time training in January. His ankle remained painful. He described the sensation as like "*walking on a brick in the sense that there was no cushion in the ankle joint*." When playing he experienced sharp pains in his ankle which limited his agility and slowed him down. He told me that he was a striker and had a "*sort of lower sense of gravity*". He told me that he would "*hold the ball up well and pivot left and pivot right and he just was not able to move functionally like he was before*." His contract with Ipswich ended in May 2014 and the claimant was without a club until December 2015 when he joined Preston North End in League 1, mid-season. As he described in his witness statement however ankle pain became "*part of the game*" for him and he required regular steroid injections to minimise it. He signed for Chesterfield in June 2015 and played for them in League 1. He experienced increasing pain along with the continued bone-on-bone sensation. By the end of the 2016/2017 season he made the decision to stop playing league football. In January 2019 he was playing a non-league game when he suffered a further left fibula fracture and following that injury he retired from the game.

The Defendant's Evidence

35. Following qualification in 1991 he became a Fellow of the Royal College of Surgeons in 1995, a Fellow of the Royal College of Surgeons (Trauma and Orthopaedics) in 2000 and a Fellow of the Royal College of Surgeons (Edinburgh) in 2007. He is also a Fellow in the Faculty of Sports and Exercise Medicine based in the Royal College of Surgeons in Edinburgh. Although originally qualifying in trauma and orthopaedic surgery the defendant quickly began to specialise in foot and ankle surgery and then further sub-specialised in sports injuries. In 2001 he was awarded the Brisbane Foot and Ankle Fellowship in Australia. He obtained a PhD from the University of Amsterdam in 2017 which was mainly to do with sports medicine and currently, and, as he told me "*for the past many years*" 60 to 80 % of his practice has involved elite athletes or professional athletes from different genres from the UK, Europe and USA. He is currently professor and lead for the Sports Injury Research Group at Imperial College. He is the co-founder and former director (2010 – 2017) of the Fortius Clinic in London, a general musculoskeletal unit involving around 200 doctors including a team of specialists in elite athletes and athletes generally. He has garnered a number of prizes and awards. He has occupied (and continues to occupy) a number of notable leadership and advisory roles, including from 2024 as Trustee of Tennis First, All England Lawn Tennis Club and honorary consultant advisor or the UK Health Security Agency for Sport and the Performing Arts. He is the author of several papers which have a bearing on the issues raised in this litigation. In addition to this private and academic work he was consultant in orthopaedic surgery at North Hampshire Hospital NHS Trust between 2003 and 2010 and

a consultant in orthopaedic surgery at Chelsea and Westminster Hospital between 2010 and 2015.

36. The parties have agreed a list of the issues and questions for my determination in this case. One important determination is the defendant's rationale for undertaking the arthroscopy and other procedures, a finding of fact to be made on the defendant's evidence. Given the way in which the submissions on behalf of the claimant were developed by Mr Maskrey in closing, it is necessary to set out in some detail what the defendant recorded first in his witness statement and what he then said at trial, in particular, in response to Mr Forde's questions in chief.

The defendant's witness statement

37. The defendant's witness statement is dated 25 June 2024. In it, he explained that the "*worrying features*" which he referred to in his letter to Dr Perry were the damage to the articular surface of the talus. He said that the cartilage defect appeared on imaging to be "*unstable with loose fragments within the joint*." In addition, he said that there were abnormalities of the syndesmosis. He recorded that there were chronic changes to the ankle joint which were associated with the previous fracture but the MRI scan of April 2013 also demonstrated an acute chondral injury in addition to the chronic changes already present.

38. The defendant said that he sought a second opinion from a consultant radiology colleague to double check his own interpretation. Whilst he had been confident in his own assessment he wanted to make sure that there was nothing that he was missing. The doctor, who was not identified in the witness statement (and from whom no witness statement has been served) apparently confirmed that there was "*an unstable acute chondral injury with clearly demarcated edges on the background of chronic changes*". I pause to note here for convenience that in evidence, the defendant told me that he had consulted with Dr Mitchell, one of two "*excellent world-class musculoskeletal radiologists*" with whom he was working at the time.

39. The statement sets out the defendant's reason for performing the arthroscopy in the following way:
    a. although not painful, the ankle was symptomatic. There was stiffness to the left ankle and the claimant had begun to develop osteoarthritis of the ankle following the fracture in 2005 along with the natural wear and tear associated with his playing elite football. There were clear chronic arthritic changes within the ankle joint.
    b. There was damage to both the syndesmotic and deltoid ligaments compatible with the previous severe fracture dislocation in 2005.
    c. Arthroscopy is a recognised procedure for the treatment of "ankle fractures" and this was particularly important for professional athletes. He described it as the "gold standard" in "*assessing potentially unstable osteochondral lesions and that it may enable differentiation of chronic from acute injuries*..". In the claimant's case, he said there was a clear indication to perform an ankle arthroscopy because there was "*an unstable OCL*" (that is an osteochondral lesion) and there was also the possibility of syndesmosis injury.

40. He consented the claimant for surgery. He advised on the acute and chronic nature of the osteochondral lesion. He said that he advised the claimant that an arthroscopy would enable him to "assess and, if necessary, treat the cartilage injury." He also explained that

if an unstable articular cartilage lesion was discovered then he would perform a debridement and microfracture, if indicated.

41. As to the surgery itself:
    a. the arthroscopy identified loose fragments of cartilage within the claimant's ankle joint which would have caused problems if they had been allowed to remain. They might cause locking of the joint during rehabilitation and further damage through third body wear, both of which would have been likely to result in acceleration of the pre-existing ankle arthritis.
    b. Debridement of the arthrofibrosis and delaminated cartilage were both clinically indicated and a necessary part of the operation.
    c. The removal of "the dense scar tissue" served two functions: first, to enable the comprehensive and safe visualisation of the joint and, second, to clear the joint of scar tissue which would have been contributing to the claimant's joint stiffness. The defendant added that the scar tissue in the front of the ankle joint did not provide any soft tissue or cushioning effect but is a recognised cause of anterior ankle impingement causing reduced movement and pain.
    d. The removal of a large and clearly unstable osteochondral lesion was necessary because without immediate treatment, the unstable cartilage would have separated from the talus and become loose within the ankle joint causing inflammation and scouring of healthy areas and potentially causing the ankle to lock and be painful. Equally there was a risk that the lesion would enlarge following return to sport. It was therefore necessary to visualise and probe the lesion to establish whether it was unstable and then to trim it back to a stable rim. Until the unstable cartilage had been removed back to a stable rim, the size of the lesion could not be identified.
    e. The purpose of the microfracture procedure was to promote revascularisation and lead to the formation of new cartilage. The new cartilage would be inferior to the hyaline cartilage which it replaced and might deteriorate over time leading to osteoarthritis. Nonetheless, the "gold standard" treatment of osteochondral lesions was, in 2013 and remains, debridement and microfracture.
    f. The management of chondral lesions depends on their size and location. He said that generally, lesions greater than 1.5cm are worse after injury. Debridement alone ie excision of the fragment +/- removal is only applicable to smaller lesions. He said that there was support for his approach in the international consensus of 2017 which set out that lesions such as the claimant's (ie lesions greater than 1.5 cm) are worse after injury and are best treated by debridement and bone marrow stimulation.

Examination in Chief

42. I permitted examination in chief covering the key issues. The defendant added to what he had recorded in his witness statement by telling Mr Forde why he believed that the ankle had sustained an acute injury during the tackle. He said that:
    a. there had been clinical signs, in particular ankle swelling, which had supported the presence of an acute injury.
    b. The nature of the trauma to the lower leg and the mechanics of the force which had been applied to the lower leg had been very severe. There had been a direct blow to the leg whilst the football boot had been firmly planted which would have caused the ankle to move sideways (a valgus force) and led to the injuries to the ligaments forming the syndesmosis.

c. The claimant's injury had been so serious that it had stuck in his memory. The fracture was 10 cm above the ankle. It was multi fragmentary and a high energy injury. This was not therefore just a mild collision and a simple break.
d. Although the fracture had occurred at the site of the previous (2005) fracture and through the screw tracks of the previous one, nonetheless the seven year gap between removal of the metal work and the 2013 injury would have required a "*significant impact*" to cause a fracture. He said that he felt at the time and still felt at trial when he reviewed the imaging that there had been an acute injury to the articular cartilage which would have been "*potentially catastrophic*" because there were "*large loose fragments within the ankle joint*" which if left would grind against other structures. So it would have been "*potentially a disaster*" to leave loose fragments around in the joint. He said that he felt that he would have been criticised if he had not undertaken an arthroscopy and removed the fragments and removed the loose cartilage.
e. On the radiology he saw large loose fragments within the ankle joint which he thought looked acute.
f. The defect in the articular surface of the talus bone had sharp edges in parts which was highly suggestive of, or compatible with, an acute injury.

43. He said that in addition to the acute injury to the cartilage over the talus, he was also concerned that the syndesmosis might be unstable which had led him to ask for a tightrope (a device that may be used to stabilise the bones) to be available. The literature which he used said that it was "gold standard" to use arthroscopy to assess the syndesmosis.

44. He told Mr Forde that his surgical findings confirmed his impression of an acute injury to the joint. There was bleeding within the joint on arthroscopy - although he later agreed that an injury or sprain to the ligament might cause bleeding within the ankle joint and swelling of the ankle. The cartilage fragments which he removed at arthroscopy were not round edged but looked fresh. Images which were taken at various stages during the arthroscopy showed very abnormal, disrupted and loose cartilage on the talus.

45. He said that if he had left everything in situ and not treated the articular defect and removed the fragments, he could quite rightly be criticised because the fragments, if left, would have been extremely painful. The claimant would have developed a catching sensation in his ankle joint. Further down the line these fragments would have rubbed on the articular surface and accelerated the arthritic process. He told me that his intervention enabled the claimant to get back to playing football for the next six years

46. The defendant relied upon a 2009 paper by Maartje Zengerink "*Treatment of osteochondral lesions of the talus: a systematic review*." This was a high powered paper with which he would have been familiar in 2013, having been an associate editor of the journal in which it had been published for some time. He digested the conclusions of the paper in the following way: "*In that systematic review, if looked at lesions overall, and if they are treated conservatively…there is a 45% good or excellent result. If you go in and do an arthroscopy and excise the lesion alone and don't do anything else, there was a 54% good or excellent result. If you go on and treat the lesion with excision and debridement there was a 77% chance of a good or excellent result*". Here debridement refers to removal of the loose fragments. He acknowledged that if the lesion is larger then it may have a less favourable outcome but that did not undermine his view that the claimant would have a good outcome from the treatment. He said that he was not aware of any literature that says that microfracture accelerates arthritis. It is used across the world as a very standard

procedure. He said that microfracture may not heal the lesion, but it is not destructive and not detrimental to the ankle joint itself. He said that the arthrofibrosis was around the front of the joint not between the joint surfaces. He said: "*we purely remove some scar tissue from the front so that we can look in the ankle joint appropriately and get a good view…There is no arthrofibrosis going across the joint's surface."*

The expert radiology evidence: Dr Patel and Dr Miller

47. Dr Himanshu Patel gave evidence for the claimant and Dr John Miller for the defendant. Both are specialist musculoskeletal radiologists. Dr Patel is currently a consultant radiologist in Wrexham Maelor Hospital in North Wales, a post which he has held since 2007. Dr Miller holds a similar position at the Western General Hospital, Edinburgh. I am satisfied that both experts have considerable relevant experience and expertise.

48. There were significant areas of agreement between Dr Patel and Dr Miller. See below:
    a. they agreed that there was an acute injury in the form of a partial tear or sprain to the AITFL. This was an "acute on chronic" injury as the ligament had previously been injured during the 2005 tackle.
    b. They agreed that the MR scan revealed evidence of a chronic osteochondral lesion at the talar dome in the left ankle. Dr Miller assessed the lesion as being 12 x 15 mm against a background of secondary bony features of osteoarthritis and associated with thinning of the cartilage and fissuring. Dr Patel assessed the size to be 13 x 14 mm and described it as being full thickness and extending to the lateral side of the dome. Dr Patel described the defect as having a tapered smooth edge laterally and a tapered smooth edge posteriorly. These changes to the cartilage were also associated with other chronic changes including sub-chondral oedema and marginal osteophytes.
    c. They agreed that in addition to the presence of a chronic lesion there was evidence of acute injury in the joint in the talar dome. They did not agree about the size. Dr Miller believed that there was a sharply marginated, full thickness cartilage loss on the central talar dome caused by the shearing off of already degenerate cartilage as a result of the tackle and that there were angular cartilage loose bodies in the joint recess. Dr Patel considered that there was a single area of sharp demarcation to the anteromedial aspect of the osteochondral lesion.
    d. They agreed that the chondral lesion of the talar dome was a combination of degenerative change and acute chondral injury and agreed that this was a 50/50 combination acute/chronic change. Dr Patel explained that he agreed 50/50 combination based on his comparison of one edge (which was sharp and in keeping with an acute injury) with another (which was smooth and in keeping with a chronic injury).

49. Dr Patel accepted that the presence of this single area of sharp demarcation could reflect an acute injury but questioned the mechanism whereby the central portion of the talus might have been injured during the tackle, explaining that the talus is a saddle shaped bone with higher shoulders and a more depressed central portion. The acute injury affected the central portion of the bone. Chondral injury in this location would have required some rotational/torsional stress which had been excluded by the defendant or some very significant axial force. He noted the absence of any significant ankle effusion which might have been expected had the ankle injury been very recent. He accepted that there was some blood on arthroscopy which could be suggestive of a very recent joint injury but said that an exacerbation of a chronic change could lead to blood in the joint.

50. Dr Miller considered that the acute injury had been caused by the cartilage having been stripped from the talar dome during the tackle, suggesting that "*that there were tangential forces at work here*." He observed that the defendant had noted contemporaneously that no rotational forces had been applied but, he said, he was sceptical of this view believing that injuries such as the claimant's involved a mixture of forces in all three planes. He accepted however that for the force to be transmitted to the ankle there needed to be a rotational element somewhere that exerts a force within the ankle joint. He believed that the presence of a bloody joint at arthroscopy was highly suggestive of an acute intra articular injury.

51. They disagreed about the nature of the so called "dark triangular shard" which appeared on the MR imaging. Dr Miller's view was that the fragment was likely to represent an acute cartilage flake or loose body. Dr Patel thought that the shard was more likely to be fibrosis given its darker signal characteristics on all sequences and its significant size which was far larger than the defect itself and so unlikely to have arisen from the defect. Dr Patel agreed with Dr Miller that there were multiple fragments of cartilage throughout the joint, some within the defect itself and some within the anterior part of the joint. However he said that they were small, difficult to characterise and impossible to date, save that some were round and were therefore old.

52. Dr Patel told me that the MR scan demonstrated the presence of arthrofibrosis extending into the joint, between the two joint surfaces at the anterior aspect of the joint and also at the medial aspect of the joint and this would potentially act as a cushion supporting the space. He said that by the removal of the scar tissue you could potentially turn a stable joint into an unstable joint.

53. As for the MR scan of mid December 2013, again there was some limited agreement. Both experts agreed that there had been progressive degeneration of the osteochondral defect on the talar dome. Dr Patel said that there had been progression of degenerate changes both distant from the site of the procedure and at the site of the microfracture. In the area distant from the operation site there was oedema and the overlying cartilage had become more degenerate with further loss and fissuring. Changes within the site of the bone marrow stimulation included extensive bone marrow oedema which was far more than he would have expected following microfracture. Dr Miller agreed with Mr Maskrey that the area of progressive osteoarthritic change was to the posterior part of the area which had been subjected to microfracture by the defendant.

The expert orthopaedic surgeons: Patrick Laing and Professor William Ribbans

54. Mr Patrick Laing gave evidence on behalf of the claimant and Professor William Ribbans for the defendant. Mr Laing is a consultant orthopaedic foot and ankle surgeon with considerable experience of managing and treating osteochondral lesions of the sort affecting the claimant in 2013 dating back to when he was working at the Robert James and Agnes Hunt Orthopaedic Hospital. He stopped practising in the NHS in 2022 and ceased private practice in 2019 Professor Ribbans has been a consultant in trauma and orthopaedic surgery since 1991 and Professor of Sports Medicine at the University of Northampton for 20 years. He has worked exclusively in the private sector within the field of sports medicine and surgery since 2012. He has held a number of posts, honorary or otherwise, for various sporting, athletic and arts organisations including the English National Ballet (for whom he has been honorary surgeon for 30 years), Northampton Town FC (for whom he has been honorary orthopaedic surgeon for over 20 years) and

England rugby. He is the Foundation Fellow of the Faculty of Sports and Exercise medicine and responsible for looking after world champions across eight different sports; he has organised three international symposiums on sport foot and ankle injuries.

55. There was broad agreement between the experts concerning the nature of the 2005 injury. They agreed that the injuries had included not only a fibular fracture but damage to the syndesmosis. Both agreed that the ligament injury had been serious. The AITFL and PITFL had been injured and the interosseous membrane had ruptured. They agreed that whilst the fibula had been repaired in 2005, the surgeon had not addressed the damage to the syndesmosis. Some fixation should have been inserted across the fibula and tibia. The syndesmosis had not healed well because of the residual instability leaving a diastasis (or gap) of 2 mm which had permitted movement of the joint giving rise to degenerative changes to the ankle joint over time. Those degenerative changes included the formation of arthrofibrosis which Mr Laing said would have developed shortly after the injury in 2005 and which would by 2013 therefore have been long standing.

56. This was the extent of the agreement between the experts who expressed fundamentally opposing views concerning the appropriate management of the 2013 injuries.

Mr Laing's Evidence

57. Mr Laing said that the mechanism causing the 2013 fibula fracture appeared from the medical records to have been a direct blow to the left fibular and lateral ankle region. He did not believe that there had been any rotational force applied to the leg and ankle during the tackle. He believed that, if there had been, then this would have led to a spiral fracture of the fibula.

58. Mr Laing accepted that there may have been some valgus or sideways force applied to the lower leg and ankle joint which caused the injury/sprain to the AITFL. But did not consider this to be a particular serious injury. It was, he said, a slight sprain. It was not associated with any local tenderness. There was no associated damage to the medial ligament, no damage to the anterior capsule and no significant ankle swelling (as on 8 April).

59. His interpretation of the MR scan suggested that there was a significant chondral lesion to the talus but that this was chronic and due to secondary changes most likely caused by the long standing syndesmotic instability. At the time of preparing his report he had not been able to see any acute component to the chondral injury and, even on review of the imaging at trial, was not able to clearly identify acute injury for himself. He could see loose fragments of cartilage lying in the bed of the cartilage within the chronic lesion itself but he did not believe that these were likely to interfere with the movement of the joint by causing catching and locking. The loose fragments did not look particularly old to him.

60. Mr Laing accepted the opinion of the radiologists that there was an element of acute injury to the talus but, like Dr Patel, he queried the mechanism whereby a central talar lesion might have occurred after a fracture of the fibula caused by a direct impact. He said that such an injury was not described in the literature. He said that for this reason he believed that the acute chondral injury was likely to be limited to a single area of sharp demarcation to the anteromedial aspect of the larger chronic lesion.

61. Mr Laing said that there was a large amount of arthrofibrosis within the ankle joint. The joint had a reduced range of movement which would have served to stabilise the fragments.

The MR scan demonstrated a dark wedge of black arthrofibrosis extending into the medial gutter of the ankle joint and into the anterior lip of the tibia. He said that there was a piece of triangular shaped black tissue in front of the ankle joint. He said that, in texture, arthrofibrosis is spongy and that it would have provided cushioning of the front of the ankle joint. It would have helped in stabilising the joint by cutting down the amount of sideways movement of the talus.

62. Mr Laing's view is that the decision to perform the arthroscopy was not reasonable.
    a. Pre-operatively there had been nothing to suggest that the claimant had suffered a significant ankle injury. There had been no rotational or significant axial force applied to the ankle joint during the tackle. There was minimal swelling.
    b. It was not reasonable to perform an arthroscopy to assess the syndesmosis because there was no evidence of any acute syndesmotic instability. The defendant had recorded that he thought that the syndesmosis was normal. Arthroscopy for any suspected subtle instability was not indicated.
    c. It was not normal practice to perform an arthroscopy for the purpose of aspirating or clearing any blood within the joint. Any blood would have been naturally reabsorbed in any event.
    d. The decision to perform arthroscopy involved a commitment to debride the extensive scar tissue from the previous injury in order to obtain a good view and access to the chondral lesion. There was a wedge of arthrofibrosis within the joint in the medial gutter and another wedge of arthrofibrosis over the front of the joint and extending under the anterior lip of the tibia This fibrosis could be seen clearly on the MR scans taken in April 2013. The surgeon would have to remove a good deal of scar tissue in the joint in order to see under the lip of the tibia and get a good view of the joint. He said that given the site of the portal of the arthroscope "*you are straight down onto it, down there in the medial gutter*."
    e. The removal of scar tissue and the articular cartilage over the central talus would alter a clinically stable situation. The scar tissue and articular cartilage (even degenerate cartilage) would have been providing some degree of stability and cushioning inside the ankle joint. The removal of the tissue would increase the stresses and loading pressure on the remaining cartilage. The scar tissue would also have been helping stabilise any delaminated articular cartilage. It would have been producing stiffness which meant that the joint was not subjected to as much movement and stress.
    f. Even though there was an element of acute damage to the talar cartilage it was not reasonable to perform arthroscopy and debridement of scar tissue and cartilage.

63. Microfracture involves the breaking up of the bony surface of the talar dome with the intention of provoking inflammation in the underlying bone and the formation of a stable layer of new cartilage on top of an area of bare bone which will either protect it from further damage or slow down the progression of further changes. The disadvantage of microfracture is that it is a destructive process which can lead to worsening of a patient's symptoms and may not induce the formation of fibrocartilage at all. Reasonable practice in 2013 reserved microfracture for symptomatic lesions.

64. The size of the lesion meant that microfracture was unlikely to have a favourable outcome. Mr Laing drew my attention to the following literature:
    a. "*Osteochondral Lesion of the Talus: Is there a Critical Defect Size for Poor Outcome*" by Choi and others in Volume 37 of the American Journal of Sports Medicine published in 2009. The purpose of the paper was to identify factors

associated with favourable and unfavourable outcomes so as to provide patients with accurate expectations of the arthroscopic marrow stimulation techniques. The study reviewed 120 ankles which had undergone arthroscopic bone marrow stimulation (or microfracture) treatment for chondral lesions of the talus and then evaluated for prognostic factors. The authors found that patients with an area of treatment (or chondral defect size) equal to or greater than 150 mm2 had significantly higher clinical failure rate. 10 out of 95 ankle (10.5%) with a defect size area of less than 150 mm2 showed clinical failure whereas in patients with an area over 150mm2 the clinical failure rate was 80%. The authors concluded that the initial size of the lesion is therefore an important, and easily obtainable, prognostic factor in osteochondral lesions of the talus which may serve as a basis for preoperative surgical decisions. Mr Laing said that the defendant should have been aware of this paper.

b. "*Treatment of Osteochondral lesions of the Talus, a systematic review*" by Zengerink and others, published in 2010. This paper emphasised that conservative treatment should always be the first choice of management for chondral lesions; that microfracture (with debridement) was the most effective treatment strategy for symptomatic lesions but that, owing to the great diversity of articles and variability of treatment results, no definitive conclusions could be drawn. The authors were not able to reach any conclusions as to the prognostic importance of the size of the lesion.

c. "*Debridement, curettage and Bone Marrow Stimulation: Proceedings of the International Consensus Meeting on Cartilage Repair of the Ankle"* by Hannon and others was published in 2018. The authors agreed that microfracture was the most frequent procedure performed for osteochondral lesions of the talus measuring less than 15 mm in diameter. 93% of consultees agreed with the statement that microfracture can be considered for surgical treatment of cartilage pathology of the ankle which has failed conservative treatment; 94% of consultees agreed that the ideal size guidelines for use of microfracture are lesions less than 10mm in diameter and less than 100mm square and that the procedure is less likely to succeed when used as a sole treatment in a lesion 15 mm in diameter or greater; historical guidelines have recommended microfracture for lesions less than 15 mm in diameter but leading experts from this consensus strongly agreed that a smaller lesion size guideline be used. This consensus was supported by recent evidence that lesions greater than 10 mm in diameter are at greater risk of failure likely due to the mechanical inferiority of fibrocartilage formed

d. "*Diagnosis and treatment of osteochondral lesions of the ankle: current concepts*" by Marcelo Pires Prado and others. The paper emphasised that the treatment of osteochondral lesions should be restricted to symptomatic lesions. The coincidental finding of asymptomatic osteochondral lesions regardless of location, type and size should be communicated to the patient and followed up. It supported the proposition that microfracture should be limited to lesions less than 1.5 cm2.

65. In summary Mr Laing found nothing in the literature to suggest that surgical treatment was indicated for asymptomatic lesions and nothing to challenge the view that it was the consensus of opinion in 2013 that osteochondral lesions of the size that the claimant had should not be treated with microfracture.

66. Mr Laing said that, on the strong balance of probabilities, but for the defendant's intervention, the claimant would have returned to his previous pain-free state. He said that: "*If he had just reduced and plated the fibular fracture then he would have been able*

to return to his pre-fracture level of activity.. There would have been a slow deterioration but would have been able to continue playing football."

67. He said that delaminated articular cartilage would gradually break up leading to the progression of arthritic changes and osteoarthritis. When asked about the time frame for the onset of ankle pain (assuming conservative treatment) he said that it was difficult to be precise, but that the claimant had been able to play football to a high level for many years in spite of his 2005 injury and its sequelae. There was however a hidden lesion which at some point was going to create a problem. He said that on balance the claimant would probably have been to continue to play at a good level for probably three to five years. He was not cross examined on this topic.

68. He said that in his view the arthroscopy and microfracture of the talar dome had led to a poor outcome and brought forward the onset of more significant degenerative arthritis. He said that the "*quite marked deterioration from an asymptomatic ankle in April 2013 to an ankle with marked articular surface irregularity, more extensive cartilage loss and subchondral bone oedema by December 2013 would suggest that intervention led to a significant acceleration of degenerative changes."*

Professor Ribbans' Evidence

69. Professor Ribbans emphasised the serious nature of the 2013 ankle injury. The mechanism had been a pronation abduction injury which had produced a shearing motion and possibly axial loading to the ankle. It had led to "*significant ligamentous damage*" as evidenced by the swelling of the ankle which had been described as a "*severe effusion*" in the Wolves notes three days post injury. He said that the medical assessment of sprains or ligament damage involves a grading of seriousness 1 – 3. Grade 2 is a moderate injury. Grade 3 involves a complete rupture. This was at least a grade 2 injury to the AITFL. There was also a possible injury to the PITFL and, in the James' radiology report, there was reference to an apparent disruption to the tibiofibular interosseous ligament. He said that grade 2 partial injuries are a "real problem." They can lead to lateral movement of the joint and he told me that elite players will return to him after such injuries complaining of pain. He will perform an arthroscopy of the ankle to check on what might be a subtle instability. He said that in this case the serious nature of the syndesmosis injury was confirmed by the blood in the joint which was likely due to the acute injury to the AITFL.

70. The tackle had also led to an acute chondral injury and the delamination of cartilage fragments from the underlying bone. The MR images, the arthroscopic record and photos all show unstable and detached fragments both centrally and anteriorly. There were fragments which were sitting at or above the level of adjacent healthy tissue. These had to be removed because once they were loosened and free within the joint they would go on to cause symptoms: pain, irritation, inflammation and swelling. They may cause locking. They act like a pumice stone, causing third party wear and tear to healthy areas. He said that the clinically stated absence of pain before April 2013 favoured the scenario that the loose fragments were new fragments.

71. Arthrofibrosis is not a "positive thing." Professor Ribbans said that in his judgement it was "noxious" and a very significant problem. It is toxic and produces cytokines which cause damage and stiffness to articular cartilage. He would have expected the defendant to have removed as much of the scar tissue as he could safely.

72. Professor Ribbans told me that arthroscopy was "mandatory" in April 2013. The claimant had suffered a serious injury caused by a high energy collision leading to swelling. There was evidence of acute damage to the cartilage over the talar dome which had shed fragments so on balance of probabilities the claimant was going to go on to have pain. However he said that the "*most pressing problem that I believe the defendant had to address was that he had an extremely abnormal ankle with regard to the ligament injury.*" The "gold standard" response to a suspected ligamentous injury was to arthroscope so that the syndesmosis could be inspected and the surgeon would be able to examine the ankle in various planes to check for instability. This is "*the absolute*" that the defendant cannot afford to get wrong and "*he needed to be absolutely sure that the injuries were intact*." He made clear that of the two acute injuries (to the talus and to the ligaments) the need to address and stabilise the syndesmosis was the more important. He said that "*stabilisation of the syndesmosis… was the critical thing that had to be determined and which justified the arthroscopy*." The chondral fragments also had to be addressed. They had been shed by the osteochondral lesion and the fragments were sitting on the joint line above the normal cartilage. They were within the joint. If not already loose then they were very unstable and if they lifted they would act like a pumice stone.

73. With conservative treatment the scar tissue would have got worse (which is associated with an extremely bad outcome) and the presence of blood "*is not great news in general*." The detachment of cartilage fragments was also a core prognostic factor for the claimant's return to elite football. By comparison, he said that those who undergo microfracture will in the overwhelming majority of cases have a positive result. If microfracture does fail then what was already bare bone (judged from the intra-operative photographs) would have remained bare. He does not think that bone marrow stimulation accelerates osteoarthritis.

74. He said that a return to elite football with an isolated osteochondral lesion of the talus following treatment with microfracture would have had a substantially reduced success rate from the 88% quoted in the literature because of the claimant's additional pathologies (within the joint and the fibula fracture) and that the claimant ought to have been told that, even with treatment, he would have had a one in three chance of not returning to elite football. He did not accept that conservative treatment ought to have been trialled first but accepted that conservative treatment did confer a chance that the ankle would not have become symptomatic. He told Mr Maskrey that the literature suggested that conservative treatment conferred (possibly) a 45/55% chance of the osteochondral lesion remaining asymptomatic but that this figure did not take into account the other features of the claimant's presentation including the subtle syndesmotic instability.

75. He said that in 2013 the recommendations for the management of chondral lesions were poor. Such literature as existed was based on lesions which were medially and laterally located. He agreed that the consensus paper confirmed that smaller lesions yielded better chances of a good outcome from microfracture. This however did not mean that patients with larger lesions should not be treated with microfracture even though the results in terms of function and comfort might be less. In any event, he doubted that it was possible to describe the exact size of the lesion using MR imaging which would or might involve distortion. But he thought that even if the area which was subjected to microfracture was greater than 1.5cm2 this did not matter because (a) the lesion was a "contained lesion" that is one with stable margins and steep/vertical sides. These allow fibrin clot to form and fibrocartilage is more likely to occur in a contained lesion; (b) the *Prado* paper confirmed

that the position of the lesion was more important than size; and (c) the *Zengerink* paper stated that once a decision has been made to perform an arthroscopy then the "*best chance of a good outcome comes from debridement, removal of loose bodies following by microfracture*."

76. Professor Ribbans said that the claimant's outcome was due to the natural degeneration of the ankle and hindfoot as part of more widespread arthritis and that his long-term sequelae were due to progressive post traumatic osteoarthritis of the ankle joint due to his medical and injury history and the inadequate surgery of 2005. The effect of the 2013 injury was to elevate the level of damage and steepen the curve of the progressive damage. By December 2013, there was evidence of osteoarthritis development in areas which had not previously been affected including the sub-talar joint and more widespread areas within the ankle joint not subjected to microfracture.

Discussion: Breach of Duty

Preliminary Points

77. I start by making some observations and findings concerning the defendant's evidence.

78. First, his evidence has evolved and developed during the course of the forensic process. His evidence in chief included a number of details which were absent from his witness statement. Whilst accepting fully that, as trial approaches and experts narrow the issues, certain aspects of any case come into closer focus, I would have expected key detail (such as the radiological features which suggested to the defendant that cartilage had been acutely injured and clinical findings confirming or supporting the presence of an acute injury) to have been included in his witness statement. The defendant's evidence at trial also significantly overstated the seriousness of the acute injuries to the claimant's ankle when compared with his earlier accounts, including his contemporaneous notes/letters. He told me that the injuries were so serious and so striking that they stuck in his memory; that the lower leg must have been subjected to a significant impact to have led to the fibula fracture; that the large loose fragments of cartilage in the joint which looked to him to be acute were potentially catastrophic or career ending for the claimant and it would have been a disaster for them to have been left in situ. Neither this message, nor anything like it, was conveyed to Dr Perry in the letter of 8 April 2013. Had the defendant, as he asserted at trial, believed that the claimant's injuries were so severe as to be potentially career ending without surgery then he would undoubtedly have wished to inform Dr Perry and, through him, the Club of this potentially dire and risky situation.

79. I cannot be sure of the reason for this evolution in the defendant's evidence. But the fact that sections of the defendant's witness statement had been cut and pasted from Professor Ribbans' report is a strong pointer in the direction of the defendant's thinking having been influenced by aspects of the expert opinion of Professor Ribbans. The defendant accepted that those sections were almost word for word identical and their inclusion could not have been accidental or coincidental. I can exclude the reverse possibility that Professor Ribbans was responsible for importing into his report those paragraphs from the defendant's statement. When this was suggested to him he could not hide his genuine outrage at such a "scurrilous" suggestion. The passages in question cover important ground such as the purpose of the removal of dense scar tissue; the purpose of the microfracture technique; the merits of debriding a large unstable osteochondral lesion of the talus and what would have been the effect of leaving such a lesion undisturbed.

80. The appearance of those sections in the statement is troubling in itself. Whilst accepting that a witness statement is likely to be assembled by lawyers (and in this case, judged by its preamble, via email correspondence) the defendant put his signature to a document which, putting it blandly, contained a justification for his management as framed by Professor Ribbans. But the concern is more general and more significant than a concern about carelessness in statement preparation. Professor Ribbans is an enthusiast for arthroscopy. He has strongly held views about the seriousness of the claimant's injuries and how those injuries should have been treated. He is strongly supportive of the defendant's management. Putting everything together I am led to the irresistible conclusion that the defendant's witness statement and his account at trial are almost certainly an amalgam of what the defendant thought and Professor Ribbans' expert opinion. The defendant's account of his reasoning and recollection has been, no doubt unwittingly, influenced by expert opinion.

81. My second preliminary point follows on from this first point. When considering the defendant's evidence and in particular his rationale for performing the arthroscopy (and subsequent procedures) on 8 April 2013, I can place little or no reliance upon the defendant's witness statement or his evidence at trial and concentrate upon what the defendant set out in his contemporaneous documentation on 8 April 2013. There is nothing particularly startling about this approach. Indeed, when some of the difficulties in his various accounts were pointed out to him the defendant agreed with Mr Maskrey that "*if we're going to find out what the thought processes and management plan was in 2013, the best thing to do is to look at the contemporaneous material*."

The Defendant's Rationale for Arthroscopy?

82. The parties agree that one of the important questions which I must ask is why the defendant performed the arthroscopy and whether it was performed to address what was believed to be an acute ankle injury or for some other reason, for example to address potential instability. This question acquired a particular importance because in cross examination, the defendant accepted that his reason for seeking out an informal opinion from Dr Mitchell was to find out whether the ankle injury was acute or chronic "*because, if it's just chronic, I don't need to do an ankle arthroscopy*." He agreed with Mr Maskrey when it was put to him: "*if this is a chronic process that's going on, then you don't need to do an arthroscopy, so your management decision hinges on your view that this was an acute ankle injury*?" This view was consistent with the claimant's case and with the opinion of Dr Miller who, in his report had said that the "*crux issue*" was whether the radiology demonstrated the presence of an acute injury or not.

83. Judged by reference to the contemporaneous documentation I address the defendant's reason for performing arthroscopy in this way. Putting it shortly, everything points in the direction that he was concerned to investigate and address instability and nothing points in the direction that he undertook arthroscopy because he believed that either of the two injuries (ligament and cartilage) were acute.

84. In his letter to Dr Perry the defendant minimises the significance of the ligament injury remarking that the ankle was "*a little swollen*" without tenderness over the medial aspect of the ankle and no tenderness around the AITFL. He observed that the abnormalities in the ligaments were old and that the syndesmosis itself was normal. Nowhere does he say

or even suggest that he believes the syndesmosis has suffered an acute injury which must be investigated by arthroscopy. Even though he arranged for tightropes to be available to address syndesmotic instability if present, it does not follow from this that he believed that any suspected instability was of recent origin. He noted the worrying feature of the presumed chondral lesion of the talus which appeared unstable and advocated ankle arthroscopy to assess the chondral lesion and potentially debride it "*if it is unstable*." Again nowhere in this letter does he say, or suggest, that his reason for undertaking surgery to the ankle joint was because the chondral lesion was an acute lesion caused or exacerbated by the recent trauma to the lower fibula. A plain reading of the letter suggests that his reason for performing the surgery was because he believed that the chondral lesion was or may be unstable. I pause to emphasise that the defendant confirmed that the fact that the chondral lesion was or may have been unstable is not indicative of it being acute: a chronic lesion can also be unstable.

85. The defendant had no good answer to Mr Maskrey's questions about the tension between his evidence at trial and the contents of the letter to Dr Perry. Further, to the extent that I am able to place any reliance upon the defendant's witness statement, it tends to support my conclusion that his rationale for operating was to address the instability of the chondral lesion. In the section of the statement in which the defendant confronts the pleaded allegation that there was no or no reasonable justification for performing an arthroscopy, he writes: "*an arthroscopy is the gold standard in assessing potentially unstable osteochondral lesions and may enable differentiation of chronic from acute injuries…*" He continues, even less ambiguously: "*In the Claimant's case, there was a clear indication to perform an ankle arthroscopy. This was on the basis that there was an unstable OCL and there was also the possibility of a syndesmosis injury*"

86. It follows from this that the defendant's rationale for operating in 2013 is not one which he would accept now, in 2025, as being reasonable or plausible. On his own evidence he should have focussed not on the stability (or instability) of the chondral lesion but upon whether the injuries had been caused during the tackle or were associated with chronic changes following the tackle in 2005. This finding is obviously of importance to the question of breach of duty. But Mr Maskrey invites me to go further and approach the question of breach of duty on the basis of my conclusions on the extent and nature of any acute injuries which would have or should have been apparent to the defendant at the time when he made his decision to operate on the claimant's ankle.

What acute ankle injuries were demonstrated on the MR imaging?

87. When considering the imaging, I am conscious that both Mr Laing and Professor Ribbans have expressed views on the topic. However on this issue, I prefer to focus on the evidence of the radiologists, the specialists and best experts in this area.

88. There was a reasonable measure of agreement between the radiology experts. They agreed that the imaging demonstrated an acute ligamentous injury, acute (or fresh) damage to the cartilage overlying the talus and the presence of chondral fragments in the joint. The difference between them concerned the extent of those acute features and their significance. Dr Patel's view was that overall the appearances in the joint were more consistent with a chronic injury than acute and that the injuries were not extensive: the ligament had been sprained; a smaller area of cartilage had been delaminated and although there were fragments of cartilage in the joint, some were small and difficult to characterise and appeared to him to have been there for some time. He was unable to age the fragments.

Dr Miller's view in contrast was that all, or virtually all, of the talar cartilage had been sheared off the bone during the tackle and that large dark fragments of loose cartilage newly loosened cartilage were in the joint.

89. There are two problems with Dr Miller's evidence. First, in support of his view that there had been an extensive delamination of the talar cartilage during the tackle he relied upon the presence of what was described as the large dark angular (or triangular) body on the MR scan which he believed to be displaced cartilage. I accept Dr Patel's evidence that this was not loose cartilage but arthrofibrosis. Even to the layperson's eye it was of a darker colour than the cartilage from which it was said to have been delaminated indicating a different signal and therefore a different substance. It was also obviously bigger than the defect from which it was said to have separated. This fragment was obviously arthrofibrosis and not cartilage. This finding undermines Dr Miller's description of the extent of the acute lesion.

90. The second difficulty with Dr Miller's evidence is that for reasons which remain opaque he sought, in his evidence, to devalue the significance of the angulation of margins as a means of assessing whether cartilage damage was old or new. He told Mr Forde that there had been an undue focus on the margins whether sharp, rounded, tapered or ragged of the chondral lesion. He said that the cartilage across the dome was already abnormal and delaminated and it would therefore be wrong to place too much emphasis on the morphology of the edges. He also emphasised the limited resolution of the scan images comparing them with the "*exquisite visualisation*" obtained by the surgeon during the arthroscopy procedure. These were both surprising comments. Neither had been heralded and neither had been put to Dr Patel. In his report Dr Miller had himself introduced the concept of "*sharply marginated*" edges and "*angula*r" loose bodies in the joint recess and when commenting on the presence of dark shards of material likely to represent recently shed cartilage flakes or loose bodies, he observed that those shards are "angular." He had done so in order to illustrate that those fragments were recent. I can only conclude from this shift in his evidence that Dr Miller had grown uncomfortable with the importance of the radiology evidence in this case and was seeking to minimise its significance by emphasising the relative value of the direct visualisation of the joint by the defendant.

91. Mr Forde submits that Dr Patel's evidence should be treated as unreliable because he has not been consistent. He refers to Dr Patel's acceptance in the joint expert meeting that there was an acute element to the chondral lesion. Mr Forde describes this as a concession as, in his report, Dr Patel had been unable to see this acute lesion. Whether correctly described as a concession, it certainly represents a change in view but not one which leads me to the conclusion that Dr Patel is unreliable. On the contrary, the purpose of joint meetings is to narrow issues and for experts to review and test their original views, to destruction if necessary. The fact that he has changed his mind is not only evidence of the process working as it should but of an expert who is objective and demonstrating his duty to the court.

92. I must also address two aspects of the defendant's evidence in this context. First his account that Dr Mitchell had reported to him that the imaging demonstrated an "*unstable acute chondral injury with clearly demarcated edges on the background of chronic changes*." I do not accept that this is reliable evidence. If this had been reported to the defendant he would have included this information in his letter to Dr Perry, particularly given that his stated reason for undertaking the procedure was to investigate possible

instability of the chondral lesion. It stretches credibility too far to suggest that a report from a world class radiologist confirming the presence of an acute unstable lesion would not have been communicated to the Club Doctor. Nor do I accept as reliable the defendant's account at trial that when he performed the arthroscopy he saw large loose fragments which he thought looked acute because they had "*sharp edges*". This finding is not recorded in the defendant's letter to Dr Perry (in which he makes no reference to flakes of cartilage) nor in his witness statement which simply records the presence of loose fragments within the joint. Mr Maskrey suggests that the account is an ex post facto reconstruction unwittingly influenced by expert views. That may be the case. It may also be a case of wishful thinking.

93. For these reasons I have concluded that the imaging demonstrated a far less serious acute picture than that presented by Dr Miller. I accept Dr Patel's evidence that there was some but not extensive delamination of articular cartilage during the course of the tackle. I accept his evidence that there were additionally some loose fragments of cartilage throughout the joint but that they were small, difficult to characterise and difficult to age. There was of course, in addition, an acute injury to the AITFL in the form of a "sprain". The next question is whether these acute injuries reasonably indicated the need for arthroscopy, debridement of scar tissue and cartilage and microfracture in this claimant.

Was Arthroscopy Indicated?

94. I start by setting out my approach to the evidence of Professor Ribbans and Mr Laing. I do so by reference to each counsel's submission that the evidence of the opposing expert was unbalanced and unreliable. I address Mr Maskrey's submissions first.

95. Professor Ribbans provided a report on liability expressing support for the defendant's management and commenting that "*the MRI scan clearly showed concerning features within the ankle joint itself*" and that "*the overall clinical, and radiological, assessment suggested that, in addition to the recurrent fibular fracture, the claimant had suffered acute injury damage to the ankle joint itself as a direct result of the tackle*." He made these comments without having reviewed the MR images himself. His supportive opinion was, it emerged, based upon the report which had been prepared by Dr James and the assumption that the contents of the defendant's witness statement and letters to Dr Perry about the imaging were correct. Not only was the James report not available to the defendant at the time of his decision to perform arthroscopy but it was common ground between the expert radiologists that there was nothing in that report which suggested the presence of an acute chondral lesion. Professor Ribbans did not, it appears, ask to see the imaging before expressing his views on the management of the claimant's injury. Nor, having seen the images after his report had been served, did he consider it necessary or appropriate to provide a supplementary report setting out his views that the MR images revealed "*sharply demarcated areas on the side walls of the chondral lesion" and "loose and unstable fragments*."

96. Professor Ribbans set out in the preamble to his report that he had not seen the relevant images and to that extent his report is not misleading. But it would have required a reasonably close reading of the document to pick this fact up. It is regrettable that, when expressing his support for the defendant, Professor Ribbans did not say that he had not seen the images and caveat his opinion by saying that he was working on the assumption that the defendant's interpretation was correct.

97. Professor Ribbans also made other assumptions in the defendant's favour. He commented that the defendant as an "*experienced ankle arthroscopist*" would share his view about scar tissue and would not feel the need to record his decision to remove as much as safely possible. He said that "*It is part of the process that we have to go through with these kind of cases*." He assumed that the defendant would share his view about haemoarthrosis saying that it "*is part of what an experienced foot and ankle surgeon knows that he is to be experiencing and finding when he is dealing with an acute fracture*."

98. There were other aspects of Professor Ribbans' evidence which were troubling. When addressing the topic of microfracture in his report, he did not draw the court's attention to literature which suggested that larger lesions such as the claimant's were associated with a less favourable outcome. Relying upon the paper by *Prado* he wrote only that there was a consensus that lesions such as the claimants are best treated by debridement and by microfracture. I agree with Mr Maskrey that any person reading this would be unaware that the 2018 consensus paper had (a) advised that microfracture be used after conservative treatment had failed and (b) microfracture is less likely to succeed when used as sole treatment in a lesion of 15 mm or greater.

99. For his part, Mr Forde invited me to find that Mr Laing lacked objectivity and that he failed to give a balanced view. He drew my attention to the fact that Mr Laing had referred in his report to the use of "*only a small K wire and drill*" when describing the defendant's microfracture technique in spite of the fact that the claim raised no criticisms of the defendant's surgical technique. He suggests that Mr Laing was a "lone wolf" in maintaining that the chondral lesion was wholly chronic and in his questioning the mechanism whereby such an injury may have been inflicted on the central part of the saddle shaped talus. He challenged the logicality of some of the views expressed by Mr Laing and submits that taking into account Mr Laing's relative lack of expertise in treating acute injuries (rather than elective surgery and especially within elite sports) when compared to both the defendant and Professor Ribbans, with the evidence that he has provided to this court, the impression is of an expert who is not taking an objective stance and advocating for the claimant.

100. I regret that I am unable to accept Mr Forde's recommendation of Professor Ribbans as a "*measured, considered and thoughtful*" expert witness. Professor Ribbans is an enthusiast for arthroscopy. He clearly has enormous respect for the defendant, a fellow specialist in treating elite sportsmen and women. He has allowed his enthusiasm for the procedure and his respect for the defendant (who Mr Forde referred to as "pre-eminent in his field" and "the leading surgeon in this field in this country") to cloud his judgement. He has been prepared to provide a supportive view on the merits of the claim without interrogating the basis upon which the defendant made the decision to operate. He has been prepared to make assumptions in the defendant's favour, assuming that the defendant's interpretation of the radiology was correct and that the defendant shared his concerns about arthrofibrosis. His report did not give a balanced picture about the merits of microfracture. Very regrettably, there is force in Mr Maskrey's submission that, overall, Professor Ribbans' evidence lacks objectivity and balance. I approach his evidence with this in mind.

101. In contrast, Mr Laing's evidence was in my judgement balanced. It is true that Mr Laing had been unable to see an acute element to the chondral lesion and that, even looking at it again at trial, he had been unable to see what others were referring to. But he accepted that there was such a lesion and provided an opinion on that basis. I am not particularly

surprised that he had difficulty in seeing the acute element, as Professor Ribbans said, "*the differences of opinion between [the radiologists] attest to the problems of interpretation in these difficult cases*." Nor was he alone in having difficulty in understanding the mechanism of injury. The defendant recorded that the tackle had not included a rotational/torsional component. Dr Miller accepted in cross examination that the mechanism of injury must have included a torsional twisting element. Dr Patel believed that a rotational force would have been needed in the absence of a really significant axial force, although accepted that a valgus force may have been sufficient given the chronic changes to the cartilage.

102. Mr Forde drew my attention to the judgment of HHJ Richard Williams in *Keane v Tollafield County Court* [2018] 8WLUK 306 in which Mr Laing had been criticised for being argumentative and hostile. I can make no comment upon the merits of those criticisms but for my part I did not find Mr Laing to be combative in his responses nor did he give the impression that he was advocating for the claimant. I found him to be a straightforward witness doing his best to assist the court.

103. Mr Forde's real criticism of Mr Laing is that he does not enjoy the same high profile as either the defendant or Professor Ribbans in the world of elite sports medicine. He is not pre-eminent in this field and may not have the same level of experience in dealing with acute ankle injuries in an emergency context. This may be true and I take it into account fully. But I am satisfied that Mr Laing has more than sufficient and appropriate expertise and experience to give an expert opinion on the merits of arthroscopy versus conservative management in this claimant. There is nothing so unique about this claimant or his circumstances as to disqualify or even undervalue Mr Laing's role as expert. His views were balanced and cogent and his opinions were logical. I accept them for the reasons which I now set out below.

Conclusion on Breach of Duty

104. I have already found that the defendant's rationale for performing arthroscopy was to investigate the potential instability of the chondral lesion irrespective of whether the lesion had or had not been caused or exacerbated by the recent tackle. To proceed on that basis was not an approach which he would, or could, support in 2025. Nor would the claimant's experts, or Dr Miller for whom the merits of intervention hinged upon whether the lesion was acute or chronic. This is relevant to the question of breach of duty but, as Mr Maskrey invites me to, I go on to examine the justification for arthroscopy in the light of my finding that there had been some ankle damage during the tackle causing a sprain to the AITFL and an element of additional damage to the chondral lesion.

105. Mr Laing has pointed out (and this is not disputed) that the decision to undertake arthroscopy in this case involves a commitment to debride scar tissue for the purpose of visualising and accessing the chondral lesion, to debride the loose cartilage from the lesion over the talar dome and to perform microfracture. No one has suggested that, following the introduction of the arthroscope, the surgery ought to have been terminated before the debridement or before the completion of the microfracture. The focus is therefore upon the decision to perform that procedure in the knowledge that it would involve those other processes; as opposed to the alternative which was to adopt a wait and see approach and if, in due course, the ankle became symptomatic then undertake arthroscopy, debridement and microfracture.

106. I accept that one of the most important considerations informing the decision to operate or manage conservatively is, or should be, the fact that the claimant had been, prior to the tackle, a professional sportsman with a pain free and functioning ankle notwithstanding his 2005 injury. He had been playing well for a Premier League team and had been that team's top goal scorer. It is not controversial that the ankle stiffness was due to the formation of scar tissue due to syndesmotic instability. I accept Mr Laing's evidence that the formation of scar tissue and associated stiffness was protecting the ankle joint, serving to limit sideways movement caused by the residual unstable syndesmosis and providing a level of cushioning of the front of the ankle joint, so enabling the ankle to function pain-free. I accept that the removal of scar tissue for the purpose of visualising and accessing the chondral lesion was in this claimant's case a relatively destructive process with the potential to de-stabilise the biomechanics of what had previously been a stable ankle joint.

107. Mr Laing and Professor Ribbans focussed upon literature when addressing the likelihood of microfracture resulting in the successful re-growth of cartilage in this case. I have found that the most useful and authoritative piece of literature brought to my attention is the 2018 consensus (following a meeting in 2017). Although it post-dates the events under scrutiny I accept that it reflected the consensus view in 2013. That report makes clear that there was overwhelming support for consideration being given to the use of microfracture for cartilage lesions which have failed conservative treatment, that is, those which were symptomatic. As to the size of lesion, the consensus view was that lesions greater than 10 mm in diameter are at greater risk of failure and the "ideal size" of lesion is considerably smaller than the claimant's, less than 10 mm.

108. Professor Ribbans raises a number of objections to the prognostic relevance of the size of the lesion. He told me that the size of the chondral lesion in this case was variously described and that it is difficult to assess the size accurately by the MR images alone. He said he had himself tried to measure the lesion over the weekend break before his evidence. He suggested that the location of a lesion and/or whether a lesion was contained or not are more important prognostic signs. Like the defendant, he placed emphasis on the *Zengerink* paper in support of the claim that use of microfracture was associated with a successful outcome in around 85% of cases. However, neither the defendant nor Professor Ribbans take into account that the *Zengerink* paper (a) emphasises that non-operative treatment should always be the first treatment to be considered; (b) that the percentage chance of a successful outcome with conservative treatment is between 45% and 53% and (c) the *Zengerink* study does not grade success by reference to the size of the lesion.

109. What I take from the literature review is that microfracture is strongly indicated as treatment for symptomatic chondral lesions but is not indicated before conservative treatment has failed. Further that microfracture for lesions of the size of the claimant's whilst not prohibited or contra-indicated is likely to be associated with a significantly higher chance of failure than microfracture for smaller lesions.

110. As to the potential benefits of arthroscopy there is no evidence before me that the element of acute delamination of the chondral lesion was bound to cause symptoms, particularly in circumstances in which the much larger lesion had been asymptomatic and pain free during the course of the previous 7 or 8 years. If it were to become symptomatic then it would be due to the lesion shedding flakes of degenerate cartilage causing third party wear and tear or impingement on other structures but no time frame was given for that process. The

loose flakes referred to by Dr Patel were small and difficult to age and cannot be said on balance to have been displaced.

111. As against this, there was a prospect that conservative treatment would be successful in the sense that the claimant would be able to return to elite football. Professor Ribbans put a figure of between 45% and 55%. He quickly qualified this percentage by pointing out that it did not take into account the other features of the claimant's presentation in particular the "subtle syndesmotic instability." But even if this was an optimistic assessment, there clearly was even on Professor Ribbans own evidence a real chance that conservative treatment would lead to a pain-free ankle for a period of time. This compared with Professor Ribbans' evidence that there was a 33% chance of microfracture failing to enable the claimant to return to elite football.

112. Professor Ribbans gave the court a list of reasons why the arthroscopy was not only indicated but mandated or "gold standard." Top of his list was the "grade 2" sprain and the suspected instability of the syndesmosis. The difficulty with his focus on the syndesmosis is that it takes no account of the fact that in April 2013 the defendant did not appear to believe that there had been a serious recent ligament injury. Nor could he have done so. I accept that during the operation the defendant had to hand a tightrope in case he found instability during his examination under anaesthesia but his contemporaneous note sets out that he thought the syndesmosis was stable. He found no tenderness around the AITFL. He noted abnormalities around the AITFL and PITFL but thought that these were old and he believed the syndesmosis was in fact normal. The stability of the syndesmosis was confirmed on EUA. I accept his evidence that he made further checks during the course of the arthroscopy but I agree with Mr Maskrey that it would be wholly illogical to perform an invasive procedure for the purpose of examining a structure which you believe clinically and radiologically to be normal and which is then confirmed to be so on EUA. Although Mr Forde makes much in his closing submission of Mr Laing's analogy of the arthroscopy being a "Ferrari" compared with the "Ford Fiesta" of the EUA, the analogy with Mr Laing made was not particularly apt. It is clear that he did not think that an arthroscopy was a gold standard Ferrari of a procedure in the circumstances of this case, rather it was an unnecessary and unjustifiable procedure.

113. Nor do I accept that arthroscopy was indicated for the other purposes listed by Professor Ribbans. I appreciate that he is strongly of the opinion that scar tissue is a bad thing because it produces cytokines and is for that reason noxious. But in this case, the scar tissue had been serving a useful function stabilising the joint. I understand that he has a special interest in the effect of blood on bone and cartilage and would have advocated arthroscopy to wash out the joint. But again, this formed no part of the defendant's thought processes. Neither of these reasons support of the defendant's case.

114. Taking all of these factors into account, I am satisfied that the decision to undertake arthroscopy (and the other procedures) was neither reasonable nor logical. I find that the defendant was in breach of duty in performing an arthroscopy on a previously symptom free claimant who had suffered a modest acute ankle fracture only. I have found that both the defendant and his experts have overstated the seriousness of the ankle injury both in terms of the forces applied to the ankle and the extent of the injuries sustained. As Mr Maskrey has submitted, the procedure to be adopted was going to (and did, according to the defendant's operation record) involve the removal of a large amount of scar tissue to visualise and access the chondral lesion and the removal of chondral tissue exposing the

bone beneath. There was therefore a clear risk of destabilising the joint by altering the talar load. Microfracture was not an advised procedure in asymptomatic patients. Microfracture was associated with a less favourable outcome in a lesion of the claimant's size. Conservative treatment had a real chance (possibly as high as 50%) of returning the claimant to elite football. I find that the only surgery which ought reasonably to have been performed on 8 April 2013 was the reduction and fixation of the fibula and that the ankle should have been left alone and monitored.

Causation

115. I turn therefore to the question of causation. Mr Maskrey invites me to conclude that the claimant has suffered pain on weight-bearing; that this pain was caused or materially contributed to by the procedure which the claimant underwent on 8 April 2013 and that the pain has prevented him from pursuing a career in elite football for a period of 3 to 5 years (following the surgery in April 2013).

116. The defendant's case is that, if the claimant did suffer ankle pain on weight bearing, then it was caused by natural degeneration of the ankle and hindfoot as part of more widespread arthritis and that his long term sequelae were due to progressive post traumatic osteoarthritis due to his medical and injury history. The effect of the injury was to steepen the curve. There was no contribution from the surgery itself. In fact, Mr Forde submits that the surgery restored the claimant to his pre-operative condition and this enabled him to play with a stiff but painless ankle initially for a football club at a League above the one to which his previous team had been relegated.

Did the claimant suffer ankle pain on weight-bearing?

117. The first question for me is whether I am satisfied that the claimant suffered ankle pain following the resumption of weight bearing in the late summer/autumn 2013. It had not been clear to me from the defendant's opening that this point was in issue but Professor Ribbans did not accept that the claimant did experience pain in his ankle in 2013. He pointed to the absence of any record in the claimant's rehabilitation notes to that effect. He remarked that if the claimant had experienced pain then it was likely to have come from the delayed and imperfect union of the fibula fracture rather than from the ankle. The point is picked up by Mr Forde in his closing submissions so I deal with it now.

118. The claimant's evidence is summarised above at paragraphs 33 and 34 above. He said that he had felt a "bone on bone" type of pain when he started weight-bearing and that this pain continued and became part of his life as a player. He was treated with steroid injections and topical pain killers. He was taken through his rehabilitation records by Mr Forde, including references to stiffness but no pain in September 2013. But his account that he felt pain on weight bearing was not challenged. Nor was it suggested to him, as later hypothesised by Professor Ribbans, that the pain did not come from the ankle but from the fracture site.

119. The claimant gave evidence to me in a straightforward fashion and I accept his evidence and find that he began to feel ankle pain on weight bearing.

What was the cause of the claimant's pain?

120. I am satisfied that the surgical procedure of 8 April 2013 caused or materially contributed to the claimant's ankle pain.

121. Like Mr Maskrey I am left wondering in what way or ways the acute injuries which the claimant suffered during the tackle could be responsible for the claimant's ankle pain. Professor Ribbans accepted that the pain could not be due to the presence of loose fragments of cartilage impinging on the ankle joint or chafing adjacent structures. Whether old or new, all of the fragments seen on imaging had been removed by the defendant on 8 April. Nor could the claimant's post-operative ankle pain be due to any syndesmotic instability because the syndesmosis was comprehensively established to be stable via three methods of testing. Neither Dr Patel nor Dr Miller suggest that the December 2013 imaging shows any residual ligament injury capable of producing pain. Nor can the degenerate cartilage be implicated because that too had also been removed by the defendant. Although Professor Ribbans was asked about possible mechanisms he was only able to say that the effect of the tackle had been to accelerate the course of the claimant's osteoarthritis generally which explained the presence of more rapid arthritic progression distant from the operation site. He also mentioned that vitamin D levels had a strong input into pain perception. But I got no clear account from him as to the mechanism by which the osteoarthritis had been accelerated by the injury in such a way as to lead to ankle pain on weight-bearing.

122. By contrast I am satisfied that there are biologically plausible mechanisms linking the arthroscopy with the claimant's ankle pain. I start with the topic of arthrofibrosis. The evidence of Mr Laing and Dr Patel was that the April 2013 MR images showed that the arthrofibrosis extended into the medial gutter in the ankle joint. Mr Laing commented that the scar tissue extended into the anterior lip of the tibia. I accept this evidence. Further, I am satisfied that scar tissue from within the joint was removed. In reaching this conclusion I am conscious that the defendant was adamant that he did not remove any scar tissue in the joint and removed only that which was necessary to visualise the chondral lesion. However, in his contemporaneous note of the procedure he recorded that he removed "*a large amount of scar tissue*" in order to "*gain access/good views*" of the joint. As Mr Maskrey points out, in order to gain access to a central lesion within the saddle of the talar bone for the purpose of assessment and debriding, it will be necessary to remove scar tissue from within the joint. The same point was made by Mr Laing who remarked that it would be inevitable that some fibrosis from within the joint would have to be removed because, as he said, given the site of the portal of the arthroscope "*you are straight down onto it, down there in the medial gutter*." It is unfortunate that the December 2013 scan was not examined in detail by the radiologists but even in the absence of a review of that scan I am satisfied that scar tissue from within the joint was removed.

123. In any event, it is not contentious that the defendant removed the degenerate cartilage which had covered the talar dome. I accept Mr Laing's evidence that its removal would have altered the loading pressures on the remaining cartilage on the shoulders of the talus. I accept Professor Ribbans' point that the remaining cartilage was degenerate and may not be connected to the underlying bone but it was, at the time of the arthroscopy, still sitting on the talar dome. He disagrees that the degenerate cartilage would have any value in stabilising the structures within the joint but provides no good reason.

124. I am satisfied that the removal of the scar tissue and/or the removal of the degenerate cartilage was to cause the claimant's pain. The removal of the scar tissue destabilised the joint by removing the cushioning and by altering the biomechanics of the joint leading to increased movement of the joint, increased instability and acceleration of degenerative

change in the ankle joint. The effect of the removal of the cartilage was to increase the pressure on the cartilage which remained on the shoulders of the talus.

125. Dr Patel identified bone oedema under the microfracture site which was more extensive than he would have expected following microfracture. He said that before the operation there had been little if any oedema at the microfracture site but that the oedema was now extensive and equivalent to where the degenerate changes had progressed elsewhere. Dr Miller made no specific comment upon the presence of oedema but Professor Ribbans told me that in his opinion the extent of the oedema was no greater than he would have expected 8 months post-surgery in combination with the natural history of the ankle. Mr Maskrey postulates that, if I were to accept the evidence of Dr Patel, the excessive bone oedema must be a response to microfracture, there can be no other explanation.

126. I accept Mr Maskrey's point that the excessive bone oedema may be associated with the microfracture technique and I agree with him that the presence of excessive oedema beneath the microfracture area is a strange coincidence. But the link between the technique and the excessive bone oedema is not spelt out in the expert evidence and I am unable to find to the requisite standard that the technique led to the oedema and that thus made a material contribution to the claimant's pain.

127. I am however satisfied on the balance of probabilities that the arthroscopy caused or materially contributed to the claimant's condition in two separate ways: by the removal of scar tissue and by the removal of the cartilage lying on the talar dome.

128. This leads me to the final topic below.

What would have been the likely course of the claimant's symptomology had there been no arthroscopy, debridement and microfracture?

129. The question here is within what timescale the claimant would have developed symptoms or pain assuming that the defendant had not performed the arthroscopy, debridement and microfracture.

130. Mr Laing confronted the topic in his evidence in chief. He confirmed his view that on the strong balance of probabilities the claimant would have returned to his previous state assuming conservative treatment. The claimant had sustained only a modest ankle injury (a sprain and an acute cartilaginous component to the talar dome lesion). He had remained pain-free for a long period of time and he had managed very well during that time in spite of the scar tissue. Although he had stiffness he had coped with that. His said that it was difficult to be precise about the timescale during which the claimant would have remained pain-free. He reasoned however that the claimant had: "*gone eight years and had no pain, and he wasn't even developing pain, so you're at a situation where you are pain-free, coping but you have a hidden lesion, a latent lesion which at some point is going to create a problem.*" He said that he thought that the claimant would probably be able to continue to play football at a good level for "*probably three to five years*" and that that remained his professional opinion. He acknowledged that it was very difficult to be precise about these things. Mr Laing was not cross examined on this point.

131. Professor Ribbans does not address the scenario which I have found established, namely, that the acute injuries to the ankle were limited to a sprain and an area of further delamination. I make no criticism of him in this regard, he was not asked for his view on

this specific point. His view (expressed in the joint report) was that if fragments had remained within the ankle joint then the outcome would have been inferior. Fragments are a source of pain and irritation for a joint. In his evidence in chief he remarked that if the arthroscopy had not been undertaken then the arthrofibrosis would have got worse; there would have been increased scarring and that the post traumatic haemarthrosis and any detachment of cartilage fragments, were "*all core prognostic indicators going forward for the claimant to return to elite football*." But this assessment was not based upon the findings which I have made.

132. I am satisfied on balance that, but for the arthroscopy, the claimant would have returned to his pre-accident pain-free state. As I have found, the ankle injuries were modest only. There was no reliable evidence before me that as a result of the further section of delamination, fresh sharp fragments of cartilage were shed and displaced into the joint. I have already explained that I do not accept that the defendant's evidence on this topic is reliable. I have rejected Dr Miller's evidence concerning the presence of large dark angular fragments and Dr Patel's evidence was only that there were a number of small fragments of cartilage in the joint which were hard to date. Even if some of the small fragments which Dr Patel was able to visualise on the scan were fresh it does not follow that they would have caused pain. As Dr Patel clarified in re-examination, fragments of cartilage are capable of causing symptoms. Whether they do will depend upon their size, location and which structures if any they impinge upon. As Mr Maskrey has said, the claimant had been pain free for 8 years notwithstanding an ongoing degenerative process causing the talar dome to fissure and delaminate.

133. In the circumstances I accept the evidence that the claimant's ankle would have remained pain free for a period of 3 to 5 years during which time he would have been capable of playing football at a high level. However, I emphasise that my findings are limited to the claimant's ankle condition in the "but for" or hypothetical scenario. Whether or not the claimant would have continued playing football at a high level and how that chance is to be quantified is a very different question indeed and one for the quantum trial. I agree with Mr Forde that there may be a host of different factors impacting upon the claimant's chances of continuing to play at elite level including other injuries, his abilities as a player, the opportunities available to him. This topic will doubtless be explored with the assistance of expert evidence in due course.

134. There will be judgment for the claimant in the terms set out above. I invite the parties to draw up the appropriate order in due course including any directions which are sought for the preparation of the case on quantum. Given my findings, I do not deal with the claimant's secondary argument on informed consent.