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Case No: QB-2020-003377

IN THE HIGH COURT OF JUSTICE
KING'S BENCH DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 31st July 2024

Before :

MR JUSTICE SWEETING

Between :

Karen Spellman	Claimant
- and -	
Portsmouth Hospitals University NHS Trust	Defendant

Cara Guthrie (instructed by Irwin Mitchell LLP) for the Claimant
Anna Hughes (instructed by DAC Beachcroft) for the Defendant

Hearing dates: 23 October 2023 - 30 October 2023

Approved Judgment

This judgment was handed down remotely at 14:00pm on 31 July 2024 by circulation to the parties or their representatives by e-mail and by release to the National Archives.

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MR JUSTICE SWEETING

Introduction

1. This is my judgment following a trial of the issues of liability and causation. Damages are agreed at £300,000.00, net of sums due to the Compensation Recovery Unit and contingent upon the Claimant proving breach of duty and causation.

2. The Claimant asserts that the Defendant breached its duty of care to her by failing to identify Cauda Equina Syndrome ("CES") during the radiological review of an MRI scan on June 6, 2017.

3. The spinal column (the backbone) has a central canal through which spinal nerves pass. Each individual bone of the spine is called a vertebra. There are 33 vertebrae in total, which, for the purpose of medical classification, are grouped into five regions: cervical (neck), thoracic (upper back), lumbar (lower back), sacral (base of the spine), and coccyx (tailbone). At each vertebral level there are spinal nerve roots which branch out to each side and facet joints which are small joints which sit between the bony process and help the spine to bend and twist. There are five vertebrae in the lumbar spine conventionally identified as L1 to L5 (where L5 is the lowest vertebra). Between each vertebra there are intervertebral discs that act as spacers and shock absorbers. These discs consist of a soft gel-like centre (nucleus pulposus) surrounded by a tougher fibrous wall (annulus fibrosus). The solid spinal cord ends at the lumbar spine and becomes the 'cauda equina'.

4. The cauda equina is a bundle of spinal nerves and nerve roots which supply the lower limbs and pelvic organs. It is named, in Latin, for its resemblance to a horse's tail. It provides motor and sensory functions to the lower extremities, as well as controlling functions of the pelvic organs. The nerve roots and cauda equina are surrounded by cerebrospinal fluid ("CSF") and contained within a membrane called the 'thecal sac' or 'dural sac' made of 'dura mater'. CSF is a clear, watery fluid that surrounds and protects the brain and spinal cord; it appears as a bright area in standard MRI scans. In a healthy spine a cross section scan (an axial or transverse scan, conventionally viewed from the feet upwards) can visualise the individual nerve roots of the cauda equina surrounded by CSF in the spinal canal. An MRI "scan" will normally consist of multiple images taken at different levels and in different planes.

5. CES is a serious condition caused by compression of the nerve roots and can cause paralysis and loss of bowel or bladder control if not treated promptly. A missed or delayed diagnosis of CES can lead to permanent and debilitating complications. CES is diagnosed through a combination of patient history, physical examination, and imaging. Prominent symptoms include back pain, numbness or loss of sensation in the area of the buttocks, inner thighs, and genitals, (the area of the body that would be in contact with a saddle when sitting on a horse and so referred to as "saddle anaesthesia"), leg weakness and difficulty urinating, incontinence, or loss of bowel control. Where there is a diagnosis of CES urgent surgery may be required to relieve pressure on the nerves and prevent permanent damage. Thus, where diagnosed, CES is treated as a medical emergency.

6. The causes of CES include:

   i) A herniated disc in the lumbar region where a disc bulges or ruptures and compresses the nerve roots.

   ii) Lumbar spinal stenosis which is a narrowing of the spinal canal in the lumbar region.

   iii) Trauma to the lower back.

7. The Claimant alleges that she suffered from CES with bladder and sensory issues on June 6th, 2017. She claims that the MRI scan performed on that day was misread as showing only mild spinal canal narrowing (stenosis) and that the reporting radiologist failed to identify significant cauda equina compression.

8. Had the MRI been interpreted correctly she argues that she would have received decompression surgery by the morning of June 7th, leading to a better outcome.

9. The Defendant denies any breach of duty in relation to the MRI report, arguing that the Claimant did not have CES. As a consequence urgent surgery was not necessary but even with hypothetical surgery by June 7th, 2017, the Claimant's condition and prognosis would not have improved.

10. There are essentially two key issues:

   i) Did the Claimant have CES, or was the initial MRI report (mild stenosis) accurate and/or reasonably reported?

   ii) Would timely surgery (following, on the Claimant's case, a correct diagnosis) have improved the Claimant's outcome?

11. The Defendant accepted that if acute cauda equina compression should have been reported on the scan of 6 June 2017 (or any scan performed later that day) then the Claimant would have undergone surgical decompression by the morning of 7th June 2017.

Background

12. The Claimant's medical records are extensive. It would be fair to say that she has struggled with obesity and addiction over a long period and has had a number of other medical problems. She was a frequent visitor to her local medical centre, at Bramham when she lived in Yorkshire and then at Bridport, after she married and moved to Southampton in around 2012. It appears that she first visited her GP complaining of acute back pain and sciatica in the course of 2003. There were similar visits in 2004 and early 2005.

13. On 1 February 2005 she was admitted to the Leeds General Infirmary with sudden onset back pain radiating down the left leg, with paraesthesia. On February 4, 2005 a lumbar microdiscectomy at the L4/5 level (removal of a herniated disc) was performed.

14. She was admitted again on 26 May 2005 with a history of back pain and urgency of urination. The discharge letter to her GP records*: "she underwent an MRI of lumbar spine which showed minor operative changes but nothing neurologically acute. On examination she was found to have back pain, there were no cauda equina features. She later on had spinal injection by one of our anaesthetists for pain control, this proved to be effective therefore she was allowed home."*

15. She was then admitted on 1 June 2005 because she had slipped whilst running on the stairs and landed on her buttocks. She complained of pain in the back and down her left leg to the knee, as well as urinary incontinence. The discharge letter to her GP records that the MRI scans were satisfactory with no evidence of cauda equina compression and concludes: *"From a neurological point of view we are satisfied that there is no cord or nerve root compression. Clinically she has no sciatica."* She was referred to the chronic pain team.

16. She was admitted again on 5 November 2005 as an acute transfer because of backpain. The admission notes indicate that she had fallen while walking in town. Revision surgery was performed to the original operation site.

17. In October 2006 she underwent an exploratory operation under general anaesthetic following the loss of a catheter (having started self-catheterisation earlier that month) after which she developed further pain in her back.

18. The Claimant experienced a number of post-surgical complications although whether they were linked to the surgery carried out in 2005 and 2006 was unclear. On 23rd of November 2006 the neurosurgeon treating the Claimant recorded:

> *"Although she never had true cauda equina syndrome she has developed urinary difficulties and has some evidence of detrusor instability.*
>
> *...*
>
> *In that I have never demonstrated pressure on her nerves nor did her urinary problems immediately follow her surgery I am somewhat mystified as to their origin."*

19. In December 2006 she was admitted for further revision surgery at the L4/5 level.

20. Urodynamic studies confirmed the development of an acontractile bladder, meaning that the bladder muscles lacked the ability to contract to expel urine. This necessitated intermittent self-catheterisation. On March 27th, 2007, the Claimant underwent an assessment at the urology department in Wakefield. During the assessment, the Claimant reported:

    i) Continued dependence on self-catheterization for urination.

    ii) Constipation.

    iii) Faecal incontinence (involuntary passing of stool).

    iv) Leakage of stool.

    v) Vaginal paraesthesia (abnormal tingling or burning sensation in the vagina).

21. Thus by 2017 the Claimant had a history of chronic back pain which had led to surgical interventions and had longstanding urological problems which had resolved to the extent that she was no longer seeking medical treatment and had returned to employment. The Claimant's evidence was that she had normal bladder and bowel function

22. She had undergone three spinal surgeries on the L4/L5 vertebrae as follows:

    i) February 4, 2005: Lumbar microdiscectomy (removal of a herniated disc)

    ii) November 9, 2005: Revision laminectomy/discectomy (reopening of the spinal canal and removal of disc material)

    iii) December 19, 2006: Revision lumbar surgery for degenerative disc disease (surgery to address wear and tear in the disc)

23. On a number of occasions the Claimant had sought medical attention for symptoms potentially indicating CES, including after falls. MRIs performed during the investigations which resulted did not reveal any cauda equina compression. It was characteristic of the scans that a number of the images were sub-optimal because the Claimant was in pain and had moved. Although there continued to be frequent visits to the GP after 2006, in relation to a variety of matters, there are then far fewer complaints of back pain with the last major episode prior to 2017 taking place in January of 2015 but with a complaint of low back pain in January 2017.

The Events of June 2017 in Summary

24. The Claimant's case is that she had a work-related fall on June 5, 2017. This triggered back pain that radiated down her legs and into her left buttock. She consulted her general practitioner ("GP"), Dr Wade, the next day (June 6, 2017). The GP notes record:

> *"Twinges of pain 3/7 ago. Decline in past 24hrs. On left side of back. No radiation to lower legs. Some perineal numbness. Felt strange when she PU. No incontinence. No bowel action (normal for PT).*
> *Cough impulse > local pain.*
> *Difficulty getting straight.*
> *Rx ibuprofen and Anadin extra, regularly tramadol nocte and trying to wean off due to past addiction.*
> *PMH: Microdiscectomy surgery ?L3/4 x 2-3, over the past few yrs. Last time 10 yrs ago. Leeds Gen Hospital. Reqd self catheterisation before and after surgery.*
> *Dw oncall orthopaedic reg in view of sacral sx.*
> *back: scar from past surgery. Stands with right tilt due to pain on left. Tender to left para spinal muscles. SLR R/L: full/40 degrees. Reduced light touch to left leg L4/5 but also @ left sacral area. Normal anal tone and power. Reflexes R/L K+/+ A+/+ plantars down*
> *Low back pain. No trigger activity."*

25. There is no reference to the Claimant having fallen in these notes which are predicated on there having been *"no trigger activity"*. Dr Wade consulted a hospital orthopaedic registrar who recommended a hospital visit. The Claimant was then seen at 11:30am on 6 June at Queen Alexandra Hospital, Portsmouth for an initial assessment by Dr. Adukia, an orthopaedic resident. Her notes record:

> *"Had previous discectomy for ?cauda equina 10 years ago at Leeds.*
> *Presented L leg numbness + low back pain + saddle anaesthesia on that occasion. Had to intermittently self catheterise then post op*
> *All symptoms resolved post surgery*
> *Now presenting 4/7 HX of sudden onset low back pain radiating to L leg + buttock + L leg numbness.*
> *Yesterday afternoon C/O abnormal sensation when passing urine ~ 3pm. 1 episode of faecal incontinence on Sunday"*

26. Again there was no reference to a fall at work. The 6 June was a Tuesday so that the reference to Sunday in the note would have been to an episode of incontinence on Sunday 4 June (prior to the fall). The timing of 3pm as marking the onset of abnormal sensation when passing water is linked to the descriptor "*Yesterday afternoon",* which is to say 5 June.

27. A physical examination led to the following findings being recorded:

    i) Left Leg:

a) Paraesthesia: Abnormal tingling or burning sensation throughout the entire leg.

b) Reduced sensation: Decreased ability to feel light touch in specific areas of the leg (L1-L4 and S1-S2). These zones correspond to specific nerve distributions in the lower back and leg.

c) Reduced power: Weakness in flexing and extending the hip and knee, although it was considered that pain might be a contributing factor.

ii) Right Leg:

a) Normal sensation and power: No abnormalities detected.

iii) Reflexes:

a) Knee reflexes: Normal.

b) Ankle jerk: Absent on the left side.

iv) Rectal examination

a) Anal tone: Normal.

b) Sensation: Reduced ability to feel a pinprick on the left side.

28. The symptoms were therefore predominantly on the left side and raised the possibility of nerve damage or compression on the left side of the lower back interfering with the nerves running down the leg.

29. An MRI scan was performed at 2:00 pm and the results were reported by Dr. Witham, a consultant radiologist, at 3:12 pm. The allegations of negligence are focused entirely on the reporting of this scan. The relevant parts of the MRI report, including the observation that there had been *"no trauma"* are:

> *"MRI Spine Lumbar (MLUSP) Clinical history: Previous discectomy 10 years ago. 4 days back pain radiating to left leg with left leg numbness and subtle anaesthesia. No trauma. Left L1-S2 numbness. Left L1-L4 weakness. Depressed left ankle reflex. Altered PU sensation 18 hours. Reduced PR sensation.*
>
> *MRI Spine Lumbar: There is much movement artefact. There is Modic type I endplate reactive change posteriorly at the L2-3 level with post inflammatory fatty endplate reactive change Modic 2 at L4-5 and L5- S1. There is multilevel lumbar disc degeneration. The canal appears slightly tight at the L2-3 level and the L3-4 level where there is less CSF seen within the thecal sac. Epidural fat playing a significant part of this*

constriction of the thecal sac. Post surgical change seen at L4- 5. The conus lies at the normal level of T12-L1. Low thoracic vertebrae are aligned and lumbar vertebrae are aligned with preservation of vertebral height

Conclusion: Poor quality spine, patient in too much pain to keep still for the scan. Modic Type I change L2-3. Disc degeneration and epidural lipomatosis 7 contributing to L2-3 and L3-4 mild central canal stenosis. Post surgical change L4-5."

30. Following a consultation with Mr. Luckos, an Orthopaedic Registrar, Dr. Adukia determined that the available clinical findings, including the MRI report, did not warrant a diagnosis of CES.

31. The Claimant was discharged from the hospital later that day, at 4:30 pm. A follow-up outpatient clinic visit was scheduled for August 3rd, 2017, for further evaluation and the formulation of a treatment plan.

32. The Claimant sought a private medical opinion on June 12th, 2017. After initially seeing another doctor who referred her on, she was reviewed by Mr. Davies, a Consultant Spinal Surgeon, at Spire Hospital (a private hospital). After reviewing the MRI scan from June 6th, Mr. Davies diagnosed cauda equina compression and proceeded, on the same day, with surgical decompression and lumbar discectomy of the Claimant's spine at the L3-L4 level. Mr Davies does not appear to have made any notes when he saw the Claimant or to have recorded a history. He did not ask for a further MRI scan to be performed and must have regarded the MRI performed at Queen Alexandra Hospital as diagnostic although both of the expert consultant spinal surgeons who gave evidence thought it was not. He did not contact the radiology department at Queen Alexandra. It does not appear that he had Dr Witham's report since he does not refer to it.

33. Prior to the operation he dictated a letter which recorded:

"Thank you for telephoning me about Karen this morning. As you know she has had multiple surgeries in Leeds and presents with significant bilateral leg pain, sensory change around the perineum and a couple of urinary losses which has been going on for a while unfortunately.

Her MRI from Portsmouth shows a two level decompression but at the level above she is stenotic and there looks like there is a big disc as well and so I suspect this is cauda equina which may have missed the boat.

She needs an urgent decompression. I have discussed the risk of non- improvement. She is bariatric which obviously makes things more difficult and challenging but I will try and get her onto my private list today at Spire. If not, we will get her transferred urgently over to the General."

34. Mr Davies's operation note records the indication for surgery *as "bilateral leg pain with disc prolapsed on MRI, perianal numbness loss of bladder sensation and single episode of urinary incontinence."* This is to be contrasted with his letter which refers to *"a couple of urinary losses."* Presumably if there had been a more extensive history of incontinence between 6 and 12 June he would have recorded it as a further indication for urgent surgical intervention. There is no indication of how "*bilateral leg pain*" was elicited as a symptom nor whether it marked a progression or deterioration.

35. Mr Davies was not called as a witness so I should make it clear that my observations are based on the limited documentation referred to and do not involve any conclusion about his professional judgement or his care of the Claimant.

36. A post-operative MRI scan was performed at the Spire Hospital on 25th July 2017. The radiologist (Dr Matthew Thomas) noted that he had no prior imaging available with which to make any comparison. It follows, curiously, that he did not see the MRI images which had been referred to Mr Davies's operation note and letter.

37. Despite this surgical intervention, the Claimant continues to experience a range of persistent sequelae (after effects) the most significant of which are:

    i) Urinary dysfunction necessitating intermittent self-catheterisation.

    ii) Bowel dysfunction requiring management with laxatives and manual techniques.

    iii) Neuropathic pain manifesting as burning or tingling sensations in the lower extremities.

    iv) Ongoing mechanical low back pain.

    v) Psychological sequelae in the form of an adjustment disorder.

38. On 24 February 2018 the Claimant was admitted to University Hospital Southampton with right sided backpain. A further MRI scan was performed which compared the position to that shown on the 6 June 2017 scan.

39. A subsequent letter to the Claimant's GP states:

> *"Patient admitted with acute chronic back pain via Southampton ED (known to chronic pain consultant Dr Hazelgrove). She reported right sided back pain - radiating down the posterior thigh and into groin and worsening urinary and bowel symptoms. She had an MRI and US pelvis neither of which explained her symptoms and she was seen by the pain team and OT and PT. She was discharged once her pain was management and MDT happy."*

40. This was therefore a complaint of radiating back pain on the right, rather than left side, with accompanying urological symptoms for which a cause could not be established on imaging. It did not progress to CES.

41. It would appear that the treating clinicians do not regard any further surgery as mandated or worthwhile.

Issues

42. The key factual issues are accordingly:

i) What is visible on the MRI scans from 6th June 2017 (pre-operative) and 25th July 2017 (post-operative)?

ii) What were the Claimant's bladder, bowel, sensory and leg symptoms prior to June 2017 and what were the specific symptoms on 6th and 12th June 2017.

iii) Was there was any progression in the Claimant's bladder, bowel, sensory, and/or leg symptoms between 6th and 12th June 2017.

iv) Was there compression of the cauda equina on 6th June 2017.

43. The allegations of breach of duty made against Dr Witham were helpfully summarised by Ms Guthrie on behalf of the Claimant in her opening note as follows:

> *"a. Wrongly described there being only mild central spinal canal stenosis at L2-L3 and L3-L4;*
>
> *b. Failed to report moderately severe central canal stenosis at L2-3 and severe central canal stenosis at L3-4 compared with the adjacent levels;*
>
> *c. Failed to identify that there was radiological evidence of gross pathology at L2-3 and L3-4 capable of causing the Claimant's clinical symptoms and signs of cauda equina compression;*
>
> *d. Failed to recommend, in the light of the Claimant's symptoms and clinical history, a further scan and/or discussion with the local spinal surgical centre."*

44. The causation issues which followed from a finding of breach of duty were then:

a) Would an MRI report identifying cauda equina compression have prompted another scan for confirmation or more precise localisation? What findings might this additional scan have revealed?

b) If the initial MRI had been interpreted correctly, would the Claimant have undergone emergency decompression surgery to relieve the nerve compression by the morning of June 7th, 2017?

c) Were the Claimant's specific symptoms on June 6th, 2017, a direct result of cauda equina compression?

d) Assuming that there was a delay in decompression surgery, did this delay directly cause the Claimant's ongoing bladder, bowel, sensory, and leg problems?

45. Ms Guthrie's opening submissions identified the central question as follows:

> *"... the single most important question for this Court is whether there is radiological evidence of cauda equina compression. If not, then it is accepted that it cannot have been a breach of duty not to report it and no damage can flow from not undertaking a decompression procedure."*

The Factual Evidence

The Claimant

46. The Claimant gave evidence. She described her history of back problems prior to 2017, her operations and her visits to her GP with pain and urinary symptoms. She was described at various points in her medical records as anxious about her health which was how she struck me. In 2017 she worked as a sales executive for a flooring company. On 5 June 2017 she said that she had slipped off a storage unit at work and landed on her bottom. She went to her first meeting at Salisbury but found she could not sit down. She called her boss and took painkillers. She woke up in the night but could not go to the toilet and thought that she must be dehydrated. The next morning she still had some issues with urination.

47. She went to see her GP but did not say that she had fallen, instead telling him (Dr Nigel Wade) that her symptoms had started three days before. She explained in her evidence that she had lied because she had panicked as she did not think that she had been taken seriously on previous occasions. Her symptoms had started only in the 24 hours following the fall on 5 of June. When she was referred to hospital under the CES pathway she accepted that the history she gave was also untruthful. She did not mention a fall and suggested that her symptoms had been going on for longer. She explained that she *"really wanted the doctor to take her seriously"*. The medical note, she said, was also inaccurate because it should refer to 3:00 am, as that was when she had woken up during the night.

48. When she was sent for an MRI scan she was in discomfort despite having been given painkillers. She could not stand properly or lie on the scanning bed although attempts were made to make her more comfortable. She was told, after the scan had been reported, that she

did not have to have surgery but said that she told the doctor that she was not prepared to leave until her pain was under control. The doctor went away and came back with morphine based painkillers. An appointment was made for her to return but she was told that she should do so before the date of the appointment if her symptoms got worse. After she had been discharged she was taking powerful painkillers and did not think she had a good memory of what had happened.

49. In her Witness Statement she described bowel incontinence and soiling in the period between 6 and 12 June 2017. At trial she was asked whether her symptoms had deteriorated after discharge. She said it was *"hard to say"*. Her boss was concerned about her pain and arranged for her to have a private consultation. When she saw Mr Davies at the Spire Hospital she told him about her problems with urinating from 5 June. She had manually evacuated her bowels on the morning she went to hospital. She was asked about the reference in Mr Davies's letter of 24 July 2017 (referring her to Miss Nugent, a pain specialist) to seeing her *"many weeks after progressive changes in her urinary symptoms"* and *"..her bladder and bowel symptoms persist"*. She said this was inaccurate, and that Mr Davies had not really taken a history but had simply looked at the scan and said that she needed an operation.

Dr Adukia

50. Dr Adukia was a Senior (Orthopaedic) House Officer ("SHO") at the Queen Alexandra Hospital and saw the Claimant on arrival. She had no recollection of her beyond what was in her notes. Dr Adukia was given a history of sudden onset low back pain radiating into the buttock and left leg which had come on over the course of four days in the form of a constant ache associated with left leg numbness. That was accompanied by a complaint of abnormal sensation when passing urine the day before and one episode of faecal incontinence two days before. She recorded the past medical history including the previous discectomy. She noted that the Claimant's symptoms appeared to have resolved after the earlier surgery. She carried out an examination with the results recorded in the clinical notes (see above).

51. She gave the Claimant pain relief, decided to ask for an MRI and then logged on to the system to see the results once it had been carried out. She noted that it was of poor quality due to movement artefact. She discussed the patient with Dr Luckos, the Orthopaedic Registrar, and said she would have read the radiological report to him if he did not have it. She was asked whether the scan, in her view, excluded CES. She thought that it did. The most common cause of CES was a large disc. It was suggested to her that patients can present with chronic changes which cause CES. She drew a distinction between a chronic condition and a critical stenosis that would require urgent surgery. She considered that they had excluded an acute CES which in turn excluded the need for surgical intervention. The cause of the Claimant's symptoms could, in her view, have simply been back pain or stenosis (she was of

course unaware of any trauma caused by a fall). She struck me as a diligent doctor who took care to try and get to the bottom of the Claimant's problems.

Dr Luckos

52. The parties provided an agreed note of his evidence which accords broadly with my own note.

53. He had not met the Claimant but he did recall the conversation with Dr Adukia, who, he said, was very concerned when she spoke to him about the patient's care. He thought that at that stage he was in the Accident and Emergency department, hence the conversation had taken place on the phone, but that he subsequently spoke to Dr Adukia again on the ward. He did not think that this latter conversation was to discuss the scan or the pain regime but was, perhaps, to consider when the Claimant should next be booked in to be seen at the hospital.

54. He thought that he had read the report but he was not 100% sure if he had seen the scan although he considered that he most likely would had done so, because that was his normal practice. If so, he would have asked for the report before he opened the scan on a workstation. His evidence was that if he had taken a different view from the radiologist, based on looking at the scan, he would not simply have deferred to her in assessing it but would have called to speak to her. He did not and he would not have taken a different course had he been asked to reconsider the scan "today". He commented: "*For 80-90% people who present, a scan will be negative for a surgically reversible cause. There is no established pathway for urgency of decompression for stenotic patients.*"

55. He had subsequently looked at the scan for the purpose of the trial and although it was hard to interpret, his conclusion was that it showed a moderate rather than a mild stenosis. He thought the stenosis was compressing the thecal sac. On the assumption that he had seen the report and scan at the time, he would not have taken a different course. He explained that the purpose of the scan was to get evidence to see if there was a problem that was surgically reversible. The question was whether there was compression and whether surgery was urgent. He agreed that he would want to ask whether the scan excluded CES and that an adjacent double level compression would have a greater impact. He agreed that the pathology should be considered and he would want to know about any fat cysts, disc bulges, the absence of CSF and the extent of the restriction. He agreed that he regarded the report as negative for CES and that it reassured him that emergency surgery was not indicated. His own view was that the scan excluded any surgically reversible cause of the Claimant's symptoms. He said that disc prolapse was the most common cause of CES although he accepted that there are other causes such as lumbar stenosis. The CES pathway was intended to ensure that there was an examination within a consistent time frame to identify whether there was a surgically reversible cause requiring urgent intervention. Because the symptoms in the Claimant's case were acute, an MRI was needed but once it could be seen

that it was not "highly compressible" it was not necessary to refer for surgery. He said that even if the stenosis had been described as moderate he would still have been hesitant to refer for surgery. However if there were acute symptoms in addition to a complete absence of CSF then that might change his view and he agreed he would then probably have referred.

Dr Witham

56. Doctor Fiona Witham is a consultant clinical radiologist at the Portsmouth Hospitals University NHS Trust and has been in that post since November of 1996 (when she became a consultant) having completed her general medical training at Kings College London and Kings College Hospital Medical School between 1982 and 1988. She had provided a relatively short witness statement but was questioned at length. I formed the impression during her oral evidence that she was, as her age and work history suggested, a highly experienced radiologist. She gave calm and thoughtful answers. She was fully aware of the purpose of the scan and its significance for the Claimant.

57. Her primary task was to interpret and report on the images. She had no contact with the patient. The SHO would have brought the request form to her, or at least that was the usual practise. As she had written on the form she thought that was an indication that she had seen and discussed the case with the SHO.

58. In 2017 she would have covered acute musculoskeletal referrals for two or more days a week and would have seen at least one set of MRI images investigating CES each day; she estimated a minimum of four a week. Having written on the form either she or the SHO then took it to the radiographer. The patient was told to come for a scan at 13.39. The first sequence was run at 13.57. Some 90 minutes passed from the form being filled in to the beginning of the scans, which she regarded as expeditious. It was a difficult scan because of the movement of the patient due to pain. It involved taking a series of images as follows:

i) Series 1 at 13.57 as a localiser

ii) Series 2 at 13.58

iii) Series 3 at 14.01 (an unsuccessful sagittal T2 scan)

iv) Series 4 at 14.09

v) Series 5 at 14.10

vi) Series 6 at 14.12

vii) Series 7 at 14.16

viii) Series 8 at 14.21

ix) Series 9 at 14.31

x) Series 10 at 14.35

xi) Series 11 at 14.40

xii) Series 12 at 14.45

59. There was a greater than expected interval between series three and four. Some scans however take longer than others. The initial localiser scan was quicker, whilst the dedicated scans took longer. Dr Witham thought that the longer interval fitted with the Claimant's account that the radiographer tried to make her more comfortable. The analgesics were given between 12:00 and 12:30 and would therefore still be increasing in effect or close to their peak during these scans.

60. The radiographer spoke to her just before 14.30 when she asked them to repeat the sagittal T2 scan. She started her report at 14.30. She thought that the radiographer had called her again to ask if it was necessary to persist as the Claimant was in a lot of pain and there would have been diminishing returns. She considered it was not required because it was not necessarily the case that further scans would have been any clearer and the Claimant was in discomfort. The clinical scenario was not one in which it was possible to simply wait a few days and redo the scans. I note that the document *"Standards for the Reporting and Interpretation of Imaging Investigations"* (January 2006), published by the Royal College of Radiologists, states "*further investigation should be suggested only where necessary, particularly when it entails discomfort or radiation exposure for the patient."*

61. The odd number scans show more movement artefact than the even numbers but all of the scans from series 7 onwards made some contribution to her interpretation. It was possible to accelerate the imaging to get a better image but she did not think there was any acceleration used, which would have been normal. A high BMI, as in the Claimant's case, does have some effect because the tissue is further away. The scans were however optimised and she considered that any problems with their quality had more to do with movement.

62. She explained that there were two red flags for CES on the referral for MRI; the reduced perianal sensation and difficulty passing urine. She was looking for a cause of acute CES. The most common cause was a large lumbar prolapse although there were, she accepted, other causes. She disagreed that longstanding canal stenosis might cause the onset of acute CES; something else would be needed and even then it depended on the degree of canal stenosis and the impact of the acute pathology. It was suggested to her that there were three potential radiological presentations; first a lumbar disc prolapse, (which was not the position here), secondly chronic stenosis with the addition of a further space occupying pathology and thirdly chronic pathology plus the acute onset of symptoms, even if a cause could not be seen on the scan, since this would suggest neural decompensation. Her response was that the scans

do not show critical or severe stenosis. There is epidural fat which would be forced out if there was severe stenosis because there would be no space for it.

63. She was asked if she was able to exclude CE compression by identifying a capacious dura, no loss of CSF and nerves floating free. She said that she was looking for an acute cause of cauda equina compression. While she accepted that someone with stenosis might be more at risk she said that she did not see an acute cauda equina compression. CES itself is a clinical syndrome (and so requires a clinical diagnosis). She said that some 90% of patients do not turn out to have a remediable cause even where there is a red flag for CES.

64. She said that the phrase *"canal slightly tight"* used in her report was a reference to the fact that this was not a capacious canal; that is to say that some people have bigger canals so she was referring to the harder structures rather than stenosis which refers to the bony and ligamentous boundary of the spinal canal.

65. It was suggested to her that her wording in this respect, in particular the use of "*slightly*", was unclear. She said that the dimensions were still one centimetre square for the thecal sac and reiterated that she was looking for acute cauda equina compression which she had not seen.

66. She said that the phrase *"mild central canal stenosis"* was indirectly telling the reader about the nerve roots. She agreed that the message of the report was that the stenosis and fat were unlikely to be contributing to the clinical symptoms. As far as the terms "*mild*" and "*moderate*" were concerned she said this was a qualitative assessment where there were no sharp boundaries. The quantity of fat ("*lipomatosis*") also influenced the description as, although not bad, it was still present.

67. She was asked about a number of academic papers that had attempted to establish a grading system for stenosis and whether her use of the term "*mild*" conformed to these. She emphasised that the purpose of her report was to establish whether there was an acute cause and that looking at the cross-sectional area of the thecal sac, given the artefact, was a reliable approach since the thecal sac remained at 82% of the cross-sectional area on the relevant slides. She made the point that the images she viewed when reporting were seen by her at full resolution on a workstation not after they had been converted to a JPEG (as they were for the purpose of trial).

68. She said that describing the epidural fat as "encircling", as it was in evidence was not a bad description. She would not have described it as profuse and had never come across an operation to remove epidural fat. It was suggested to her that there was a cyst at L2/L3 which was not referred to in her report. She accepted that there was a darker area on images 8 and 9 at this level but she thought this was a hypertrophic facet joint. She accepted that others, with the benefit of subsequent scans, had identified this area as a facet joint cyst but made the point that she did not have the later images and was not sure in any event if she agreed with that interpretation of the dark area. She thought there was a mixture of evidence, which

was not uniform, that the thecal sac had been impinged by a cyst at L2/L3. It did not alter her opinion as to whether a surgically remediable cause of CES was present. She again said that the thecal sac was 82% of the cross section including the impingement. That was not sufficient to explain the presentation so she remained equivocal about the dark area and would have to say that she was unsure about its presence or significance.

69. She was asked whether she agreed that the scans showed no CSF at the L3/L4 level. She thought that although the quality was reduced she could see bright fluid between the nerve roots. She agreed that contrasting images 14 and 13 with each other showed a reduction in CSF but it was still there. She repeated that the cross-sectional area assessment was OK; the signal return would be affected by lots of factors so that she would say there was less CSF not no CSF.

70. She was asked about other possible pathologies. She did not think there was a possible disc bulge at L3/L4 and thought that what could be seen was the longitudinal ligament which was not indented by a disc or at least was not significantly indented. She was asked to look at image 14 and axial T2 and it was suggested to her that there was a disc extrusion which had bisected the epidural fat. She disagreed. She agreed that CE compression at two adjacent levels was worth reporting, as she had done, because that was important information for a surgeon. She disagreed that the conclusion to her report was to be read as saying that there was no crowding of nerve roots. She was asked to look at the diagrams in the literature bundle at page 28. It was suggested to her that her scans were close to grade C. She disagreed and thought they were more likely to be between B&C and pointed out that the literature was accompanied by much clearer scans.

71. She disagreed that the use of the term "*mild*" was misleading to the surgeons and pointed out that fat is a much softer constricting force than, for example, a disc bulge. It was suggested to her that there was nothing in her report to explain what the extent of the compression was. She said it was normal for there to be some fluidity in the descriptions used.

72. It was suggested that if there was any uncertainty the Claimant should have re-imaged after analgesia. She said that she did not think that would have been possible given the patient's pain and the hospital protocol which did not allow for sedation. There was no guarantee that better images would be obtained. It was suggested that she could have sought a second opinion from the radiological department at Southampton but she said this was not a pathway and she could not get an opinion from that hospital when she was dealing with a clinical referral from within her own hospital. She said this was an emergency and an urgent pathway. She could not see anything that needed emergency surgery and did not consider there was any lesion that could be remedied by surgery.

The Scientific Literature

73. The Point made by Dr Witham and Dr Luckos in their evidence as to the low correlation between the number of those referred for an MRI scan on a CES pathway and the identification of acute symptoms requiring urgent surgical intervention was born out by the scientific literature:

i) *Scan-Negative Cauda Equina Syndrome A Prospective Cohort* Hoeritzauer, Neurology 2021:

> *"Cauda equina syndrome (CES) is a surgical emergency caused by compression of the cauda equina nerve roots. It is suspected when patients present with bladder, bowel, or sexual dysfunction or saddle numbness with or without new back or leg pain. An MRI scan is required to demonstrate cauda equina compression and it is recommended that this occurs within 1–4 hours of presentation to hospital, creating significant pressure on emergency care, neurosurgical, orthopedic, and radiology staff to provide a responsive 24-hour service.1,2 However, a mean of 81% of patients referred to neurosurgery with CES have normal or nonexplanatory imaging—"scan-negative" CES3 —despite having similar rates of pain and bladder and neurologic dysfunction."*

ii) *Shades of Grey – The challenge of 'grumbling' cauda equina symptoms in older adults with lumbar spinal stenosis* ("LSS") - Musculoskeletal Science and Practice (2020):

> *"When a patient presents with clinical symptoms that a clinician feels warrants investigation, MRI is considered the gold standard for confirming a diagnosis of both LSS and of CES. There are, however, notable challenges with MRI interpretation, particularly in relation to LSS. There is no standardised and reliable system for interpreting and reporting LSS on MRI, and a vast array of classification systems is in current use (Schroeder et al., 2016). Whilst MRI sensitivity may be as high as 96% for diagnosing LSS, specificity might be as low as 68% (Wassenaar et al., 2012; Lurie and Tomkins-Lane, 2016), and it is (C. Comer, et al. Musculoskeletal Science and Practice 45 (2020) 102049 2 0016) widely accepted that symptoms and disability correlate poorly with the severity of degenerative narrowing seen on MR imaging (Kalichman et al., 2009; Genevay and Atlas, 2010). Indeed, MRI findings of stenosis are commonly found in people with no symptoms (Lurie and TomkinsLane, 2016). Very few imaging studies have focused on CES symptoms in LSS (Deen et al., 1994; Kawaguchi et al., 2001), and these suggest only a weak correlation between severity of stenosis on MRI and bladder dysfunction (Tsai et al., 2010). There is currently no consensus on the degree of spinal canal restriction that would be expected to cause bladder dysfunction."*

74. The classification systems mentioned in the latter of these two papers include categories identified as "mild" and "moderate" which do not easily read across to other classification frameworks, bearing out the observation that there is no standardised or reliable system (see *Qualitative Grading of Severity of Lumbar Spinal Stenosis Based on the Morphology of the Dural Sac on Magnetic Resonance Images.* Schizas et al 2010 and *A New Grading System of Lumbar Central Canal Stenosis 33 – 39 on MRI: An Easy and Reliable Method*. Guen et al 2011).

75. Ms Guthrie asked questions in cross examination by reference to the *Schizas* paper (above) which suggested a grading system going from grade A to grade D, where grade A was defined as no or minor stenosis, B as moderate stenosis, C as severe stenosis, and D as extremes stenosis. Whilst a similar grading system was suggested in *Guen* (above) the attributes of a scan which could be interpreted as minor, mild or moderate differed; the differences reflecting the extent to which the exercise involved a quantitative exercise or focussed on the impact of stenosis on the thecal sac and spinal cord/cauda equina. For that reason I did not find the reference to a grading system (one of a number) of particular assistance; the central question was whether there was acute compression of the cauda equina. Had there been a fixed standard applied and understood by other clinicians then questions of nomenclature and grading might have assumed more importance, but that was not the case here.

76. The *Hoeritzauer* paper (above) suggests that in a substantial group of patients with a complex medical background, of the sort shared by the Claimant, there may be other pathologies which explain what appears to be a CES presentation but in which there is a negative scan:

> *"Our study highlights that patients with scan-negative CES are group with high rates of chronic pain, psychiatric comorbidity, bladder dysfunction, and impaired social functioning. We propose several clinical implications:*
>
> *1. Urgent neuroimaging is required in all CES presentations. Although we have demonstrated some clinical features that may help differentiate scan-negative from scan-positive CES at presentation, an urgent MRI scan continues to be essential, as none of them allows clinical separation with sufficient confidence.*
>
> *2. Some clinical features should no longer be considered to have any specificity for a structural cause for CES including anal tone, saddle numbness, and urinary retention. There is an argument for abandoning examination of anal tone unless otherwise indicated.*
>
> *3. Providing positive diagnosis and treatment pathways for scan-negative CES: At present, patients with CES are rushed into hospital, but then when the scan is normal, generally given no explanation for their symptoms. Clinical features we have found including preexisting bladder dysfunction, particularly stress incontinence, chronic*

widespread or back pain, panic, and dissociation at the onset of CES symptoms and positive signs of FNDs should raise expectations of a negative scan. More explicit discussion, both before and after imaging, about the possible mechanisms of CES symptoms (with consideration for other neurologic disease causes) can give patients and health professionals an explanatory model compatible with rehabilitation treatment. Ingredients may include management of constipation, reduction of opiates, use of flip flow catheters with early trial of removal of catheter, physiotherapy directed towards chronic pain or FND issues, and follow-up within a multidisciplinary team including psychological input where appropriate."

The Legal Framework

77. There was no dispute that the standard of care for radiology reporting is to be determined in accordance with the general, *Bolam/Bolitho*, clinical negligence principles albeit that Ms Guthrie submitted that the factual distinction between a case involving a treatment choice and the reporting of an MRI allowed for a more flexible approach to the *Bolitho* test (see *Bolam v Friern Hospital Management Committee* [1957] 1 WLR 583, *Maynard v West Midland Regional Health Authority* [1984] 1WLR 634 and *Bolitho v City and Hackney Health Authority* [1998] AC 232).

78. There are two stages. The interpretation of image content is a factual matter whilst the subsequent determination of whether a report is reasonable (non-negligent) requires a court to assess whether the radiologist's actions were consistent with the expected standard of care in interpreting and communicating imaging (*see Penny v East Kent Health Authority* [2000] Lloyd's Rep Med 41 and *Brady v Southend University Hospital NHS Foundation Trust* [2020] EWHC 158 (QB)).

79. In *Penny* the claims related to four cervical smears, taken as part of the national cervical screening programme, each of which was reported by the screeners as being negative. Having cited the judgments in *Bolam* and *Bolitho*, Lord Woolf MR, delivering the Judgment of the Court, said at [26] to [28]:

> *"26. Both before the judge and before this court counsel were agreed that the approach indicated in the passages which have been cited should be applied to these cases. We agree. The screeners were exercising skill and judgment in determining what report they should make and in that respect the Bolam test was generally applicable. Later authorities make clear that this it is the appropriate standard to apply. However, as we will explain, the fact that two sets of competent experts genuinely hold differing opinions as to whether or not at the relevant date, which is the date of the examination, the screeners could without being negligent have diagnosed the smears as negative does not necessarily provide the solution to the dispute on liability in these cases.*

> *27. There is the qualification which Lord Browne-Wilkinson identified in the passage already cited from his opinion in Bolitho. In addition the Bolam test has no application where what the judge is required to do is to make findings of fact. This is so, even where those findings of fact are the subject of conflicting expert evidence. Thus in this case there were three questions which the judge had to answer:*
>
> > *What was to be seen in the slides?*
> >
> > *At the relevant time could a screener exercising reasonable care fail to see what was on the slide?*
> >
> > *Could a reasonably competent screener, aware of what a screener exercising reasonable care would observe on the slide, treat the slide as negative?*
>
> *28. Thus, logically the starting point for the experts' reasoning was what was on the slides. Except in relation to the slide known as Palmer 2, as to which there was a striking conflict, as a result of a meeting which took place between the experts they were in substantial but by no means total agreement. In so far as they were not in agreement, the judge had the unenviable task of deciding as a matter of fact which of the experts were correct as to what the slides showed. This was a task which required expert evidence. However the evidence having been given, the judge had to make his own finding on the balance of probabilities on this issue of fact in order to proceed to the next step in answering the question of negligence or no negligence. Having come to his own conclusion as to what the slides showed, the judge had, therefore, then to answer the 2nd and 3rd questions in order to decide whether the screener was in breach of duty in giving a negative report. Whether the screener was in breach of duty would depend on the training and the amount of knowledge a screener should have had in order to properly perform his or her task at that time and how easy it was to discern what the judge had found was on the slide. These issues involved both questions of fact and questions of opinion as to the standards of care which the screeners should have exercised..."*

80. In other words, having made findings of fact as to what was shown on the scan the question as to whether or not the scan was reported in a reasonable manner must be determined by reference to the *Bolam* test, subject to the *Bolitho* qualification. Indeed, in *Brady*, despite finding against the defendant and holding that the scan did in fact show an infection, the Judge still concluded that the radiologist reporting on the scan (who had not identified infection) had nonetheless reported in a reasonable manner so that the allegations of breach of duty were dismissed.

The Expert Evidence

81. The parties called the following experts to give evidence at trial:

i) Dr Spratt, the Claimant's expert radiologist

ii) Dr Rankine, the Defendant's expert radiologist

iii) Prof Greenough, the Claimant's expert spinal surgeon

iv) Mr Thorpe, the Defendant's expert spinal surgeon

82. In addition there were expert reports from urology experts, Mr Hetherington (Claimant) and Mr Reynard (Defendant) and colorectal experts Mr Gudgeon (Claimant) and Mr Hartley (Defendant). These experts did not give oral evidence and were not in a position to provide definitive opinions on whether the Claimant had CES on 6 June 2017. While they acknowledge that CES could have caused the Claimant's urinary and bowel symptoms the determination of whether CES was present turns on the evidence of the spinal and radiological experts.

The Radiologists

83. In their joint report the radiologists agreed that the scans reported by Dr Witham showed that:

i) at the L2-3 and L3-4 discal levels there are moderate central canal stenoses from facetoflaval hypertrophic degeneration with additional epidural lipomatosis posteriorly impinging on and constricting the thecal sac

ii) there is a left intracanalicular facet joint cyst at L2-3 which contributes to substantial cauda equina impingement

iii) there is a moderate degree of constitutional stenosis with additional epidural lipomatosis constricting the thecal sac.

iv) there is a small superior left paracentral disc extrusion at L3-4.

84. The presence of the facet joint cyst relied upon a retrospective analysis. In relation to whether it could be demonstrated on the 6 June scans, Dr Rankine expressed similar reservations to those voiced by Dr Witham.

85. Dr Rankine considered that the small disc protrusion at L2-3 was not compressing the cauda equina. Dr Spratt agreed with his assessment and confirmed in his evidence that it was not. Thus there was no breach of duty by omitting to mention the disc protrusion in the radiological report because, in effect, it was not of clinical significance.

86. Both experts agreed that although there was movement artefact there was *".. a responsible and reasonably competent body of radiologists who would have considered the scan to be of*

sufficient diagnostic quality such that it was appropriate to provide a definitive report and not to convey doubt as to the findings or to recommend a further scan".

87. Both experts agreed that measuring the size of the spinal canal is a subjective process lacking standardised, quantifiable methods. The commonly used descriptive terms for spinal stenosis (mild, moderate, severe) are not based on precise measurements of the canal's cross-sectional area.

88. Dr Rankine's view was that that the descriptors used in the report, of *mild* stenosis and *slight* constriction are those which would be used by a reasonably competent body of radiologists. Dr Spratt disagreed and considered that that the term "slight", used to describe the thecal sac constriction at L3-4 and L2-3 inaccurately minimised the degree of cauda equina compression. His view was that the report should have used terms such as "substantial," "marked," or "severe" to emphasize the critical condition of the cauda equina. This would have alerted the surgeon to the urgent need for decompression surgery given the patient's symptoms, consistent with cauda equina syndrome. This criticism of the language used in the report was accordingly based upon a prior conclusion as to the extent of compression of the cauda equina.

89. Dr Witham had identified "less" CSF at the L2-3 and L3-4 level. Dr Spratt thought that there was no CSF at these levels at all. There is certainly an absence of bright signal particularly at the L3-4 level but I preferred Dr Rankine's interpretation of the relevant scans as showing some CSF signal at the relevant point (Axial T2, Series 1, Image 13) and the presence of nerve roots (rather than artefact) in the adjoining axial image (Axial T2, Series 11, Image 14). Whilst the assessment was complicated by the poor quality of the scans, his explanation, that a spinal canal with the width shown could not have had all of the CSF "squeezed" out of it, struck me as a logical interpretation of what could be seen. I note that Dr Thomas's radiological report of 25 July 2017 (post-surgery on 12 June) observes:

> *"..., there is clear evidence of surgical intervention around the lower lumbar spine with posterior decompression of at least L4 and a little local epidural fibrosis on the left from L3 to the L4-5 disc. There is degenerate disc change and scattered small disc protrusions, most marked in the left paracentral location between L2 and L4. This does cause narrowing of the thecal sac between L2 and L4 with quite marked reduction in intrathecal CSF signal intensity."*

90. The nub of the disagreement between the expert radiologists was that Dr Rankine concluded that there no acute findings supportive of severe cauda equina compression, since all the findings on the MRI were, in his view long standing constitutional, degenerative changes whilst Dr Spratt's view at the time of the joint statement was that *"a radiologist should be able to diagnose all surgically correctable causes of cauda equina syndrome and highlight their nature and location to the surgeon. In this case surgical targets for decompression are moderate constitutional central canal stenoses and epidural lipomatosis at both L2-3 and L3-*

4 levels in addition to the L2-3 facet joint cyst and the L3-4 disc extrusion all pathologies contributing to substantial constriction of the thecal sac at both levels".

Dr Spratt

91. In his oral evidence Dr Spratt sought to demonstrate, by reference to the scans, that there was a significant narrowing of the thecal sac at the L2-L3 level which with the absence of CSF indicated, in his view, that there was compression of the cauda equina. The purpose of the scan was to identify an acute cause of CES which he termed "a surgical target". The pathology could be acute or chronic but the question was whether it could be reversed by surgery. There was no dispute that this was the correct general approach.

92. Dr Spratt initially identified a significant disc extrusion at L3-4 as the cause of severe cauda equina compression, criticising Dr Witham for failing to identify this. However, upon further review and discussion with Dr Rankine, Dr Spratt realised he had misidentified a facet joint cyst at L2/3 as a disc extrusion at L3/4. A subsequent report identified a new disc protrusion at L3/4, which was not mentioned in his original report. Ms Hughes submitted that this indicated that there were three critical errors in his initial assessment: 1) misidentifying the spinal level, 2) misidentifying a facet joint cyst as a disc protrusion, and 3) failing to identify the disc protrusion now claimed to be at L3/4.

93. Dr Spratt explained that he had to consider the images which were made available to him, which were jpegs supplied to him on a CD rather than medical grade images viewed on medical software. It might be observed that Dr Rankine was supplied with the same images and did not fall into similar error. Ms Hughes drew attention to the fact that in another case in which Dr Spratt had given evidence the judge had concluded that he had made a similar error in interpreting scans. She also criticised Dr Spratt's subsequent letters which sought to add to his report or in effect, in the Defendant's submission, to ameliorate deficiencies. This included reliance on the post-operative MRI scan from July 25, 2017, to bolster his interpretation of the pre-operative scan from June 6, 2017, which Ms Hughes argued was problematic in a number of ways:

    i) First, and despite having access to the July 2017 MRI, Dr Spratt inexplicably omitted any reference to it in his initial report and subsequent joint statement.

    ii) Secondly, the inherent limitations of using post-operative imaging to infer pre-operative conditions are well-established. Surgical interventions inevitably induce tissue alterations, rendering accurate comparisons between pre- and post-operative scans challenging. Professor Greenough himself acknowledged the interpretive difficulties posed by post-operative changes in the July 2017 images.

    iii) Thirdly, Dr Spratt's misdiagnosis of a normal anatomical structure as a residual disc protrusion on the July 2017 MRI underscores the unreliability of drawing definitive conclusions from post-operative imaging. This error, it was argued, further

undermines the validity of his attempt to correlate findings from the later scan with the earlier one.

94. Dr Spratt initially described the stenosis at L2/3 and L3/4 as "severe". However, he then agreed with Dr Rankine's assessment that the degree of stenosis at both levels can properly described as "moderate".

95. During his oral evidence he conceded that it was reasonable not to identify the pathology at L2-L3 as a facet joint cyst or report it as such. He also confirmed that he had changed his mind regarding the need to report a disc prolapse at L3-L4 where he suggested that the main issue was related to fat and stenosis. He did not, any longer, support the contention that there was a need for further scans or discussions with the local spinal surgical centre.

96. Dr Spratt agreed that CES was a clinical diagnosis and more often than not one would not find a cause on a scan; in fact in his written report he had pointed out that over 95% of those referred for an urgent scan for CES at a busy district general hospital will show no causative pathology.

Dr Rankine

97. Dr Rankine's analysis of the MRI images obtained on June 6, 2017, lead him to the conclusion that there was no evidence of acute cauda equina compression. In contrast with Dr Spratt's findings, he considered that the purported disc extrusion at L3/4 was in fact a misidentification of the thecal sac. The substantial cyst at L2-3, while present, was simply part of the overall moderate spinal stenosis and would not independently exacerbate cauda equina compression. This conclusion was, in his view, reinforced by the presence of CSF at both L2-3 and L3-4, indicative of an unobstructed spinal canal.

98. In short, his opinion was that the stenosis and the facet joint cyst were long standing, constitutional degenerative changes and did not provide an acute, surgically correctable cause of cauda equina compression.

99. In his oral evidence he explained that the nerves floated within the thecal sac surrounded by CSF, with ample space, so that a constriction of the thecal sac did not affect them until it was sufficient to clump them all together and then exert pressure on them as a mass. He did not think that there was any point at which nerves surrounded by CSF even where the thecal sac had been constricted were under compression. This was why even where there was extensive stenosis there could be no effect on nerve function. He demonstrated this by reference to the scans, which he said were not unusual and like scans of the sort that he came across routinely in the course of his practise without there being a diagnosis of CES.

100. Ms Guthrie criticised this approach as being assertive rather than reasoned and based upon his own idiosyncratic grading system. For my part I found Dr Rankine's evidence helpful and considered that he was entitled to give an opinion by reference to his own clinical

experience as to whether the appearance of the scan was unusual or commonly encountered and, if so, how he would classify the scan in the context of a process that was agreed to be subjective.

101. Dr Rankine is a very experienced radiologist so I found his explanation of the passage in the joint statement where he had referred to his opinion as relating to the entirety of the presentation as falling within the term "*moderate stenosis*" to be puzzling. The language of the joint statement does not suggest this. Ms Guthrie made the point that if the facet joint cyst and epidural fat are to be regarded as additional constrictive features then they would represent further impingement on an already moderately stenosed spine justifying, in her submission, the label of *severe* (or *moderately severe).* But the central issue is whether there was a compression of the cauda equina, which was a surgical target, that was causing symptoms.. It does not seem to me that that question ultimately depends upon whether moderate stenosis is used in the joint report simply to refer to the bony and ligamentous dimensions of the spinal canal or has a wider meaning. Dr Witham's report does seem to be to be clear in identifying lipomatosis (fat) and disc degeneration as additional features *contributing* to existing L2-3 and L3-4 mild central spine stenosis. With the exception of disagreement as to whether the term "mild" was apt that does not appear to be incorrect even on the basis of the Claimant's case.

The Spinal Surgeons

102. Both experts concurred that moderate spinal stenosis at the L2/3 and L3/4 levels would typically warrant conservative outpatient management rather than immediate further investigations. The consequence of this was, as the Defendant submitted, that even if the initial radiological report had been in error in characterising the stenosis as mild instead of moderate, a causal link between this alleged negligence and the Claimant's subsequent condition could not be established. The absence or otherwise of acute spinal cord compression at these vertebral levels was the pivotal issue.

Prof Greenough

103. Professor Greenough thought that the radiological report was quite reassuring because it did not mention severe compression to the extent of the exclusion of CSF. This view therefore depended upon a factual conclusion as to what the scan in fact showed. As to that, his view was that there was significant stenosis but the scan was of too poor quality to give any detail about the extent of the narrowing. He deferred to the radiologists to give a detailed interpretation of the images.

104. His opinion and thought process was essentially that because, in his view, no CSF could be seen on one of the slides that indicated compression; CES could not be excluded and therefore there was an urgent need to operate. He also thought that the scan was not of diagnostic quality but had done the best he could to indicate what he thought the scan

showed. He considered the scan should have been redone in order to obtain a better quality image. However if it could not be, what was shown on the poor quality scan was sufficient to mandate surgical intervention. Whilst he could not see evidence of a disc prolapse he did think that compression was shown and would have mandated surgery if another scan had not been available.

105. His view was that the past history including surgery meant that the nerves were likely to have lost some of their reserve and hence their ability to recover from any impingement or compression. He made the point that CES was a clinical diagnosis which did not necessarily depend on a scan. He accepted that a possible explanation for the symptoms in 2017 was that they had been triggered by a fall but said that that could not be relied upon if there was a scan showing no CSF and the other pathology which he regarded as significant. He considered that the claimant had CES "in evolution". He did however accept that the previous history of surgery may have rendered the Claimant more vulnerable to injury of the cauda equina in future. Further he accepted that there is a cohort of patients, with whom the claimant shared a number of risk factors, who are more likely to have scan negative back pain and bladder problems which could not be accounted for on imaging.

106. Ms Hughes submitted that there was an inevitable tension between the contention that the scan was not of diagnostic quality for surgery but was nevertheless diagnostic of acute cauda equina compression. In cross examination Professor Greeough accepted that there was essentially only one image which he considered demonstrated no CSF at L3-4.

107. It seems unlikely on the basis of his evidence that he would have taken the same course as Mr Davies who identified acute pathology which went beyond what the scan could be relied upon as demonstrating, given Professor Greenough's view that the scan was not optimal for surgery and that a further scan should have been obtained.

Mr Thorpe

108. He accepted that it was possible that CES could be caused by existing stenosis with a small change in pathology. He said it was possible to present with CES as a result of stenosis but it was rare. He accepted that the first question that needed to be answered was whether there were clinical symptoms. The request for a scan was to determine whether cauda equina compression was shown. After that one needed to know what the acute and chronic pathology was although that was an ideal situation and it was not always straightforward in practice.

109. He was asked to look at the MRI scan of the 6th of June 2017. He thought it was of poor quality but series 7 to 11 could be interpreted. He drew such conclusions as he could but he emphasised that the scan was not in his view of diagnostic quality. He said there appeared to be constriction at L3 and L4. He could not say it was compression because it was very difficult to interpret. He disagreed with the suggestion that there was an absence of CSF on

the scan which indicated compression. He said it could be due to compression or scarring or artefact particularly, as here, if the scan was poor.

110. When asked whether he could say that the scan excluded CE compression he said that he would not want to rely on it one way or the other; one explanation would be compression so it could not be excluded. It was suggested to him that there was more constriction shown at L3-4 but he said it was not possible to quantify it. When asked about compression at two adjacent levels he said it was worrying at any level but it was worse if there was compression at 2 levels although there was not always a linear relationship between compression and the consequences; that is to say it was not twice as bad because there was compression at 2 levels.

111. He said that there was fibrosis of the thecal sac and the walls of the dura as a result of surgery. Because surgery had taken place at the L4-5 level he would anticipate it at that level but thought that it could also affect L3-4. That would be because the surgeon would take a wider approach and had to go past the thecal sac in order to get to the disc. He agreed that dural fibrosis is irreversible. He could not point to dural fibrosis in the notes but there is scarring around the disc so one can assume there is dural scarring. He noted that in the second operation the surgeon had taken off the complete L4 laminate so that this would have an effect on the L3-L4 level. He was asked whether fibrosis can cause clinical symptoms. He said this was controversial but, in his view, yes it could.

112. He was asked to comment on the fact that there had been 10 years of stability after the three operations. He said that was not inconsistent with the position in this case because not everyone has symptoms; that was the case even in relation to marked stenosis. He agreed that Dr Witham did not note fibrosis in her report. It was pointed out to him that in the post 2017 scans the canal was noted to be capacious. He pointed out that in 2017 the scan was still reporting constriction even after decompression so that it might take some time for the sack to expand. Fibrosis was likely to result in tethering of the sack to part of the surrounding anatomy but the rest of the sack remained elastic. He was asked what he would do if confronted with report including the reference to *mild*. He said he would not expect that to cause clinical symptoms but it was not necessarily reassuring because there was epidural fat present so he would have read it as indicating that there was mild or moderate stenosis and constriction but not compression.

113. He had agreed that there was an absence of CSF 'signal' but that was different from an absence of CSF. He thought that there was no compression. Because the areas which were said to be indicative of an absence of CSF were confined to a slice he thought that this was more likely to be artefact because one would have expected the scan to show a gradual loss of CSF in the levels adjacent.

114. He was asked about the bladder symptoms worsening after the June 2017 operation. He said if that was the case then he could not say that compression was the likely cause; it could equally be operative traction. He thought that the urological problems could be due to pain

and distress at the time of her presentation. He had drawn attention to the fact that one would expect improvement if there was an operation to decompress because it was logical that if the compression is relieved that would ameliorate the symptoms but he accepted that ongoing symptoms of a urological type and neuropathic pain could also be consistent with CES. He thought that it was possible that the fall was an index event. But he disagreed that there was no real alternative to compression as the explanation. He said that after her surgery the Claimant had gone through a difficult period which can be the cause of urological disorder as contrasted with the earlier period of operations when she had been well supported. He thought it was not possible to use urodynamics to diagnose what the position was. Her presentation had been very similar on previous occasions when she had almost identical symptoms but there was no CE compression or CES. He accepted that if the scan had shown compression it would have led to an operation and that if the scan on the 6th of June identified compression then another scan on that day would have shown it as well.

Conclusions

115. The radiological report was in support of a clinical diagnosis which led to the Claimant being discharged on the basis that the clinicians, Dr Aduki and Dr Luckos did not diagnose CES. The breach of duty alleged is not in relation to that diagnosis and the decision to send the Claimant home but is confined to the reporting of the MRI scan. It is said that Dr Witham failed to identify compression which required urgent surgery and that her report was reassuring when it should have been to the opposite effect. The central issue is therefore primarily within the discipline of radiology.

116. The scan on 6 June 2017 was subject to movement artefact. Dr Witham was plainly well aware of the significance of the scan and its urgency. She commenced her report before scanning had been completed and asked the radiographers to obtain better scans if possible. It was reasonable not to re-scan given her decision that it may not have provided any better quality scans. It was nevertheless diagnostic for a consultant radiologist (and agreed to be so by the expert radiologists); Dr Witham was entitled to treat it as such. The Claimant was on an urgent CES pathway and there was no reason in these circumstances to seek to repeat the scan.

117. The radiology experts jointly concluded that the MRI findings demonstrate moderate central canal stenosis at the L2-3 and L3-4 levels, primarily attributed to facet joint hypertrophy and epidural lipomatosis. There is a significant left-sided intracanalicular facet joint cyst at L2-3 which has a compressive effect on the cauda equina. Additionally, there is constitutional spinal stenosis with further thecal sac compromise due to epidural lipomatosis (fat build up) with a small disc extrusion noted at L3-4. This was inevitably a retrospective analysis but I conclude as a fact that these features were shown on the 6 June scan with the caveat that the facet joint cyst is not distinctly shown and there may be room for interpretative differences on the face of the 6 June scan alone.

118. Dr Spratt made a number of errors in his interpretation of the scan including identifying pathology at the wrong level. This did not inspire confidence and where there was a difference of opinion as to what was shown I preferred the evidence of Dr Rankine in reaching factual conclusions.

119. This was the case in relation to the presence or otherwise of CSF which I conclude was present at all levels notwithstanding that there is less CSF signal, and so less CSF to be seen on the images, at the L2-3 and L3-4 level. I note that in the post-operative MRI report of 25 July 2017, Dr Thomas observed *"There is degenerate disc change and scattered small disc protrusions, most marked in the left paracentral location between L2 and L4. Agreed. This does cause narrowing of the thecal sac between L2 and L4 with quite marked reduction in intrathecal CSF signal intensity."* Whilst Professor Greenough thought there was one image showing the absence of CSF and Mr Thorpe regarded that "slice" as explained by artefact I consider that the radiologists were best placed to give definitive evidence on the issue particularly when faced with a poor quality scan where individual images may have been differentially affected by patient movement or lordosis.

120. I also preferred, and accept, Dr Rankine's evidence that the imaging does not identify any point at which there is sufficient constriction for the spinal nerves to be clumped together and subject to compression. Having carefully considered the images I found that explanation to be compelling. It reflected his evidence as to the relevant anatomy and why stenosis and intrusive pathology is not of itself bound to cause interference with the spinal nerves.

121. Dr Witham was confident in her evidence that she accurately assessed the MRI scan and did not overlook any significant pathology indicative of cauda equina compression, which was the purpose of the MRI imaging. She emphasised her careful adherence to the Cauda Equina Pathway guidelines and her long and frequent experience in reviewing scans to identify whether there was CE compression. She confirmed the presence of CSF at the relevant spinal levels according with Dr Rankine's view, and contradicting any suggestion of artefact.

122. I do not consider that I can rely on the onset of the Claimant's symptoms or the position between the 6 and 12 June as supporting the presence of compression. Although I accept that the Claimant was seeking to be candid in her evidence to me I approach her account with caution since the medical notes reflect an untruthful history and, after she had been sent home, she was on strong painkillers.

123. The Claimant had previously experienced symptoms after a fall, of the sort she presented with in June 2017, without CES being demonstrated. Her medical history is complicated and her urological and pain symptoms have never been shown to be definitively attributable to CES. A similar sequence of events unfolded in early 2018 involving back pain, extending downwards, as well urinary difficulties; again with nothing to explain her symptoms or to suggest CES. It is plausible, as Ms Hughes submitted that her fall had triggered the same condition for which she had been treated before without patent signs of CES.

124. Mr Davies appears not to have taken any history from the Claimant and his letter and operation notes are inconsistent in an important detail in relation to episodes of incontinence. The reference to "bilateral pain" as marking a progressive symptom seems to me to be questionable if it depended on the Claimant's account given that she was obliged to say that his reference to symptoms evolving over many weeks was incorrect. Mr Davies only operated on L3-L4 so that no decompression was carried out at the L2-L3 level in circumstances where what he considered the scan to show and to be diagnostic of is at best unclear.

125. Therefore in answer to the central question posed by Ms Guthrie in her opening submissions I conclude there is no radiological evidence of cauda equina compression. Since it was not there to be found the fact that it was not identified represents an accurate assessment of the scan and not a breach of duty.

126. The question of whether the scan was otherwise reasonably reported is unlikely in those circumstances to have any causative significance. Even if the case were to be advanced on the basis that, irrespective of lack evidence from the scan, there was nevertheless underlying cauda equina compression and that the reassuring nature of the report contributed to a misdiagnosis or underestimation of other symptomology by the clinicians I do not conclude that the scan was not reasonably reported.

127. For the reasons discussed earlier there is no fixed standard so that, inevitably, there is a degree of subjectivity in reporting scans. The difference between the use of the term *mild* and *moderate* does not appear to me to convey a significant difference in assessment so as to be outside of what it was reasonably open to the radiologist to report in this case; nor does the description of the canal at the L2-3 and L3-4 level as *slightly tight* underplay what is shown on the scan given the explanation from Dr Witham and the immediate reference in the report to less CSF being seen and the role of epidural fat in the constriction being significant.

128. For these reasons the Claimant has not established that there was any breach of duty on the part of Dr Witham and I dismiss the claim.

129. I have made a number of references to the Claimant being untruthful, as she herself accepted, and I have doubted her reliability in some respects. I should nevertheless make it clear that she was an engaging and pleasant witness who was seeking to tell the truth in court. The outcome will not be what she hoped for but I wish her well in future.

130. I am grateful to both counsel for the quality of their written and oral submissions.

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