Neutral Citation Number: [2026] EWHC 1574 (KB)

1IN THE HIGH COURT OF JUSTICE
KING'S BENCH DIVISION

Claim No. QB-2022-NCL-000023

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 29/06/2026

Before :

CHARLES BAGOT KC, sitting as a Deputy High Court Judge

- -

Between :

LIAM DICKINSON

Claimant

-and-

NEWCASTLE UPON TYNE HOSPITALS NHS FOUNDATION TRUST

Defendant

- -

Henry Pitchers KC instructed by **Irwin Mitchell LLP** for **the Claimant**

Nadia Whittaker instructed by **Weightmans LLP** for **the Defendant**

Hearing dates: 2-6, 9 & 13 February 2026 and further documents received by email and email exchanges thereafter.

Draft circulated: 24 June 2026

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Approved Judgment

Note: The trial bundles ran to 5,108 pages. They were contained in a core bundle (consisting of 4 volumes within the same pdf), the references to pages of which in this judgment follow the format (p. page number), plus a bundle of DWP and Local Authority Records (volumes 5 and 6), together with a complete bundle of medical records (volumes 7 to 12) and an authorities bundle. Numbers alone in square brackets [5] are to the paragraphs of the report or authority being discussed. In addition the parties provided written opening and closing submissions, a Claimant's chronology of records, copies of medical literature, experts' CVs, a further letter from Dr Achinivu and various other documents which were handed up and subsequently emailed to the Court. The Court also subsequently received searchable bundles after the trial concluded and there were subsequent email exchanges with the parties.

CHARLES BAGOT KC, Deputy High Court Judge:

This judgment is in 9 parts as follows:

I. INTRODUCTION

1. The Claimant in this Clinical Negligence claim is now a 47 year old man who has developed Functional Neurological Disorder ("FND") and Functional Cognitive Disorder ("FCD")[1] and a depressive disorder. There is no dispute that these give rise to profound physical disability and also functional cognitive and memory disorder. With minor differences, the consensus is that the prognosis is bleak for any meaningful improvement.

2. The issue in this case is how the Claimant came to have those conditions and whether they are related to the Defendant's admitted breach of duty. It is accepted by the Defendant that when the Claimant was admitted to its Freeman Hospital for treatment of a facial lesion, in August 2016, the Defendant failed to administer a course of prophylactic Thiamine (Vitamin B1) in the form of Pabrinex, given he was a known alcoholic being treated with Chlordiazepoxide for symptoms of withdrawal from alcohol.

3. The Claimant's case is that the Defendant's breach caused him to develop Thiamine deficiency which caused a Wernicke's Encephalopathy ("WE"), and then a disabling functional disorder affecting him physically and cognitively, and giving rise to major reactive depression.

4. The Defendant does not materially dispute the Claimant's current condition but denies causation. Its case is that the symptoms arose coincidentally or more likely were pre-existing, but worsened markedly, albeit unrelated to the failure to administer Thiamine.

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[1] The Claimant's Neuropsychiatrist, Dr Achinivu, prefers the term Functional Neurological Symptoms Disorder ("FNSD") as an umbrella term which encompasses FND and FCD, but differences around labelling were not thought significant by the experts. I will use the umbrella term FNSD for ease.

As part of that denial, the Defendant also disputes the notion that the Claimant ever developed WE.

5. As well as the issue of causation, there are also some quantum issues which fall to be determined. The Claimant's claim on quantum as pleaded is for past and future financial losses of £5,820,292.71, were the claim to be resolved on a lump sum basis, plus general damages for pain, suffering and loss of amenity ("PSLA"). The parties have commendably been able to narrow the issues on quantum somewhat and agree a number of heads of loss, subject to liability, as set out below.

6. Although it is not realistic or necessary to mention or rule upon every aspect of the extensive evidence or every point of the many raised in oral and written submissions, in this judgment, I have taken them into account when reaching my conclusions.

7. The Claimant is represented by Mr Henry Pitchers KC and the Defendant by Ms Nadia Whittaker. I am grateful to them both for their obvious hard work in preparing this case for trial and for the skill and care with which they presented their respective cases. I am also grateful to their respective wider legal teams for their considerable help, particularly with the logistics. For reasons of judicial availability locally, the case was transferred at short notice from Newcastle District Registry where it had been due to be heard, to the Royal Courts of Justice, in London.

II. THE BACKGROUND

8. The background and chronology are drawn from the medical records. The Claimant provided a helpful chronology which was largely agreed, although the Defendant referred to some entries which were not contained in that document. As it is (quite properly) lengthy, I have not formally annexed it to this judgment. But I have drawn upon it in giving an overview and reaching my conclusions although I make clear that I

have taken all the entries I was referred to into account. For the following summary I have drawn upon both the records and the chronology, as well as the narrative summaries in the skeletons, suitably adapted and amended by me.

9. The Claimant's date of birth is 19 June 1979, hence he was aged 37 years at the relevant time and by the time of trial was aged 46 years. Prior to the events in question, his medical history was characterised by long-standing poor mental health, chronic alcoholism, spinal surgery in 2006, surgery to the right knee in 2011 and some left leg symptoms, the aetiology of which was not definitively established at the time.

10. I will review later in this judgment some parts of the previous medical history which are relevant to causation and the Defendant's case that the FNSD long pre-existed the relevant events. Focussing for now on the lead up to the breach of duty, the first pertinent entry is the Claimant's attendance at his GP surgery on 1 June 2016, with his then partner and now wife, Mrs Michelle Dickinson (to whom I will refer as "Mrs Dickinson"[2]). The main focus was the Claimant's ongoing substantial alcohol consumption: 10-20 cans [of lager, at c.2 units per can, so 20-30 units] at the time [A2/37].

11. It is appropriate to summarise some of the relevant entries about alcohol consumption as there was some difference between the parties about the level of consumption at the relevant time:

    a. 1 June 2016 (as summarised in the preceding para.): GP: 10-20 cans per day.

    b. 17 June 2016: alcohol key worker: 8-20 cans daily; shakes and sweats; been drinking like this "*for the past year*" [1653].

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[2] The Claimant's mother also provided a witness statement in this matter and for clarity, I will refer to her as Mrs Valerie Dickinson.

c. 18 August 2016: alcohol support worker: had drunk for many years; from 7 cans to 20 cans daily; reduced to 7 "*shandys*" and 3 cans a day after assessment (presumably after 17 June 2016); but since 18 July (when as will be seen, the Claimant's wife's evidence was that she left home for regular periods to care for her father who passed away a few weeks previously) "*increased back to 20 cans daily*". This would seem to be the last contemporaneous record of his drinking prior to the day of his admission to hospital on 26 August.

d. 26 August 2016:

   i. GP: 9-10 cans per day and awaiting help gradually to reduce [1367].

   ii. Hospital: 10 cans per day [1381-2] and [1417].

e. 30 August 2016: alcohol liaison nurse: 10-year history of daily alcohol consumption at heaviest 20 cans lager daily (40 units), but with work over previous 3-4 months, reduction to 10 cans (20 units) [1387-8].

12. Other potential indications around the relevant time of the effects of excess alcohol consumption include:

a. 2 August 2016: GP: vomited blood last week; referred for gastroscopy [A2/36].

b. 30 August 2016: endoscopy: gastric ulcer/erosions at the gastric antrum; generalised gastritis [1505]. Oesophagitis was diagnosed and advice given to start proton pump inhibitors (PPI) [1388].

c. 5 September 2016: histopathology: report on gastric ulcer biopsy showed appearances in keeping with reactive gastritis [1509].

13. There was a GP attendance with a left sided facial infection on 23 August 2016 [1367] and an attendance at the Emergency Department ("ED") without being seen that day

[1368]. There was a further GP attendance on 26 August [1367], when a diagnosis of cellulitis of the face was made. On the same day, the Claimant was admitted to the Defendant's Freeman Hospital [1381-2].

14. Following that admission, it was appropriately noted that the Claimant was alcohol dependent, would need CIWA scoring (to assess alcohol withdrawal – see [1436-40]) and chlordiazepoxide (a benzodiazepine drug used to manage acute alcohol withdrawal) [1383]. A cannula was inserted but apparently fell out [1443]. The Claimant was correctly prescribed Pabrinex, which was mandated under NICE Guidelines to mitigate the risk of Thiamine deficiency and WE. But the consensus between the experts, based on the records, is that even the first dose was not properly administered (discussed further below) [1452]: Neurologists' joint statement *"a complete vial of Pabrinex was not given"* [282].

15. In any event, the Defendant agrees no further doses of Pabrinex were administered during the admission and has admitted that this was a breach of duty.

16. There is nothing of particular significance to note in the first part of the Claimant's time as an inpatient. There was discussion about the Claimant being unable to "vape" on the ward or to consume alcohol [1426]. He was documented to be awake and orientated. There is no suggestion in the records of unsteadiness or confusion at this point. He was mobile and off the ward at times. He discussed HIV testing on 27 August [1384].

17. At 1700 on 28 August, a facial abscess was identified by ultrasound [1409] and the plan was for the Claimant to go to theatre [1385]. He underwent drainage of the left facial collection that evening [1386] and [1397]. The next day, he continued with antibiotics and a drain was in situ [1386].

18. On 29 August, the records document the Claimant expressing some concern about how he would cope after discharge without chlordiazepoxide and there was a plan to contact the alcohol liaison nurse [1427].

19. On the ward round of 30 August, the Claimant felt groggy with pain around the wound. On examination there was no sign of facial weakness, and he was noted to be alert, orientated and eating breakfast [1387]. The plan was for a discussion with microbiology regarding antibiotics and with the alcohol liaison nurse, with discharge home "*later if no issues*".

20. The alcohol liaison nurse made a detailed record of her discussion with the Claimant at 1000 on 30 August 2016 [1387-8]. She noted his history of alcohol consumption (already summarised above). The specialist nurse recorded "*no evidence of acute alcohol withdrawal symptoms – some anxiety which he agrees is a chronic condition he manages by "walking his dog" and leaving ward for "fresh air" for an hour or so at a time*." The specialist nurse was of the view that his detoxification was nearing completion, and it was not appropriate to discharge on chlordiazepoxide for anxiety. She encouraged engagement with his key worker to sustain his abstinence on discharge. The plan was for discharge when medically fit and CIWA score of less than 8 for 24 hours. In this regard, she advised, "*please take into consideration chronic anxiety and agitation associated with his anxiety*".

21. It was noted by nursing staff that the facial drain was removed around 1100 on 30 August, who also recorded in relation to CIWA that "*Liam gets agitated which he is scoring on but that is his personality & not his withdrawal*". There was a plan for follow up in 3 or 4 weeks after discharge [1428].

22. On 31 August, it was noted that the Claimant had been for a wander in the hospital grounds [1428]. He was seen on the ward round at 0810. No cellulitis or new collection

was found. There were complaints of constipation, but no abdominal pain, nausea or vomiting. The gastroscopy results were explained and the plan was for follow up in 3 to 4 weeks. There is no reference to confusion or unsteadiness in this entry [1389]. At 0910, the negative HIV result was communicated to him [1389].

23. At 1240, the following nursing entry was made:

> "*R/v on ward round. Patient fit for discharge…patient went off ward for 2* [hours?] *this morning & on return c/o felt drunk when outside & had to sit down. D/w alcohol liaison nurse who stated that this should not hinder d/c plans. Pt & his partner aware to contact support worker to arrange community follow up. Discharged from ward.*" [1428-9]

24. The Claimant was discharged on 31 August at approximately lunchtime and this generated various discharge documents summarising his inpatient stay and advice [1369] and [1511-12].

25. The Claimant's wife's evidence will be discussed in the following section, but her statements and oral evidence dealt with her recollections about the events between that discharge and the readmission to the Defendant's Cramlington Hospital on the evening of 1 September.

26. As discussed further below, Mrs (Michelle) Dickinson described the Claimant's confusion on returning home, becoming upset before going to bed. She described researching the Claimant's symptoms, sitting up all night (of 31 August to 1 September 2016). She says she tried to call the "*alcohol liaison officer*" but they were not available. She came across a reference to Thiamine deficiency, and the condition Wernicke's encephalopathy. There is an account of an untimed telephone call to the ward in which she discovered that the Claimant had not been given his Thiamine medication. There is also an untimed call to the GP.

27. It is not possible to determine the precise timing and sequence of the early clinical contacts on 1 September 2016. Subject to that caveat, there were the following:

    a. A timed record was made in the Progress Notes, by the alcohol key worker at 1133 [1655], who had spoken to Mrs Dickinson and noted matters including:

        i. Mrs Dickinson had been "*by his side since discharge*" and confirmed that he had remained abstinent, with there being no alcohol in the house.

        ii. She reported "*increasing concern*" about the Claimant and "*sounded very distressed and concerned on the phone*".

        iii. She described the Claimant's condition as "*like he is 'brain damaged'*".

        iv. The details she provided were that he could not walk in a straight line, kept falling over and was impulsive.

        v. The record confirms that by this point she had informed the GP and spoken to the ward, who apparently advised her to speak to the key worker. The key worker felt this matter was not for them as they were not medically qualified, so recommended telephoning the GP or taking the Claimant to hospital.

        vi. There is no reference to WE, Thiamine or to Pabrinex, although this differs from Mrs Dickinson's recollection in her witness statement.

    b. The Claimant was taken for wound care following surgery, but the appointment is not timed, although it would seem to come before the GP's assessment [1367].

i. The nurse also confirmed that the Claimant had not had any alcohol for 6 days. She described him as "*very unsteady shaking etc.*". Mrs Dickinson had apparently left a message for the key worker. The nurse spoke to the GP.

ii. The GP, the first clinician to see the Claimant after his discharge from hospital, also recorded that the Claimant had had no alcohol. The note of the presentation includes "*Hasn't been himself today – confused this morning, dizzy, 'not himself'. Sleepy.*"

iii. The examination seems to have been normal save for a "*mildly ataxic gait*". No tremor or ophthalmoplegia was noted.

iv. In terms of diagnosis, the GP wondered "*about Wernicke's encephalopathy*".

v. There is no documented awareness about the failure to administer Pabrinex during the hospital inpatient stay, at this stage.

vi. It appears that the GP initially prescribed oral Thiamine, but later reconsidered and felt that parenteral (not oral) Thiamine may be required so telephoned and spoke to Mrs Dickinson. She was then to take him to A&E.

28. So it was that the Claimant arrived in the Emergency Department ("ED") of The Defendant's Cramlington Hospital at 1920 on 1 September 2016 [1371]. The ED Assessment Documentation included the following summary at 1950 and a recognition at that point that the Claimant had not been taking Thiamine:

> "*Recent detox while in hospital having an abcess removed. C/o acute confusion and feeling generally unwell. Has not been taking thiamine. Seen by GP today who advised A+E. Acute confusion ?Wernicke*" [1460]

29. Medical clerking was documented between 2245 and by 2340 [1462-70] that day. The matters noted included the following:

    a. The doctor was told that no Pabrinex had been given during his recent admission to hospital, although chlordiazepoxide had been.

    b. The doctor recorded two presenting complaints:

        i. Confusion; and,

        ii. Balance problem.

    c. In the presenting history, a two-day history of "*problems with memory/confusion*". A rudimentary bedside test of cognition (the six-item screener or "SIS") was undertaken and the Claimant scored 5/6, having wrongly answered as to the day of the week).

    d. It was noted that the Claimant felt drunk without drinking.

    e. The Claimant complained of his left eye flickering and of paraesthesia (abnormal sensation) in his hands and legs. He had suffered a single episode of faecal incontinence that night.

    f. The note relating to the examination of eye movements is not entirely legible, but suggests some blurred vision was reported.

    g. The doctor made a note of "cerebellar signs" using the DANISH mnemonic (a test designed to identify signs of cerebellar dysfunction). All appeared to be negative/normal save for a specific note of "*Ataxic* [with] *no heel toe walk*".[3]

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[3] Ataxia is a term for a group of disorders and conditions that effect co-ordination, balance/walking and/or speech. It can also impact swallowing, motor control and vision. The exact symptoms and their severity vary depending on the type of ataxia a person has.

h. The conclusion of this medical clerking, like the GP, was of "- *Possible Wernicke's – Vit B1 deficiency*". The plan was to admit the Claimant and amongst other things start intravenous Pabrinex, which appears to have commenced at 0020 on 2 September [1629].

30. A further medical review was performed by CT1 Shevlin at 0120 on 2 September 2016, so about an hour after IV Pabrinex had started [1471]. The presenting complaints were recorded as "*confusion, ataxia and paraesthesia in glove & stocking distribution*". The Claimant was noted to be feeling a little better. He was described as alert and orientated, scoring 9/10 on another rudimentary test of cognition (forgetting his address, apparently). The doctor noted that the Claimant's fingertips and toes "*still feel tingly*". It was recorded that the Claimant was "*still ataxic on walking*". The working diagnosis was still "*? Wernicke's*".

31. A nursing note at 0130 confirmed the Claimant "*remains confused*" [1477].

32. At 0650, the Claimant "*appears less confused this AM…*" according to nursing notes [1477]. CIWA scoring started at around 0800 [1548].

33. Dr Bennett, the Consultant, conducted the Post-Take Ward Round at 0935 on 2 September [1473]. The presenting complaint was documented to be "*confusion & ataxic*". The Claimant was noted to feel sore and tired and frustrated by being in hospital. A history recorded "*unsteadiness, forgetful and confused, came on over the last few days*". He "*seemed drunk*" and had difficulty walking. An abnormal neurological sign was found on examination; namely left side "*past pointing*", a clinical sign of cerebellar dysfunction. The Claimant was noted to be "*unsteady on standing*" and "*pins & needles*" in both fingertips and toes were highlighted. The Consultant's provisional differential diagnosis was "*Vitamin B1 deficiency ? Wernicke's*". Of note is

that this was the third doctor in hospital consecutively to make this diagnosis, a point discussed further below.

34. At 1100 on 2 September, ASN Laura Brown had an apparently detailed discussion with the Claimant which she wrote up as follows [1477-8]:

> "*...Liam settled at present no signs of alcohol withdrawal at present. States abstinence for 8 days, as recently detoxed in hospital. Liam stats a long history of heavy alcohol use 20 cans daily for approx 10 years. Denies any withdrawal symptoms. Is aware of medical condition, long discussion re thiamine + nutrition highlighting the importance to health. Liam is aware of the danger of sudden cessation + implications to health of continuing drinking. He plans to abstain on discharge and engage with key worker Amy at NTRP. Message left with Amy to inform her of admission. Liam lives at home with partner + her two kids aged 13 + 14. Discussed concerns re alcohol dependency and duty of care to social services. Will discuss with Amy when returns call...*"

35. At some point on 2 September a CT head scan was performed which was reported to show normal intracranial appearances [1508].

36. The next ward round documented was on 5 September 2016, in which Dr Warren noted "*Confusion improving*" and in a discussion with Mrs Dickinson "*explained [the] probable diagnosis of Wernicke's encephalopathy*" [1478-9], which was a "*clinical diagnosis based on kind of ataxia, ophthalmoplegia, confusion*". By this point, Mrs Dickinson was documented as expressing concerns about "*memory impairment and neuropathy, which is new*". Dr Warren explained that Pabrinex had been started [to be administered] and symptoms might improve but it was likely to take weeks or months rather than days. Mrs Dickinson said she had written a letter of complaint.

37. A series of physiotherapy appointments reveal significant ongoing problems with mobility:

    a. 5 September: [1642-3] and [1480]

b. 6 September: [1481]

c. 8 September: [1486]

d. 9 September [1445]

e. 12 September [1491]

f. 13 September [1493]

38. On 6 September, the Claimant told the assessing clinician that he was "*struggling with physio as cannot remember the commands & steps*" required and was concerned Pabrinex had been stopped the previous night [1480]. He expressed concern about his memory to nurses at 1110 on the same day [1480]. In the same entry there is an update about the referral to children's services "*no further action*".

39. On 7 September, physiotherapy could not be undertaken *"...In bed, [patient] asked if we could leave it today asked p[atien]t why, p[atien]t pulled sheets over his head and started to cry."* The Claimant seemed unable to answer questions other than wanting the physiotherapist to leave [1483]. On the same day, the doctors noted that he felt upset about everything that was going on; felt confused, but able to remember the name of a doctor who reviewed him on the preceding day [1482]. A discussion was had about WE and his condition [1483-4].

40. At the ward round on 8 September, it was noted that the Claimant had been upset on the previous day, was of low mood and wanted "*to die*" [1484-5]. His mobility was limited by burning pins and needles in his legs, like a dead weight. The doctor recorded the following impression: "*No clinical evidence of Wernickes, ?cause of [illegible]...if not improvement... MRI spine, May be behavioural [therefore] hold.*" [1485]

41. On 9 September, the note of the ward round indicates that the Claimant was mobilising to the toilet with a Zimmer and felt very unsteady on his legs. There is reference to a normal neurological examination. The Claimant's frustration about poor mobility was noted. [1486]

42. On 12 September, an elevated test of liver function (ALT) was noted and the cause queried, including, "*??alcohol over weekend/end of last week as was off ward for period of time…*". However, this was later confirmed to be likely secondary to antibiotics (see discharge letter of 14 September [1521-2]).

43. On 13 September, the Claimant was encouraged to use crutches around the ward rather than a wheelchair [1491-2].

44. The Claimant was discharged on 14 September 2016, with the following summary in the discharge letter [1374]:

> "***Presenting Complaints and Clinical Summary***
> *This 37 year old was admitted with confusion, unsteadyness, forgetful. He has a history of alcohol excess. He was very recently discharged by the Freeman ENT team for abcesses (?parotiditis). He was treated with possible Wernickes with a long course of Pabrinex. He was also seen by the alcohol nurse whilst an Inpatient. He improved clinically and was discharged. He was seen by the physio and on discharge could mobilise with crutches and was safe on the stairs. He had some equipment put into his house including rails. His abcesses were still an issue so will be seen by the ENT team on 14/9/16. We noted an ALT of 288 but this is likely secondary to antibiotics. We would be grateful if the GP could check this in 1 week to ensure it is settling. Liam will have an outpatient gastro appointment - date and time TBC.*
>
> ***Diagnosis***
> *Possible Wernickes - high dose pabrinex given*
>
> *Discharge medication included Thiamine tablets; 100mg x 2 per day orally*."

45. Whilst further clinical entries for later in 2016 are set out in the Claimant's Chronology, the above provides a sufficient picture of the relevant background and what is recorded in the medical records. There is largely a consensus about the Claimant's steady decline to a profoundly disabled state.

III. THE FACTUAL EVIDENCE

46. For practical reasons, the Claimant's wife, Mrs Michelle Dickinson gave evidence by video link from the Claimant's Solicitors' offices in Newcastle. She was given valuable help navigating the bundles by a trainee Solicitor with the Claimant's Solicitors, as she has some health problems of her own. But I was completely satisfied that she was giving her evidence unprompted and independent of that assistance. There was a witness statement from Ms Valerie Dickinson, the Claimant's mother, but the Defendant did not require her to be tendered for cross-examination. The Claimant himself did not serve a witness statement or give oral evidence.

47. None of the treating clinicians involved in the contemporaneous treatment provided statements or were called to give evidence. The Defendant called no factual witnesses.

48. Mrs Valerie Dickinson's witness statement was candid that she had a limited recall of some of the events, as she was sadly dealing with the serious ill-health of her aunt at the time and shuttling between her and the Claimant. In her unchallenged evidence, she noted that some knee problems meant the Claimant had been unable to work for a time. He was seeking help for his drinking and also off work whilst doing that. What she does recall is that at the time, the Claimant was mobile and did not need any help or assistance with his care, as well as having no problems with his memory of which she was aware.

49. Valerie Dickinson recalled the Claimant telling her about a fall he had experienced whilst in the Freeman Hospital although she was unclear precisely when. Later, she recalled being told by her daughter-in-law, Mrs Michelle Dickinson, on 1 September, that following his discharge from the Freeman Hospital the previous day, the Claimant was unwell and that the GP had told Michelle Dickinson to take him to A&E urgently due to a concern that he needed IV vitamins. She recalls Michelle Dickinson saying that the Claimant was unsteady on his feet and she needed help, Valerie Dickinson then states "*I went with Liam and Michelle in the car to Northumbria Specialist Emergency Care. I remember Liam was confused and struggling to walk and that we needed to use a wheelchair to get him to A&E where he was admitted to hospital for treatment.*" She was frank that she has no clear recall of the events of the following days.

50. Mrs Michelle Dickinson had provided witness statements dealing with liability in August 2024 and quantum in March 2025. She was robustly but appropriately cross-examined about her recall of the relevant events, not least as in the absence of the Claimant giving evidence, hers was the sole factual witness account of the contemporaneous events. Save, that is, for the details upon which Mrs Valerie Dickinson could comment. Mrs Dickinson's evidence traced the history of their relationship, dating back to 2009 and their domestic arrangements. She was candid about his pre-existing unemployment from his joiner work, due to knee pain, his other health issues and alcohol use. Whilst noting his long-standing knee pain, she was clear that prior to the index events he was mobile, his vision and speech were normal and he had no issues with his vision, speech, memory, concentration and coordination. She described giving the Claimant an ultimatum in around June 2016 about his excessive drinking and was proud that he had been able to cut down his drinking from around 20 cans to more like 4 to 8 cans per day.

51. Mrs Dickinson described the events leading up to the Claimant's admission to the Freeman Hospital, whilst he was there and upon discharge. Amongst other details she noted:

   a. That the day after the operation, hence 29 August 2016, that the Claimant was not his usual self but the nurses reassured her and that it was the after-effects of morphine from the operation, although she felt that was not a reaction he had had before. The Claimant also described to her feeling groggy and like he was drunk.

   b. He started to appear confused and she observed and heard the Claimant's account of emerging mobility issues.

   c. He was not himself upon discharge and came across as confused and with ongoing mobility issues which required some help from her.

   d. At home, she managed to get him out into the garden to watch the birds. "*However, he was confused, did not know where he was and became really upset. In the end I managed to get him to bed, calmed him and left him to sleep.*" (my emphasis as this point became the subject of debate)

   e. She was increasingly concerned and sat up all night and researched the Claimant's symptoms. She came across a reference to Thiamine deficiency and WE. She had never heard of this condition but "*it did seem to fit with his symptoms.*"

   f. She described telephoning the ward at the Freeman Hospital and discovered via a nurse that the Claimant had not been given Thiamine. She started to realise that this might be why he was unwell. The following morning she spoke to the GP, explained his symptoms and also her concerns about possible WE. "*The GP explained that this condition is caused by a vitamin deficiency of thiamine* *following alcohol withdrawal. The GP then prescribed thiamine tablets for Liam to take at home… [subsequently] the GP rang me and said that I needed to take Liam straight to hospital as he was concerned that he needed IV thiamine.*"

g. She described the arrival at the Cramlington Hospital and the need to use a wheelchair to get the Claimant into A&E. He was admitted and put on a Thiamine drip.

h. The following day he was transferred to the North Tyneside General Hospital where he stayed for a further 3 weeks. She describes him as less confused but still having some memory problems, still not being able to walk properly and needing a wheelchair and nursing help for toileting. He had physiotherapy and used a Zimmer frame.

i. She described his condition upon discharge from hospital on 14 September, "*He had blurred and double vision. He still had pins and needs and could not walk properly and was using crutches. We had some adaptations made for him to come home- a stair rail and an over the toilet commode. He struggled to even walk with crutches and so he mainly stayed in bed*."

j. After a further admission due to a recurrence of the abscess she described (see para.31 on p.146) the gradual deterioration in the Claimant's condition over the following 8 years and his significant degree of disability requiring help with all activities of daily living and being largely bedbound. "*Liam had none of these problems prior to the events of August 2016 and the failure to give him the medication he needed to manage his for alcohol withdrawal whilst he had surgery.*"

52. Mrs Dickinson's statement as to quantum describes in detail the significant level of his disability and the extensive care he has needed since these events. I will return to those

matters in more detail when considering quantum, but it paints a sadly bleak picture of his situation and the practical consequences. "*I am increasingly concerned about that fact that Liam is bedbound. I am frightened that in an emergency if Liam is upstairs I would not be able to get him out of the house.*"

53. Whilst there are issues around care needs, his condition as described is in line with the expert evidence. One additional point which his pertinent to liability is the Claimant's mental health and the terminal illness of Mrs Dickinson's father: "*He did suffer from a low mood at times before August 2016 as a result of not being able to work, his alcohol dependency and the pain he was in with his knees. My Dad [who Mrs Dickinson described elsewhere as having been an alcoholic] had been diagnosed with terminal cancer in around June 2016 and he sadly passed away on 26 July 2016 which really affected Liam's mental health. However now Liam's mood is much worse…there are times where he is extremely low and cries all the time…There are times when Liam openly says that if he had the ability to end his life he would do which is really upsetting and distressing to hear.*"

54. In Mrs Dickinson's oral evidence, she clarified her statement to comment on a progress review with the alcohol liaison nurse, dated 18 August 2016. That contextualised the apparent reduction in alcohol consumption to 7 shandies a day and three cans, "*… however on 18/7 his partner had to leave the home to care for her father who sadly passed away a few weeks ago. Since then Liam increased back to 20 cans daily. He stated that it was due to no one being there to help him or tell him not to.*" Mrs Dickinson described spending time away from the family home for about 2 weeks before her father died (on 26 July), to care for him. For about 3 weeks after he died, she was having to empty his flat, which had to be done within 4 weeks so "*there was quite a lot to do*".

55. She clarified her recollection that the Claimant rarely had lunch, unless the children were off school. In the months leading up to the relevant events, she cooked daily at teatime and the family ate round the table.

56. As noted above there was robust and extensive cross-examination.

57. Mrs Dickinson was challenged on a number of the aspects of the Claimant’s physical, cognitive and mental health presentation that essentially those were not different from how he had been on occasions in the past, but she did not accept that. The thrust of her evidence was that the situations were not comparable.

58. She was challenged on the impact of a number of other life events around that time, including the Claimant’s brother’s imprisonment in May 2016, financial issues with benefits entitlement and potential local authority safeguarding involvement stemming from the alcohol use. She did not accept the implication that these were destabilising for the Claimant, rather than concerns he had, or might have been the source of the change in him, in contrast to the events around his hospitalisation.

59. Mrs Dickinson’s response to the apparent change in her account from one of a successful and reducing picture of alcohol consumption to one of that picture being interrupted by her absence caring for her father, was that the statement was a summary and did not explain every detail. She accepted that there were different amounts recorded in different entries in the records. She noted that the picture was up and down and was not consistent. Her best recollection was that following the detox his consumption initially went down and then went up again either side of her father’s death, whereas in the final days prior to the admission to hospital it probably went down again to nearer 4 to 8 cans per day. She denied repeated suggestions that she has been trying to paint a picture that the Claimant would have recovered and been absolutely fine, contrary to the real world situation at the time and/or that she was now changing her evidence.

60. Mrs Dickinson was challenged extensively about her recollection of how the Claimant presented in hospital, potential contradictions with, e.g. his playing of Pokémon on his mobile telephone and other potential explanations for what she observed. She largely maintained her account of what she observed and her concerns.

61. There was a good deal of focus in cross-examination on the contrast between Mrs Dickinson's account that the Claimant rarely ate lunch prior to these events and her account as recorded by Dr Goulding, "*[Michelle Dickinson] told me that she made sure he had a good lunch and an evening meal each day.*" Mrs Dickinson's explanation was that this was an error and there must have been some confusion. Perhaps she had said that this was following his hospitalisation but she maintained that previously he did not have a good lunch. She was pressed on other entries in the records and reports tending to suggest that the Claimant ate relatively well and normally. She was pressed on entries in Prof Carson's report about the Claimant eating normally and having a good diet. Mrs Dickinson pointed out that Prof Carson had made other errors as he said the Claimant was studying and was muscled. It was her who was studying, not the Claimant and he could not be described as muscled. She felt that the records indicating the Claimant ate more in hospital fitted with a change in him, as he also switched from drinking coffee, as he had done before, to drinking tea, "w*hat he ate [in hospital], that is not the Liam I recognise*". In re-examination she clarified that in hospital he was not drinking alcohol, implying that the volume of what he was consuming in liquid was much less.

62. She was pressed about differing accounts of the state of the Claimant's confusion, memory and cognitive ability both during the admissions and since. Mrs Dickinson explained how she saw the situation day-to-day and there could be fluctuation, as he could remember a detail for about 3 days but would forget other information. This meant he could cope with some tasks such as using a computer but could not retain information

beyond those few days, albeit his longer term memory of events prior to the Freeman admission was better.

63. There was exploration of the incident at home on 31 August, specifically in the garden. This took on some importance in the experts' evidence so I will record my contemporaneous typewritten note of what she said in evidence. Whilst it is not verbatim, it records the key aspects, "*[I] heard him scream, [he did] not know who I was, he [did] not know where he was, [I] brought him in and calmed him down. Then [the] episode was over. Then I put him to bed. He slept through [the] night, [there was] no further panic or confusion.*" In re-examination she gave the same account in its material respects.

64. As for the interaction with the GP, she said she told the GP she thought the Claimant might have Thiamine deficiency and the GP said he wondered if he had WE.

65. She rejected repeated suggestion she was a liar or trying to bolster the Claimant's case on various points, e.g. after discovering that establishing malnourishment would help his case. Her account regarding quantum was also probed but on balance, Mrs Dickinson's account remained consistent. She described his current problems as wide-ranging and severe mobility, pain, mood and cognition difficulties, as set out at (p.153/10).

66. She was asked about the Claimant not wanting carers in the house. She explained there were a few reasons, going beyond the Claimant not trusting them. Other reasons were money, as the prices went up and they were not able to afford it. It would have been better if it was the same people all the time, but it was different people in succession and they were unable to handle the Claimant properly. They were extremely young. The Claimant's request for them to leave was in the context of having a series of different carers with whom he was not familiar.

67. She clarified that since her statement, the Claimant had moved downstairs. She was challenged about the time it took to care for the Claimant's birds and her capacity to do it with her own disabilities. She stood firm that it was 2 hours each day to do the bare minimum to keep them alive. She maintained that notwithstanding the limited walking ability recorded in the DWP records, the Claimant had been taking the dog for a walk. He did it in stages, including rests, by going to the field at the end of the street, stopping to talk to people on the way. She was challenged about the Claimant's willingness to engage with therapies and said, where appropriate he was willing. They had only seen physiotherapists and he had done his best to engage. They tried to get FND rehabilitation, the Claimant wanted to go but they did not think he was suitable. That was last year, she thought.

IV. THE EXPERT EVIDENCE

68. There was extensive expert evidence before the Court from sixteen experts.

69. I heard oral evidence from seven of those experts: the parties' Neurologists, Dr Goulding for the Claimant and Dr Wills for the Defendant; the parties' Neuropsychiatrists, Dr Achinivu for the Claimant, as to causation, and Dr Symeon for the Claimant, as to condition and prognosis ("C&P"), and Prof. Carson for the Defendant on both issues; and the parties' Care and Occupational Therapy experts, Ms Woolcomb for the Claimant and Ms O'Connell for the Defendant. The other experts were not in the event called as the issues had been sufficiently narrowed or agreed in respect of their fields of expertise, although there were written reports commissioned by the parties from neuropsychologists (Drs Leng and Evans for the Claimant; Dr Hull for the Defendant), life expectancy experts (Dr Davies for the Claimant; Dr Cohen for the

Defendant), physiotherapists (Ms Richards for the Claimant; Ms Browning for the Defendant) and accommodation experts (Mr France for the Claimant; Ms Twentyman for the Defendant).

70. **Dr Goulding**, the Claimant's Neurologist, had a career in the NHS, as well as stints in research through to his retirement as a Consultant in 2018. He summarised his views on the various issues:

a. He thought the Claimant's Thiamine status was likely very low at the end of August 2016, directly caused by heavy drinking. That heavy drinking had a range of effects, including on the absorption and transport of Thiamine notably to the brain and its activation to become a constituent part of allowing brain cells to produce energy.

b. There were three particular effects which excess alcohol had on absorption. It increased the speed of transit of food through the gut, resulting in less contact with the bowel mucosa; the lining of the bowel became inflamed which reduced the passage of Thiamine and alcohol impacted the transit through to the blood stream.

c. Irrespective of the amount of food consumed, excessive alcohol would have affected how much Thiamine was being absorbed and the other mechanisms would be affected.

d. The metabolism would also have been impacted. The large amount of alcohol would have needed to be metabolised so it can be absorbed and excreted. Up to 20 cans was an energy dependent mechanism. The body would also have needed to repair the damage to the bowel and liver. This is energy dependent and needs Thiamine. Excess alcohol would cause the body to burn through more Thiamine.

There would also be excessive excretion of Thiamine due to the diuretic effect of excessive alcohol.

e. For the brain to have access to Thiamine it would have needed to pass the blood brain barrier. That would then need to be activated into Thiamine Pyrophosphate to allow the brain to metabolise glucose to allow the cells to function. If that process does not work appropriately it leads to a situation where WE develops.

f. The reported vomiting of blood and later gastritis was evidence that the Claimant was drinking heavily for this to happen. Many people with reactive gastritis have indigestion and this may have impacted the Claimant's appetite. This would in turn potentially have affected Thiamine intake and levels.

g. The NICE guidelines identified the recognised concern around those abusing alcohol presenting in an emergency hospital setting. The first is that patients drinking heavily are not able to drink in hospital so are likely to develop alcohol withdrawal syndrome. Also patients go to hospital because of a medical condition generally and those need the body to produce energy to combat them. Any energy process needs Thiamine and so Thiamine levels will drop. If they are already low due to excessive alcohol consumption, this can precipitate WE. The NICE guidelines indicate that there should be treatment with Thiamine and if there is concern about nutrition, this should be IV treatment to bypass concerns around absorption. Those guidelines indicate that there should be 3 doses over subsequent days and it is unclear if even a single dose was administered.

h. The Claimant had a concurrent illness and need for surgery under general anaesthetic for the facial abscess, requiring energy. He was having IV antibiotics indicating a level of concern and his inflammatory markers were elevated which required energy. There was also the alcohol withdrawal syndrome: at one point his CIWA score, the measure of alcohol withdrawal, went up to 20, which is severe. This results in irritability and tremor/sweating and the depletion of Thiamine in the brain cells.

i. It is most likely that the Claimant developed a WE on 1 September, shortly before Mrs Dickinson contacted various people to report her concerns that he seemed brain damaged, impulsive and was walking unsteadily. Ataxia and confusion were classic as two of the three hallmarks of WE and the reported symptoms at the time were consistent with those two being present. Ataxia and walking with a wide-based gait were documented by three doctors. The Consultant also noted pass-pointing, a sign of cerebellar dysfunction.

j. The WE here did not cause permanent organic brain damage. He was rapidly treated with IV Pabrinex which is a powerful way of increasing brain Thiamine levels. If someone is mildly affected, a good recovery would be anticipated. The confusion was not an issue a day after the admission, given he was able to have a discussion with the alcohol nurse.

k. The notes do not indicate that the unsteadiness improved very much and it was all quickly subsumed by FND. By 6 September there was evidence of the Claimant getting worse and becoming emotional. That is most likely where the FND symptoms were developing.

71. Dr Goulding was subject to a well-planned and lengthy cross-examination across the full range of his opinions. He was challenged about his experience as an academic and clinician of WE. He contextualised the limited nature of that by the fact that WE is uncommon given the prophylactic use of Pabrinex in those who are vulnerable to it. He had had cause to diagnose it in his practice and to see those vulnerable to it. He had studied alcohol related ill-health and in his clinical work it was quite common to see

patients with, for instance, seizures from large alcohol binges or related brain injuries. He felt that hospital doctors would have a lot of contact with patients with alcohol related problems.

72. Dr Goulding was clear that contrary to Mrs Dickinson's account, he was not confused about the account of that although he did not have breakfast, she made sure the Claimant had a good lunch and dinner. He was challenged about various entries not specifically referenced in his report, including the GP entry for 1 June 2016, (A2/37) and indicated that it was difficult to decide what to include. His report was 60 pages and he tried to be thorough but not over thorough. He rejected the notion that this was a tactic to ignore records inconsistent with his line of thinking or that in any part of his report he was making a conscious decision to exaggerate or eagerly support the Claimant's case.

73. He maintained that even if the Claimant was having one or two good meals a day, he still was likely to have low thiamine levels caused by his heavy drinking and problems with absorption. He accepted that intake of Thiamine was key to likely levels of it. But he stood by his views that the Claimant's food intake would have been poorly absorbed (and hence the Thiamine from it) and rapidly metabolised in the circumstances.

74. Even if he had stopped drinking entirely prior to the admission, which was not the evidence, it would take a few weeks for bowel problems to recover and Thiamine levels to improve. He felt that the good appetite recorded in hospital would have been reflective of the fact he had been drinking heavily for years, had been detoxed and it was the first time he could taste his food.

75. Whilst he accepted that if the Claimant was eating well, that he would be absorbing some Thiamine, even if there was some bowel damage, the key factors after intake of Thiamine were absorption, storage, transit and metabolic demand. Those underpinned his views on likely Thiamine levels.

76. He accepted that there were millions of alcoholics and they did not all get WE, indeed that was a very rare condition. He explained that WE tends to be precipitated by an external factor, such as illness, fasting and withdrawal syndrome and other alcoholics usually avoid WE, especially given the use of prophylaxis. If someone has very low Thiamine levels but enough to allow the brain to function, they will not develop WE, but if something lowers it further to a critical level then WE will develop.

77. Although there was a normal liver function test, this did not indicate whether the liver was normal. He felt it likely that there were issues with the liver and hence storage of Thiamine, not least given the amount he had been drinking over a long period; alcohol was the most common toxin to cause liver disease. There was a test of liver function a year later which showed changes suggestive of fatty liver.

78. He was challenged on the cited literature, "*Time to Act on the Inadequate Management of WE in the UK*", Thomson et al, 2012, focusing on the causes of WE; and "*WE: new clinical settings and recent advances in diagnosis and management*", Sechi and Serra, 2007; and "*A study of the nutritional defect in Wernicke's syndrome",* Phillips et al, 1951. In relation to the latter paper, he accepted that for the patients in the study, the improvement from confusion took days, not perhaps 12 hours as in the present case. However he felt the Claimant's mild presentation could not be compared to the seriousness of the patients in that study who had severe WE and "*these poor people had no chance*".

79. He was accused of adopting a reductive logic which was not scientific. He contended that there was compelling evidence that chronic alcohol misuse does reduce the Thiamine available to brain cells.

80. It was put to him that someone who is mobile, eating and drinking normally, going for walks and outside to vape, did not have a metabolic mechanism out of the bounds of

ordinary. He highlighted the other processes at play, despite challenges about them, including the alcohol withdrawal (which at times was severe, even with the drug treatment received), the facial abscess causing pyrexia (raised temperature) and raised inflammatory markers and the metabolic effects of surgery, i.e. healing from the wound of surgery.

81. He was challenged about the NICE guidelines, given the mere fact that he was an alcoholic admitted with an acute illness did not require parenteral Thiamine unless he was malnourished or at risk of that or had decompensated liver disease. Dr Goulding pointed out that the treating team must have thought the Claimant was at risk as they prescribed IV Thiamine. He also questioned why breach of duty was admitted otherwise. He did not accept that Pabrinex was widely overprescribed and thought the opposite was true. He pointed to the paper with his causation report based on an audit in Birmingham where only 14% of patients who should have received Pabrinex actually received it. The conclusion was that it should be more widely advertised that anyone with alcohol abuse and a surgical or medical problem should receive it in IV form. He felt that the fact that the clinicians thought he needed to be prescribed Pabrinex was confirmatory of him needing it. Had this been done, he would not have got WE.

82. Whilst accepting that a healthy individual would have 18 days of stored Pabrinex, that did not apply to the Claimant as he was not healthy and was an alcoholic. He felt that the Claimant probably received about 1/30 of the first dose. He then developed overt symptoms on 1 September when levels became low enough for his brain not to function properly; he may have been starting to develop subtle symptoms in the day or so leading up to that. Mrs Dickinson’s report on 1 September to the alcohol nurse was significant as she described confusion, without any alcohol being in the house to be consumed, appearing like he had brain damage, being unable to walk straight and being impulsive.

83. He rejected the notion that the symptoms the Claimant presented with could, at that stage, be functional. A functional presentation is normally of a very striking disability not in keeping with the other facets of the presentation. It is very unusual for a functional disorder to be a very mild disorder the doctors are struggling to find. That is not at all like a functional presentation. The physical symptoms from the earlier part of 2016 were consistent with knee pain. They did not include unsteadiness on standing which was the heel-toe ataxia noted in hospital. The same applied to the much earlier records going back to 2011, which was a wide-based gait in the context of stiff legs. In 2015 there was an antalgic gait (a limp), not ataxia which is quite different and a medical student could tell the difference.

84. It is also noteworthy that the clinicians in hospital were also using a working diagnosis of WE and not a functional condition. Once the Claimant's WE receded, which it did rapidly as expected given the treatment using Pabrinex, he then went on to develop a new onset functional condition, which he had by 8 September and which was developing by around 6 September when his symptoms started to get worse again. It continued gradually to get worse thereafter.

85. **Dr Wills** gave neurological evidence for the Defendant. He had also had a career in the NHS, at Consultant level since 1998 and ongoing, as well as stints in research and extensive publications in his name. His teaching had led to an appointment as Honorary Clinical Associate Professor by the University of Nottingham. He considered that to understand the case it was necessary to understand general medicine as well. He had moved into that field 10 years ago. Most Wednesdays he was in A&E seeing an unselected medical take and lots of alcohol related problems and on the acute medical wards he saw similar patients. Other aspects of his background and work he also felt provided him with relevant experience.

86. His evidence about the rarity of WE chimed with that of other experts. Even with his experience of acute medical wards, he had seen a handful in his career. If it was suspected, it is correct that it should be treated.

87. He then outlined, for the first time, that for the purposes of this case, he had done an audit last year of Thiamine administration in his hospital. approaching the pharmacy and using the records of 40 patients, to do a personal audit.

88. The Claimant took objection to this line of questioning given it was not something trailed in his evidence or which was known about and essentially was unfair. There was no information against which to explore the issue with him in cross-examination. If it was important, it should have been set out in writing, with access given to the information and opinions expressed. I ruled at the time that I would allow the questioning but consider what weight, if any, to apply to it following submissions.

89. In further questioning, Dr Wills appeared to change tack, saying that the audit was not actually for the purpose of this case but because there needed to be periodic audits. It took place a couple of years ago and he had not mentioned it in, for instance, the joint statement which post-dated it, as he did not think the case would go to Court. Of the 40 cases given Pabrinex, none was shown to have WE. His conclusion was that the drug was often over-prescribed as the consequence of not doing so was so profound.

90. Returning to his reports, he corrected the assertion, at p.251 of the bundle as the report is not page numbered, to point out that he was wrong to say, "*He was given Chlordiazepoxide but apparently only one dose of Pabrinex*." In the Joint Expert Report ("JER") with Dr Goulding, he agreed that a complete vial of Pabrinex had not been given, but could not say how much. He agreed with Dr Goulding's analysis which led him to the view that it was at most only about 1/30th of the prescribed dose.

91. He agreed in his evidence with Dr Goulding that humans are not able to synthesize Thiamine, it has to come from diet and that it is an important vitamin which is important to many energy producing processes. His view was that of the four mechanisms discussed, by far the most important mechanism was nutritional intake. In order to become deficient and for that to affect the brain, there had to be some kind of disruption from food getting into the system and thereby Thiamine into the nervous system. He felt that nutritional intake was adequate in the Claimant's case, having read and heard the evidence.

92. He did not consider that the gastrointestinal symptoms described were significant to the nutritional intake. Vomiting was only mentioned once in the records. Thiamine needs to be absorbed by the small bowel which starts after the stomach. It is known from the endoscopy that the duodenum was normal and it was likely that the jejunum (the preceding section) was also normal.

93. As regards storage, Thiamine is stored in a number of organs, the biggest being the muscles. Contrary to Mrs Michelle Dickinson's evidence, he thought the Claimant must have been muscled as she had said he had a high BMI but was not obese. The muscle stores must have been normal. He was sceptical about the suggestion of his liver being otherwise than normal. Whilst the absence of abnormal liver function tests cannot exclude problems with the liver, there was lots of other evidence that the Claimant's liver was normal and none of the other stigmata of liver disease were present.

94. As for excretion, whilst it is true that alcohol is a diuretic, Dr Goulding did not seem to elicit a history of diarrhoea or polyuria (excessive urination usually at night) which undermined that theory. As for transport of Thiamine through the body, most of it is transported in red blood cells and he described the mechanism. Liver function is key to this process and there was a normal test of liver function and normal clotting "*when readmitted with so-called WE*".

95. He highlighted the difficulty of definitively diagnosing WE. This led to treatment being given, as it was cheap and the consequences of not doing it if there truly was WE were grave, so that in many suspected cases, the diagnosis was never actually made.

96. Whilst accepting the consensus that alcohol has the ability to disrupt the blood brain barrier, there were many other conditions which do that where there is no Thiamine deficiency. He doubted the catabolic state theory, that the Claimant was so ill from his facial abscess that his metabolism was burning up Thiamine in a short space of time, was tenable on the records made of observations and vital signs. He was sceptical about a number of the aspects of the neurological presentation being seen to be indicative of WE.

97. He underlined that despite the widespread excessive use of alcohol, WE remained rare, as there needed to be other factors in addition to excess alcohol. The alcoholics which cause concern are those who are wasted physically, as that is a sign of nutritional deficiency; nutrition was key.

98. If the Claimant had no or very low Thiamine, he would likely manifest with symptoms within 5 or 6 days, which was compatible with the timescale here, but he did not believe the Claimant had no Thiamine or that he ever had WE.

99. Here the MRI scan, which could pick up abnormalities in about 50% of cases, was done many weeks later and any structural changes would have resolved by then. So it was not proof one way or the other.

100. If the Claimant was showing signs of WE, which he did not accept, the timescale of improvement did not fit with the expected outcomes. He had been taught in a department with particular expertise in vitamin deficiency, that nystagmus got better quickly, within hours or a day or two; ataxia and confusion took longer (as they were from structural

changes), with confusion taking weeks to resolve. It did vary somewhat between patients. But other causes of confusion could resolve more quickly.

101. Dr Wills was subject to a skilful cross-examination. He reiterated that the audit of cases of suspected WE in his hospital he had carried out was in fact nothing to do with this case. It was done before he finalised his causation report, but "*did not feel [the audit] was sufficiently relevant to include in the report or joint statement, [because] he did not think the case would go to trial.*" When asked whether that was a good reason to omit relevant evidence from his report, his answer was "*I do not know if that was a good reason.*" Having done medico-legal work since 1998, he had given evidence at trial 6 or 7 times and his experience was that trials usually do settle after the JER, "*I did not think I would be sat here today."* The reason for doing the audit was that Pabrinex was becoming more difficult to obtain from the manufacturer and seemed a reasonable time to take stock and get resident doctors to be more alert to the symptoms and signs of WE. The motivation was not to do with this case. He confirmed he did not do an audit into patients who were prescribed it and did not receive it. He looked at those who received Pabrinex but did not go on to develop WE.

102. When challenged on his views on nutritional status, he confirmed that he felt that the Claimant's stores of Thiamine were the same as the average healthy person, i.e. 18 days' storage. This meant he could not have developed deficiency and WE within the relevant 5 day window whilst an inpatient.

103. In June 2016, on referral to the Recovery Partnership, the Claimant admitted that he was drinking 12-40 units of alcohol a day (p.250). Dr Wills accepted that most alcoholics understate the amount they are drinking. Even 12 units a day was excessive and he did not doubt for a minute that the Claimant was drinking excessively.

104. Dr Wills confirmed that the record Dr Goulding had been criticised for not mentioning, the GP record for 1 June 2016, was not mentioned in his report either. Dr Wills had not interrogated the records further than the paragraph about the 12-40 units to see how much the Claimant was drinking prior to admission. He did not think the effort was worth it as estimates were likely unreliable, people often drink secretly and the Claimant had been drinking heavily for many years. The level of consumption had to be looked at in combination with nutritional status. If someone took a lot of alcohol but was getting sufficient Thiamine, then they would not get WE. He accepted that absorption was more likely to be impacted in someone drinking 40 units/day than someone drinking 8. But here there was no evidence of mal-absorption.

105. He could not explain why he had not discussed the endoscopy report in his report, which was a minor part albeit important. He did not know why he had not included it.

106. He accepted that his record about the Claimant having 2 meals a day came from Dr Goulding's C&P report. He had recently read the entry (p.1654) for 18 August 2016 from the Alcohol Service, about the Claimant's then partner having left the home on about 18 July to care for her father and the Claimant increasing his consumption back to 20 cans daily, due to no one being there to help him or tell him not to. He could not remember whether or if he had seen this entry before. He agreed it was an important contemporaneous record as to what the Claimant was drinking in July and August 2016; if accurate it did indicate an increase in consumption as shown. However, taken with assessments of his nutritional status, he still doubted the Claimant was nutritionally compromised.

107. Whilst he accepted the references to gastrointestinal problems, he relied upon the absence of a specific gastrointestinal diagnosis. He accepted that an endoscopy in 2012, had shown duodenitis. The Claimant clearly had chronic stomach problems related to alcohol but Thiamine was absorbed elsewhere in the gut, mainly the jejunum. The

contemporaneous endoscopy did not exclude an impact on absorption through the small bowel.

108. He was challenged on his conclusion (p.283, para.2) in the JER that there was no evidence of Thiamine deficiency and it was at normal levels. He focussed on the food intake and made no mention of the excessive alcohol consumption. He accepted that the latter was a relevant factor.

109. Looking at the "*Time to Act*" paper, he agreed with the reason chronic alcohol misusers are at particular risk of WE included because "*Absorption of Thiamine is reduced by both alcohol and malnutrition acting separately or together (Cooke et al, 1988).*"

110. He accepted that he had not asked to examine the Claimant and commented "*[The Claimant] had never volunteered to do it.*" This meant he had not been able to ask about his bowel transit and there was no evidence about whether it was increased. Thiamine was in many foods, so if eating normally, a person had far more than they needed. Either someone was not taking enough in or vomiting it out, that was the crux of the matter when considering the development of a deficiency.

111. He accepted that the diuretic effect of alcohol can contribute to depletion. It was not known whether the Claimant had polyuria or nocturia (nighttime urinary frequency). If that was the case then it would get someone a bit closer to deficiency on the balance of probabilities. He accepted in principle that someone consuming 20 cans of lager/ day, that volume of liquid had to go somewhere and would lead to excessive excretion.

112. He agreed that the *"Time to Act*" paper had another bullet point reason for alcoholics developing WE as being "*ethanol neurotoxicity causes impaired utilization of thiamine (Sechi and Serra, 2007).*" In relation to that latter paper, he accepted the role that genetic and environmental factors play but also highlighted the centrality of dietary intake. He also accepted what that paper said (p.1237) that whatever factors might be at play, the

risk of WE in patients with alcoholism is high, albeit seeing the condition was rare. When asked if the NICE guidelines and prophylactic use had reduced the frequency with which it was seen, his comment was "*One would hope so*".

113. He accepted that Mrs Dickinson may be wrong about the Claimant not having been fat, that the BMI score had deficiencies, and if the Claimant was not physically active, he may have been overweight rather than muscular to explain the BMI. If someone consumes 20 cans of lager that would give calories well in excess of the recommended daily intake, with no Thiamine in it.

114. There was discussion about the significance or otherwise of the various measures of nutritional status, or which testing other things recorded aspects relevant to this, in the records. He thought those remained significant and alcoholics did not generally lie about their food intake.

115. Dr Wills was taken to the NICE guidelines (p.1215). It was put to him that a dependent drinker should receive prophylactic oral Thiamine irrespective of their nutritional status. He interrupted to say that Counsel was looking at the wrong section. But he then accepted that section 1.2.1.2, indicating the need for oral Thiamine, irrespective of nutritional status if undergoing a medically assisted withdrawal, there being no requirement in that part of the guidance to need harmful drinking or malnourishment (or the risk thereof). He did not agree that he was ignoring other mechanisms of deficiency, there was an important hierarchy, with nutrition at the top. He stood by the opinion that if the Claimant was adequately nourished, as he thought he was, and not BMI obese (as Mrs Dickinson said he was not), then it was impossible for him to go into Thiamine deficiency.

116. He accepted that if the Claimant's nutrition was adequate but the other aspects of his presentation were impaired to more than a minor degree, e.g. insufficient stores,

insufficient intestinal absorption, the Claimant could develop WE. But he did not think those factors applied or that he had WE.

117. As for the second part of the NICE Guidelines at 1.2.1.3, he accepted that he was at risk of malnourishment given his drinking history, but it was a very low risk. He did not agree that the Claimant was actually malnourished. If Dr Wills had been looking after the Claimant he would not consider him to be malnourished or at risk of malnourishment. He did not know who made the decision to give him Pabrinex and there were no witness statements from any clinicians, but he doubted that an ENT doctor was best placed to make those judgments. That said, he accepted that under the NICE guidelines, there was a risk (albeit a low one) of malnourishment so he fell within the bracket of the guidelines to receive Thiamine.

118. He was taken to the following passage in the Sechi paper (p.1239) and there was nothing in it he disagreed with, "*Unbalanced nutrition: Because the body's reserves of thiamine are sufficient for up to 18 days, in a healthy individual, any condition of unbalanced nutrition that lasts 2-3 weeks may lead to WE. In individuals with marginal stores of thiamine, the disorder may occur earlier, particularly if the diet has been very rich in carbohydrates.*"

119. He accepted that lager was high in carbohydrate but felt that this was balanced by other parts of the diet and disagreed that on 26 August the Claimant's Thiamine levels were probably greatly diminished, whether he had two meals/ day or not. He accepted the possibility that the levels were abnormal but if they were they were not sufficiently abnormal to cause WE.

120. He was taken through the various observations, indicative of at times significant alcohol withdrawal but he did not consider that this would be imposing a high metabolic load or was particularly significant. He agreed that a localised infection, from the abscess

requiring surgery, would increase metabolic demand to some extent. Trying to portray the Claimant as very or moderately well, holistically, was not tenable.

121. He agreed that someone presenting with low Thiamine and this experience over the course of the admission could go from very low Thiamine to WE and that is the purpose of the NICE Guidelines to avoid this. If he had very low Thiamine, the mechanisms described could be sufficient.

122. In discussing the diagnoses of ataxia, Dr Wills was sceptical about whether it was actually present and in a form relevant to WE. The Claimant had not been under the care of neurology but under acute physicians. The standard of assessment would not have been to the standard of a neurologist or might not be. He worked a lot with acute physicians and their clinical acumen had deteriorated, with clinicians over relying on tests. Most acute physicians were pretty bad at neurology, although he had no knowledge of Dr Bennett. He had no basis to think these doctors do not do things properly.

123. The two other aspects of the triad for WE were confusion and ocular symptoms. A significant minority of patients would have all 3, about 16%. It was relatively common for WE patients to have only symptoms affecting one or two aspects of the triad.

124. He agreed with the assessment on discharge (p.1387-8) that the detoxification of alcohol was nearing completion. He agreed that the note of the incident on 31 August (p.1428-9) of feeling drunk when outside and needing to sit down could be a sign of onset of WE, taken in isolation, but viewed holistically he disagreed that this was the case.

125. As for the incident in the garden after discharge, he accepted that could be evidence of confusion attributable to WE, but there were lots of reasons to make him think it was not. This included playing Pokémon early in the day. He referred to the Claimant not

knowing who he was and that *"if you do not know who you are, that is non-organic, as even severe dementia patients retain a sense of self."*

126. Looking at the record on 1 September at 11.33 (P.1655), he accepted that the note seemed reassuring about alcohol consumption. He agreed the only way to read the note was that Mrs Dickinson was significantly concerned. There was nothing to suggest just a continuation of how he had always been and he agreed something had changed. Her description of appearing brain damaged and being unable to walk in a straight line, falling over and being impulsive included symptoms which could be attributable to WE, including those suggestive of truncal ataxia (although a functional presentation could explain it). He was sceptical of the subsequent GP record of an ataxic gait as, "the average GP would not be able to differentiate between an ataxic gait and a functional disorder." He was challenged to the effect that a competent GP would describe what was clinically found, not what the GP had been told by a relative what the patient may have. He accepted he had no reason to go behind what the GP had written, but he was questioning the GP's ability to make a differential diagnosis, such as a functional condition.

127. On admission on the evening of 1 September, he accepted that if the presenting complaints were present, namely confusion and a balance problem, then that was two of the triad for WE. He felt that the tests for cognition used were rudimentary and nothing like a neurologist would do. It was suggestive of mild confusion and *"for a man who the night before did not know who he was, this is not the natural history of WE."*

128. The reference to feeling drunk without drinking would be consistent with WE taken in isolation. The working diagnosis of the doctor was WE. But this was a FY2, a very junior doctor, saying he had ataxia. He did accept that he was making an assumption about the competence of the doctor who he agreed had the benefit of examining the

Claimant at the time. He was saying that whilst the doctor had *"done all the right stuff, it is the interpretation of the signs which I query"*.

129. Dr Wills accepted that in his report (p.252), the entry about the Claimant being given IV Pabrinex was out of chronological sequence, after the Ward round, whereas it was started at 0020. He was challenged about the brevity of his review of the ward round through to his discharge on 14 September, being dealt with in a few short phrases. He accepted that he may have had some loss of focus there. It was said in his report he had moved very quickly from the conclusion about normal nutrition to concluding that the Claimant did not have WE. He accepted that he was not forensic enough in his analysis at the beginning. But he since had had a lot of information and his opinion remained the same.

130. Looking at the entry for 0120 (p.1471), about an hour after Pabrinex started, he accepted that there was mild confusion, although commenting that it was a rudimentary test. The CT1, more senior doctor than before, identified that the Claimant was still ataxic on walking. Dr Wills agreed that this was another doctor describing 1 of the 3 triad. He agreed that at 0130, the Claimant on admission to the ward was noted to remain confused (p.1477). He felt that the entry for 0650 was important and was not mentioned in his report. The Claimant was noted to be "*less confused this morning*". Dr Wills placed a lot of weight on the garden incident which he did not know about before coming to Court, "*I argue that [his presentation at 0650 on 2 September] was a massive improvement from the garden incident [on the evening of 31 August] when he did not know who he was.*"

131. He accepted that the ward round by Dr Bennett at 09.35 on 2 September (p.1473) there was on examination still apparently ataxia and still a description of pins and needles in the fingers and toes, as well as past-pointing. He agreed that the only differential diagnosis listed was WE. He agreed with Dr Goulding that putting "? Wernicke's" did

not necessarily raise an element of doubt in the clinician's mind and agreed that no other differential diagnosis was noted. Having no other conditions mentioned other than WE indicated less doubt in the clinician's mind. He *"totally agreed"* that contemporaneously, the clinicians believed this was WE.

132. He agreed that WE was a reversible condition if treated properly and promptly. He accepted what was said in the "*Time to Act"* paper (p.1204) that "*Initially, the pathophysiological changes inherent to WE are reversible with administration of parenteral thiamine as long as necrosis has not occurred. Without adequate treatment, the changes become irreversible (Sechi and Serra, 2007).*" Also what was in the Sechi paper (p.1233) that with parenteral (IV) administration of Thiamine this allows a rapid correction of Thiamine deficiency. Whilst it was not impossible, it was very unusual for symptoms to resolve so quickly. The pattern of going from very confused to not confused was highly atypical.

133. It was put to him that considering the level of confusion at the time the Pabrinex was started, apparent improvement over the following 11 hours was plausible, but he pointed to the Phillips paper from the 1950s where it took weeks for the confusion to resolve. He accepted that it was a study of only 9 patients all of whom seemed to be much worse than the Claimant ever was. His observation was that "*most reasonable neurologists would say that the recovery in this patient was too quick [for it to be WE].*"

134. He disputed the notion that the Claimant developed WE on 1 September or perhaps 31 August and that was the explanation for his presentation with a functional picture only emerging in the subsequent days. By disputing the working diagnosis of those treating the Claimant, he accepted this begged the question of what had happened and that his theory was FND, coincidental to the failure to provide prophylactic Thiamine. The WE diagnosis did not fit with some of the other symptoms the Claimant was presenting with such as upper limb paraesthesia, faecal incontinence and flickering.

135. He was asked in re-examination about the status of the Sechi paper. He commented that this was a review article not a randomised controlled trial and, "*It is from Italy and I do not know these authors. I have had one article published in the Lancet Neurology, so they must know what they are talking about.*"

136. He felt that the threshold of moving from oral to parenteral prophylaxis, in the context of the NICE Guidelines was quite low.

137. He gave an account that he had "*thought of a better answer about how long stores of Thiamine last*" and proceeded to describe a calculation he had since done. This led him to think that even if one ignored that the Claimant had a bit of Thiamine administered and assuming generously that his metabolic rate was 25% above normal, he would still have taken 10 days to run out of Thiamine. This was too long to become deficient compared to what actually happened.

138. The Claimant objected to this new evidence which was not put to Dr Goulding for him to comment. It was applying a level of arithmetic, to the evidence of values we did not know and levels of Thiamine which were not recorded, which it did not warrant.

139. **Dr Achinivu** gave neuropsychiatric evidence for the Claimant on liability. Whilst he undoubtedly had fewer years of experience than Prof. Carson for the Defendant, he had worked as a Consultant Neuropsychiatrist from 2009 to 2020, both in the NHS (to 2018) and in a private setting at the Olive Carter Unit, in Birmingham. In 2015 he had founded Simply Neuro Ltd. providing specialist neuropsychiatric services to both private and NHS bodies as its Clinical Director. This had been his sole focus since 2020. He also had various publications under his name.

140. By agreement between the parties, in addition to his reports and contribution to the JER, a supplemental letter was admitted in evidence, dated 23 January 2026. In it, Dr Achinivu wished to correct what he considered was the misrepresentation of his views

on causation as to when the Claimant started to suffer symptoms of FNSD, in the Counter Schedule. He also took the opportunity to correct an error he had made in the JER, to record that the Claimant was discharged from the Freeman Hospital on 31 August rather than 30 August as he previously stated. As to causation he observed variously:

"*In the joint statement with Professor Carson dated 21 December 2016 (sic), I stated that the exact date and time of when the Claimant started to suffer from a Functional Neurological Symptom Disorder (FNSD) is difficult to ascertain from the records. I remain of that view. What is clear is that the FNSD was clearly established by 8 September 2016 [and declined thereafter]…*

I also remain of the view that there was a psychological stressor for FNSD which was triggered when the Claimant became aware that he was not provided with IV Pabrinex during his hospitalisation in August 2016 which had the potential to lead to severe and permanent neurological and neuropsychiatric consequences. I believe that this knowledge of the admitted negligence triggered a "sense of grievance" in the Claimant leading him to experience deep-seated feelings of resentment and bitterness due to his belief that he had not been treated correctly while in hospital. It is, however, difficult to clearly establish when the Claimant became aware of the negligence which triggered this "sense of grievance".

I wish to clarify that it is my view that the Claimant's initial symptoms around the time of his discharge from the Freeman Hospital on 31 August and 1 September 2016 were unlikely to have been "functional" in nature but rather, he was experiencing clear symptoms of confusion and mild ataxia, which were probably related to an underlying Wernicke's encephalopathy, though I defer to the neurology experts on this issue. I disagree with any suggestion that his apparent deterioration after discharge on 31 August 2016 was a Functional Neurological Disorder which simply coincided with the *failure to provide Pabrinex. As I explain below, the "psychological framework" which led to the development of FNSD only arose after the Claimant had deteriorated and after he became aware of the failings in his medical care and the association with his presentation to medical professionals on 1 September 2016…*

It is likely that the Claimant remained symptomatic of an underlying organic condition, though it is my view that the "psychological framework" for the full establishment of the FNSD was already in place during his hospitalisation and certainly by the time of the ward round of 5 September 2016 when his wife reported new onset memory and sensory symptoms.

I wish to clarify that if the Court finds that the Claimant did suffer a mild Wernicke's encephalopathy it is my professional opinion that, on the balance of probabilities, the cause of the Claimant's FNSD, was the knowledge of the admitted negligence which triggered a "sense of grievance" in the Claimant, causing him to experience deep-seated feelings of resentment and bitterness… [the] "psychological framework" for the development of the FNSD "started" around the time of his presentation to hospital late on 1 September or early 2 September 2016 and the symptoms slowly evolved over the course of his admission such that by 8 September 2016 even his treating doctors became clear that there was a non-organic component to his presentation… If the Court finds that the Claimant did not suffer a mild Wernicke's encephalopathy, but some other organic neurological pathology (as seems to be suggested as an alternative by the Defendant), it remains my professional opinion that, on the balance of probabilities, the cause of the Claimant's FNSD, was the knowledge of the admitted negligence which triggered a "sense of grievance" in the Claimant causing him to experience deep-seated feelings of resentment and bitterness."

141. Whilst everyone agreed that the current diagnosis was FND (or in his view a better term was the umbrella FNSD), his view was that the Claimant did not have that condition

prior to the admission in September 2016. He highlighted the difference in the Claimant's presentation in the first week of September, struggling with the physiotherapy sessions, having previously engaged, not wanting to have the session and by 7 September, pulling the covers over his head and starting to cry when the physiotherapist came to see him. He felt it likely that his knowledge acquired on about 1 or 2 September that there had been a wrongful failure to provide Pabrinex, leading to the risk of a serious condition, set off a complex process of rumination, dwelling, focus on symptoms and disconnect between the function of the brain and leading to functional symptoms not compatible with a medical condition.

142. Dr Achinivu was subject to an extensive and exacting cross-examination. He currently saw 2 or 3 patients per month, mainly referred by case managers for treatment. The bulk of his work was medico-legal, but that was against a background of more than 10 years of full-time clinical practice in the NHS and privately. In the NHS he did a functional clinic for 10 years, which included seeing patients with FNSD. He had wide clinical experience across the range of neuropsychiatric practice. He accepted that Prof. Carson had an academic role, but that had not been a route of interest for him. He considered the academic literature in the context of being a full-time clinician.

143. He was challenged robustly in relation to the letter he had produced subsequent to the JER. He was not seeking to pass the blame onto someone else or correct an error (save in relation to the date of discharge) but wanting to clarify what everyone agreed were complex matters having seen the Counter Schedule and considering that his views had been misunderstood. He wanted to clarify when the FNSD started as it depended on what one called the start, whether this was when the stressor started or when the condition started. He was using the terms from the DSM V criteria, where, at p.319 the clinician was directed to specify whether it was "*with psychological stressor or without*"

144. The disorder starts when there is a stressor present, which is the precipitating event. The same applied to PTSD where the start is when the patient is exposed to a significant stressor, that is the onset of the condition, whereas the symptoms may start manifesting later. In FNSD in the meantime, from the point of the stressor, there is rumination and physical symptoms start to manifest days later. FNSD fell into a broad spectrum of somatic conditions, where someone manifests physical symptoms in response to particular life stressors. Here it is useful to have the physiotherapist noting the interaction on 7 September, which was a step change from anything before, and the CT2 describes the Claimant feeling upset about everything. He did not accept that he was wrong that the condition started when there was a stressor rather than actual symptoms. It was fully established by 8 September, when one was dealing with overt FNSD. One could go back to 5 September when Mrs Dickinson noted new onset memory and sensory symptoms and say it started then. However one looked at it, it took a stressor to precipitate it, which led to an evolution of symptoms.

145. He was challenged on why the Claimant presented in this way at this time when he had always had stress, with lots of other factors such as his prior ill-health, his brother's imprisonment, problems with his benefits, his father in law's illness and death. Previously he had resorted to alcohol as his coping mechanism, but he had been detoxified and then, he learnt from the doctors that he had not been given Pabrinex and was either suffering WE or they believed he had WE (if he did not). WE was for the neurologists to diagnose, although Dr Achinivu had seen the condition in patients in its acute phase and when Wernicke Korsikov ("WK") had developed. He had also seen lots of patients with complex FNSD. Even though the Claimant was confused on readmission to hospital, he had sufficient awareness of what was going on to be subject to the stressor.

146. He did not accept that on 31 August the Claimant's symptoms were consistent with the DSM V diagnostic features for FNSD, as that highlighted that the symptoms needed not to be better explained by another medical disorder. The Claimant failed on diagnostic criteria (C). One has to rule out an organic cause before FNSD comes into play. It was better explained by WE. Whilst he accepted that the Claimant had significant pre-existing vulnerability, what he looks for is the vulnerability and the stressor, there needs to be a reason "*why now*".

147. It was put to him that he "*got it right*" in the JER when he said that FNSD was already present on 31 August or 1 September. He did not accept that this was what he said or at least meant. He clarified that he said (or meant) that the psychological trigger started at the point he became aware he had not been given Pabrinex; when he started to manifest symptoms is more difficult, but that was by 7 September and probably 5 September.

148. He was challenged on the sentence in DSM V (p.320) that whilst an assessment for stress and trauma is important, the diagnosis should not be withheld if none is found. Dr Achinivu said he had only seen one patient, and that was in his work in Nigeria, where he could not find a psychological stressor. He did not consider that Prof. Carson had looked for one, but he had.

149. He did not consider that any cognitive effects from alcohol misuse, which were very mild and non-negligent, would have affected his ability to work or live independently.

150. He accepted that in the JER (p.297) his comment about not robustly treating it, that should have referred to the first admission to hospital and it was a mistake to refer to the second one.

151. He considered that had the Claimant received appropriate treatment in hospital he would probably have continued to function in the way he functioned pre-incident. He might have improved somewhat had he reduced his alcohol intake, although the risk of relapse

was high. He might have been a bit better; how he was before was the worse-case scenario. He previously had a tendency to symptoms of pain and somatisation but he did not have a functional neurological disorder and there was not a basis to diagnose FNSD previously. He did not agree with Prof. Carson's view that there was FND going a long way back into the pre-admission history. In Dr Achinivu's view, the Claimant had somatic symptoms, focussed on pain. He did not have the motor weakness and frank disability that he manifested after the admissions, which was completely different. There may be a range of opinion about whether he had a somatic symptoms disorder previously, although he thought the symptoms came and went. There was a close temporal relationship between acquiring the knowledge of the failure to give Pabrinex and its consequences which led to the change, becoming FNSD. This was a completely different picture from what had come before. It was not merely a coincidence or a continuation or worsening of what had gone before.

152. Whilst he recognised the concept of the cumulative allostatic load (the wear and tear on the body) which leads to a worsening, that concept is much more complex than the way in which Prof. Carson described it and he did not consider it an explanation here, despite the various other stressors going on in the Claimant's life. For Dr Achinivu, the importance of the stressor was not what it was, but the impact on the person. Where 2 people are made redundant, one gets depressed and the other may use it as an opportunity. It is the appraisal of the stressor by the individual which is important, leading to rumination and anger. Otherwise, why was there not this same step change before? Here the timing could not just be put down to coincidence or 'the final straw'.

153. He did not agree that there was not a jot of evidence that the Claimant was aggrieved about not being prescribed Pabrinex or that this factor was not material in the development of worsening functional symptoms.

154. In re-examination, Dr Achinivu noted the Claimant's concerns on 6 September around his Pabrinex being stopped the previous night (p.1480). He confirmed that was one of the records he had in mind about the Claimant's concerns. That concern was also highlighted in the entry (p.1483-4) at 1520 on 7 September. On 7 September there was the reference to pulling the sheets over his head and starting to cry when the physiotherapist came and also the following day to wanting to die and the clinician is querying whether it may be a functional condition. It was clear at this point that the symptoms were evolving. It was quite clear that he was psychologically distressed on 7 and 8 September, at the time he was concerned about Pabrinex being stopped (again).

155. **Dr Symeon** gave neuropsychiatric evidence for the Claimant limited to the issue of C&P. For reasons of scheduling this was interposed between Dr Goulding and Dr Wills, although it is more logical to summarise his evidence at this stage of the judgment. His main work was in the NHS, alongside a private practice. There was a large measure of agreement between him and Prof. Carson on the seriousness of the Claimant's condition and prognosis. He had previously expressed some views on causation but it was made clear, which he readily accepted, that he was no longer being asked to deal with that issue.

156. He accepted that the Claimant's long-standing history of depression meant he was likely to have continued to have depressive episodes in any event. He had a major depressive disorder. The FND was overlaid on this.

157. He did not consider a private day patient programme to be practical for the Claimant and he would need inpatient rehabilitation. A willingness to engage had been expressed and that was key. Whilst the prognosis was poor and he would not expect significant changes, the Claimant's quality of life could be improved, resulting in more pleasurable activities, without him making significant improvements to his actual condition. It

would be unlikely to change his care needs. There was a chance of some improvement in his activities of daily living ("ADLs") but that was extremely slim.

158. He highlighted a mild to moderate risk of deterioration without treatment. There was the risk of deconditioning and atrophy when not moving. A primary risk was the risk to self, from suicidal thoughts and low mood. That came from both the depression and the FND. If the Claimant was his patient, he would want him to be somewhere where he had 24 hour input in his own property, not just for direct personal care, given the risks of accidental injury from trying to move or inappropriate transfers, but also for managing the risk of harming himself. The next step down from 24 hour care was care 4 times per day, which would leave too many gaps where the risks already identified would not be mitigated. The care needs were likely to stay the same.

159. **Prof. Carson** gave neuropsychiatric evidence for the Defendant on both causation and C&P. As noted already, it was evident that he had greater years of experience than Dr Achinivu and, in contrast to him, a much greater focus on academic research, alongside clinical work. He described in his evidence having received various prestigious awards in the UK and overseas, including the British Neuropsychiatric Society's medal in 2025. He had set up a Society with others for functional neurological disorders and had had dedicated training in alcohol use disorders under the tuition of world authorities in these conditions. He felt well qualified to comment on the issues across both WE and FND/FCD.

160. In summary, his opinions were that:

a. The Claimant did not have WE at any point but probably had had FND since around 2012.

b. If the Court found that the Claimant did have WE, then he would accept that the occurrence of that illness had the potential to trigger a worsening of his FND.

Any stressor on the cumulative allostatic load had the potential to cause a worsening. He was doubtful that it would have made much difference to the long-term outcome.

c. He did not consider that concerns of missing a dose of Pabrinex were the cause of a functional disorder and that a link could only be made if there was a true encephalopathy.

d. He did not consider that there was evidence of Thiamine deficiency, based on the body habitus and the Claimant's wife's description of his diet, but he would defer on that issue to neurologists.

161. In a probing cross-examination, he confirmed that he unequivocally deferred to neurologists on the concept of Thiamine deficiency. There was the separate question of the diagnosis of WK. It was generally accepted under the recognised criteria that for WE, there either needed to be marked dietary deficiency or two standard deviations under standard weight. It was very rare to get WK syndrome in the absence of those factors. WK and WE were used interchangeably in the medical literature. It was possible but rare to get WE if of normal body habitus. He accepted that this was why the NICE Guidelines said to give prophylaxis orally for problem drinkers who have a normal diet.

162. He was taken to his report at p.6/ lines 221-234 (p.264). He accepted that saying *"at the time of the accident*" was an error as this case was not about an accident. At the time the Claimant was 37 not 32 years as stated. It was not intended to be a verbatim note. When challenged on the basis that it was not the Claimant who was studying but his wife, his response was *"that is what she told me".* Asked to explain what the phrase *"foredge waste*" meant, his response was *"I have no idea what that is."* It was put to him that Mrs Dickinson denied using the phrase "*very muscled*" about the Claimant or that he

ate normally and had a good diet. He denied picking those points up from Dr Goulding's report. He did not accept that his report was so riddled with mistakes that one needed to be careful about relying on it. His response was that there would be no reason for him to make those words up and he cannot speak to their accuracy.

163. His account was that in his hospital in Edinburgh, a patient presenting with an acute presentation of potential WE would be seen by neurology or liaison psychiatry or neuropsychiatry. He would defer to neurology on ataxia and ophthalmia but altered mental state was within his domain and was present in 90% of cases.

164. He was doubtful that there was real confusion at the time, based on the rudimentary testing. It was difficult to have an encephalopathic state and produce those results. He accepted that in about 1 in 10 cases, WE could present without confusion. If he was right that there was not a confusional state, this was not incompatible with there being WE. Whilst he would defer on the diagnosis of ataxia, he had relevant experience to distinguish functional ataxia. It was relevant that at the time no one was highlighting that the Claimant had a CRPS, the impact that already having and its overlap with FND type symptoms.

165. His view was that if the treating clinicians had known about the CRPS they would have weighed up what they found on testing differently. He was taken to the entry at 01.20 on 2 September (p.1471) that did document CRPS. His response was that this was not necessarily put into the neurological examination, even though it was in the history. He accepted that the clinicians were not being called to give evidence to answer this.

166. It was suggested to him that a competent doctor, GP or acute medicine clinician should be competent to identify ataxia rather than a limp from CRPS or antalgic gait. His response was that research showed GPs do not do much better than chance. He accepted that an inability to walk in a straight line was a classic presentation of ataxia, not CRPS.

He was not disputing that some of these signs were somewhat typical of ataxia, the nuance was whether the ataxia is functional or neurological.

167. When asked by the Court, why it might be that if the clinicians were so bad at identifying it why they were getting it wrong in the same direction and why some were not saying it was functional. He was not seeking to say they were so bad at it but that it was a difficult skill to work it out. If they were thinking this might be WE, they started to see things through that lens.

168. He was challenged on p.19 of his report which appeared to suggest that all 3 of the triad had to be present to diagnose WE. He said he had been clear that the full triad is only present in 20% of cases, at about 90% of cases, confusion was the commonest symptom. He was also challenged on p.14 (p.803) of his report which only mentioned one test showing unsteadiness. He accepted that this was not the only evidence of ataxia on 1 and 2 September. When reminded of the phone call where Mrs Dickinson said about the Claimant being unable to walk in a straight line, he said he focussed more on the medical findings. He was challenged on his point in the JER (p.304) that one of the reasons there was not WE was that there was no evidence of pathology on imaging. He accepted the premise that this was a neutral point as to WE as in 50% of cases the imaging would be normal.

169. He was taken through the history of the diagnosis of CRPS initially being made, via its precursor condition RSD, in November 2011, by Orthopaedics (A4/114). But when seen by a Consultant in Pain Medicine, on 10 September 2012 (A4/127) the conclusion was *"I do not think he has CRPS…"* Whilst there is further mention of it in the GP records, that appeared to be the most recent authoritative view. He had read this aspect of the history rather differently as there were ongoing complaints and treatment which appeared to be attributed to CRPS and gait disturbance. It was put to him that what one cannot find here is clear evidence of a functional neurological disorder since 2012, as

he has suggested. Prof. Carson felt that there were ongoing references in the records which suggested a functional disorder. If one wanted to say it was not functional, then it is somatoform. It may not make a difference.

170. He accepted that there was a distinct change in presentation from 1 September onwards. Unquestionably the Claimant worsened and there was an ongoing deterioration to his current very disabled state. It was put to him that there was not just a change in severity but a change in the nature of the presentation. Having had unexplained or non-organic pain and an orthopaedic like presentation, within a few days from 1 September he had significant memory impairments, bizarre symptoms in the lower limbs rendering him reliant on a wheelchair. His view was that it was not a complete change in nature. To a lay person it would undoubtedly look different but to him, clinically, if he already had symptoms in that limb then it represents a worsening.

171. It was put to him that if the explanation for the step-change deterioration is an evolution of FND, it would have to be an entirely coincidental matter that it happens 6 days after the failure to administer Pabrinex. It was wrong to state it was entirely a coincidence as he had an awful lot going on in his life and for the FND to worsen was not a surprise. If he was wrong and Dr Goulding was correct that there was a WE, that would contribute to the allostatic load that might help produce a worsening. If he had WE it would be a contributory factor but it would not be the trigger.

172. He was challenged on the lack of evidence, such as anyone suggesting he smelt of alcohol, in records in relation to his theory (p.278) about the covert consumption of alcohol after the detoxification. He did not abandon the theory, but said he did not want to overemphasise the point. He did not know whether it had happened.

173. If there was a finding of WE, he accepted the potency psychologically of the Claimant finding out that he has a serious neurological condition and that he got it as the hospital

failed to give him the thing they should have given him. It would contribute but it did not negate all the other difficulties and it was not the sole factor which altered the trajectory.

174. **Ms Rachel Woolcomb** gave evidence on matters of care and occupational therapy, for the Claimant. She qualified as an OT in 1992 and held a post-graduate certificate in posture management of the severely disabled. She outlined that she had experience with patients with similarities to the Claimant. She started working in the community in 1999, with those with long term neurological disorders, and from that point onwards with those with complex needs, such as 24h care and also their rehabilitation. She left the NHS in 2017, since when she has been in independent practice. Within her normal day to day practice she continued to work with those with FND and also in the rehabilitation of those with complex needs.

175. She did not have the neuropsychiatric C&P JER at the time of preparing the care JER. There was nothing in that document or in the evidence of Dr Symeon, which she had been in Court to hear, that caused her to change her opinions. Without information on any care needs in any event, she had calculated the care needs in their entirety. She had since had a chance to review the evidence and DWP records. She noted that prior to the index events, Mrs Dickinson was making her husband's appointments, but was not supporting him to go to them. She would not necessarily describe that as care. She noted he was not driving at that stage so presumed if he went out, someone was taking him. Those were the two things she noted but did not feel the costings needed changing to reflect that. She explained the slight difference between her hourly rates for future care and those of Ms O'Connell for the Defendant. She explained why she felt Liberatum was more appropriate for the type of care she was recommending, as an agency used to providing complex care packages to those with physical and cognitive issues, at an aggregate rate of £31-32 per hour. She felt that Ms O'Connell's recommendation of

using Helping Hands was not appropriate for the care needed. That was more suitable for sessional care for short periods, providing personal and formulaic care. It was less used for complex care as the carers did not have that level of expertise and there would be less continuity of carers where the same person was unlikely to be attending on a repeat basis.

176. Ms Woolcomb was cross-examined skilfully on a number of points. She was probed about why she approached her task as assessing all of the Claimant's needs, given indications he already needed some assistance prior to the index events. She confirmed that this seemed appropriate on the information she had. She subsequently reviewed the additional information, such as the DWP records. The refinements to reflect Mrs Dickinson needing to sort out his appointments for him and the fact that the Claimant did not drive, were then factored in.

177. As for the assumptions she made around the condition the Claimant had, her current understanding was the WE was a short term issue and he went on to have FND. Her role was not to discuss causation. She knew he had low mood before the relevant events, but subsequently he had had suicidal ideation which she could not recall having seen evidence of him having before.

178. She confirmed she had taken account of the Claimant's complaint of having only 3 days of immediate working memory, although he could remember things further back. She noted that this was quite unusual, but that was the account she was given , in the context of a person with FND, so it was factored into the assessment. She noted in her report at p.28/4.1.4 (p.583), Dr Symeon's view that the Claimant's cognitive impairment is due to non-organic factors which have resulted in FND. She accepted that she had not specifically referenced other views, such as Dr Evans' view (p.485) or Dr Goulding's (p.354) that there were some disparities in his cognitive presentation, but she had read all of that evidence and taken it into account. Her experience of working with those with

FND was that their level of function can be changeable so the Claimant's presentation made absolute sense to her.

179. She did not accept the proposition that she had simply been taking everything that the Claimant told her at face value without looking at the medical evidence inconsistent with that. She recorded what the Claimant presented with. She understood that a non-organic presentation, such as FND, will mean that for example there will not be anything causing the eyes to struggle to track. When challenged about whether there was a distinction to be made between what the Claimant said and the actual true level of function, her assessment was that he could do some things and not others and, as Dr Symeon explained, this was very much in keeping with a FND diagnosis. She did not feel that the Claimant was holding things back, for instance, he told her he watched TV, despite his account of eye tracking problems.

180. She accepted that the state of health and needs of Mrs Dickinson, who had fibromyalgia, and her daughter, who had mental health issues, would be relevant to the assessment. She was asked about Mrs Dickinson's evidence that even when she told him she was tired, he said he did not want help. Ms Woolcomb felt that this should be seen in the context of this being sitting support. 6 hours over 2 sessions to sit with him in the same room to enable Mrs Dickinson and the children to go out. The Claimant was struggling to accept that, notably as the person was not always consistent and his difficulty trusting people. That was exacerbated in Covid as the care was cut to just two 2 hour visits per week.

181. She rejected the notion that she had not taken account of Mrs Dickinson's health challenges when assessing her input at around 50 hours/ week, which was more than a full-time job for a healthy person. She had borne that in mind and Mrs Dickinson has been providing care possibly to the detriment of her body which is failing her. She finds it exhausting, but did not accept that this ought to be reflected in a reduction in hours. It

was a true reflection of her care input. It was noteworthy that the local authority had been providing a sitting service, from which it followed that they had assessed him as needing 24 hour care or they would not have provided that service.

182. She was taken in some detail through the tasks involved and the time spent. In relation to meals, she had factored in that before these events, Mrs Dickinson had made a meal for him, her and the family. She had allowed for the extra time over this and the need not just to make things, but to encourage him to eat. At times he struggled to swallow and sometimes he could not use his hands. There was also quite a lot of ad hoc support for which she needed to be on hand, such as his need for regular repositioning. In including time for providing comfort and emotional support, she had considered what was already being provided before August 2016. There was support arranging appointments but nothing like the intensity of emotional support later required. As making appointments did not count as a care need, there was not a need to reduce the subsequent provision.

183. She was challenged on the notion that this was 24 hour care. After her exhausting day of providing care, Mrs Dickinson did have to sleep herself. What she was providing was supervisory care. If he called out, she was next to him and would provide care. That was not part and parcel of conjugal life. What was being described was dealing with a distressed husband at night, helping him to regulate his distress and tasks such as moving pillows. She believed that was care and was variable night on night. She had not seen evidence that this level of nighttime intervention was needed before the accident, albeit she accepted there were periods of time where the GP described him as struggling to sleep due to issues at that time. The adjustable bed he has only raises his head and knees. He needs manual pillow adjustments and he is sensitive to touch so asks for assistance, which is not a quick fix.

184. Although she accepted that the evidence indicated the Claimant had given up activities like going fishing, in 2012, she felt that a powerful wheelchair for uneven terrain was still reasonably required to facilitate beneficial activities out in the community. She had understood that he did not drive prior to these events, so that part of childcare he would not have been involved in, in any event. The fact that he needed to be driven might result in a need to adjust the care provision a bit as she had not taken anything off for that.

185. The allowance for dog care did not include dog walking. The Claimant previously did that but now the family let the dog out into the garden. She was challenged about the extent of bird care provided for. There were 100 birds and having seen them and with some experience of bird care and with her OT expertise, she felt the allowance made of 1.5 hours/day was correct, even to do the bare minimum. Prior to the events, the Claimant would work with bird breeders so the family were not doing as much as he did previously. The birds were no longer there when she returned in 2024.

186. In making the overall assessment, she was challenged about whether she had taken into account the medical opinion, DWP records and the extent of pre-existing health problems (and likely future ones). She had and mentioned Dr Symeon, as well as e.g. Dr Achinivu's opinion (p.391) that "*he would have continued to present... with symptoms of major depression, anxiety as well as chronic pain*". She also highlighted Dr Achinivu's opinion on the same page that on the balance of probabilities, but for the index events, "*the need for care and support would not have been required.*" In addition, she used her own experience of providing rehabilitation to those with FND. She maintained, when challenged, that he required a 24 hour regime, including night care, so that he was not solely dependent on his wife whose health is deteriorating. He was a man who was very disabled, highly dependent, vulnerable, needed help to move and had been mostly in bed for 9.5 years. In addition to his care for his basic needs, he requires access to rehabilitation to find purposeful activity outside his sleeping

environment. Her assessment was that he was not receiving or requiring care prior to these events. Although not working, he was taking the dog out, managing his birds, moving freely around the home and that did not translate into Mrs Dickinson giving her physical time to give him care. Previously decorating and DIY would have been possible but would probably have taken a long time and been painful.

187. Despite extensive probing, Ms Woolcomb did not believe that the Claimant previously required hands on care for his ADLs which he was doing independently. In response to questioning, she maintained her position that the equipment recommended, including the extra powerful wheelchair were reasonably required for the Claimant's needs. He was quite a big man, who had been in bed a long time, with very specific sensory needs, pressure issues and needing support for his posture and to cope with his weight. Based on his account of some visual acuity problems, she had added an attendant control so someone could manoeuvre him. Even taking any cognitive issues out of account, she would still recommend this wheelchair both for going out but also within the home if he was in the right environment. As for vehicle provision, whilst there is a wheelchair enabled vehicle through the Motability scheme, the latest budget had made some changes to that scheme introducing uncertainty about future provision.

188. In re-examination, she highlighted the difference between services and provision for care needs. She explained that references in the records to the pre-incident needs were examples of him experiencing symptoms when doing tasks, but did not indicate he needed care and assistance to complete those tasks. His current nighttime needs, whilst not all arose every night but could do so unpredictably, included repositioning if uncomfortable as he cannot move himself and he is highly sensitive to touch; adjusting pillows; providing support to manage his pain and distress upon waking in the night; tending to his sheath catheter if it comes off (and changing bedding) or changing the bag; bowel care as his bowel needs are irregular and he could not get out of bed

unassisted, to use the toilet. His needs were more than that which a sleeping night carer could do which was a brief disturbance up to twice per night. For more than that the carer would be expected to be paid as a waking carer.

189. She explained that the previous reluctance to engage with care probably reflected the fact that the current property is unsuitable for paid carers and to have a life at the same time; he was stuck upstairs at the time of her assessment although he was now downstairs. He has not got access to normal facilities or an area where care can be provided and he needs to be able to leave the property for outings. Previous carers had been paid to carry out domiciliary visits and sit and look at him, which is very hard to tolerate. What Ms Woolcomb was proposing was very different to that and based around single level accommodation and being able to go out. It would not work with sessional care in fixed time slots, which would not necessarily fit with when he needed or wanted to do things.

190. She maintained that case management was needed in the long-term, beyond the MDT period. Although a specialist care agency would have a clinical lead to do rosters, the Claimant's needs were complex and alongside that, there would need to be case management support in the process, to look at risk assessment and ensure he had the right equipment. There was not an extensive need, but she would not recommend having no case management.

191. From her studies and teaching in posture management, she felt that the powered wheelchair recommended by Ms O'Connell was not suitable for someone with complex needs.

192. **Ms O'Connell** gave evidence about care and assistance needs on behalf of the Defendant. She had a Master's degree in Occupational Therapy and had worked in the field of neurological rehabilitation for 20 years. This was both in the acute and

rehabilitation settings as well as community based care. She had worked with those with FND routinely both acutely and in the ongoing phase.

193. She agreed in cross examination that when producing her report and JER she had not had sight of the neuropsychiatrists' JER as to C&P, in which (p.1005), they agreed "*FND: His FND causes profound physical and cognitive limitations. He is largely bed bound, requires assistance with all transfers (using a sliding board), and is unable to walk. He has severe pain and sensory disturbance in his lower limbs, preventing weight bearing and even contact with bedsheets. He also has issues with bladder control, requiring a sheath.*"

194. She accepted that this was the core medical opinion when making recommendations to the Court on future care. Having seen that document, she confirmed that she maintained her recommendations in the JER. She recommended on a daily basis one 1hr visit and two 30 minute visits and in addition 9 hours/week to access the community. This was sufficient to meet his disabilities as noted by the neuropsychiatrists, in her view.

195. Looking at (p.936/ 5.2.13) in her report, she maintained the opinion in that to return her from carer to partner, the Claimant would need assistance with personal care and transfers via a commercial care package. She remained of the view expressed (p.937/5.2.18), that the Claimant should not be relying on Mrs Dickinson to meet his needs, but needs a commercial package of support indefinitely.

196. If the Court were to accept that the Claimant requires a 24 hour package of care, which is not her view, she would accept that Ms Woolcomb's costings are reasonable in that her hours of care and hourly rates recommended would be reasonable in that scenario.

197. She felt that provision via Helping Hands, for her recommended care package, would be reasonable for someone with FND. Such individuals can become care dependent. They should be provided with enabling care to avoid becoming too dependent on that

particular provider. She accepted that in a 24 hour care regime it would involve a team of 6 or perhaps 8 carers, which would mitigate the development of a dependency on one person.

198. One of the difficulties to date had been a lack of continuity in carers. But as the Claimant was not really accepting of any care, he did not have an opportunity to build a relationship with the carers.

199. She thought that with an FND there would be more fluctuation in the condition than dealing with a spinal or brain injury, albeit they all have fluctuations. The mental health aspect would also have greater volatility.

200. She did not accept that the Claimant had effectively been afforded the equivalent of a 24 hour package for the last 7 to 8 years, with someone present in the property for 24 hours and being in receipt of care for those 24 hours. She referred to Mrs Dickinson's account that on some days he did not get washed, there being different levels of care need.

201. She was taken to Dr Symeon's C&P report (p.438/6.23). She had heard his evidence in line with that saying that he required access to a carer 24 hours a day and was asked why she did not accept that. She felt that his recommendations were based on 3 needs. He is vulnerable and cannot leave the property in an emergency, he is at risk of accident harm in falling out of bed etc. and at risk of deliberate harm. Although a vulnerable individual, she pointed to the fact that he can use a phone. In his current accommodation he could not leave but could be alerted to emergency services in case he needed to leave urgently and technology could be provided for falls, such as an emergency pendant. The risk of accident harm did not include any history of pressure injuries; moisture lesions were very different from pressure injury. He could adjust his own position on the bed and push himself up. He can move on the bed with limited or no assistance and can pull

up his own shorts, so he is able to move within the confines of his bed. He had not fallen out of bed in the last 9 years. His transfers could be made more predictable. He gets out of bed to shower. He is not transferring every day, perhaps 2 or 3 times per week. With the establishment of a routine, these transfers could become more predictable. As for his bowel regime, with routine care provision he could establish a bowel regime one would hope. She accepted that the risk of self-harm was an argument in favour of a 24 hour package.

202. With his level of disability he was likely to get out of bed and sit in a recliner chair for short periods or access the community. That was why she was recommending an additional 9 hours per week. He has not been in a routine about that but this should be able to be established.

203. She was asked to consider circumstances where Mrs Dickinson was not there for a weekend. Would she expect him to wait to transfer until a carer was scheduled to arrive and has just 30 minutes for him. He would not have needed to do that before he had FND and did this not raise a concern about his autonomy? Her response was that she and Ms Woolcomb had taken two different approaches. One could ask what a carer would be doing whilst he was making a decision about whether or not to get out of bed. He was limited in his activity levels before, by establishing a routine, he would be given the opportunity to engage, without a carer being present at his home all the time. She did not accept that her proposals involved a rigid timetabled ability to access the community, without the flexibility to cater for fluctuations in mood and what he wants to do. She felt the additional 9 hours a week would allow for this. She was asked, what if the second half hour visit was at 7.30pm and he did not want to get into bed. She felt if he did that, it was his choice. She agreed that if he needed a bag changing or there was a spillage from e.g. the catheter , or he soiled himself, after the last of the 30 minute visits, he would have to wait until the following morning. It was put to her that there

was a long list of things he needed assistance for where the timing was not predictable. What about obtaining food and drinks or snacks; if he needed something fetching, he would have to wait for the next visit. When asked whether that was reasonable, she pointed to the pre-existing pain in his knees and the ad hoc need for assistance. It was put to her that his leg pain was never such that he was stuck and had to wait until someone could hand him something. She agreed that the provision of services, such as booking his appointments, was different from care and that there was no evidence of him previously needing assistance with personal care.

204. She pointed to multiple references in the notes to suicidal ideation pre-incident. She was challenged on where in the psychiatric opinion it indicated that he was likely to experience suicidal ideation in any event, let alone to the same extent as now. She said, *"I am not able to tell you without going back through it."*

205. As for case management, she relied on Mrs Dickinson taking some household management tasks, and some case management being needed to establish a suitable care regime, then Mrs Dickinson could then manage it as she would have managed things in any event. It was put to her that once one acknowledged that he cannot rely on his wife to provide the support, the Claimant is not in a position to liaise with a care agency, with other therapists and medical practitioners and he would need ongoing professional support. Her view was that this could be restricted to a contingency to cover changes in need.

206. She agreed that there was no evidence of him previously needing care at night. With a suitable regime, he did not need help re-positioning as she had observed him doing that himself. He would not need help with his bowels if in a bowel regime. If he was waiting distressed from pain at night, she felt this reflected his previous account to the DWP of pain waking him at night. When asked if this was to the same extent as now and needing

same level of reassurance from his wife, she felt this was likely to involve waking his wife in any event with them sharing a bed.

207. As for the powered wheelchair, she accepted her recommendation was at the bottom end of the range. She recognised that he might need the one recommended by Ms Woolcomb upon assessment but she did not believe he would. She agreed it would give him more flexibility as to what he could do outside the home. But before his activities involved mainly walking the dog, he was not walking up mountains or going to the beach. She felt her riser recliner chair was sufficient. Ms Woolcomb's was a version of what he already had but apparently, he had not used it for 2 to 3 years.

208. She was challenged on various aspects of her past care provision. She did not accept that her assessment was a very significant underestimate of what his wife actually provided. It reflected a discount for his care needs in any event.

209. In re-examination she explained that to justify a 24 hour regime, the Claimant would have to have utterly unpredictable care needs across the 24 hours needing to be attended to there and then. This would mean he never had any time on his own, such as engaging in 3D printing or watching a movie. It would be to accept that he needs supervision across the 24 hour period to maintain safety and emotional well-being. It would be better for the Claimant to be together with his wife and family without someone else in the property all the time. It would be of benefit to his condition. The consensus of people working with those with FND is that the focus should be reablement.

V. LEGAL FRAMEWORK

210. Given the admission of breach of duty, the focus here is on whether the Claimant has satisfied the burden of proof upon him to prove causation, on the balance of probabilities. If so, questions of the quantum of damages then arise.

211. The causation question is resolved by considering the experts' opinions as well as making findings drawing on the contemporaneous records and witness evidence.

212. It is open to the Court to draw inferences as to causation, where appropriate. In *Jones v Great Western Railway Co.* (1930) 47 TLR 39, at 45, Lord MacMillan,

> "*The dividing line between conjecture and inference is often a very difficult one to draw. A conjecture may be plausible, but it is of no legal value, for its essence is that it is a mere guess. An inference in the legal sense, on the other hand, is a deduction from the evidence, and if it is a reasonable deduction it may have the validity of legal proof. The attribution of an occurrence to a case is, I take it, always a matter of inference. The cogency of a legal inference of causation may vary in degree between practical certainty and reasonable probability. Where the coincidence of cause and effect is not a matter of actual observation there is necessarily a hiatus in the direct evidence, but this may be legitimately bridged by an inference from the facts actually observed and proved.*"

213. In *Bailey v MOD* [2009] 1 WLR 1052; [2008] EWCA Civ 883, Waller LJ neatly summarised the relevant legal test for causation:

> "*In my view one cannot draw a distinction between medical negligence cases and others. I would summarise the position in relation to cumulative cause cases as follows. If the evidence demonstrates on a balance of probabilities that the injury would have occurred as a result of the non-tortious cause or causes in any event, the claimant will have failed to establish that the tortious cause contributed. Hotson's case exemplifies the situation. If the evidence demonstrates that "but for" the contribution of the tortious*

cause the injury probably would not have occurred, the Claimant will (obviously) have discharged the burden. In a case where medical science cannot establish the probability that "but for" an act of negligence the injury would not have happened but can establish that the contribution of the negligent cause was more than negligible, the "but for" test is modified, and the claimant will succeed."

214. The Defendant helpfully reminded me of the useful encapsulation of the standard "but for" causation test, which it is agreed applies here, with the burden of proof naturally upon the Claimant, as described by Lord Brown-Wilkinson in *Bolitho Appellant v City and Hackney Health Authority Respondents* [1998] AC 232*,* where at [239-240] he explained:

"*Where, as in the present case, a breach of a duty of care is proved or admitted, the burden still lies on the plaintiff to prove that such breach caused the injury suffered: Bonnington Castings Ltd. v. Wardlaw [1956] A.C. 613 ; Wilsher v. Essex Area Health Authority [1988] A.C. 1074 . In all cases the primary question is one of fact: did the wrongful act cause the injury?*"

215. The "but for" test of causation is also pithily described in a short passage from *Clerk & Lindsell on Torts 24th Edition* at [2-09]:

"*The first step in establishing causation is to eliminate irrelevant factors, and this is the purpose of the "but for" test. This test asks: would the damage of which the claimant complains have occurred "but for" the negligence (or other wrongdoing) of the defendant? If the damage would have occurred in any event the defendant's conduct is not a "but for" cause.*"

216. Neither party thought it likely that the Court would need to determine the case drawing on principles of material contribution. As I have not needed to resort to that approach, I have not set out the related legal principles in this judgment.

217. As for adverse inferences which the Court might draw from the absence of a witness, the Defendant reminded me of the formulation by Lord Leggatt in *Efobi v Royal Mail Group Ltd* [2021] UKSC 33; [2021] 1 WLR. 3863. I draw from this that the assessment of whether to draw an inference, or not, is particularly context and circumstance specific rather than being subject to specific rules. Lord Leggatt observed at [41]:

> "*41. The question whether an adverse inference may be drawn from the absence of a witness is sometimes treated as a matter governed by legal criteria, for which the decision of the Court of Appeal in Wisniewski v Central Manchester Health Authority [1998] PIQR P324 is often cited as authority. Without intending to disparage the sensible statements made in that case, I think there is a risk of making overly legal and technical what really is or ought to be just a matter of ordinary rationality. So far as possible, tribunals should be free to draw, or to decline to draw, inferences from the facts of the case before them using their common sense without the need to consult law books when doing so. Whether any positive significance should be attached to the fact that a person has not given evidence depends entirely on the context and particular circumstances. Relevant considerations will naturally include such matters as whether the witness was available to give evidence, what relevant evidence it is reasonable to expect that the witness would have been able to give, what other relevant evidence there was bearing on the point(s) on which the witness could potentially have given relevant evidence, and the significance of those points in the context of the case as a whole. All these matters are inter-related and how these and any other relevant considerations should be assessed cannot be encapsulated in a set of legal rules.*"

218. Where liability is established, the basis for the assessment of damages that the Court will make, is that set out by Lord Woolf MR in *Heil v Rankin* [2001] 2 QB 272:

> *"The principle is that 'full compensation' should be provided… this principle of 'full compensation' applies to pecuniary and non-pecuniary damages alike…. the compensation must remain fair, reasonable and just. Fair compensation for the injured person. The level must also not result in injustice to the Defendant, and it must not be out of accord with what society as a whole would perceive as being reasonable."*

219. The Claimant is entitled to damages to meet his reasonable needs arising from such negligently caused injuries as he establishes. Reasonableness applies to both the nature and the amount of the head of loss claimed.

220. Reasonableness in these contexts was considered by Swift J in *Whiten v St George's Healthcare NHS Trust* [2011] EWHC 2066 (QB):

> *"In considering what is "reasonable", I have had regard to all the relevant circumstances, including the requirement for proportionality as between the cost to the defendant of any individual item and the extent of the benefit which would be derived by the claimant from that item."*

VI. ANALYSIS AND FINDINGS ON LIABILITY

221. I start this analysis by clearing the decks of two points.

222. The Defendant pressed the Court to draw an adverse inference from the fact that the Claimant, a man who does not lack mental capacity, had not provided a witness statement or given evidence in his own claim, although his account was recorded in notes and expert reports. His wife's evidence was that he had recall of events leading up to his admission, even if not after that and even if his shorter term memory was now

said to be limited to a few days. The thrust of the Defendant's case was that the Claimant could give relevant evidence and answer for what he had told the experts and the Court should infer that he had chosen tactically not to do so but to leave his wife and mother to be the sole witnesses.

223. To some extent, that submission did not come without jeopardy for the Defendant. On its own side, none of the treating clinicians, all employed by the NHS[4], involved in the Claimant's care gave statements or oral evidence. This was notwithstanding the scrutiny of the experts focussed on those clinicians' contemporaneous records and the scepticism expressed by the Defendant's experts about some of their findings.

224. Having considered the guidance in *Efobi,* that whether to exercise my discretion to draw inferences depends entirely on the context and particular circumstances, I have little doubt that I should not, as against either the Claimant or the Defendant.

225. Whilst adjustments could have been made to accommodate the Claimant giving evidence, it is uncontroversial between the parties to say that he is clearly vulnerable. He is currently largely bed bound, severely disabled, with fragile mental health and at risk of self-harm. Calling him to give evidence, even remotely, would have been logistically challenging and might well have been destabilising and harmful. I did not find the decision for him not to give evidence particularly surprising. His wife was present at the relevant events and was well placed to give first-hand evidence. There are detailed records for that period. If his absence has had an effect, it is more to complicate the presentation of his case than provide some tactical or evidential advantage.

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[4] All clinicians with material involvement in the Claimant's care were employed by the NHS. Those involved in the admission prior to discharge on 31 August were employed by the Defendant Trust. Those involved in the admission starting on 1 September were not employed by this Defendant but by a different NHS Trust.

226. In the Claimant's closing submissions, Mr Pitchers' focus was, rightly in my view, on resisting any suggestion of an adverse inference being drawn against the Claimant rather than pressing for one to be made against the Defendant, although he had floated that question in his opening note. The point he did make was that given the Claimant was happy to rely on all the contemporaneous records, it made it difficult for the Defendant to invite the Court not to accept those records when it was not calling the NHS clinicians who created them. For completeness, I also conclude that I should not draw adverse inferences against the Defendant for the NHS clinicians' absence. I will consider further later in this analysis whether I accept the Defendant's experts' scepticism about the content of some of the records, but that is not a question of adverse inferences.

227. The Defendant's experts, especially Dr Wills, focussed on a supposed account by Mrs Dickinson that during the garden incident at home on 31 August 2016, the Claimant had experienced a loss of sense of self. Their recollection was that, in addition to her statement saying (p.144/21) that he did not know where he was, she had said in oral evidence amongst other things "*he did not know who he was*". That was important additional detail for them, for instance Dr Wills opining *"if you do not know who you are, that is non-organic, as even severe dementia patients retain a sense of self."* i.e. this was an indication of a functional presentation at that stage. It was also totally inconsistent with a recovery to a much milder state of confusion by the re-admission on 1 September and the review at 0650 on 2 September, in Dr Wills' view. However, as I outlined later in the trial, that was not my note of her evidence, which I have recorded at [51(d) & 63] in this judgment. Her account was that he did not know who *she* was and *where* he was, not who *he* was. So this was a non-point which the Defendant's experts mistakenly latched onto in my view. It is noteworthy that Dr Wills conceded that the confusion in the garden could have been an indication of WE. The extreme

nature of the loss of sense of self was a key component in his view that it was on balance functional.

228. Another step in unlocking the outcome in this case is to determine both approximately how much alcohol the Claimant was consuming in the lead up to 26 August 2016 admission and also his diet and nutritional state. At first blush, the Defendant successfully opened up a rift between Mrs Dickinson's evidence and the account Dr Goulding recorded in his report that "*she made sure he had a good lunch and an evening meal each day.*" Dr Goulding was adamant that was what she told him. However, in her oral evidence, Mrs Dickinson felt Dr Goulding was confused about that. The Defendant understandably cross-examined Mrs Dickinson on the basis that her recollection when she met Dr Goulding in 2020 was probably better than when giving evidence in 2026. That would normally be a powerful basis to question her account at trial that, (a) the Claimant did not normally eat lunch; and (b) that she was absent from the home for around 2 weeks prior to and 3 weeks after the death of her father, on 26 July, caring for him and clearing his flat, hence she was not preparing meals for the Claimant. See this judgment at [54-55 & 59].

229. Her response in cross-examination that her statement was a summary and did not deal with every detail, might well not have been convincing in other circumstances. That is even though I found her to be a straightforward witness aiming to help the Court in so far as she could (or could not, as the case may be) remember the details. I reject the suggestion she was deliberately trying to re-frame her evidence to fit the Claimant's case.

230. However, in my judgement, Mrs Dickinson's clarification is in fact much more consistent with the contemporaneous records than might be thought, based on the account she gave to Dr Goulding in 2020. In Mrs Dickinson's oral evidence, she clarified her statement to comment on a progress review with the alcohol liaison nurse, dated 18

August 2016. That contextualised the apparent reduction in alcohol consumption to 7 shandies a day and three cans, after the North Tyneside Recovery Partnership ("NTRP") treatment programme started at the start of June 2016, noting the Claimant's account "*…however on 18/7 his partner had to leave the home to care for her father who sadly passed away a few weeks ago. Since then Liam increased back to 20 cans daily. He stated that it was due to no one being there to help him or tell him not to.*"

231. In my view, that is a key entry in the contemporaneous records and was also the last reference to the level of drinking before the admission in August. It came before the events in question and any breach of duty, let alone realisation that there had been a breach. So it could not have been a merely self-serving account. Notably, it chimes with the main thrust of Mrs Dickinson's account at trial. That entry helps me to resolve the confusion around this point.

232. I find as a fact that, on the balance of probabilities, when Mrs Dickinson was present in the family home, she probably did see to it that the Claimant ate at least one proper meal a day. However, that was not the position when she was absent in the second half of July and the early weeks of August, prior to the admission to hospital. In that period, she was absent for extended periods, even if it is perhaps likely she went back to see her children and the Claimant, perhaps overnight on occasions. I find that two things happened during that period. First, given his upset about his father-in-law's terminal illness and death and the absence of his wife, the Claimant's drinking, which had reduced following the ultimatum and him starting on the reduction programme, increased back to at or around 20 cans (so 40 units) per day. Second, as Mrs Dickinson was not there for extended periods, certainly during the daytime, she was not preparing good meals and making sure he ate well. Hence whilst he would have been eating something, alongside his heavy drinking, he was not eating during that period "a good lunch and an evening meal." That is consistent with the contemporaneous entry on 18

August describing there being "*no one there to help him*". That would have applied both to helping him to maintain his reduced drinking but also someone to ensure he ate well.

233. Dr Wills was adamant about the overwhelming importance of nutrition and Dr Goulding accepted it was important, alongside the four factors thereafter of absorption, storage, transit and metabolic demand. In my judgement, the combined impact of diminished nutrition and a return to maximal alcohol consumption (consuming lager, which was rich in carbohydrates and had no Thiamine, both of which impacted the levels of Thiamine) from mid-July onwards, left the Claimant's stores of Thiamine depleted, alongside him being at risk of malnourishment, by the time of the admission on 26 August. It follows that this left him at risk of developing WE quite quickly, in the absence of receiving prophylactic Pabrinex. This is also consistent with the Sechi paper, with which I note that Dr Wills agreed, see this judgment at [118], "*Unbalanced nutrition: Because the body's reserves of thiamine are sufficient for up to 18 days, in a healthy individual, any condition of unbalanced nutrition that lasts 2-3 weeks may lead to WE. In individuals with marginal stores of thiamine, the disorder may occur earlier, particularly if the diet has been very rich in carbohydrates.*" I also note that, even if prior to Mrs Dickinson's absence, the Claimant had normal reserves of Thiamine, which I am doubtful about given his significant (albeit somewhat reduced) drinking and gastro-intestinal symptoms, even if his diet was better at that point, her absence and the interruption to his nutrition from mid-July onwards likely resulted in the depletion of the Thiamine stores, putting him at risk of WE.

234. Returning to the expert evidence, in a complex case with extensive records, it was not that surprising that all of the experts had made one or two errors. They had also overlooked or failed to mention entries which the other party thought significant. That applied to Dr Goulding who accepted he had not noted the GP entry for 1 June 2016

which it turned out the Dr Wills had also not recorded in his report, see [104]. What was instructive for me was the contrast between how they dealt with those matters.

235. Dr Achinivu made a date error in the JER (p.301/9a) when he recorded the date of discharge as 30 August, rather than 31 August 2016. He accepted that he did not routinely provide letters of clarification after the JER, but he did in this case and it was dated 23 January 2026. By the time of trial, the parties had agreed it should go into evidence. He said he wanted to correct the error in the date of discharge. He said in the letter that the Counter Schedule "*appears to misrepresent my views*" about when the Claimant started to suffer FNSD. That was probably a poor choice of words which implied blame. When pressed in cross-examination he said that it was a misunderstanding of his view and he wanted to clarify what he had said in the JER to help the Court to understand the progression of this complex condition and explain his view on when it started. That was a sensible concession, properly made, recognising that these were complex matters, but that it appeared his views had been misunderstood and needed clarification. It would have been better if he had recognised this from the outset, but that he did ultimately do so in his oral evidence was to his credit.

236. By contrast, whilst recognising their eminence and experience as clinicians and expert witnesses, in this particular case I found the way that Prof. Carson and Dr Wills dealt with errors and omissions in their reports and evidence less satisfactory.

237. I felt that Prof. Carson had been uncharacteristically less careful than my previous experience of him, in the preparation and presentation of his evidence in this case. I refer to [162] in this judgment. There were the odd references to *"at the time of the accident*", which a proof read would have identified as an error. At one point Prof. Carson tried to refer back to an accidental fall in the medical history, when the reference was clearly to the index admissions in August and September 2016, which had nothing to do with an accident. Rather than accepting that he had probably misunderstood who

was studying and that it was the Claimant's wife, not the Claimant, his response was *"that is what she told me"*. Asked to explain (p.264/p.6 of the report) what the phrase *"foredge waste*" meant, his response was *"I have no idea what that is."* I found it curious that his response was not to apologise for the error or recognise it as such, but to express his lack of understanding of it. Almost as if it was something in a document which he had not created, let alone put before the Court.

238. Dr Wills accepted some sequencing errors and a lack of detail on important parts of the chronology in his report, see for instance [129] in this judgment. At (p.252), the entry about the Claimant being given IV Pabrinex was out of chronological sequence, after the 2 September ward round, whereas it was started at 0020. He was challenged about the brevity of his review of the period from the ward round through to his discharge on 14 September, this being dealt with in a few short phrases. He accepted that he may have had some loss of focus there. I agreed with the Claimant's observation, put to him, that in his report he had moved very quickly from the conclusion about normal nutrition to concluding that the Claimant did not have WE. He accepted that he was not forensic enough in his analysis at the beginning. His response was that he since had had a lot of information and his opinion remained the same. I felt that this lack of rigour in his analysis of the key parts of the chronology was a lacuna in his evidence which deserved greater explanation, rather than a readiness simply to stand behind the confident assertion that nothing he had since seen changed his view. This glossing over of detail and high degree of confidence in his conclusions, notwithstanding a lack of attention to detail, did not sit well with his readiness to undermine the skills and conclusions of the treating clinicians, especially those who were more junior in their careers (see further below).

239. What was surprising was less the making of errors and omissions themselves, as we all can and do make errors and omissions, but the reaction to them being pointed out. This

gave an impression of loftiness on the part of the Defendant's experts which gave me pause for thought about the cogency of their overall opinions.

240. Two other aspects of Dr Wills' evidence which I did not find impressive were, (a) the way he introduced for the first time in his oral evidence-in-chief the audit he said he had carried out in his hospital of WE cases; and (b) the calculation of how long the Claimant's reserves of Thiamine would have lasted, which he introduced for the first time in re-examination. Unlike Dr Achinivu, he did not seek to deal with points of clarification, correction or supplemental opinion by a follow-up letter prior to trial, he left it to his oral evidence. However, he knew about the audit prior to finalising his report and dealing with the JER, but he did not mention it. I found his explanation that he did not think the case would go to trial, so had not mentioned the point sooner, difficult to understand and an insufficient explanation. As I recently observed, albeit in different and much more troubling circumstances than apply here, at [78] in *Mew v The General Dental Council* [2026] EWHC 1116 (Admin) it is not open to an expert consciously to hold back matters relevant to their opinion until they give oral evidence. Such a matter should be set out in their report or, if it arises later, included in a promptly prepared and disclosed supplemental report or letter.

241. The introduction in re-examination, of his calculation of how long the Claimant's stores of Thiamine would have lasted, had the air of an after-thought about it. One might be tempted to say colloquially, that this had a whiff of a '*back of a fag packet calculation*' about it. The way it was introduced, at that stage and without being put to Dr Goulding struck me as giving rise to likely unfairness to the Claimant. I felt that if important and helpful, it could and should have been introduced sooner in the case or at the least earlier in the trial. Similar concerns arose from mention of the audit for the first time in his evidence-in-chief. I have not put any weight on either the audit or the calculation for those reasons.

242. Prof. Carson raised in his report his theory that there might have been covert consumption of alcohol (p.278). That seemed to be based more on his general experience of the regularity of relapse than any sound evidential foundation in the case, beyond a question being asked in a single entry in the records, much later in the chronology on 12 September (p.1490) and which was never corroborated. There was no suggestion that this could explain earlier symptoms, such as anyone smelling alcohol on the Claimant's breath upon examination, whereas there was plenty of evidence of abstinence being maintained from the August admission onwards through the relevant events. Whilst he did recognise the limitations of the theory, given the lack of evidence, one might have expected him to concede in oral evidence that the Court could not realistically conclude on the balance of probabilities that this was the explanation for what was wrong during the admission on 1 September. He did not do so.

243. I also found Dr Wills and Prof. Carson's attitude to the contemporaneous notes and conclusions of the treating clinicians unconvincing. There was a sense that their eminence led them to two unconvincing positions. First, they had what came across as an excess of confidence in their ability retrospectively to second-guess the contemporaneous notes, when they did not fit with their theory on diagnosis and causation. During his evidence, I asked Prof. Carson, as noted at [167] in this judgment, why if the treating clinicians were "*so bad at [identifying ataxia]*" they were all getting it wrong in the same direction and why some were not saying it was functional, on re-admission and in the early days of September. He clarified that he was not seeking to say they were so bad at it (which see below had been Dr Wills' view) but that it was a difficult skill to work it out. If they were thinking this might be WE, they started to see things through that lens.

244. Second, at times they also displayed an unfortunate and undue scepticism about the competence of the treating clinicians at all levels of seniority, none of whom were in

Court to comment. Even the treating Consultant, Dr Bennett (p.1473) who conducted the post-take ward round on 2 September, did not escape this approach. Dr Wills sweepingly observed, as discussed at [122] in this judgment, that "*most acute physicians are pretty bad at neurolog*y", although he had "*no knowledge of Dr Bennett*". I also refer for instance to this judgment at [94; 126; 128; 131; 166-167].

245. For Dr Wills, this attitude apparently extended to the authors of one of the academic papers cited, see [135] in this judgment. The way he dealt with the status of that paper, published in the *Lancet Neurology*, had more than a hint of academic *de haut en bas* about it which, coming from him, was as uncharacteristic as it was unfortunate.

246. There was also another relevant theme in some of the Defendant's expert evidence of undercutting the admission of breach of duty. Entirely properly in my view, there was no attempt made prior to or at trial to withdraw that concession. But there was a sense at times that the Defendant's experts were not entirely comfortable with or accepting of it and/or that the Defendant itself perhaps regretted it. I refer for instance to [117] in this judgment and Dr Wills' observation that he did not know who made the decision to give the Claimant Pabrinex and there were no witness statements from any clinicians, but he doubted that an ENT doctor was best placed to make those judgments. The implication was that if he had been the treating clinician, he would not have prescribed it. Whilst he was driven to accept that it was in accordance with the NICE Guidelines to do so, this was general guidance in his view and he gave the impression of thinking that it was defensive medicine and/or not really necessary in this case.

247. The audit he belatedly referred to appeared to be grounded in his belief that Pabrinex was overprescribed. He mentioned that as Pabrinex was becoming more difficult to obtain from the manufacturer, this seemed a reason to take stock and get resident doctors to be a bit more alert to the symptoms and signs of WE, by looking at those who had Pabrinex and did not go on to develop WE. I refer also to [81] and the cross-examination

of Dr Goulding. He was challenged about the NICE guidelines, given the mere fact that the Claimant was an alcoholic admitted with an acute illness did not require parenteral Thiamine unless he was malnourished or at risk of that or had decompensated liver disease. Dr Goulding pointed out that the treating team must have thought the Claimant was at risk as they prescribed IV Thiamine. He also questioned, reasonably, why breach of duty was admitted otherwise. He did not accept that Pabrinex was widely overprescribed and thought the opposite was true.

248. Prof. Carson's firm view was that the Claimant had had FND since long before the index events, dating its onset to 2012. Whilst not exclusively on this basis, it was largely predicated upon (a) the diagnosis of CRPS in 2012 and references to that condition and related symptoms since then in the medical records; and (b) his view that the treating clinicians in 2016 had not known about the pre-existing functional condition so had mistaken a functional presentation for WE. The difficulty I have accepting those opinions arises from the records which call into question those conclusions. As noted at [169], Prof. Carson was taken through the history in cross-examination. The diagnosis of CRPS was initially made, via its precursor condition Reflex Sympathetic Dystrophy ("RSD"), in November 2011, in the Orthopaedic clinic. But when seen by a Consultant in Pain Medicine on 10 September 2012, who would be better placed to make the diagnosis, the conclusion was *"I do not think he has CRPS…"* Whilst there was further mention of it in the GP records, perhaps also overlooking the Pain Consultant's view, the 10 September 2012 opinion that this was not CRPS appeared to be the most recent authoritative view. Whilst Prof. Carson had included that pain medicine reference in his records review in his report (p.273), I was not convinced that he had taken it into account. I did not find his explanation for why, notwithstanding that, and despite not having seen the Claimant prior to the index events, he was able to second guess the Consultant in Pain Medicine.

249. On point (b), his view was that if the treating clinicians had known about the CRPS they would have weighed up what they found on testing differently. He was taken to the entry at 01.20 on 2 September (p.1471) that did document CRPS in the history, contrary to his assertion. His response was that this was not necessarily factored into the neurological examination, even though it was in the history. He accepted that the clinicians were not being called to give evidence to answer this. Again, I felt this was part of the tendency to doubt the treating clinicians and second guess what they did and their conclusions. I consider that much more weight can and should have been placed on the consistent picture which emerges from those contemporaneous records.

250. I prefer Dr Goulding's more plausible opinion, as discussed at [83]. He rejected the notion that the symptoms the Claimant presented with could, on re-admission, be functional. A functional presentation was normally of a very striking disability not in keeping with the other facets of the presentation. It was very unusual for a functional disorder to be a very mild disorder the doctors struggled to find. The 1 September presentation was not at all like a functional presentation. Nor did such a condition pre-date the index events. The physical symptoms from the earlier part of 2016 were consistent with knee pain. They did not include unsteadiness on standing which was the heel-toe ataxia noted in hospital. The same applied to the much earlier records going back to 2011/12, which were of a wide-based gait in the context of stiff legs. In 2015, there was an antalgic gait (a limp), not ataxia which is quite different and, in Dr Goulding's view, a medical student could tell the difference. Dr Achinivu was also of the view that whilst there was a tendency to somatise prior to the index events, there was no functional condition until the onset of FNSD in September 2016. I find the analysis of Dr Goulding and Dr Achinivu more plausible and I prefer it.

251. I found Dr Goulding's description of the impact of very heavy drinking and (where relevant to the factors) the facial abscess and surgery, on the key four factors after input,

namely absorption, storage, transit and metabolic demand to be compelling and logical. For the reasons set out above, the approach Dr Wills chose to take to a number of issues undermined my confidence in him in this particular case, notwithstanding that he is an undoubtedly eminent and experienced expert in this field. This made me cautious in adopting his views more widely. As noted below, where they differed, I prefer the views of Dr Goulding.

252. The factual conclusions I have reached are also crucial to my findings on the key questions in this claim. Those, combined with my analysis of the expert evidence, cause me to prefer the conclusions of Dr Goulding and Dr Achinivu, where they differ from Dr Wills and Prof. Carson.

253. In my judgement, the repeated examination findings, coupled with what Mrs Dickinson was reporting, were consistent with symptoms of WE. The consistency with which the treating clinicians at a range of different levels of seniority, from the most junior to Consultant level, were mentioning only WE as the working diagnosis was a compelling picture.

254. I reject the Defendant's suggestion, through its experts and in closing, that Mrs Dickinson's research overnight on 31 August into 1 September, "*contaminated the thinking of the clinicians who assessed the Claimant afterwards.*" Mrs Dickinson's riposte when this was put to her in cross-examination was understandably pithy, "*My research saved his life as if he had not been given Thiamine, he would not be here now*." There was also a sense from the Defendant's experts that they felt Mrs Dickinson had sowed the seed with the clinicians from the morning of 1 September onwards and this explained their focus on WE. See [94] in Dr Wills' evidence, "*he was readmitted with so-called WE*". Similarly, Dr Wills and Prof. Carson suggested that it was hardly surprising that the clinicians who were directed to think about Thiamine deficiency and WE, interpreted some of the signs on 1 and 2 September as ataxia. Again, I consider

that all of this does less than justice to the treating clinicians. I do not accept that they did or would simply pick up and run with a diagnosis of a condition mentioned by a non-medically qualified relative of their patient or find ataxia from their own examination, where it was not present, simply because of being told about prior symptoms they had not witnessed. The knowledge that Pabrinex had not been properly administered, coupled with the reported and observed symptoms and the examination findings recorded, provided a logical underpinning for the consensus amongst all the clinicians who saw him upon re-admission that the only working diagnosis was WE.

255. I also reject the implication that this was a completely mistaken characterisation of what were actually functional symptoms. In my judgement, the functional symptoms only emerged later after the WE receded, namely from 5 September and were well established by 7 and 8 September. This picture from the contemporaneous clinicians' notes, chiming as it did with the views of Dr Goulding on this point, which I accept, leads me to conclude, on the balance of probabilities, that the Claimant did develop WE on 31 August through into 1 September. Given this was relatively mild, albeit that is not to diminish the seriousness of the condition, I find that it responded quickly to the rapid administration of high doses of IV Pabrinex, which the literature confirmed can – and did here I find- rapidly reverse the condition and symptoms. I accept Dr Achinivu's opinion that the picture then became one which evolved into a symptomatic and deteriorating FNSD. It had its genesis in the significant psychological stressor for the Claimant of discovering on 1 or 2 September that he had developed a serious neurological disorder, with potentially grave and permanent consequences, by reason of the Defendant's failure to administer Pabrinex. This was not a pre-existing condition which became worse or some implausibly coincidental (but entirely unrelated) new onset functional condition. In my judgement, there is a clear thread of cause and effect

to be followed through the records in this period, backed by the Claimant's experts' opinions.

256. I do not accept Prof Carson's theory that this was merely another part of an increasing allostatic load, leading the Claimant inexorably to the same nature and degree of functional disability. The Claimant had been the subject of multiple different life events in the past and had consistently dealt with them by resorting to consuming alcohol and, at times, somatised symptoms manifesting themselves in unexplained pain, rather than the florid functional symptoms he subsequently displayed. Challenges around his benefits were something the Claimant had experienced for a while. The death of his father-in-law undoubtedly affected him but, again, he retreated into his usual dysfunctional coping mechanism of excessive drinking prior to the admission and detoxification. Likewise the imprisonment of his brother was noted on 1 June, by the GP, so was not a new factor; despite it he had managed to cut down his drinking for a month or more. By early September 2016, he had had a medically assisted alcohol withdrawal, which would have left him vulnerable and without the crutch of self-medicating with alcohol. The reference to a safeguarding referral, by reason of his alcohol use, was quickly resolved with no further action. Having rejected the theory that there was pre-existing FNSD, I do not accept that the WE episode, his fear and realisation that it was negligently caused, was purely coincidental in the onset of FNSD. I prefer Dr Achinivu's opinion that it was the cause, in the context of the psychological stressor he identifies.

257. Drawing the strands together, I accept that the issues in the Claimant's closing note appropriately encompass the questions I should consider, in reaching my findings. I confirm that I have placed the burden of proof on these matters on the Claimant and the standard is the balance of probabilities. For the reasons discussed in this judgment, I answer those questions and make findings as follows:

a. **What was the Claimant's likely alcohol consumption prior to the admission on 26 August 2016?** This had on balance gone back up to approximately 20 cans of lager per day or 40 units, consistent with the last contemporaneous entry in the records prior to that admission, on 18 August 2016. The expert consensus was that if individuals did not give an accurate account of their drinking, which was common, they understated the amount, not overstated it. So this can probably be seen as a likely minimum consumption at the time.

b. **What was his likely nutritional status prior to that admission?** There was disruption to his diet, given Mrs Dickinson's absences from the home in the 2 weeks before and 3 weeks after her father's death on 26 July. The contemporaneous record on 18 August, which was in no way a self-serving document given it preceded the index events, corroborated her account at trial that she was not there to help him during this period. This was coupled with the rise in his alcohol consumption to maximal levels again. I therefore find that his nutritional status was poor at the point of admission and in the preceding weeks. At the very least he was at risk of malnourishment, which eating well in hospital, likely reflecting a return in his appetite once not consuming a large volume of lager every day, could not reverse sufficiently quickly, in the absence of Pabrinex.

c. **Were his thiamine stores at the point of admission (a) normal or (b) impaired and to what extent?** They were significantly impaired for the above reasons, putting him at risk of WE if IV Pabrinex was not administered.

d. **How much Pabrinex was likely administered on 26 August 2016?** At most 1/30th of the 250mg vial.

e. **Were the Claimant's thiamine stores depleted by the admitted failure to provide him with Pabrinex between 26 August and 31 August 2016 to the extent that he was at risk of developing WE?** Yes.

f. **Did the Claimant develop a WE and, if so, when did this occur?** Yes, he did, likely starting on 31 August and developing into 1 September 2016.

g. **When did the Claimant develop the functional disorder (later diagnosed variously as FNSD, FND and/or FCD)?** By 1 or 2 September 2016, the Claimant was subject to a significant psychological stressor, one of the DSM-V diagnostic criteria for FNSD. He became aware that he was facing a significant neurological condition, likely flowing from the negligent failure to administer appropriate Pabrinex. This stressor laid the groundwork for the development of overt symptoms of FNSD over the following days. These symptoms were probably present by the latest on 5 September and certainly by 7 and 8 September 2016, with a gradual worsening thereafter. This was not a pre-existing condition, whether dating to 2012 or otherwise.

h. **What caused the development of the functional disorder and would it have been avoided with the proper administration of Pabrinex?** It was caused by the significant stressor outlined in the preceding sub-paragraph and it would have been avoided by the proper administration of Pabrinex. The timing was not merely a coincidence or a milestone on an inexorable process of an allostatic load. The episode of WE and effect on the Claimant was the cause of his FNSD on a balance of probabilities.

258. It follows that, in my judgement, but for the admitted breach of duty, the Claimant would not have developed the functional disorder and liability is established.

VII. ANALYSIS AND FINDINGS ON QUANTUM

259. The parties' laudable efforts to narrow the issues meant that various heads of loss were agreed, subject to liability, either prior to or by the end of the trial. There remain various major items which I am asked to determine.

260. The helpful summary table in the Defendant's closing submissions on quantum described the agreed (in bold and shaded green) and disputed (the remainder) heads of loss:

Head of Loss	**Claimant**	**Defendant**
PSLA	TBC	TBC
Interest	TBC	TBC
PAST LOSS		
Care and assistance	£174,220.66	£29,626.00
Increased costs	£5,272.31	£0.00
Travel expenses	£487.90	£0.00
Miscellaneous	£2,060.84	£0.00
Total past loss	**£182,041.71**	**£29,626.00**
Interest	**£20,121.16**	**£2,681.15**
FUTURE LOSS		
Loss of earnings	**£0.00**	**£0.00**
Care and assistance	£3,847,541.57	£780,782.29
Case management	£138,669.72	£5,667.00
Physiotherapy	**£15,000.00**	**£15,000.00**
Physiotherapy equipment	**Included above**	**Included above**

OT	**£30,000.00**	**£30,000.00**
Psychological therapy	**£25,000.00**	**£25,000.00**
Services	**£2,500.00**	**£2,500.00**
Aids and equipment	£128,959.80	£31,754.51
Transport	£131,654.36	£18,927.20
Accommodation	**£700,000.00**	**£700,000.00**
Total future loss	**£5,019,325.45**	**£1,609,631.00**
GRAND TOTAL	**£5,221,488.32**	**£1,641,938.16**

261. There is agreement between the parties' experts that his life expectancy is markedly reduced to age 63.5 years. As discussed in closing submissions, I am invited to determine the appropriate multiplicands for future losses and then give the parties time, after the handing down of the judgment, to seek to agree the appropriate form of award, whether capitalised amounts or periodical payments, for the various heads of future loss.

262. In addition to the matters set out earlier in this judgment drawn from the witness, the expert evidence and the pre- and post-index events medical records, there is a useful summary of the neuropsychiatrists' joint statement as to C&P (p.1103-9) at para. 73 of the Claimant's skeleton argument:

"The following points emerge from the neuropsychiatric joint statement as to condition and prognosis [1103-9]

c. The Claimant suffers from a "*severe FND, which is the primary cause of his profound physical disability*" and a functional cognitive disorder "*with the addition of opiate medication*".

d. Dr Symeon diagnoses a Major Depressive Disorder, with ongoing low mood, diminished interests, feelings of guilt and low self-worth and suicidal thoughts. Prof. Carson felt some of those features were present but could not get an accurate history as to how pervasive they were, nor the extent to which they are better explained by excess opiate medication.

e. Very importantly, the experts <u>agree</u> the following functional limitations arise:

    i. From FND: profound physical and cognitive limitations. He is largely bed-bound, requires assistance with all transfers (using a sliding board), and is unable to walk. He has severe pain and sensory disturbances in his lower limbs, preventing weight-bearing and contact with sheets. He has issues with bladder control, requiring a sheath.

    ii. From NCD: functional cognitive symptoms affect memory and everyday function, but the extent is difficult to assess. It does not seem to prevent any daily activity he is physically capable of.

    iii. From depressive symptoms: impacts engagement with activities, contributes to social withdrawal and effects his overall quality of life and could affect his willingness to participate in rehabilitation.

f. As to prognosis, both <u>agree</u> the overall prognosis is poor and return to employment is improbable. Even with treatment, full recovery is not expected and he would still have "*significant ongoing disability and care needs*"; requiring long-term care. Deterioration remains possible. There is subtle disagreement:

  i. Dr Symeon is slightly more optimistic, holding that *some* improvement in *quality of life* is possible with appropriate rehabilitation, "*but this is unlikely to translate to a change to care needs*". Fluctuating psychiatric and FND symptoms would persist indefinitely.

  ii. Professor Carson believes "*…on the balance of probabilities, it is unlikely he will benefit from treatment, and his condition will remain the same*."

g. If there is to be rehabilitation, it should be intensive, MDT and inpatient. Professor Carson is sceptical of the benefit from such treatment but states that it is "*reasonable that he has access to in-patient*" [rehabilitation]. There is agreement as to the need for future psychiatric/neuropsychiatric treatment but with some differences as to cost and detail."

263. In his evidence, Dr Symeon accepted that the Claimant would continue to experience recurrent episodes of depression, irrespective of the index events. But he highlighted the degree of suicidal ideation and self-harm risk that he now has. I refer also to the pre-existing medical history and conditions, such as knee pain. Whether he would have remained abstinent from alcohol, but for these events, is far from certain. There was a real risk that he would once again have become a problem drinker.

264. I refer to Mrs Dickinson's description of the Claimant's condition, as discussed at [65].

265. I accept the evidence that prior to the index events the Claimant was not receiving any care and assistance. He was self-caring and did his own ADLs. He had some limitations in walking distance, but managed to take the dog to a local field, pausing on the way.

266. **Pain, Suffering and Loss of Amenity:** shortly after the trial, the 18th Edition of the Judicial College Guidelines was published. I take account of the uprated figures for the corresponding brackets referred to by the parties.

267. The Claimant sought a figure of £230,000. This was by reference, for his physical injuries, to the brackets for an incomplete spinal cord injury, as he cannot walk, but with more painful extreme sensory pain. The range for paraplegia is £289,420 to £375,540 and for a severe neck injury with incomplete paraplegia is in the region of £195,970. His FCD could be compared to an acquired brain injury, albeit the symptoms are functionally mediated, the moderate brain injury category for a moderate to modest intellectual deficit, would be £119,860 to £198,320. There are also the chronic pain awards, including severe, for CRPS of £69,360 to £110,990.

268. The Defendant contended for a figure of £100,000 by reference to the bracket for severe psychiatric injury which, in the 18th Edition is £72,440 to £152,900. Its position was that reference to the brain injury section would not be appropriate as it is agreed that this is not an organic condition, there is greater cognitive and physical function on the evidence, than the Claimant perceives himself to have. The Defendant referred to the need for the Court to consider whether there was an exacerbation of the recurrent depressive episode, which I consider there was.

269. I factor in the parties' submissions. Bearing in mind the highest bracket in the chronic pain section, at chapter 9, which does refer to FND, is for CRPS suggesting £69,360 to £110,990. The features that describes do not fit with the severe disability experienced by the Claimant. Whilst it does envisage a poor prognosis, significant care needs and psychological problems, it envisages one limb being affected with, at the top of the bracket, symptoms spreading to other limbs. That does not do justice to the Claimant's condition which is significantly worse and more disabling in both the physical and cognitive domains, with a need for 24 hour care (as I have found). Whilst this injury

does not fit into the spinal or brain injury categories, as it is functional, I have found reference to those brackets useful to calibrate where this case fits on the spectrum of disability, pain and loss of amenity flowing from those injuries.

270. Although I have not tied this assessment to any particular bracket, for the reasons explained, I have nevertheless borne in mind the slight uplift for RPI which would be applicable on the existing brackets in the 18th Edition, given those are figures updated to August 2025 (RPI figure 407.7): see p.xiv of the Introduction. The figure for May 2026 is 415.3. So an uplift of 1.86% to those brackets is applicable.

271. The Defendant referred me to some quantum reports involving FND. The largest PSLA figure identified for FND was £121,000 (updated for RPI) in *WU v CB* (2022). I must exercise some caution about this as it was a figure extrapolated by the parties in that case, from an overall settlement, rather than a Court award. It involved FND in a 29-year-old that resulted in "*Virtually no sensation in lower body, poor lower limb strength, double incontinence, sexual dysfunction*". The report of that case also includes the following narrative:

"C was hospitalised for several months and underwent intense inpatient rehabilitation which led to a significant improvement in his symptoms. Approximately 10 months post-accident he was able to transfer independently, stand and mobilise very short distances with crutches. C was extremely motivated with his rehabilitation and made further improvements over the course of the next two years. He regained some independence in his daily life and was able to socialise. He did however continue to rely on the help and support of his family as his mobility was still significantly compromised. C's mood was affected by his injuries and particularly on the impact they had on his ability to lead a fulfilling and fully independent life. He continued to take pain and sleep medication.

C remained off work for almost four years following the accident. He was only able to return to his role as a professional sports coach on a part-time basis and was unable to *fully resume coaching due to his inability to stand unaided for any length of time. He therefore had to undertake more administrative and office-based duties. He was also unable to enjoy his pre-accident sporting activities and hobbies and had lost the opportunity to compete in high-level competitions with associated potential lost earnings."*

272. Importantly, in the quantum report, it is said that "*The settlement was agreed on the assumption that C was likely to make some improvement if he underwent further inpatient rehabilitation.*" Sadly that is not the position in Mr Dickinson's case. But it provides important context for the award of the updated amount of £120,000 in a case where the outcome was a significant improvement to a much better level of function, in contrast to Mr Dickinson's serious and enduring level of physical and cognitive disability.

273. Taking all of the relevant features into account, given the severity of the physical and cognitive disability here, the painful sensory symptoms, the need for 24 hour care and significantly restricted independence, the Claimant's middle age (albeit with a reduced life expectancy to age 63.5 years) and bleak prognosis, but factoring in the pre-existing limitations, I consider that an award of **£185,000** for PSLA is appropriate.

274. I invite the parties to agree the appropriate figure for interest at 2% p.a. from the date of service of proceedings, as part of the discussions about the consequential Order.

275. **Past care:** as set out above, the parties' competing positions are £174,220.66, for the Claimant and £29,626.00 for the Defendant. This reflects the contrasting assessments of Ms Woolcomb and Ms O'Connell. The former determined that Mrs Dickinson has devoted herself to the Claimant since the index events, providing 40 to 50 hours of care a week, at the aggregate rate, notwithstanding her own limitations and probably at the cost of her own health. For Ms O'Connell, the reasonable care over and above pre-

existing needs, varied between 10.5 and 23.25 hours per week, proposing the standard (basic) rate as this was care provided in Mrs Dickinson's own home.

276. As with her evidence as to liability, I considered that Mrs Dickinson was doing her best to assist the Court with a representative picture of the care she has provided and the Claimant's wide-ranging needs. I accept that she has soldiered on in providing largely full-time care, at a significant health cost in terms of her own disabilities. I largely prefer Ms Woolcomb's assessment over Ms O'Connell's as a more realistic assessment for someone who, despite his pre-existing conditions, I have found was not in receipt of care. That is not to say that I did not consider that aspects of the care were probably assessed at an overly generous level. There would have been times when Mrs Dickinson was not available or was limited to some extent in what she could provide when her own conditions flared up. I felt that the level of bird care claimed whilst the birds were still present at their home into 2020 was implausibly high if as described the bare minimum was being done. I do make an award for companionship and emotional care, bearing in mind the guidance that in appropriate cases this is recoverable: see Yip J's (as she then was) judgment in *Welsh v Walsall Healthcare NHST* [2018] EWHC 1917 (QB) at [109-113] and the decision of Langstaff J in *Warrilow v Norfolk NHST* [2006] EWHC 801(QB), referred to in that section. Here, there are similar features, namely the Claimant's need for reassurance, especially at night, his fragile mood and vulnerable mental state, plus his suicidal ideation and the risks of self-harm identified by Dr Symeon, who I found to be a measured, erudite and helpful expert. That is something that Mrs Dickinson was well placed to provide and has done devotedly, in my assessment. It is not something upon which her own physical limitations imposed any material limitations.

277. I do accept that this is one of the unusual cases where the aggregate rate is justifiable, given this included personal care and at times physically and emotionally demanding

care, including at anti-social times of night. Making an appropriate downward adjustment, I assess past care at **£140,000**. That amount is awarded on trust for Mrs Dickinson and any other gratuitous care provider(s), in accordance with well-established principles.

278. **Past increased costs:** £5,272.31 is claimed, broken down as £15 per month (£1,682.85 in total) for additional bedding because the Claimant spends the majority of the day in bed. The balance of this head of claim is for increased heating costs, as it is said the Claimant and his wife spend longer than they would have done at home, incurring additional costs of between £6.47 and £7.57 per week in increased energy bills. A total of £3,589.46 is claimed for that aspect. The claims are also touched upon in Mrs Dickinson's statement at (p.160). I consider that the Defendant's objection in the Counter Schedule to the increased heating costs claim is a valid one and I conclude that causation is not established. Prior to the index events, the Claimant was not working and had limitations to his mobility meaning he likely spent a good deal of time at home already. In addition, Mrs Dickinson's own disabilities would likely have resulted in her being at home much of the time and incurring similar costs in any event. So I do not award any sum for increased heating costs. The Defendant's objection to the bedding costs claim is that the Claimant should give credit for the saving in clothing that he no longer wears. I consider that to be a valid point to some extent, but the Claimant will still have needed clothes on most days. Making an appropriate adjustment for those factors, I consider that **£1,000** for the bedding claim is an appropriate award in the circumstances.

279. **Past travel expenses**: these are modest in scope, claimed at £487.90. The Defendant makes the point that the Claimant's pre-existing conditions would have necessitated regular medical appointments and the HMRC rate of £0.45 per mile exceeds the actual loss which is better represented by a rate of £0.25 per mile going back to 2016. Making

adjustment for journeys in any event and a lower rate, I consider that **£300** would be reasonable compensation under this head.

280. **Past miscellaneous expenses:** I bear in mind the sums claimed in the Schedule totalling £2,060.84 which for the reasons set out in the Counter Schedule, the Defendant denies are recoverable, partly due to credit being required for similar costs or costs saved. The parties did not make formal submissions on these modest items but ask me to resolve them on the witness evidence and written positions. The laminate flooring allowed the Claimant to circulate more easily in his wheelchair and replaced the carpets. It appears modest in scope in terms of cost and the value of the gratuitous labour to install it. Likewise the materials and labour to install a patio to facilitate him accessing his aviary by wheelchair. I award those two items in the sums claimed. I agree with the Defendant that the fans and landline telephones are everyday domestic expenses which might well have been incurred in any event for the same or similar items, so they are not recoverable. I award **£1,945.84** under this head, before interest.

281. **Future care:** I have been asked to assess the multiplicands, leaving the parties to consider the appropriate form of award, whether lump sum or periodical payments. I refer to the description of the competing evidence from Ms Woolcomb and Ms O'Connell in the expert evidence section of this judgment at [174-209]. There was a stark difference between them.

282. Ms O'Connell considered that the Claimant's reasonable needs would be met on a daily basis by one 1 hour visit and two 30 minute visits and in addition 9 hours per week to access the community. This was sufficient to meet his disabilities as noted by the neuropsychiatrists, in her view. But, if the Court considered that the Claimant reasonably required a 24 hour care regime, Ms O'Connell agreed with Ms Woolcomb's costings, whether the hours or rates, namely a multiplicand of £248,843.40 p.a.

283. I have found it helpful in resolving this dispute, to consider the evidence of Dr Symeon. As noted above, I found him to be a measured, erudite and helpful expert. As set out above at [155-158], his views on overall care needs can be summarised as follows: "*If the Claimant was his patient, he would want him to be somewhere where he had 24 hour input in his own property, not just for direct personal care, given the risks of accidental injury from trying to move or inappropriate transfers, but also for managing the risk of harming himself. The next step down from 24 hour care was care 4 times per day, which would leave too many gaps where the risks already identified would not be mitigated. The care needs were likely to stay the same.*"

284. This overview chimed with my own impression of the reasonable needs here, having heard the totality of the factual and expert evidence. I have little hesitation in deciding that the Claimant's reasonable needs, in view of the severe physical and cognitive disability here, can only be met by a 24 hour regime of the type described by Ms Woolcomb. I recognise that this is considerably more than the care Mrs Dickinson has been providing and is being compensated for in the past. But, dedicated as that has been, the Claimant's needs are such that only a 24 hour professional regime will keep him safe, allow Mrs Dickinson to step back from being a carer to being a spouse, reflect the fluctuating and unpredictable needs he has, whilst also providing opportunities to broaden his horizons beyond, largely, his bedroom and home. This is not the same as saying he needs constant care and that is not the threshold test for a 24 hour regime.

285. I agree with the Claimant's submissions that the fixed and limited hours proposed by Ms O'Connell would not provide adequate, flexible and safe support. It implicitly relied upon Mrs Dickinson's availability in the background, although she has her own health issues, should no longer be having to be a carer rather than a wife and is entitled to take time away from the home (or may need or want to). The Claimant's personal autonomy would not be provided for adequately by such a regime. I agree that he should not have

to wait for one of the three brief scheduled visits for assistance with transfers and/or personal hygiene and/or getting dressed/undressed and/or catheter management and/or emptying his bowels and/or being provided with meals, snacks or drinks. Likewise opportunities to access the community when his wish and his capacity to do so should drive the decision, not pre-determined visiting hours. In addition, importantly, there are the real risks of accidental injury and self-harm which would not have arisen but for the index events and cannot be appropriately mitigated save by a 24 hour regime.

286. I conclude that the appropriate multiplicands, agreed between the experts if I determine as I do, contrary to the Defendant's case based on Ms O'Connell's views, that a 24 hour regime is reasonably required, are **£104,936.83 for Year 1** and **£248,843.40 p.a. for Year 2 onwards.**

287. **Case management:** given my decision on the appropriate care regime, I do not consider that partial or total reliance on Mrs Dickinson in the longer-term is reasonable or practical, as a substitute for long-term case management.

288. As for years 1 and 2, as it will be necessary to get a 24 hour regime up and running, as well as facilitating a move of property, I consider that figures closer to those costed by Ms Woolcomb are appropriate. I award **£20,000 for year 1 and £10,000 for year 2**.

289. After the first two years, a contingency allowance of 40 hours for his lifetime, to cater for changes in his condition or circumstances, as Ms O'Connell suggests, is insufficient in my view.

290. I consider that the appropriate award to facilitate oversight of the care package and therapies in the longer term, is the sum claimed by the Claimant of **£7,404 p.a**.

291. **Future aids and equipment:** the dispute on which I am asked to rule, is whether Ms O'Connell's provision for a heavy duty powered wheelchair in addition to a self-propelled wheelchair with a power pack is appropriate or whether Ms Woolcomb's You-

Q Luca XL Powered Wheelchair with attendant controls in addition to the manual wheelchair, is reasonably required.

292. The Claimant's mobility was quite limited before the index events. He could just about manage to take the dog to the field at the end of the street, by having regular breaks on the way. He had long since given up fishing. The approach of the Court in tortious damages is to put the Claimant back into the position he would have been in, but for the accident. It is important for his dignity, wellbeing and independence that the Claimant is facilitated to access the community. That will be possible with the 24 hour regime I have allowed for. But I must have an eye for the sort of activities he would and would not have carried out, but for these events, within the limitations he would in any event have had. I consider that the provision for a powered wheelchair and a self-propelled wheelchair with a power pack, recommended by Ms O'Connell amounts to reasonable compensation in terms of the nature of the need and the amount it will cost: see the legal principles discussed at [218-220] above.

293. By contrast, I consider that the carer's bed is an appropriate item which will allow the Claimant to sleep next to his wife. Similarly, the tilt-in space armchair reflects the one he currently uses when sitting out of bed. He should be facilitated to do more sitting out (and going out) with a 24 hour regime, so provision for that item is appropriate.

294. Having ruled on what I understand were the disputed items, I invite the parties, as part of the consideration of the consequential Order, after handing down of the final judgment, to agree the appropriate award for the above items of future equipment loss.

295. **Future transport:** Ms Woolcomb gave evidence (and I am aware) that there have been some changes to the Motability scheme to restrict the scope of vehicles available somewhat, to exclude perceived luxury brands. I am not aware that there is any other suggestion that the scheme itself is in jeopardy or that vehicles suitable for this Claimant

will no longer be within its scope. The Claimant has already obtained a vehicle through the scheme, suitable for his needs. I understand that he has made little use of that vehicle for the past 2 or 3 years and it has been adapted for Mrs Dickinson to use. That should not be held against him. It is likely a reflection of his isolation and proper commercial care provision will allow him to gain some independence and access the community.

296. In my judgement he will probably continue to use the Motability scheme, as he already has. It would be reasonable for him to continue to do so, in the circumstances of this case, rather than opting for a private purchase. Any costs, if any, associated with the scheme would be recoverable, but I understand that he has not been liable for the advance payment to date. Assuming his damages are suitably managed, so as not to impact his means-tested entitlement, that would appear likely to remain the position.

297. It would be reasonable for the additional cost of carers' insurance to be recovered to enable him to access the community. Given the Claimant's pre-existing health issues, I anticipate that he would have paid for car cleaning in any event so I do not make an award for that.

298. Having ruled on what I understand were the disputed items, I invite the parties, as part of the consideration of the consequential Order, post handing down of the final judgment, to agree the appropriate award for the future transport claim.

299. In the event that the parties need me to rule upon any additional details on quantum, not dealt with above and which cannot be agreed as a consequence of my rulings, no doubt this will be addressed in succinct written submissions as part of the post-judgment hand down process towards agreeing a consequential Order. I refer to the postscript below.

VIII. CONCLUSION AND OUTCOME

300. For all the above reasons, but for the admitted breach of duty, the Claimant would not have developed the functional disorder. I refer to the summary of my findings on the key issues at [257] in this judgment. There will be judgment for the Claimant on liability.

301. The quantum issues upon which I was asked to rule at this stage are resolved as set out above, in Part VII of this judgment.

302. The parties agreed at trial that once they had my findings on the quantum issues, including relevant multiplicands for the future heads of loss remaining in dispute, including care, in the event of a judgment for the Claimant, it would be helpful if the Court permitted the parties some time to seek to resolve issues around the form of award. Namely whether there should be an award of periodical payments or a lump sum only award for future loss, as well as other consequential matters. The mechanism for this is set out in the postscript below.

IX. POSTSCRIPT

303. I invite the parties to seek to agree an Order consequent upon this judgment. As provisionally agreed during closing submissions as being appropriate, in the event of judgment for the Claimant, I will grant the parties time beyond the handing down of the judgment to discuss and see if they can agree the terms of that consequential Order. That would include whether the form of award should be by way of Periodical Payments or solely a lump sum. In the light of further helpful email input from the parties' Counsel following circulation of the draft judgment, I will set an initial deadline of 4.30pm on **14 August 2026** for the consequential Order. If closer to that deadline, the parties agree that they need additional time, again, they should email jointly to that effect. I will be

amenable to extending the time period for providing the draft consequential Order if told that this is likely to allow all issues to be resolved consensually.

304. If, despite using their best endeavours to agree the consequential Order, there are any remaining issues which need a ruling from the Court, the parties should provide succinct written submissions by the deadline (or extended deadline as applicable) for the consequential Order and the Court will make a ruling on paper or consider whether a further hearing is required.