FORENSIC REPORT

CLIENT

Name:

Address:

Date of birth:

Age at examination:

EXPERT WITNESS

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Date of examination:

Date of report:

Instructed by:

Solicitors' reference:

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EXPERT'S RESUME

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INTRODUCTION

1. This medical report is based on my examination of Mr. * at 85 Wimpole St., London W1G 9RJ and the following evidence:

    1.1. Medical records from Generalist Physician (GP), in 4 parts; updated records in 3 parts; from [date] to [date] in 2 parts

    1.2. Medical records from [Hospital name] B1 to B833, of various dates

    1.3. A medical report from Dr. X, dated [date]

    1.4. Four medical reports from Professor P, dated [date], [date], [date], and [date]

    1.5. Two medical reports from Professor J, dated [date] and [date]

    1.6. Medical records from [Hospital name], [date]

    1.7. The witness-statement of Mr. *, dated [date]

    1.8. [Hospital name] Records, in 4 parts, of various dates

    1.9. Two witness-statements of Mr. Y, dated [date] and [date]

    1.10. Two medical reports from Dr. T dated [date] and [date]

2. All opinion below is elaborated as to reasoning and to the evidence that supports it. All published evidence is from books produced by creditable publishers or from peer-reviewed journals of adequate or greater quality, written by clinicians at minimum but preferably by clinicians with scientific qualifications at the doctoral level, particularly for technical and scientific research applied to medicine.

SUMMARY OPINION

3. In addition to Complex Regional Pain Syndrome affecting the left foot, Mr. * has Wernicke-Korsakoff Syndrome (WKS), a major neurocognitive disorder. This disorder is irreversible. Evidence for alternative diagnoses of vascular dementia (including strategic-infarct dementia and subcortical or Binswanger's dementia), inflammatory and autoimmune encephalopathies, and toxic encephalopathy due to poisoning was assessed and found to be inadequate to explain the symptoms and their timeline.

4. WKS is a consequence of the treatment of the crush-injury to his left foot with narcotic analgesics, in tandem with digestive-tract disease and depression, which caused malnutrition. Malnutrition resulted in a thiamine-deficiency, which was causative of the Wernicke-Korsakoff Syndrome.

5. Mr. *'s cognition is impaired across a broad range of functions. His primary problems are a severe anterograde amnesia, a partial retrograde amnesia, and fluctuating but severe problems with orientation. He has moderate problems with executive functions, primarily with adjusting to task-demands, behavioural and problem-solving flexibility, emotional self-management, foresight and monitoring his social behaviour, planning, organising, and initiating activity (including problem-solving), and sustaining working-memory. There are no noteworthy problems with impulsiveness, nor with organising his environment and materials. Also, Mr. * seems to have good awareness of his problems at times but poor insight at other times. Other problems include a minor and very specific paraphasia (an impaired ability to use language properly) and problems with visuospatial processing.

6. Mr. * also suffers from a Major Depressive Disorder of moderate-to-severe intensity with significant risk of suicide. This is as a result of the crush-injury's effects on his life as well as the effects of his neurocognitive disorder. His depression does not account for his cognitive deficits.

7. Recommended treatment involved multidisciplinary approaches in a residential rehabilitative unit that provides comprehensive and high-quality rehabilitation, integrating cognitive therapy, group-based psychotherapy with family-carers, occupational therapy, medical hypnotherapy, acupuncture, neurofeedback and/or fMRI-based neuromodulation, and pharmacotherapy. This will need to be privately funded due to the restricted scope of services available on the NHS.

8. Mr. * is permanently disabled from his condition and unable to work in the future.

9. He requires day-to-day care and attendance with personal needs and tasks as a result of the injuries, which is medically justified in the report below. These needs may reduce somewhat with successful treatment but he is highly unlikely to become independent to any meaningful extent due to the nature of his neurocognitive disorder. The success of support-services provided to him will depend on their integration with the outputs of his rehabilitative treatment.

10. A report as to costings and availability should be obtained from a rehabilitative case-manager, most likely a consultant nurse, as this lies outside my area of expertise.

ASSESSMENT

11. I assessed Mr. *'s mental status in reference to the instructions received.

12. I assessed Mr. *'s cognitive function using the Neuropsychological Assessment Battery (Stern and White, 2001). Please see the Appendix for more specific information. The application and interpretation of the cognitive tests were modified to a minor degree to account for visual problems. Mr. Y, Mr. *'s husband, had not brought the latter's glasses but had brought a magnifying glass instead.

13. I also sent the Behavior Rating Inventory of Executive Function, Adult version (BRIEF-A) by email to Mr. Y using an online assessment-system provided by Psychological Assessment-Resources Inc. The BRIEF-A is a standardised measure of an adult's executive functions or self-regulation in his or his everyday environment. Both a self-report and an informant report are used; Mr. Y had Mr. * fill in the self-report and filled in the informant-report himself.

14. I also sent the Personality Assessment Inventory (PAI) of Morey (1991; see Appendix), to screen for problems with mood, personality-disorders, psychoses, *et sim*.

15. Findings were evaluated against the diagnostic criteria of the:

    15.1. World Health Organization's International Classification of Diseases and Related Health Problems (ICD-10), Chapter V (Mental and Behavioural Disorders)

    15.2. American Psychiatric Association's Diagnostic and Statistical Manual 5 (DSM-5)

FINDINGS

16. I was careful to look at any inconsistencies in the interview, records, and the psychometric findings. No notable inconsistencies were found.

17. **Cognition**:

    17.1. I screened for function related to Attention, Executive Function, Spatial Function, Language, and Memory, and also used the in-depth modules for assessing the last three in particular. Both Attention and Executive Function were considered to be sufficiently impaired as to not be worth assessing using the in-depth module. However, The Screening Module's assessment of Executive Functions was affected by visual problems; consequently, a second assessment was done using a different method in the BRIEF-A, as described above.

    17.2. **Attention**: Mr. *'s Attention was shown to be moderately impaired overall, primarily due to problems with vision and working memory. However, his awareness of his self in relation to his environment was severely impaired. This indicates a significantly increased risk in self-management.

        17.2.1. Technical specifics: Mr. * had above-average selective attention and concentration but moderate-to-severe impairments in efficiency. His auditory attentional capacity was observed to be normal; working memory for orally presented information was impaired; his divided attention and information-processing speed seemed to be normal but affected by visual issues.

    17.3. **Language**: Mr. * has a specific language-problem (a paraphasia); this particular paraphasia is unlikely to affect his function day-to-day. However, the language-associated daily-living task was also mildly-to-moderately impaired, but primarily due to problems with memory, attention, and executive function. This suggests a reduced ability to self-manage in tasks using these functions.

        17.3.1. Technical specifics: screening for language-function showed mild-to-moderate impairment in language in the presence of intact comprehension. His speech output and fluency are above average and auditory language-comprehension is average. Visual confrontation-naming and word-finding is mildly-to-moderately impaired, and his reading comprehension is moderately impaired.

        17.3.2. A word-generation test was marked by fluent and perseverative phonemic but non-semantic verbal paraphasias, indicating a dysfunction specifically related to the head of the caudate nucleus; however, this type of language-problem is associated typically with the head of the *left* caudate nucleus (in which there is no evidence of infarct on MRI) and/or in the posterior temporal region (Lewis, 2000).

        17.3.3. His moderate impairment in reading-comprehension is insufficient to suggest problems with the angular gyrus in the left parietal lobe currently, however perseverations suggest left frontal systems'

involvement, while intrusions of new words suggest a combination of frontal-lobe systems' impairment and left mesial temporal lobe dysfunction.

17.3.4. These findings suggest specific injury to the left frontal lobe, left temporal lobe, as well as the basal ganglia.

17.4. **Spatial Functions**: Mr. * has some mild-to-moderate impairments in his visuo-spatial cognition that also affect his ability to self-manage on a daily basis, including finding his way to and from locations.

17.4.1. Technical specifics: he shows mild-to-moderate impairment in visual discrimination and design-construction, with average figure-drawing organisation and fragmentation on copying but mild-to-moderate impairment on immediate recall, moderate impairment on immediate recall and severe impairment in retention. He also shows below-average map-reading, including severe mis-estimation of distance. He also showed asymmetrical drawing in both copying and immediate recall, and some minor impulsivity.

17.4.2. This suggests that his left and right parietal lobes are mostly intact, though affected by frontal lobe dysfunctions and also visual function. A lack of identifiable problems with fragmentation in copying show that right frontal-lobe functions may be less affected than those of the left.

17.4.3. Again, the particular areas suggest injury primarily to the left frontal lobe.

17.5. **Memory**: Mr. * showed impairments in all aspects of memory assessed, including working-memory. Recall consistently showed severe impairment. This indicates an inability to form significant new memories and constitutes an anterograde amnesia. This implies a significant risk to self-management on a daily basis.

17.5.1. Technical specifics: Immediate recall of word-lists showed mild-to-moderate impairment, whereas delayed recall showed severe impairment. Shape-learning showed mild-to-moderate impairment overall, with performance affected by his lack of glasses. Story-learning showed mild impairment in learning of both phrases and themes at short-term but severe impairment in learning both phrase-units and thematic units with delayed recall. Daily Living Memory test showed mild impairment on immediate recall but severe impairment on delayed recall.

17.5.2. Visual recognition is somewhat less impaired than visual recall, despite the effects of not having glasses; verbal learning is severely impaired.

17.5.3. The nature of the impairments in list-learning show sensitivity to retroactive interference, rather than encoding or forgetting, and which is often associated with frontal systems dysfunction. However, the visual/non-verbal learning system is also impaired, indicating that the right mesial temporal lobe is also affected.

Shape-learning also suggests problems with the right mesial temporal lobe, although to a lesser extent than with the left mesial temporal lobe.

17.5.4. These problems in the right mesial temporal lobe are in addition to those found in the left mesial temporal lobe and in the frontal lobe, supported by the primacy and primarily anterograde nature of the problems with memory. There is likely damage to the fornix or hippocampus and connections with the hypothalamic mammillary bodies.

17.6. **Executive Functions**: Mr. * shows severe impairments in certain executive functions. As a whole, these are defined in terms of **Organisation:** attention, planning, sequencing, problem-solving, working memory, cognitive flexibility, abstract thinking, rule-acquisition, selecting relevant sensory information; and **Regulation:** initiation of action, self-control, emotional regulation, monitoring internal and external stimuli, initiating and inhibiting context-specific behaviour, moral reasoning, and decision-making. His impairments relate primarily to planning and foresight, and working memory. A number of the remaining functions are intact.

17.6.1. Technical specifics: the Screening Module showed problems with planning and foresight, as well as visual scanning. The Mazes problems suggest dysfunction in frontal-lobe systems, particularly the dorsolateral prefrontal cortex.

17.6.2. Specific aspects of executive function were assessed more in-depth using the Behavior Rating Inventory of Executive Function (BRIEF), which is described in more detail in the Appendix. Both Mr. * and Mr. Y filled out a form, with Mr. Y providing his opinion on his husband's function, which provides a better estimation of Mr. *'s issues like awareness and insight of his problems.

17.6.3. In summary, there are significant problems with executive function. Comparing direct testing to self-report and information-report, the primary problems are with adjusting to task-demands, behavioural and problem-solving flexibility, emotional self-management, foresight and monitoring his social behaviour, planning, organising, and initiating activity (including problem-solving), sustaining working-memory. There are no significant problems with impulsiveness or inhibiting it, organising environment and materials. Also, Mr. * seems to have good awareness of his problems with largely intact insight.

17.7. Overall, these findings do not reflect deterioration from the nadir of [date] (as also evidenced by Dr. S's report on [date]), which suggests that the condition is likely to be at least stable.

18. Emotional function

18.1. Mr. * completed an assessment of personality and mood called the Personality Assessment Inventory (PAI), a very thorough, 344-item measure of psychiatric status that has been standardised against several

thousand respondents from both medical and community-based samples. This measure provides the clinician with an ability to provide a nuanced interpretation of clinical findings as well as to achieve a differential diagnosis in shorter time. I relied on the extensive documentation that comes with this psychometric for the interpretation of the various scales. This is described more fully in the Appendix.

18.2. This psychometric assessment was used to provide perspective on Dr. J's interview-based assessments.

18.3. **Validity**:

18.3.1. Mr. * responded without any evidence of malingering or purposeful distortion of the results in any of the several measures of distortion. His responses and the findings are considered reliable.

18.4. **Symptomatology**:

18.4.1. Mr. * has an extremely negative view of himself and his life. His responses and his profile as a whole indicate someone who is severely depressed. He does not report irritability, which suggests that his outbursts are not fully mood-related but also to his frustration with his neurocognitive disorder or problems with memory of his feeling-state. In contrast, while his depression is significantly related to his cognition, it is not explained by the latter. His self-esteem is badly affected and he is very socially withdrawn. He also has significant symptoms of anxiety as well as emotional instability, which is affecting his relationships.

18.4.2. Most probably due to his neurocognitive disorder, Mr. * reports having had symptoms such as delusions or hallucinations indicative of a thought-disorder more typical of psychosis, as well as having problems with his identity or sense of self, most likely as a result of the devastating effect of his illness on his life and sense of self. He reports an aggressive attitude, associated with physical aggression rather than verbal aggression. His risk of violence is somewhat raised, but not highly.

18.4.3. While he has no symptoms suggestive of impulsive behaviour that has potential for self-injury, he has a high risk of suicide.

18.4.4. The PAI psychometric assesses historical information that reflects major milestones in the development of an alcohol-problem, which is typically associated with social and occupational failures related to drinking, along with episodes where the person was intoxicated for prolonged periods. This is complemented by statistical strategies involving other subscales typically associated with alcohol-problems to assess possible alcoholic denial. Mr. *'s profile fitted that of someone who is severely depressed (coefficient of fit: 0.851) but not of someone who is alcoholic (coefficient of fit: 0.378). The one report of moderate alcohol-use (4 bottles of wine per week) is in the nursing notes following [date] when he was admitted for a disorder characterised in part by confabulation (the fabrication of

imaginary experiences to compensate for the loss of memory) and so is not a reliable self-report.

18.4.5. Mr. * shows a clear openness to engage in treatment but, at the same time, that the treatment-process will be complex and challenging.

19. **Differential diagnosis**:

19.1. Mr. * has a moderate-to-severe Major Depressive Disorder (ICD-10 code F32.1 or F32.2) with anxious features and a notable risk of suicide. This disorder predated the onset of WKS, therefore it is not possible to attribute it to the effects of WKS; further, Mr. *'s psychotic symptoms (e.g., hallucinations) can be attributed to the onset of WKS, and there is also no evidence to suggest that his depression has resolved since the crush-injury.

19.2. Mr. * also has a Major Neurocognitive Disorder Due to Another Medical Condition (DSM code 294.1, E51.9 using ICD10-CM for thiamine-deficiency) also known as Organic Amnesic Syndrome, not induced by alcohol and other psychoactive substances (ICD-10 code F04).

19.2.1. The possible DSM code referring to a major neurocognitive disorder due to multiple aetiologies was preferred against on the grounds that the neurocognitive examination suggested little identifiable contribution of the possible vascular disease to the current symptoms other than perseverative paraphasia (see below). This conclusion is likely on the balance of probabilities but may be a moot point.

19.3. The process of differential diagnosis was narrowed initially to:

19.3.1. vascular dementia (including strategic infarct dementia and subcortical or Binswanger's dementia)

19.3.2. inflammatory and autoimmune encephalopathies

19.3.3. toxic encephalopathy due to poisoning

19.3.4. Wernicke-Korsakoff Syndrome (WKS)

19.4. The alternatives were ruled out on the basis of the evidence available.

19.5. The approach taken was to determine the most parsimonious explanation of:

19.5.1. all Mr. *'s relevant symptoms recorded, including a widened and dyscoordinated gait and walking-problems with neuropathy ([date]), amblyopia and suspicious optic discs (loss of visual acuity without known cause, [date]), cognitive deficits ([date]), digestive-tract disease, mood and related problems, hallucinations, confabulation (fabrication of imaginary experiences to make up for loss of memory), systematised delusions, cardiac problems, hormonal abnormalities, urinary incontinence, alterations in specific white blood-cells (monocytes), etc.

19.5.2. the absolute and relative severity of symptoms

19.5.3. the development of symptoms over time

19.5.4. the sensitivity and specificity of measured symptoms in relation to a specific diagnosis

19.5.5. the absence of specific symptoms considered specific to or sensitively indicative of alternative diagnoses

19.5.6. the convergent probability of evidence towards a diagnosis and causative factors, independently and jointly

19.6. Firstly, the possibility of Mr. *'s disorder being explained by **vascular encephalopathy**:

19.6.1. Specifically: While Dr. T identifies the MRI showing "moderate small vessel disease", he opines that the "degree of vascular change does not necessarily equate with the degree of decline described in respect of Mr. *'s cognitive function" (report [date], p3).

19.6.2. These facts would tend to deprecate the possible contribution of vascular dementia to the symptoms, and Binswanger's dementia in particular (requiring white matter atrophy with subcortical dementia) as causative of the cognitive problems, despite the findings of subcortical pathology.

19.6.3. Vascular dementia can occur without major or multiple minor strategic infarcts (i.e., dead tissue due to lack of blood-supply) through lowering the threshold for concomitant pathology (Mortimer, 2013). It is reasonable to consider whether the development of Mr. *'s current neurocognitive disorder may have been contributed to in a similar way by background vascular pathology, particularly in reference to possible subcortical symptoms such as depression, clumsiness, repeated falls, irritability, and one or more instances of urinary incontinence.

19.6.4. In particular, there is a question of the role of the observed infarct in the right caudate nucleus. The right and left caudate nuclei are relay-stations between the limbic system and the frontal lobes, particularly the dorsolateral prefrontal and lateral orbitofrontal cortices, with functions relating to planning and sequencing, attention, recall of episodic and semantic items, recognition-memory, sleep, social behaviour, spatial learning and especially working-memory (Mendez, 1989).

19.6.5. Infarcts in the right caudate nucleus as a whole are related particularly to visual amnesia and contralateral neglect (there was no evidence of the latter in Mr. *); infarcts in the left are related to alterations in monitoring of language-production (of which there was indeed evidence in Mr. *, but no visible damage to the left caudate nucleus on the MRI). Infarcts in the **head** of the caudate nucleus are more specific: they are typically associated with restlessness (akathisia) for a few months after the infarct, which can

then settle down. The head of the caudate nucleus is connected to the nigrostriatal pathway, which contains dopaminergic neurons and is implicated in Parkinsonism and particularly the degree of dementia associated with Parkinsonism. However, there were no Parkinson-like symptoms noted in Mr. *, which would suggest that an alternative, related diagnosis of Lewy Body dementia could also be ruled out.

19.6.6. These facts contribute to the idea that a more systemic problem is occurring than can be explained by the infarct in the head of the right caudate nucleus alone, even though it is a potentially strategic infarct because of its role in multiple systems of the brain.

19.6.7. Additionally, it is unclear when the infarct in the head of the right caudate nucleus occurred; however, it was not reported as likely to have been contemporaneous with the onset of the hallucinations *et sim*. occasioning hospitalisation in [date].

19.6.8. On the balance of probabilities, Mr. * is unlikely to have been disabled to any degree by this possible vascular pathology in itself, since Mr. * had changes in the brain that are probably related to age, hypertension, polycythaemia, and smoking, which were apparently prior to [date] and probably causative of the infarct of the right caudate nucleus, but which were not associated with any overt neurocognitive disorder prior to [date].

19.6.9. Binswanger's disease is associated with normal declarative (episodic and semantic) memory, which Mr. * shows severe deficits in. Cognitive deficits in vascular dementia more generally are patchy and do not form an identifiable cluster in terms of functional or structural abnormalities of the brain.

19.6.10. At worst, vascular pathology could explain a minor portion of Mr. *'s symptoms, but not all of them without evidence of other disorders, such as Lewy Body dementia, but which would likely entail other symptoms that Mr. * does not have.

19.6.11. Thus, vascular disease and particularly the infarct in the head of the left caudate nucleus might explain one minor symptom and, on the balance of probabilities, vascular pathology may be a minor contributing factor through its lowering the threshold for the development of a more directly-caused pathology.

19.6.12. However, overall, both the known and potential vascular pathologies are unable to address the issues identified in §§19.5.1-6 above.

19.7. Dr. T also mentions the possibility of an underlying **inflammatory disorder**; the presence of oligoclonal bands in the cerebrospinal fluid should be noted at this point.

19.7.1. Such an inflammatory disorder could relate causatively to a subclinical vascular disorder or to immunological activity in the rest of the body or specifically in the brain (these are two separate

compartments of the immune system). Mr. * has a long-standing autoimmune disorder of the skin (atopic dermatitis), which can be related to changes in brain-function in principle; however, no clear possible mediators, such as humoral autoimmune factors, were found during the rather thorough work-up in [date]. Autoimmune encephalopathy has been associated with severe but nonspecific slowing of the EEG (Flanagan, 2010), which was also not identified when Mr. * was inpatient in [date]. Furthermore, autoimmune encephalopathies tend to have a rapidly progressive and often fluctuating course, whereas Mr. *'s disorder had a subacute onset and has not been noticeably progressive nor fluctuating since his stabilisation as an inpatient following [date].

19.7.2. Again, both the known and potential autoimmune and inflammatory pathologies are unable to address the issues identified in §§19.5.1-6 above.

19.7.3. The timeline and dynamics of the symptoms are therefore suggestive not of autoimmune encephalopathy but of a different disorder. While there is a possible contribution from systemic autoimmune disease, its nature would likely be facilitative rather than causative.

19.8. The tests carried out in [date] also explored the possibilities of **toxic encephalopathy**:

19.8.1. The possibility of a **toxic amblyopia** had been raised on [date] when Mr. * had experienced problems with his vision. Toxic amblyopia can be caused by poisoning or by malnutrition. I address the possibility of poisoning here and of malnutrition below.

19.8.2. Raised levels of the toxic metals cadmium and arsenic were noted during the period of hospitalisation in [date]; Mr. *'s exposure to these toxic metals is most likely to have been through heavy smoking, since high levels of cadmium and low levels of arsenic are found in cigarette-smoke. These metals have been associated with deficits in attention and memory, for instance in adult males with occupational exposure (Hart, 1989), but the slightly increased levels reported during his hospitalisation in [date] (e.g., cadmium at 15 nmol per litre) are not adequate to explain Mr. *'s specific cognitive deficits; on the basis of epidemiological reasoning, I believe they would be unlikely to explain the visual deficits either, else there would be an epidemic of visual loss among heavy smokers, which does not exist.

19.8.3. The possible contribution of poisoning is unable to address the issues identified in §§19.5.1-6 above. Thus, poisoning by heavy metals is not considered to be causative of Mr. *'s problems as a whole and his neurocognitive disorder in particular.

19.9. Dr. S raised the possibility of **Wernicke-Korsakoff Syndrome** (WKS) in his neuropsychological report on [date], on the basis purely of cognitive findings.

19.9.1. WKS is characterised specifically by a triad of signs: ocular abnormalities, including an otherwise unexplainable loss of visual acuity (as in toxic amblyopia) and/or problems in the movement of the eyes (as in nystagmus); ataxia (loss of voluntary coordination of muscle-movements, with gait abnormalities), and neuropsychiatric abnormalities (notably anterograde amnesia, hallucinations, and confabulation).

19.9.2. This syndrome matches Mr. *'s symptoms well, which started to develop in [date], well over a year before his hospitalisation. Just prior to his hospitalisation in [date], Mr. *'s symptoms met the Caine-criteria for Wernicke's encephalopathy (Caine, 1997) and this progressed to symptoms of the full syndrome in the ensuing period, which led to hospitalisation.

19.9.3. Only 16% of people with WKS exhibit the full triad of symptoms (Harper, 1986); while neuroimaging can be of help, such findings are not necessary or specific to WKS (Jung et al, 2012). It is helpful to note that Mr. * exhibited all the diagnostic signs of WKS, as follows:

19.9.3.1. On [date], Mr. *'s medical records by his Generalist Physician noted left-eye deterioration. On [date], Mr. * was reported to have suspicious optic discs, with possible "toxic amblyopia". Optic atrophy can be ischaemic; however, toxic amblyopia is believed to reflect damage to the papillomacular bundle of the optic nerve (i.e., near the eyeball) with vision blurring and dimness typically developing over days to weeks, with a blind-spot (scotoma) that slowly enlarges and progressively interferes with vision. The dynamic of this visual loss is not typical of optic ischaemia. It is also specifically recorded as part of the visual-field tests in Mr. *'s ophthalmic notes on [date], where it is also noted to be bilateral, although the left eye is notably worse.

19.9.3.2. Professor J's report of [date] §52 noted continuing bilateral nystagmus (involuntary eye-movements) as well as aniscoria (unequal size of pupils, with the left smaller than the right), the former which is a relatively common oculomotor abnormality in WKS and the latter which is a relatively uncommon but indicative oculomotor abnormality in WKS (Sechi and Serra, 2007).

19.9.3.3. On [date], Mr. * was reported (vid., T report [date] pp.2-3) as having been unwell for a few weeks, that his legs would not work, and that he stayed in bed. In hospital, Mr. * was found to have problems with balance and gait, involving a moderately severe symmetrical mixed sensorimotor primary axonal polyneuropathy, with chronic loss of motor-nerves below the knees on both legs. This is consistent with Wernicke's encephalopathy.

19.9.3.4. The problems with his legs was then followed in [date] by hallucinations, confusion, confabulation, and other cognitive

problems, which precipitated hospitalisation. This is consistent with Korsakoff psychosis and is a typical trajectory of deterioration to the full syndrome of WKS leading to inpatient-treatment.

19.9.4. Thus, the most reasonable and most sure diagnosis is that of Wernicke-Korsakoff Syndrome.

19.10. This diagnosis is a clinical one and it explains both the variety and specificity of symptoms, as well as the temporal development of symptoms. The diagnosis of WKS addresses well the criteria laid out in §§19.5.1-6 above.

19.11. The other clinical evidence is also consistent with WKS rather than any other disease and correctly explains the great majority of symptoms recorded, the order of their development over time, and the lack of symptoms that could be associated with the existing ones but aren't, deprecating or ruling out alternative diagnoses. All the recorded problems from multiple specialties also converge well on this diagnosis.

20. **Causation**:

20.1. Given the nature of WKS and its causation, Mr. *'s disorder could and would have developed as it is without any contribution from poisoning, or vascular, autoimmune, or inflammatory disease and therefore none of these possible pathologies is considered to be a necessary or sufficient cause of his neurocognitive disorder.

20.2. In order to determine whether the onset of the Wernicke-Korsakoff Syndrome (WKS) was related in any way to the Complex Regional Pain Syndrome (CRPS), it is necessary to look at the causation of all factors (including the infarct of the caudate nucleus) in the correct timeline.

20.3. Specifically, the analysis of causation must determine if there are:

20.3.1. factors that are in common with the causation of CRPS and of WKS, or

20.3.2. if the development or treatment of CRPS gave rise to factors that were causative of WKS

20.4. Firstly, none of the records report a date for the infarct of the head of the right caudate nucleus. This is not necessarily problematic but should be noted. Infarcts of the caudate nuclei are not directly causative of either CRPS or of WKS.

20.5. Causation of the Complex Regional Pain Syndrome (CRPS):

20.5.1. The crush-injury to the left foot was necessary and also sufficient for the development of the CRPS.

20.5.2. CRPS is thought to develop through a number of synergistic mechanisms, including central sensitisation (developed hypersensitivity) of the central nervous system, altered function of

the sympathetic nervous system, and increases in local inflammation disconnected from proper regulation by the brain.

20.5.3. Mr. * had a number of factors that would have indicated a significantly increased risk for CRPS. These risk-factors include immobilisation (which was necessary for healing the crush-injury), increased vasomotor tone due to systemic hypertension and polycythaemia (which Mr. * has), anti-hypertensive medications of the ACE-inhibitor-class (Birklein, 2009) such as Rifampril (which Mr. * was taking), and autoimmune mechanisms (Goebel, 2013; possibly related to Mr. *'s existing autoimmune atopic dermatitis), which also increase pain-sensitivity in mouse-models of CRPS (Li, 2014) and are associated with long-standing and intractable CRPS in humans (Dubuis, 2014).

20.5.4. Additionally, there is a primary pain-system in the brain that runs between the rostroventral medulla, hypothalamus, caudate nucleus, cingulate gyrus, anterior cingulate cortex, and prefrontal cortex. If the infarct in the caudate nucleus occurred before or contemporaneously with the crush-injury, it may be that the lesion in the caudate nucleus contributed to the promotion of the development of the CRPS through its effects on up-regulating the perception of pain and modifying the ability of the sympathetic nervous system to dampen the inflammatory response.

20.5.5. Thus, we can identify that, along with the necessary condition of the crush-injury, Mr. * had a number of additional risk-factors that increased the likelihood of his developing CRPS and which explain, on the balance of probabilities, why he developed CRPS following his crush-injury.

20.5.6. None of these risk-factors is implicated in the causation of WKS. Thus, the causation of WKS and CRPS is distinct.

20.6. Causation of the Wernicke-Korsakoff Syndrome (WKS)

20.6.1. There is also no evidence to support a claim that WKS is caused directly by a crush-injury to a limb.

20.6.2. WKS is associated with malnutrition, specifically in inadequate thiamine (vitamin B1) availability to the peripheral nerves and the brain through malabsorption of nutrients at the levels of inadequate nutritional intake, decreased absorption in the digestive tract, and impaired thiamine utilisation within cells.

20.6.3. This syndrome is most commonly seen in alcoholics and excessive alcohol-use can exacerbate thiamine-deficiency by intensifying sugar-metabolism in the brain. However, there is no evidence of alcoholism in Mr. * and alcoholism is neither necessary nor sufficient to cause WKS in that it does not induce WKS in the presence of adequate nutrition.

20.6.4. Malnutrition associated with thiamine-deficiency causes specific damage to areas including the frontal cortices (involved in

executive functions), the thalamus (involved in memory-formation), the cerebellum (involved in walking and movement), and the mammillary bodies (involved in both memory-formation and the production and regulation of hormones, such as prolactin and sex-hormones, which relate to sex-drive among other things and were also found to be abnormal in Mr. *). It also causes damage to the optic nerve.

20.6.5. Damage to the caudate nucleus is not specifically noted in thiamine-deficiency, indicating, again, that that pathology is distinct.

20.6.6. Malnutrition is considered to be causative of Mr. *'s WKS for the following reasons:

20.6.6.1. Mr. * already had a history of not eating in response to pain or specifically narcotic analgesics, as noted in his GP records on [date] when he had severe lower-back pain and was prescribed the narcotic painkiller codydramol; it was noted at that time that he was not taking his anti-inflammatories due to not eating.

20.6.6.2. Following the crush-injury in [date], Mr. *'s medical records show that he was treated with narcotic painkillers.

20.6.6.3. Narcotic painkillers such as morphine (opiate) and ketamine (NMDA receptor-antagonist), are significantly associated with nausea, vomiting, a loss of appetite, and thus reduced nutrition (vid. Merck Manuals Professional Version online—http://www.merckmanuals.com).

20.6.6.4. Failure to eat adequately would have resulted in nutritional inadequacy, which would have been exacerbated by inflammatory problems of the digestive tract, adding the problem of nutritional malabsorption.

20.6.6.5. I am unaware of any records showing that Mr. * received the necessary services in his pain-treatment from a nutritionist or health-psychologist to prevent malnutrition.

20.6.6.6. On [date], a concern was recorded in his GP records that he might have been overusing morphine due to the excruciating nature of the pain from the crush-injury. With an infarct of his right caudate nucleus, it is possible for there to be a heightened sensitivity to pain, making the experience of CRPS even more excruciating. As a consequence, Mr. * may have increased his use of narcotics, which would have resulted in further inhibition of appetite.

20.6.6.7. On [date] he was reported as being tired all the time and losing weight.

20.6.6.8. On [date] he was recorded as having dysphagia (problems eating) with associated weight-loss, related to an oesophageal

hernia, oesophagitis, gastritis, and duodenitis. I have not identified the causes of these digestive-tract disorders.

20.6.6.9. On [date], he was recorded as being nauseated after taking morphine.

20.6.6.10. On [date], he was also reported as having a history of abdominal pain, vomiting, and weight-loss.

20.6.6.11. In [date], it was noted that he was not eating or drinking (vid., Dr. T's report of [date] pp2-3).

20.6.6.12. In his witness-statement of [date] (prior to hospitalisation), Mr. * records that he had lost 2.5 stone in the last six months. This was corroborated in the hospital record of [date], where he was noted in his hospital records as having a history of losing 2-3 stone in weight, as well as vomiting. The same problems were noted on his hospital records following admission to hospital in [date].

20.6.6.13. In his witness-statement of [date] (§13), Mr. Y reported that his husband had refused to eat or drink, said that he was useless and wanted to die. It had already been noted that Mr. * was depressed. Depression very likely exacerbated his lack of eating, which was a consequence of the crush-injury and would itself have been exacerbated by inadequate nutrition.

20.6.6.14. It is reasonable to conclude from this history that malnutrition developed over time, including a thiamine-deficiency.

20.6.6.15. There are a cluster of closely related syndromes due to thiamine-deficiency, namely beri-beri and epidemic Cuban neuropathy, symptoms of which include weight-loss, impaired sensory perception, weakness and pain in the limbs, difficulty in walking due to a sensory ataxia, and vomiting, all of which symptoms Mr. * exhibited.

20.6.6.16. These symptoms are identifiable even in people without any signs of overt malnutrition due to the very specific nature of the thiamine-deficiency and the resulting damage to nerves, firstly in the main body and later in the brain.

20.6.6.17. These symptoms are also diagnostic of Wernicke's encephalopathy. If left untreated, it is established that Wernicke's encephalopathy leads to Korsakoff psychosis; jointly, they constitute Wernicke-Korsakoff Syndrome (WKS).

20.6.6.18. Furthermore, if treated inadequately, using only calorie-supplementation without high-dose thiamine-replacement, Wernicke's encephalopathy progresses to Korsakoff psychosis much more quickly and thus the full Wernicke-Korsakoff Syndrome.

20.6.6.19. In Mr. *'s case, his GP responded to his complaint of weight-loss with prescribing Nestlé's Build-Up, a drink with high carbohydrate-content; Build-Up is no longer available but is similar to Complan, which is 33% sugars and 31% other non-sugar carbohydrates with thiamine-supplementation that is inadequate in the presence of Wernicke's encephalopathy. The reasons for the GP's failure to identify and record the symptoms of Wernicke's encephalopathy are unknown.

20.6.6.20. Using Build-Up would have upregulated glucose-oxidation, which is a thiamine-intensive process, thereby further depleting Mr. *'s already severely depleted reserves of thiamine, exacerbating neural injury, particularly in the brain, and accelerating disease from Wernicke's encephalopathy to WKS. This is evidenced by the progression from walking-problems to symptoms including cognitive deficits and hallucinations following the use of Build-Up.

20.6.6.21. While Wernicke's encephalopathy is partially reversible, once it has advanced to WKS, it is largely irreversible due to the nature of the damage to the parts of the brain, although small improvements may be noticeable. Treatment then focuses on management of the consequences of disease for the patient.

20.7. Summarising the findings, the nature of the cognitive problems as a whole reflects damage to the head of the caudate nuclei (probably both, not just the right nucleus), both mesial temporal lobes, both sides of the frontal lobe, and hypothalamus. While the problems with language and visuospatial function are likely due to vascular injury to both caudate nuclei, the clustering of functional symptoms related to the mesial temporal lobes, the frontal lobes, and the hypothalamus is consistent with known pattern of injury due to thiamine-deficiency.

20.8. The evidence of these factors is adequate to show, on the balance of probabilities, a sufficient cause for Mr. *'s present Neurocognitive Disorder that is a direct result of the treatment for the consequences of the crush-injury to the left foot.

TREATMENT

21. Mr. *'s neurocognitive disorder requires extensive rehabilitative treatment; this will not return him to his baseline condition but will give him the best chance to return to as high a degree of function as possible. The suggested treatment is, indeed, extensive, but one that is appropriate to his degree of dysfunction. In addition, his condition is likely to affected by ageing in the future and, although it will not make him independent in living, rehabilitation will minimise his dependence long-term, including when he encounters problems associated with ageing, such as (further) reduced mobility and falls.

22. Mr. * presents a difficult challenge to any treatment-process, including a degree of lack of co-operation due to frustration and reduced insight; this problem is part of his condition.

23. Psychological interventions for the neurocognitive disorder and the pain-disorder should be integrated with physiotherapeutic services around use of the left foot. I am unaware of any such services having been provided previously, despite their known clinical effectiveness.

24. Given the particularly difficult challenges posed by Mr. *'s condition, it would be wise to maximise the chances of effective services by integrating these services to the fullest extent possible, including co-application (e.g., acupuncture and neurofeedback). The specific therapies recommended are as follows:

    24.1. **Cognitive therapy** for those who suffer from anterograde amnesia focuses on compensatory techniques and intensive training-programmes involving the active participation of the individual along with their supporting network of family and friends, and including environmental adaptation-techniques (Gordon, 2006; Champion, 2006). In severely injured individuals, effective interventions rely more on external aids, such as reminders, in order to facilitate particular knowledge or skill acquisition, along with reality-orientation techniques, enhancing orientation using stimulation and repetition of basic orientational information (Wilson, 2003).

    24.2. Progress in this treatment will subsequently need to be integrated with **occupational therapy**, to promote adaptation to daily living tasks. Occupational therapy without cognitive adaptation through psychological treatment will not be effective long-term.

    24.3. Emotion-oriented psychotherapy for both the depression and the pain-disorder is of limited applicability. The most applicable approach to psychotherapy in this area is cognitive-behavioural and mindfulness-based therapies, which are largely not applicable to Mr. * due to his neurocognitive disorder. However, **medical hypnotherapy** has been shown to be effective (Derbyshire, 2009) and it does not rely on conscious learning over time. This should be explored as a treatment-option.

    24.4. Other neuromodulatory methods, such as **neurofeedback**, rely on standard learning-methods, which are likely to be a highly increased challenge for someone with WKS due to the learning-deficits involved. However, they are effective when combined with other cognitive adaptation-techniques that remind the person with anterograde amnesia what to do, which require him to learn associated behavioural procedures, even though he can not remember that he has learned it.

    24.5. There are also visual-learning modalities, but these are difficult to access due to their relative lack of availability. In tandem with behavioural-procedural methods involving **fMRI**, with or without brain-computer interfacing, these have been shown to be effective in pain-treatment (Chapin, 2012) and may be a way of navigating the problems posed by anterograde amnesia since they access a form of procedural learning not affected as much by WKS. These approaches should be used in the instance that Mr. *'s pain-disorder proves not to be amenable to medical hypnotherapy.

    24.6. The major depression should be addressed clinically but within the context of a neurorehabilitative environment at first.

24.7. **Medication** for depression should be chosen that is likely to enhance emotional function without increasing sensitivity to pain. Depression in the instance of brain-injury (through nutritional inadequacy or infection, for instance) requires a more complex approach than depression in the absence of known brain-injury, not least in the initial dosing and dose-escalation. With the additional problem of CRPS, this is a highly specialised area and I will defer to a rehabilitative team on the medications to be proposed.

24.7.1. **Group-based psychotherapy** should be provided with his family and in liaison with his case-manager and should be integrated with his **cognitive therapy and occupational therapy** in order to translate the benefits to daily living long-term, particularly around adaptations requiring other people. This will also allow treatment for the effects of Mr. *'s injuries on his husband and their daughter, to maximise the ability of the family-unit to provide maximally effective support for Mr. * long-term, not least by reducing stress and burnout on family-carers.

24.8. **Acupuncture** is an obvious suggestion as repeated meta-analyses have identified its effectiveness in pain-treatment (Patel, 1989; Vickers, 2012). What form of acupuncture would be most effective lies outside my area of expertise and I would defer to the rehabilitation-team's opinion if it contains a fully qualified doctor of Chinese medicine, not just an acupuncture-technician. It is likely to have additional effectiveness if its application is integrated with medical hypnotherapy, neurofeedback, and/or cognitive therapy.

24.9. **Physiatric rehabilitation** with the services of physiotherapists and physicians addressing the gait abnormalities may have been adequate to date, but this remains to be determined by a consultant physiotherapist. If there are possible gains still to be made through treatment, this should be integrated with the cognitive and other treatments for the pain-disorder and use of the left foot.

25. Given the complexity of the treatment required, the known challenges with a treatment-process for Mr. *, and the multidisciplinary nature of adequate treatment, I would recommend that this treatment be in a private, intensive **residential rehabilitative unit**. Access to the highest-quality services—that provide all these services specified, with fully qualified specialist clinicians, not just some of the services or with clinicians inadequately trained in the specialist modalities—would probably take 3 months; services of a lesser quality might take 9 months to approximate the same gains, if at all. This will need to be privately funded due to the inadequate services available on the NHS.

26. A report should be provided to any agency providing day-to-day services so that their services can be tailored and effective, by providing services that maintain and possibly consolidate the interventions developed for Mr. * during his future neurorehabilitation.

ADDITIONAL REPORT

27. I am unable to identify the costings for the treatment proposed, due to its complexity and multidisciplinary nature. A report should be sought from a specialist rehabilitative clinician and case-manager, most probably a consultant nurse, who could also provide a short-list of potential units. This would likely include units internationally (e.g., Germany, USA) due to the restricted scope of treatment available in Britain.

ABILITY TO WORK

28. Mr. * has a severe and lifelong disability affecting almost all areas of his function physically, mentally, and socially. He is unable to work due to the combined effects of his neurocognitive disorder, his pain-disorder, and the loss of use of his left foot, and will remain unable to work for the rest of his life (Palmer, 2013).

SERVICE-REQUIREMENTS

29. Mr. * currently requires day-to-day care and attendance with personal needs and tasks as a result of the injuries.

30. This is medically justified, as should be identifiable from this report.

31. It is likely that his service-requirements may reduce somewhat in the instance of highly successful rehabilitative treatment and in the presence of fully professional and effective support-services; however, such a degree of success is both unlikely, on the balance of probabilities, and would also not remove the need for such care and attendance entirely. Of particular concern are the problems with orientation, memory, and executive function that affect Mr. *'s ability to be alone safely throughout the day and night. Effective treatment with effective support-services will minimise the challenges posed but not eliminate them.

APPENDIX: PSYCHOMETRICS

A. Neuropsychological Assessment Battery (NAB; Stern, 2001): The NAB is a comprehensive, modular battery of cognitive tests developed for the assessment of a wide array of cognitive skills and functions in adults, aged 18 to 98 years, with known or suspected disorders of the central nervous system.

   A.1. It screens for both impaired and normal performance across a comprehensive range of functional domains

   A.2. It combines the strength of flexible and fixed battery approaches to assessment and avoids ceiling and floor effects

   A.3. The entire battery is normed on a large, single standardisation group ("co-ordinated norming") and also has demographically corrected norms

   A.4. It has a Screening module, followed by modules assessing Attention, Language, Memory, Spatial, and Executive Functions in greater depth

   A.5. The Screening module allows users to determine which patients perform so poorly or so well on sections of the Screening Module that the administration of the corresponding and more thorough domain-specific module is unnecessary; patients would be expected to achieve similarly impaired scores on the respective modules

B. The Behavior Rating Inventory of Executive Function®–AdultVersion (BRIEF®-A; Gioia 2000) is a standardised rating-scale developed to provide a window into everyday behaviours associated with specific domains of the executive functions in adults ages 18 to 90 years.

   B.1. The BRIEF-A consists of equivalent Self-Report and Informant Report Forms, each having 75 items in nine non-overlapping scales, as well as two summary-index scales and a scale reflecting overall functioning (Global Executive Composite [GEC]) based on theoretical and statistical considerations.

   B.2. The Behavioral Regulation Index (BRI) is composed of four scales: Inhibit, Shift, Emotional Control, and Self-Monitor.

   B.3. The Metacognition Index (MI) is composed of five scales: Initiate, Working Memory, Plan/Organize, Task Monitor, and Organization of Materials.

   B.4. There also are three validity-scales: Negativity, Infrequency, and Inconsistency.

   B.5. The BRIEF-A can serve as a screening tool for possible executive dysfunction, as an index of the ecological validity of laboratory or clinic-based assessments, and as an indicator of individuals' awareness of their own self-regulatory functioning, particularly when both Self-Report and Informant Report Forms are used.

B.6. The Informant Report Form provides information about an individual's functioning in the everyday environment based on an informant's observations.

B.7. The Self-Report Form provides an understanding of the individual's perspective regarding their own difficulties in self-regulation — information that can be critical to the development of interventions.

B.8. Explicitly assessing, valuing, and providing feedback about an individual's viewpoint can facilitate rapport and the development of a collaborative working relationship that can, in turn, serve as a starting point for intervention.

B.9. Determining the degree to which an individual is aware of their executive dysfunction can be helpful in gauging the amount of support he or she will require. For those who possess a high degree of awareness, as well as motivation, the intervention process can be facilitated. For those with limited awareness, a greater degree of external support may be required.

B.10. Although response patterns on self-report behaviour rating scales such as the BRIEF-A can range from strong agreement with other informants to complete denial of any problems, rich clinical information can be gleaned from directly assessing self-reported opinions.

C. The Personality Assessment Inventory (PAI; Morey, 1991) is a very thorough, 344-item measure of psychiatric status that has been standardised against several thousand respondents from both medical and community-based samples. This measure provides the clinician with an ability to provide a nuanced interpretation of clinical findings as well as to achieve a differential diagnosis in shorter time. I relied on the extensive documentation that comes with this psychometric for the interpretation of the various scales.

C.1. It includes several distinct measures of distortion in responding, making it difficult for respondents to manufacture results or to produce results that would be taken as reliable when they are not. These measures of distortion (Rogers, 2008) include:

C.1.1. creating a negative impression (i.e., that one is suffering more than one actually is)

C.1.2. creating a positive impression (that one is suffering less than one is)

C.1.3. malingering

C.1.4. inconsistency in responses

C.1.5. carelessness in reporting

C.2. Specifically, the PAI provides an assessment of the following factors:

C.2.1. somatisation and excessive concern over one's physical health

C.2.2. depression generally and in specific reference to diagnostic criteria relating to its cognitive, emotional, and physical aspects

C.2.3. anxiety generally and in specific reference to diagnostic criteria relating to its cognitive, emotional, and physical aspects

C.2.4. anxiety-related disorders, specifically phobias, obsessive-compulsive disorders, and traumatic stress disorders

C.2.5. mania generally and in specific reference to diagnostic criteria relating to activation, irritability, and grandiosity

C.2.6. paranoia generally and in specific reference to diagnostic criteria relating to hyper-vigilance, persecution, and resentment

C.2.7. schizophrenia-type problems generally and in specific reference to diagnostic criteria relating to delusions, social withdrawal, and thought-disorder

C.2.8. borderline personality disorder generally and in specific reference to diagnostic criteria relating to emotional instability, identity-problems, self-harming, and relational problems

C.2.9. antisocial personality disorder

C.2.10. aggression

C.2.11. stress from life-changes

C.2.12. openness to psychiatric treatment

C.2.13. perception of social support

C.2.14. suicidality and suicide-potential

C.2.15. alcohol- and drug-use

C.2.16. dominance in interpersonal relationships

C.2.17. positive orientation towards social relationships

C.2.18. violence-risk

C.2.19. treatment-potential

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DECLARATION