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Kemp, S., Kapur, N., Graham, C.D. et al. (2022) Functional cognitive disorder : differential diagnosis of common clinical presentations. Archives of Clinical Neuropsychology, 37 (6). pp. 1158-1176. ISSN: 0887-6177

https://doi.org/10.1093/arclin/acac020

Abstract

Background: Cognitive symptoms in the absence of neurological disease are common. Functional cognitive disorder (FCD) has been conceptualised as a cognitive subtype of functional neurological disorder (FND). Although FCD is understood as different from exaggerated or feigned cognitive complaints, previous accounts have provided little practical advice on how FCD can be separated from factitious or malingered cognitive complaints. Also, the distinction of FCD from other medical or mental health disorders that impact on cognition is an area of ongoing study and debate. Diagnostic precision is important to prevent iatrogenesis and for the development of needed treatment protocols.
Method: We summarise the current literature and present seven anonymised case vignettes to characterise the challenges in this area and develop proposals for solutions.
Results / conclusions: Recognising the limitations of categorical diagnostic systems, we position functional cognitive disorder as distinct from feigning and cognitive symptoms of psychiatric disorders, although with overlapping features. We set out typical clinical features and neuropsychological profiles for each category of cognitive disorder and a statistical method to analyse performance validation tests (PVTs) / effort tests to assist in determining feigned or invalid responding.

Introduction

Subjective cognitive symptoms are common in the general population and not exclusively a manifestation of brain pathology. Benign cognitive symptoms are experienced by 5 to 32% of healthy young adults (McCaffrey, Bauer, Palav and O'Bryant, 2006). Some of these individuals present to primary care or memory clinics, where a majority of patients currently referred (around 70 per cent) are not found to be suffering from dementia (Bell, Harkness, Dickson & Blackburn, 2015; Menon & Larner, 2011; Larner, 2014). The generally accepted position among dementia professionals is that clinical history, symptoms and neuropsychological profile can distinguish between different types of dementias and other cognitive complaints. Here neuropsychological examination plays a key role. In the context of brain imaging and other investigations, neuropsychological profiles enable the formulation of clinical diagnoses and inform clinical management. Indeed, research demonstrates that it is possible to distinguish conditions characterised by cognitive impairment on clinical grounds with a high degree of accuracy (Snowden et al, 2011). Among those with mild cognitive impairment (MCI), a state associated with mildly disabling cognitive symptoms, which may not necessarily progress to dementia, the conversion rate to dementia (i.e., Alzheimer's disease) has been found to be 10 to 15% per year (Janoutova, Sery, Hosak & Janout, 2015). In the literature on traumatic brain injury and particularly at the mild end of the spectrum, it is understood that subjective symptoms alone should not be taken as evidence of structural brain damage (Baxendale, Heaney, Rugg-Gunn & Friedland, 2019) since the extent to which someone reports symptoms may reflect the cumulative effects of multiple variables including genetic factors, mental health, medical problems, pain, fatigue and other psychosocial and environmental factors, including the presence of litigation processes. Van den Bergh, Witthoft, Petersen & Brown (2017) proposed an integrative explanatory model for how symptoms and objective physiology can diverge, and how subjective observation and expectation / belief driven inference

can produce symptoms that can be discordant with or independent of bodily dysfunction. There are then, a large group of patients with subjective cognitive complaints whose symptoms are not explained by any identifiable neurological disease and with a benign or non-specific neuropsychological profile.

Various terms have been applied to subjective cognitive symptoms unexplained by dementia or neurological disease including – pseudodementia (Kang et al, 2014) benign senescent forgetfulness (Kral, 1962) depression-related cognitive dysfunction and depressive pseudodementia. The assumption underpinning these terms being that subjective cognitive symptoms reflect normal physiological age-related decline (for example in verbal memory) or have a mental health explanation, primarily depression (Ahern & Semkovska, 2017) or generalised anxiety (Langarita & Gracia-Garcia, 2019) or other mental health disorder. More recently, cognitive symptoms in the absence of underlying organic or other psychiatric cause have been placed within the framework of functional neurological disorder (FND), and termed functional cognitive disorder (FCD) (Teodoro, Edwards & Isaacs, 2018; McWhirter, Ritchie, Stone & Alan Carson, 2020[a]). This has happened at a time when the field of FND more generally has moved away from the previous dominance of the conversion model of Freud and Breuer (1957) towards a neurocognitive understanding of FND as a disorder involving altered attention, sensation and beliefs that can be influenced by many antecedent risk factors including injury, trauma and stress (Edwards, Adams, Brown, Parees & Friston, 2012; Espay et al, 2018) and formulated within a biopsychosocial framework (Reuber, 2009). This is a welcome transition, positioning FCD as a topic for scientific and multidisciplinary study, with testable predictions and potential for much needed new treatment approaches.

The focus of this paper is FCD as it has come to be defined and the recent literature, how FND more widely is understood in the Diagnostic and Statistical Manual of Mental Disorders-Fifth Edition (DSM-V) and how FCD typically presents clinically, with concerns primarily about memory, differentiating FCD from related phenotypes, and in particularly charactering FCD as distinct from feigning and malingering. Within or related to FCD we include fugue state, psychogenic or dissociative retrograde amnesia (Kopelman, 2000), and conditions that have been proposed outside of formal mental health classification systems including cogniform disorder (Delis & Wetter, 2007) and neurocognitive hypochondriasis (Boone, 2009).

The relationship between FND and its cognitive subtype, functional cognitive disorder (FCD) has been outlined in the recent helpful publications by Teodoro et al (2018) and McWhirter et al (2020[a]). Teodoro et al (2018) reviewed the literature on FND generally, fibromyalgia and chronic fatigue symptoms and proposed a unifying theory to explain the differences they found between subjective cognitive complaints and objective neuropsychological deficits. Subjective cognitive symptoms were common and included forgetfulness, distractibility and word-finding problems, but objective deficits on neuropsychological tests were inconsistent. The inconsistent objective deficits on formal testing were attributed to pain, fatigue and excessive interoceptive monitoring detracting from working memory, attention and slowing of cognitive processing. Perfectionism and over-interpretation of cognitive lapses were seen as part of the psychopathology of FND. McWhirter and colleagues (2020[a]) developed this line of reasoning further, presenting a systematic literature review and adding helpful clinical characterisation to differentiate FCD from degenerative brain

disease to assist with diagnosis. For example, FCD patients being more aware of cognitive problems and giving detailed descriptions in clinic compared to patients with degenerative brain disease. Using video recordings of how patients with 'functional memory disorder' present their memory complaints to neurologists, Alexander, Blackburn & Reuber (2019) found that patients typically gave detailed and eloquent accounts of symptoms and disrupted functioning, so detailed as to be incongruous with objective memory and cognitive deficits. The authors likened the inconsistency between cognitive competence in clinic and complaints about memory dysfunction with the Hoover sign (i.e., involuntary extension of a "paralysed" leg when flexing the contralateral leg against resistance). Importantly, the review by McWhirter et al (2020[a]) notes the likely iatrogenic harm associated with an incorrect diagnosis and states that FCD is likely to be under-recognised in memory clinics. At what is an early phase in understanding FCD, they adopt a broad terminology of subjective cognitive symptoms, and cluster various non-neurological cognitive symptoms such as; high awareness of cognitive difficulties and giving a detailed account in clinic, as an indicator of the presence of FCD, and presenting this approach as a necessary preliminary step for future research.

Poole, Cope, Bailey & Isaacs (2019) characterised FCD and related psychiatric conditions, but the authors do not set out the neuropsychological features or offer guidance on FCD as distinct from non-credible clinical presentations.

Ball et al (2000) helpfully discuss the differential diagnosis between dementia and functional neurological disorders (FCD specifically) as causes of cognitive difficulties. This is area of on-going clinical characterisation. Kapur, Kemp & Baker (2021) have challenged Ball et al (2000) for overstating 'internal inconsistency' as a defining clinical feature of FCD, for insufficient consideration of the role of neuropsychological examination in differential diagnosis and for omitting to address the difference between FCD and feigning / exaggeration. Another recent review by McWhirter, Ritchie, Stone & Carson (2020[b]) suggested that this differentiation could not be reliably achieved with current effort tests / performance validation tests (PVTs). They found no reliable differences in the rates of performance validity test failures between various clinical populations. However, significant weaknesses in the conceptualisation, methodology and analyses has generated critique from groups of neuropsychologists in the U.K. (Kemp, Kapur, Bunnage, Dorris, Moore & Friedland, 2020) and U.S. (Larrabee, Boone, Bianchini, Rohling &, Sherman, 2020).

The purpose of the present paper is to characterise the differences between FCD and established mental health and anxiety based clinical presentations more clearly and to clarify the distinction of FCD from malingered, factitious or exaggerated clinical presentations. Feigning / malingering is a topic of particular interest for neuropsychology, with a rapidly increasing effort and symptom validation test literature (Lippa, 2018) and recent clinical guidelines in the United States and U.K. (Bush, 2005; Heilbronner, Sweet, Morgan, Larrabee & Millis, 2009; Sweet, et al. 2021; British Psychological Society, 2009 and 2021). Prior work has provided helpful clinical descriptions of FCD to assist physicians working in memory clinics, to clarify links between FCD and other manifestations of FND and to produce an understanding of FCD that is grounded in modern concepts of cognitive psychology and biopsychosocial modelling (Wakefield et al, 2017; Blackburn & Reuber, 2019).

However, these previous papers say relatively little about the differentiation of FCD from feigned cognitive symptoms.

The need for a clear distinction of these phenomena may not be a prominent issue in memory clinics, but it is a common and important problem in a medico-legal context, with patients in litigation, particularly after head trauma, presenting in multiple clinical settings. Aside from addressing a matter of societal importance, the clarification of the separation from feigned symptoms will also make a contribution to the conceptualisation of FCD. As diagnostic imprecision between dementia and FCD can cause iatrogenic harm, incorrect classification between functional cognitive problems and feigning may be no less harmful, with a false opinion of the presence of feigning likely to be particularly damaging. The distinction between FCD and feigning has, to date, largely been overlooked in the emerging literature on FCD and is one important focus of this paper. As the basis for discussion of the concept of FCD, we present seven case studies to illustrate what we regard as typical features of health anxiety involving 'fear of forgetting', FCD and feigning / non-credible presentations to assist with diagnosis, treatment approaches and on-going research. We outline a conceptual framework of overlapping conditions (Table 1) but with some distinct clinical and neuropsychological features.

The seven vignettes are based on real patients from clinical and medico-legal practice, but other than the essence of the clinical presentation and neuropsychological test performance required to serve the aims of this paper, all the details have been changed substantially to ensure anonymity and safeguard patient confidentiality. A summary of neuropsychological test data in key cognitive domains are included for each vignette.

Table 1: Framework of cognitive symptoms ranging from neurological disorders to feigning / exaggeration

1. Neurological disorders, brain trauma and medical disorders	2. Mental health disorders	3. Health anxiety involving 'fear of cognitive lapses'	4. Functional Cognitive Disorder (FCD)	5. Feigning / exaggeration / malingering / factitious disorder
		The recent literature tends to classify both groups as FCD		
Cognitive symptoms which are the direct result of structural brain pathology or clearly identified pathophysiology, arising from: a) brain injury, dementia, brain tumor, progressive neurological disease etc. This would include conditions such as; seizures, transient global amnesia, transient ischaemic attacks. b) Also, brain / cognitive dysfunction in the context of medical disorders (e.g., endocrinological disorders, diabetes, cardiac and respiratory disorders). c) In this group we also position delirium and drug induced cognitive symptoms.	Cognitive symptoms as part of a clinical mental health disorder including anxiety, depression, PTSD, dissociative disorder without a primary cognitive focus, and somatic symptom and related disorders as defined in formal mental health classification systems.	Mental health symptoms that involve a specific fear of illness associated threat monitoring, with patients becoming particularly anxious about their cognitive performance, generally memory, sometimes overusing strategies to help cognitive functioning. Can occur in the context of normal lapses, anxiety about dementia, or following head injury, with disproportionate subjective cognitive concerns relative to objective neuropsychological testing. General mental health and daily functioning is intact.	Sub type of functional neurological disorder (FND) characterized by: a) excessive attention on cognitive performance, maladaptive beliefs, meta-cognitive errors, and repetitive illness behaviors conforming to a biopsychosocial functional disorder. Or b) Fugue state and dissociative retrograde amnesia involve more marked symptoms, but occurring less commonly. Psycho-genic language and visual disorders.	Non-credible neuropsychological presentations, with Performance Validity Tests / effort test failure and a symptom profile lacking neuropsychological and psychological coherence, which is inconsistent with any diagnostic classification. Feigning / exaggeration is motivated by external incentives such as avoiding work or financial compensation, where factitious disorder is motivated by the internal incentive of adopting the sick role. While malingering and factitious disorder will usually have a major conscious component, some degree of unconscious and automatic forms of responding may occur.

Note: We have focussed on scenarios where cognitive symptoms are the primary concern for patients, and that concern is generally focussed on memory. We have not included all conceivable clinical presentations that include cognitive symptoms, for example caused by fatigue or sedative medications, and we have not included diffuse and pervasive cognitive symptoms such as 'brain fog' that patients in all the above categories could report and are common in clinical groups such as CFS / ME, and in the emerging evidence on long COVID.

Case A: History and clinical presentation:

Mr A was given an accidental overdose of Caffeine (30g instead of 30mg) when taking part in a study. He was initially tremulous and unstable with ventricular tachycardia and was taken to hospital. Glasgow Coma Scale (GCS) score was 15/15 at initial assessment in Accident & Emergency (A&E). There was no loss of consciousness. He was anxious and agitated. He was cared for on the Intensive Care Unit and given dialysis to remove caffeine from his blood, along with other medical treatments for arrythmia and hypertension. He was transferred from ICU to a

renal ward. In spite of going on to successfully complete his medical degree, successfully completing an intercalated degree, and securing a job as a junior doctor, Mr A continued to report cognitive symptoms, in particular with anterograde memory and sustained attention. 3T MRI (brain) scan was normal. Neurological examination was normal. Detailed neuropsychological examination at 4-years post-accident showed well above average premorbid general intellectual ability and an entirely intact cognitive profile, other than occasional dips in performance that were attributed to test anxiety and was within the base rate incidence of low scores (Iverson, Brooks & Holdnack, 2012). Performance validation testing (PVT) was intact. There were no neurobehavioural symptoms suggestive of brain injury. The psychological component of the neuropsychological examination was more revealing in showing protracted adjustment symptoms with low mood, anxiety and mild PTSD symptoms. He was initially nauseous in clinical settings and generally vigilant for threat. In spite of impressive career achievements, he continued to doubt himself, had dysthymic level low mood and had become focussed on cognitive lapses possibly threatening the safety of his clinical work and his career as a doctor. Mr A was using various memory support strategies, but not to a level that was discernibly different to his peers. He had responded partially to Eye Movement Desensitization and Reprocessing (EMDR) therapy.

Neuropsychological data:

Cognitive domain	**Test**	**Index score / pooled Z score**
Premorbid ability:	TOPF	Predicted full-scale IQ=115
Obtained ability:	WAIS-IV: Verbal IQ Performance IQ Full-scale IQ	 116 121 118
Performance validation tests (PVT):	WMT TOMM Embedded PVT (RDS)	Pass Pass Pass
Verbal memory:	BMIPB (List Learning + Story Recall)	Z = 0.86
Non-verbal memory:	BMIPB (Design Learning + Figure Recall	Z = 1.10
Information processing:	WAIS-IV	119
Frontal-executive tests:	Hayling Test Brixton Test D-KEFS: Letter Fluency D-KEFS: Category Fluency D-KEFS: Colour-Word Interference	Average to Good Good Age Scaled Score=10 Age Scaled Score=11 Age scaled scores=11,10,12,13
Psychological measures:	HADS	Anxiety = Normal Depression = Normal

TOPF: Test of Premorbid Function / WMT: Word Memory Test / RDS: Reliable Digit Span / BMIPB: BIRT Memory and Information Processing Battery / D-KEFS: Delis-Kaplan Executive Function System / HADS: Hospital Anxiety & Depression Scale.

Formulation: There was no neurological, neuroimaging, neurocognitive or neurobehavioral evidence of brain injury. This potentially fatal caffeine overdose triggered a protracted period of psychological adjustment symptoms of low mood, partial PTSD, general anxiety and anxiety about cognitive lapses impacting on his career. Mr A graduated from medical school with a first class degree and his career was progressing well. The cognitive symptoms were formulated as subjectively real (i.e., genuine, but discrepant from the objective neuropsychological test findings and without evidence of a structural basis) and secondary to an adjustment disorder. We position Mr A in category 2 in Table 1, albeit with clinical features of category 3.

Case B: History and clinical presentation:

Ms B was knocked off her bicycle on her way to work as a full-time Personal Assistant at a busy engineering firm. The ambulance records state no loss of consciousness and a GCS score of 15/15 throughout. Careful examination in the neuropsychology clinic two years post-accident revealed no retrograde amnesia (RA) and responses on the Rivermead Post-Traumatic Amnesia (PTA) Protocol indicated no PTA. She was seen in A&E, treated for facial lacerations and dental injuries and discharged. She did not undergo acute CT head scan and later MRI brain scan (conducted because of subjective cognitive complaints) was normal. In clinic, 2-years post-injury, Ms B was alert and articulate. Speech was fluent and structurally normal. There were no neurobehavioural signs suggestive of brain injury. She returned to work after 8-weeks, without making a phased return. Her line manager was on long-term sick leave on her return. She returned to extra duties, minimal support, had some cognitive lapses and struggled with low mood, anxiety and loss of confidence. After an occupational health assessment, she commenced a phased return to work and managed to increase to 24-hours per week, with considerably reduced duties and this was the position 2-years post-injury. At 2-years, Ms B reported good physical recovery and a good dental repair. She reported on-going problems with sustained and divided attention, poor anterograde memory with on-going lapses such as forgetting to pick up the children from school and forgetting appointments. She was tearful in clinic when talking about her cognition. She did not report problems with mental speed or frontal-executive functioning, and her subjective cognitive complaints were not typical of traumatic brain injury. She had become extremely reliant on various memory support strategies including Apps, Google Calendar, multiple reminder lists and several diaries for work and home. She reported sometimes going into meetings at work without pre-prepared notes and panicking. She worried about underlying structural brain damage, possible later dementia and had phases of Googling brain injury symptoms and recovery times, but her anxiety about day-to-day cognitive functioning and fear of lapses were more consistent clinical features. She was otherwise in good psychological health, but worried about the next day at work and sleep could be poor. The neuropsychological profile showed average premorbid IQ and was intact other than the odd lapse that was within the base rate incidence of low scores. Performance on PVTs / effort tests was close to ceiling. There was no formal pre-injury mental health history, but Ms B was a frequent GP attendee with various physical symptoms and health anxiety.

Neuropsychological data:

Cognitive domain	Test	Index score / pooled Z score
Premorbid ability:	TOPF	Predicted full-scale IQ=94
Obtained ability:	WAIS-IV: Processing Speed Index (PSI)	 111
Performance validation tests (PVT):	WMT TOMM Embedded PVT (RDS)	Ceiling level pass Pass Pass
Verbal memory:	BMIPB (List Learning + Story Recall)	Z = 0.51
Non-verbal memory:	BMIPB (Design Learning + Figure Recall	Z = 0.32
Frontal-executive tests:	Hayling Test Brixton Test D-KEFS: Letter Fluency D-KEFS: Category Fluency D-KEFS: Colour-Word Interference	High Average (Total) Average Age Scaled Score=10 Age Scaled Score=12 Age Scaled Scores=13,13,10,11
Psychological measures:	HADS	Anxiety = Mild Depression = Mild

TOPF: Test of Premorbid Function / WMT: Word Memory Test / RDS: Reliable Digit Span / BMIPB: BIRT Memory and Information Processing Battery / D-KEFS: Delis-Kaplan Executive Function System / HADS: Hospital Anxiety & Depression Scale.

Formulation: There was no clinical indication of traumatic brain injury. Ms B could remember the moment of injury, which excludes PTA, and later 3T MRI (brain) scan reported by a consultant neuroradiologist was normal. At 2-years, Ms B was in essentially good psychological health. There was mild vigilance about road safety, which is a common psychological consequence of road traffic accidents, short of PTSD. Of more relevance was moderate anxiety about forgetting, over-use and excessive dependence on memory support strategies. Sleep remained disturbed. She was under-functioning at work comparted to her premorbid ability. We position Ms B in category 3 in Table 1.

Case C: History and clinical presentation:

Ms C was involved in a road traffic accident when her partner collapsed at the wheel. Her child was in the car. She sustained orthopaedic injuries including a spinal fracture, and a right scapula fracture and a nasty scalp laceration. She was not comatose. Glasgow Coma Scale (GCS) score was 13-15 at the scene and consistently 15/15 once at hospital. Acute CT (head) was normal and later MRI (brain) was normal. In the neuropsychology clinic 3-years post-injury, there was no retrograde amnesia. Careful administration of the Rivermead PTA Protocol indicated a likely PTA probably less than 1-hour, but duration was difficult due to Morphine and psychological distress. At 3-years, Ms C's clinical presentation was characterised by an interplay of pain, anxiety, mild low mood and poor sleep. She was frequently tearful, but entirely amenable and she engaged well with neuropsychological examination. There were no neurobehavioural signs of significant brain damage. She reported good and bad days with her attention, anterograde memory, mental processing speed and frontal-executive functioning, with no overall improvement over 3-years. She also reported loss of remote autobiographical memories. When asked about skills, she reported sometimes forgetting where files are stored on her computer, and on occasions, forgetting her bank PIN number. She once forgot what the dials mean on her microwave oven and this was not due to visual agnosia. She had lost confidence in her memory and gave a clear account of excessive reliance on list, notes and checking. She had difficulty reporting her beliefs, but worried about

her future physical and mental health, and about the possibility of later dementia. She hoped that she was not brain damaged, and did not present with the fear-avoidant coping style typical of pain disorder. Scores on the Pain Catastrophizing Scale were not particularly high. But there was marked avoidance of situations involving cognitive demand. She had returned to her job in the insurance industry, but on half-time hours. She worried about making mistakes, had developed an elaborate system of notes and regularly asked colleague to check her work. Her work appraisals were positive, but she failed some work-related training due to anxiety about having to retain new information. She required support to pass the training. The trainer reported that the prior failure was due to anxiety and not Ms C's ability. She presented with a number of PTSD symptoms, short of full-blown PTSD (DSM-V). She had a pre-injury history of irritable bowel syndrome, and the GP note suggested a tendency to somatise her distress. The neuropsychological profile showed low end of average premorbid IQ and no impairment in any domain of ability. She passed PVTs. The qualitative aspect of neuropsychological testing was more significant than the data. Ms C was highly aware of her cognitive performance, anxious about making mistakes and she would not guess. She did not particularly respond to reassurance that the neuropsychological tests are generally designed so that people do not get all the answers correct.

Neuropsychological data:

Cognitive domain	**Test**	**Index score / pooled Z score**
Premorbid ability:	TOPF	Predicted full-scale IQ=92
Obtained ability:	WAIS-IV: Verbal IQ Performance IQ Full-scale IQ	 93 97 101
Performance validation tests (PVT):	WMT TOMM Embedded PVT (RDS)	Pass at ceiling level Pass Pass
Verbal memory:	BMIPB (List Learning + Story Recall)	Z = 0.12
Non-verbal memory:	BMIPB (Design Learning + Figure Recall	Z = -0.24
Information processing:	WAIS-IV	110
Frontal-executive tests:	Hayling Test Brixton Test D-KEFS: Verbal Fluency D-KEFS: Category Fluency D-KEFS: Colour-Word Interference	Average (total) Good. Age Scaled Score=10 Age Scaled Score=11 Age Scaled Scores=10,11,9,10
Psychological measures:	HADS	Anxiety = Mild Depression = Mild
Pain	Pain Catastrophizing Scale	Z = -0.71 (Relative to a clinical sample)

TOPF: Test of Premorbid Function / WMT: Word Memory Test / RDS: Reliable Digit Span / BMIPB: BIRT Memory and Information Processing Battery / D-KEFS: Delis-Kaplan Executive Function System / HADS: Hospital Anxiety & Depression Scale.

Formulation: Ms C sustained a mild traumatic brain injury and 3-years later presented with some PTSD symptoms, some generalised anxiety, but her anxiety was focussed on her cognitive performance. This was clear from the clinical interview, and obvious to observation on neuropsychological tests. There were some subjective symptoms inconsistent with known responses to brain trauma, but she did not present with established patterns of illness behaviour and was functioning at a reasonably good level. She was determined to improve further, flexible in her

problem-solving and amenable to psychological advice. In Table 1, we position Ms C in category 3, but with some features of category 4.

Case D: History and clinical presentation:

This Professor of Mathematics was knocked down whilst crossing the road sustaining a fractured right femur and multiple lacerations. He did not lose consciousness and GCS score was 15/15 throughout, but he possibly struggled to verbalise his address at the scene. Acute CT (head) scan was normal. In the neuropsychology clinic 4-years later, Professor D's presentation was uncommon from the outset when he reported being unable to concentrate for period of longer than 15-minutes or so, after which he would pause, stop speaking and look out of the window for a few minutes. Speech was prolix. He was anxious and sardonic at times. In spite of effort to build rapport, he was difficult to interview, although not frankly uncooperative. Clinically, there was no RA and no PTA. Professor D reported multiple physical and neuropsychological symptoms, including diffuse cognitive complaints. He reported developing a habit of verbalising thoughts as soon as they came to mind in case he forgot. He had stopped reading books, stopped driving and given up his career in academia. Four years post-accident, he presented with a dramatic fear-avoidant style of coping, and a strong belief in brain injury. He was doing voluntary work in a school. He reported going from the top of the tree to the bottom of the heap. His wife was excessively supportive, and both were extremely reliant on brain injury cognitive approaches. Both understood his symptoms as brain damage, and some doctors had diagnosed post-concussional syndrome. The cognitive profile showed superior premorbid IQ (98th centile). He passed a range PVTs and symptom validation tests (SVTs), covering different cognitive domains, with all scores above cut-off. Given the absence of PTA, there was not a brain injury reasons to administer neuropsychologist tests, although a brief battery of tests was administered to determine his abilities and for qualitative purposes. The profile was patchy with reverse gradients in performance (i.e., the opposite of the pattern of scores expected with brain damage – sometimes referred to easy / hard difference or as 'profile analysis' on the Word Memory Test (WMT) / Medical Symptom Validation Test (MSVT); Green, Montijo & Brockhaus, 2011) and some scores too low to be explained even by severe brain injury, including a decline in general IQ of 28 points. As with Ms C, the qualitative aspects of neuropsychological elicited clear anxiety about cognitive performance, excessive awareness of the vicissitudes of test performance and various maladaptive coping techniques, such as fixation on subvocalization and rehearsal on verbal tests, frequently pausing to look out of the window, deliberately working extremely slowly on timed tests due to anxiety about making errors and declining to do some tests. This was consistent with Professor D's cognitive style characterised by dichotomous / black and white reasoning and characteristic of low mood, self-criticism and low self-esteem. He had gone from a Professor of Mathematics to someone doing voluntary work in a school and struggling to explain basic maths to teenagers. The history indicated a perfectionist individual from a high achieving and authoritarian family and a strong predisposition to develop psychological symptoms at times of stress and adversity (obtained from the medical and mental health records).

Neuropsychological data:

Cognitive domain	Test	Index Score / pooled Z score
Premorbid ability:	TOPF	Predicted full-scale IQ=131
Obtained ability:	WAIS-IV: Verbal IQ Performance IQ Full-scale IQ	 92 121 103
Performance validation tests (PVT):	WMT TOMM Embedded PVT (RDS)	Pass Pass Pass
Verbal memory:	BMIPB (List Learning + Story Recall)	Z = -1.23
Non-verbal memory:	BMIPB (Design Learning + Figure Recall	Z = -0.74
Information processing:	WAIS-IV	82
Frontal-executive tests:	Hayling Test Brixton Test D-KEFS: Verbal Fluency D-KEFS: Category Fluency D-KEFS: Colour-Word Interference	High Average (total) Superior Age Scaled Score=10 Age Scaled Score=5 Age Scaled Scores=4,1,7,8
Psychological measures:	HADS	Anxiety = 1 Depression = 5

TOPF: Test of Premorbid Function / WMT: Word Memory Test / RDS: Reliable Digit Span / BMIPB: BIRT Memory and Information Processing Battery / D-KEFS: Delis-Kaplan Executive Function System / HADS: Hospital Anxiety & Depression Scale.

Formulation: There was no clinical or radiological evidence of traumatic brain injury, even at the mild / concussive end of the spectrum, although some doctors had diagnosed PCS and treatment had reinforced this belief. Given the striking clinical presentation and neuropsychological test performance, we position this case as a good example of FCD, category 4 in Table 1.

Case E: History and clinical presentation:

Ms E was a 27-year old nursery nurse involved in a minor road traffic accident; Her young daughter was in the car. Neither Ms E nor her daughter initially attended A&E, but Ms E attended some days later reporting right-sided tingling and numbness. CT (head) scan was normal. Over several months, she developed more pronounced right-sided symptom and unusual visual problems. The physical symptoms resolved, but she remained off work, although motivated to return, and approximately 1-year post-accident she was seen in the neuropsychology clinic with unusual memory complaints. There was no RA and no PTA, but the GP had diagnosed post-concussion syndrome. She gave an emotional account of normal memory for the course of 1-day, but waking-up each morning with no recall of the previous day. She reported no memory of recent events, although her mother, who accompanied her to all consultations, reported examples of contrary evidence. She also reported patchy remote autobiographical memory, but without a classic temporal gradient. Ms E was unable to give an account of her daily functioning beyond the events of that day. Her mother reported that her employer was sympathetic and had placed her on long-term sick leave due to safeguarding concerns, and were awaiting a neuropsychological opinion. Ms E presented with normal speech. There were no neurobehavioural signs. Affect was variable in the consultation and attributed to psychological factors (the referring neurologist had not diagnosed any structural basis for this, for example

pseudobulbar affect). There was no formal thought disorder. It was not possible to assess for PTSD because Ms E reported no post-accident recall other than for the events of that day. The mother reported some mild pre-accident work stress. Neuropsychological testing showed average premorbid and an intact profile, although there were some reverse gradients (i.e., a pattern of scores the opposite way around to brain damage) in the profile. Ms E was not in litigation regarding the road traffic accident.

Neuropsychological data:

Cognitive domain	Test	Index score / pooled Z score
Premorbid ability:	TOPF	Predicted full-scale IQ=104
Obtained ability:	WAIS-IV: Verbal IQ Performance IQ Full-scale IQ	 109 100 106
Performance validation tests (PVT):	WMT TOMM Embedded PVT (RDS)	Pass Pass Pass
Verbal memory:	BMIPB (List Learning + Story Recall)	Z = -0.32
Non-verbal memory:	BMIPB (Design Learning + Figure Recall	Z = 0.19
Information processing:	WAIS-IV	101
Frontal-executive tests:	Hayling Test Brixton Test D-KEFS: Verbal Fluency D-KEFS: Category Fluency D-KEFS: Colour-Word Interference	Good High Average Age Scaled Score=10 Age Scaled Score=11 Age Scaled Scores=9,11,5,7
Psychological measures:	HADS	Anxiety = Moderate Depression = Mild

TOPF: Test of Premorbid Function / WMT: Word Memory Test / RDS: Reliable Digit Span / BMIPB: BIRT Memory and Information Processing Battery / D-KEFS: Delis-Kaplan Executive Function System / HADS: Hospital Anxiety & Depression Scale.

Formulation: Clinically, Ms E sustained a whiplash injury. She initially manifested physical symptoms unexplained by physical changes on investigations, which resolved, but she developed FCD, with subjective cognitive symptoms consistent with portrays of amnesia in film and the arts. She engaged with a treatment programme of psychoeducation and a Cognitive Behaviour Therapy-based intervention involving graded activity to challenge her beliefs and made a slow phased return to activity and work. There was no evidence of exaggeration or external incentive. We position this case in category 4 in Table 1.

Case F: History and clinical presentation:

Mr F, a senior accountant, was a cyclist involved in a collision with a pedestrian, sustaining a fractured left radius and ulna. He was hospitalised for 1-night and discharged home. He did not lose consciousness and GCS score was 15/15 at the scene. He was given Morphine and non-narcotic inhalation analgesia (a mixture of nitric oxide and oxygen also used in childbirth). Acute CT (head) scan was normal and subsequent MRI (brain) scan also reported as normal. Clinically, there was no RA. At clinical interview prior to testing, Mr F appeared evasive, giving atypical and inconsistent responses to questions. This response style did not settle over the course of a full-day examination with two different clinicians. It was not possible to determine PTA via the standard retrospective technique because of poor quality responses and complains of on-going deterioration in anterograde memory and in

general awareness. At 2-years post-accident, he had returned to work with the same firm, but on part-time hours and with considerably reduced duties. He reported elaborate and diffuse physical and neuropsychological symptoms. The neuropsychological complaints were inconsistent with known responses to brain trauma. He reported loss of many previously well-learned skills, but this was not consistent with the records or his daily activities. Mr F had a history of depression, anxiety and the GP notes referred to perfectionist tendencies. There was a history of work stress and a 'nervous breakdown' 4-years pre-accident. The neuropsychological profile showed average premorbid IQ (62nd centile) and a decline of 21 IQ points (comparing estimated with obtained full-scale IQ). On standard tests, Mr F could state his name and address, but was not fully orientated in time or place. He travelled to the examination alone on public transport, but got lost several times going from the consulting room to the waiting room and asked for a map of the clinic. There were multiple reverse gradients in the cognitive profile (i.e., instances of poor performance on low-demand tests, but good performance on high-demand tests, which is the wrong way around for brain damage). There was marked inconsistency between the test performance and Mr F's subjective cognitive complaints, such as reporting severe word-finding problems, but performing at the 98th centile on a confrontation naming test. He failed serial PVTs / effort tests, with markedly poor scores, and the PVT data were subject to binomial calculations (see box 1 for the method).

Neuropsychological data:

Cognitive domain	**Test**	**Index score / pooled Z score**
Premorbid ability:	TOPF	Predicted full-scale IQ=105
Obtained ability:	WAIS-IV: Verbal IQ Performance IQ Full-scale IQ	 96 110 84
Performance validation tests (PVT):	WMT TOMM Embedded PVT (RDS) Coin-in-Hand Test	IR=35% / DR=20% (failed profile analysis). 14/50 and 17/50 Fail 3/10 (Fail)
Verbal memory:	BMIPB (List Learning + Story Recall)	Z = -1.25
Non-verbal memory:	BMIPB (Design Learning + Figure Recall	Z = -0.98
Information processing:	WAIS-IV	68
Frontal-executive tests:	D-KEFS: Verbal Fluency D-KEFS: Category Fluency D-KEFS: Colour-Word Interference	Age Scaled Score=9 Age Scaled Score=8 Age Scaled Scores=1,1,1,2
Naming	Graded Naming Test (GNT)	98th centile
Psychological measures:	HADS	Anxiety = Severe Depression = Severe

TOPF: Test of Premorbid Function / WMT: Word Memory Test Test (IR-Immediate Recognition – DR = Delayed Recognition) / RDS: Reliable Digit Span / BMIPB: BIRT Memory and Information Processing Battery / D-KEFS: Delis-Kaplan Executive Function System / HADS: Hospital Anxiety & Depression Scale.

Formulation: Mr F broke his arm when involved in a cycling accident. At 2-years post-accident, although working part-time at reduced capacity, he reported many symptoms that did not conform to a pattern consistent with neurological disease or brain injury. Mr F was unable to do basic cognitive tasks such as count from 10-1 reliably and he failed multiple PVTs, with instances of under-chance performance. We have positioned this case in category 5 in Table 1.

Box 1. Suggestions for evaluation of under-chance PVT results:

Although relatively uncommon, given the clinical importance of statistically significant below-chance performance, it is important that clinicians are familiar with a test method. Of the methods available, the easiest is probably binomial theorem using the on-line binomial probability calculator at the Vassarstats website: http://vasserstats.net/textbook/ch5apx.html.

We use patient F's test results to illustrate this method:

On a 40-item forced-choice test, Mr F obtained scores of 14/40 and 8/40. Both are significantly under chance (binomial probability significance levels P <.05 and P <.001 respectively). In other words, the probability that a score of 8/40 could have arisen out of 'unlucky guessing' was less than 0.001 per cent. Hence, on balance, the under-performance was deliberate. With the lower score of the two trials (i.e., 8/40), if one converts the binomial probability to a fraction and reduces the fraction, this series of calculations gives a probability of obtaining a score of 8 or fewer correct responses on a 40-item forced-choice test as $91/10^6$. In other words, a score of 8 or fewer would be seen 91 times in 1 million trials of the test.

Note:
Under-chance PVT performance is relatively uncommon, and it is important to remember that PVTs generally emphasize specificity over sensitivity (have a minimum specificity generally of 90 per cent), hence PVT failure probabilistically rules in invalid responding, but a pass might not rule out invalid responding.

Case G: History and clinical presentation:

Ms G was a 22-year old woman referred to the neuropsychology clinic 1-year after falling off a step ladder and sustaining a likely mild concussive head injury. She did not attend A&E, but consulted her GP a few days later complaining of mild, but typical post-concussional symptoms. Ms G reported a PTA of around 7-days, but this was inconsistent with having returned to college after 2-days, and completing a National Vocational Qualification (NVQ) in Hair and Beauty. There was brief PTA. MRI (brain) scan was normal. Neurological examination acutely and at 1-year was normal. In the neuropsychology clinic, the mother was adamant that due to attentional problems and fatigue, Ms G would not tolerate more than 45-60-minutes, and unusually even after severe brain injury, the neuropsychological examination spanned four brief consultations. Speech was normal and Ms G did not present with neurobehavioural signs of brain trauma. She was difficult to engage at examination and gave an imprecise account of multiple and severe physical and cognitive symptoms, but was driving and proud to own a convertible car. There was much variability in self-reported symptoms to different doctors. She drove alone to see friends and without difficulty. She had not worked since completing the NVQ course several months post-accident. There was a history of shyness and anxiety during the school years. At 1-year, she presented with mild low mood, anxiety, a passive coping style and was not working. A counsellor had suggested a disorder of diminished motivation secondary to brain injury. Neuropsychological examination showed low end of average premorbid IQ (25^{th} centile). She failed serial PVTs including stand alone and embedded tests, with scores typically at-chance. The cognitive profile contained many instances of test performance inconsistent with daily functioning and reverse gradients in performance. She failed 'profile analysis' on the Word Memory Test (i.e., the profile was consistent with insufficient test effort and inconsistent with a

significant memory disorder). There were instances of normal performance on some standard memory tests, which are more demanding tests than PVTs / effort tests. Ms G was in litigation and subsequent surveillance raised significant concerns about her genuineness.

Neuropsychological data:

Cognitive domain	Test	Score / pooled Z score
Premorbid ability:	TOPF	Predicted full-scale IQ=90
Obtained ability:	WAIS-IV: Verbal IQ Performance IQ	 76 75
Performance validation tests (PVT):	WMT TOMM Embedded PVT (RDS) Coin-in-Hand Test	IR 55% and DR 60% (failed profile analysis). 32/50 and 32/50 Fail 9/10
Verbal memory:	BMIPB (List Learning + Story Recall)	Z = -1.23
Non-verbal memory:	BMIPB (Design Learning + Figure Recall	Z = -0.58
Information processing:	WAIS-IV	70
Frontal-executive tests:	D-KEFS: Verbal Fluency D-KEFS: Category Fluency D-KEFS: Colour-Word Interference	Age Scaled Score=10 Age Scaled Score=10 Age Scaled Scores=3,2,1,1
Psychological measures:	HADS	Anxiety = Severe Depression = Severe

TOPF: Test of Premorbid Function / WMT: Word Memory Test (IR-Immediate Recognition – DR = Delayed Recognition) / RDS: Reliable Digit Span / BMIPB: BIRT Memory and Information Processing Battery / D-KEFS: Delis-Kaplan Executive Function System / HADS: Hospital Anxiety & Depression Scale.

Formulation: Ms G fell from a ladder in a shop and probably sustained a brain injury at the mild end of the mild spectrum. Although there were clinical features that would place her in category 4 in Table, 1, her presentation lacked psychological coherence, she failed PVTs including 'profile analysis', presented inconsistently at different examinations and surveillance footage revealed significant inconsistences between her self-reported difficulties and her observed daily functioning. We have positioned this case in category 5 of Table 1.

Table 2: Clinical characteristics of (i) Health Anxiety with Fear-of-Forgetting (ii) FCD (iii) Exaggeration / Malingering

Clinical feature / subjective complaint / neuropsychological and effort / symptom validation test performance.	**Health anxiety involving 'fear of cognitive lapses'.**	**Functional neurological disorder-cognitive subtype (FCD)**	**Feigning / exaggeration / malingering.**
Subjective cognitive complaints	Common	Common	Common
Diverse and elaborate subjective cognitive complaints	Quite common	Common	Common
Wider diffuse symptom complaints	Rare	Common	Common
Subjective reports of on-going post-illness-injury / post-accident cognitive deterioration	Rare: but patients typically state that their	Sometimes	Common

	memory hasn't improved.		
Discrepancy between subjective cognitive complaints and daily / occupational functioning or 'Internal inconsistence' (e.g., reporting severe attentional problems, but performing complex cognitive tasks day-today).	Common	Common	Common and often dramatic (e.g., failing simple bedside orientation questions including present location when travelled to the consultation alone).
Frequent Google searching / information seeking about symptoms and feared diagnoses	Common	Probably quite common	Rare
Subjective cognitive symptoms inconsistent with known neuropsychological responses to brain trauma or neuropathology	Rare – patients tend to have anxiety and vigilance to performance in one or two cognitive domains	Common	Common
Discrepancy between neuropsychological test findings and known patterns of brain function	Sometimes – test anxiety can be a feature and can impact on test performance	Common	Common
Discrepancy between neuropsychological test findings and daily and occupational functioning (e.g., memory performance <5th centile, but no indication of amnesia in clinical presentation or daily functioning)	Rare	Common	Common
Discrepancy between self-reports and the medical records / documented history	Rare	Sometimes	Common
If possible traumatic brain injury, not being able to establish an end point to post-traumatic amnesia (PTA) when assessed via the standard retrospective clinical technique (Rivermead Protocol).	Rare	Sometimes	Common
Presence of substantial external inventive	Rare	Rare	Common
Observational or video evidence of discrepancy between presentation in	Unknown / presumed rare	Essentially unknown / presumed rare	Sometimes / common

clinic / subjective complaints and daily functioning			
Complaints of loss of well learned skills, such as unable to use washing machine, unable to sign a cheque, unable to log onto a computer.	Rare	Rare	Sometimes / common
Wider implausible symptoms such as; failing simple bedside orientation questions, forgetting how to sign your name, forgetting to eat, forgetting how to use familiar domestic appliances, waking up and seeing different coloured shapes, forgetting how to lock the front door, experiencing a tingling sensation that starts at the tip of the nose and radiates to both ears etc.	Rare	Sometimes	Common
Overuse / excessive reliance on cognitive strategies / assistive technology.	Common	Common	Rare
Odd behavior at the examination (e.g., asking for directions back to the waiting room, asking for a map to go to a local café for lunch when the patient travelled to the consultation independently.	Rare	Sometimes	Common
A characteristic fear-avoidant psychological style	Common	Common	Rare
A generally odd clinical presentation and constellation of subjective complaints that lacks psychological coherence for a biopsychosocial formulation.	Rare	Rare	Common
Single PVT / symptom validation test failure or borderline performance	Rare	Rare / sometimes	Common
Multiple effort / symptom validation test failure, with a 'reverse gradient' of performance across tests	Rare	Rare	Common
At-chance / under-chance PVT / SVT performance, as determined by binomial theory.	Rare	Rare	At-chance is not uncommon. Under-chance performance occurs sometimes and is definitive evidence of deliberate under-performance.

Discussion

Subjective cognitive symptoms are common in the general population and clinical practice, and not specific to neurological and psychiatric diagnoses. The previous literature has only provided limited detail on the neuropsychological characterisation of patients with FCD, and especially their differentiation from those with feigned cognitive difficulties. Research comparing cognitive profiles of patients with functional memory disorder versus with those with amnestic-mild cognitive impairment is scarce (Wakefield et al, 2018). The purpose of this paper is to develop the small and emerging literature on the differential diagnosis of functional cognitive disorder (FCD) by suggesting a more detailed clinical characterisation positioning an anxiety-based explanation and feigning / non-credible explanation either side of FCD. We place particular emphasis on the clinical and neuropsychological features of FND as distinct from feigning / exaggeration, which is relevant for both clinical and legal settings. We present these three categories as distinguishable on clinical and neuropsychological grounds, but with overlapping features.

Health anxiety involving 'fear of forgetting':

Whilst cognitive complaints are common in mental health disorders, there is a group of patients with a more focal anxiety or phobic-type of clinical presentation relating to their cognitive abilities. This is driven by a fear of having specific disease, illness, or injury, generally self-monitoring for symptoms, thus focussing on their attentional skills and anterograde memory. We have termed this clinical presentation ‘health anxiety involving fear of forgetting’ and present cases A and B as examples of how such patients typically present in clinic and at neuropsychological testing. In terms of the Diagnostic and Statistical Manual of Mental Disorders; Fifth Edition (DSM-V) these patients could be classified as having Unspecified Anxiety Disorder, as a type of health anxiety with a cognitive focus. Health anxiety is generally understood as arising when bodily sensations or changes are misinterpreted as indicative of possible or likely serious underlying disease, with severe symptoms often classified as hypochondriasis (Asmundson et al, 2010) Somatic Symptom Disorder or Illness Anxiety Disorder (DSM-V). Patients in the ‘health anxiety involving fear of forgetting’ group can fit these diagnostic categories and present in the context of worry about possible dementia, for instance because of a family history of dementia.

Perhaps even more commonly such patients present following mild brain injury or possible mild concussive head injury. Characteristically, patients have a focal anxiety about day-to-day cognitive performance and the psychological, social and often career implications of cognitive lapses. It is common for such patients to avoid cognitively challenging situation, or endure-with-dread situations such as meetings at work or having to do a presentation, having become over-reliant on and overuse cognitive support strategies such as lists, notes or assistive technology. Generally psychological upset with anxiety and low mood is common in this patient group, but phenomenologically there is focal health anxiety, preoccupation with cognitive lapses and a phobic type coping style, but without the wider illness behaviours and illness beliefs common in FCD. Patients rarely report the type of diffuse and elaborate symptoms common in FND or feigning, although ‘internal inconsistency’ is common; such as Case B forgetting to pick-up her children from school, but working in a busy

engineering firm, and Case C forgetting what the dials on her oven mean (in the absence of visual disturbance), but having positive work appraisals. Discrepancies between self-reported history and the records, and self-reported symptoms inconsistent with known neuropsychological responses to injury and illness are uncommon. Information seeking about a feared diagnosis is common. The recent literature (Teodoro et a, 2018; McWhirter et al, 2020[a]) define FCD in broad terms and what we have classified as health anxiety with a cognitive focus falls into their definition of FCD. Previous authors appear to have done this because an excessive attentional focus on particular experiences or cognitive lapses is regarded as one of the drivers of Functional Neurological Symptom Disorder (FND), and also as a starting point for later work to finesse the clinical characterisation of FCD, which is an aim of this paper.

On neuropsychological testing, the profile of this group is generally intact, but can contain discrepancies from the premorbid level not conforming to an established pattern and better explained by anxiety. Test anxiety is common and generally observable, with patients often intolerant of errors and reluctant to guess. Patients can utilise strategies during testing detrimental to performance, such as working deliberately slowly to avoid errors on timed test. Performance validation test (PVT) / effort test performance is intact (Table 2).

Functional cognitive disorder (FCD)

While FCD can include many of the features described above, we propose that an FCD presentation is often more extreme and complex and goes beyond normal cognitive lapses, and beyond the explanations that could be provided solely by either mental health problems or a focal health anxiety.

In a detailed analysis of the literature on cognition in FND, fibromyalgia and chronic fatigue syndrome Teodoro et al (2018) report common features and propose a unifying theory extrapolating to FND. Key findings are support for the generally accepted discrepancy between subjective cognitive complaints and objective cognitive impairment on neuropsychological testing, with the latter being fewer and not conforming to a recognised cognitive profile of other neurological / neurodegenerative disease. It is well understood that subjective cognitive symptoms are frequently disproportionate to objective test performance, in that patients can report severe cognitive symptoms, but perform well on objective testing (Park et al, 2019; Reid & Maclullich, 2006; Rasouli et al, 2019) although there is evidence that subjective sense of cognitive decline could be an indicator of memory impairment and early Alzheimer's disease (Dardenne et al, 2017) and in neuropsychological test interpretation care needs to be taken to place memory performance in the context of premorbid general intellectual ability (Hawkins & Tulsky, 2001). Teodoro et al (2018) report a discrepancy between prominent subjective cognitive symptoms and patchy objective neuropsychological test performance, with problems on attentional and timed tests in some patients without a generalised deficit and without consistent evidence of poor test effort. Key clinical features of FCD from Teodoro et al (2018) and McWhirter et al (2020[a]) are subjective cognitive symptoms, no structural brain damage, memory perfectionism, overinterpretation of lapses, high self-monitoring for cognitive lapses and low confidence in memory, detailed complaints in clinic including bringing a list, 'internal inconsistency', being able to offer a detailed past

history often in spite of reporting recent and remote autobiographical memory complaints, and variable clinical course not suggestive of decline seen in neurological disease. Using conversational analysis, Alexander et al (2019) found that in giving detailed accounts of perceived memory problems in clinic, patients provided objective conversational evidence of intact memory, hence undermining their claim of an objective problem.

Recent writings on FCD have applied the FCD label to a range of phenotypes including health anxiety with a cognitive focus. We have separated these profiles and more narrowly defined FCD on the basis of clinical and neuropsychological features. However, future work will be required to address the intersection of FCD with medical and psychiatric disorders, other functional disorders and feigned presentations, better conceptualising the phenotypic overlap and developing improved treatment pathways.

In our collective clinical observation and neuropsychological test experience, people presenting with FCD tend better to fit into a biopsychosocial framework of understanding (White, 2006) with predisposing, precipitating and perpetuating factors than patients with health anxiety focussed on cognition and the feigning / non credible group. FCD patients tend to be polysymptomatic reporting diverse and elaborate symptoms, often showing discrepancy with known neuropsychological responses to brain injury and neurological disease to a higher degree than health anxiety patients. We propose that whilst health anxiety and FCD patients share high self-monitoring for cognitive lapses and over-interpretation, and perhaps low confidence in their cognition functioning and have lower expectations of performance, the FCD group have more elaborate and negative illness beliefs and faulty illness behaviours, integrating these beliefs into their sense of self to a greater extent. Illness beliefs and metacognition in FCD are an area for much-needed research. Unlike in patients who feign, if there is prior head trauma, it is generally possibly to establish post-traumatic amnesia (PTA) using the Rivermead Protocol for retrospective PTA assessment (King et al, 1997) in the health anxious and FCD group. In the absence of witness reports, this is generally difficult or impossible in the feigning group. At formal neuropsychological examination, the qualitative / observational component of neuropsychological testing is generally informative in terms of test behaviours elicited. FCD patients tends to struggle to engage with testing, often requesting short test sessions and giving-up easily on tests in favour of reiterating cognitive symptoms. In our collective experience, testing can elicit more extreme and odd behaviours such as excessive rehearsal, subvocalization, odd manipulation of the test materials due to concerns about glare or seeing too much information, markedly slow performance on timed tests, avoidance and total collapses in performance. In short, formal neuropsychological testing often elicits more than test anxiety, often producing a ‘noisy’ cognitive profile with dips or collapses in performance not consistent with a neurological cause, with generally intact or no worse than borderline performance on formal PVTs / effort tests (Kemp, Coughlan, Rowbottom, Teggart & Baker, 2008). Extreme cognitive symptoms can occur, but tend to be isolated and not part of a wider non-credible presentation. We present Cases D and E as examples of FCD.

Feigning / exaggeration / malingering

Cognitive testing is done in many clinical settings as part of a wider neuropsychological examination to understand a patient's presenting complains as a basis for treatment or rehabilitation. In routine clinical practice, the validity of the data and the fact that the patient has applied sufficient test effort are usually assumed, although this assumption may not be appropriate in many clinical and medicolegal contexts. It was apparent from the early studies on performance validation that test effort exerted a larger effect size on neuropsychologist test scores than even severe brain injury or neurological disease, and explained approximately 50 per cent of the variance in a large sample of patients examined in some medico-legal contexts (Green, Lees-Haley & Allen, 2003). Although clinicians generally feel able to detect poor effort and exaggeration, the evidence does not support this without the use of PVTs or other validation methodology (Dandachi-FitzGerald, Merckelbach & Ponds, 2017). Consequently, the literature on PVTs / effort testing expanded exponentially with guidance for clinicians from the British Psychological Society (2009) the National Academy of Neuropsychology, NAN (Bush, 2005) and the American Academy of Clinical Neuropsychology, AACN (Sweet et al, 2021), with the NAN guidance stating that symptom exaggeration or fabrication occurs in a sizable minority of patients at neuropsychological examinations, and that assessment of response validity is medically necessary. This quickly evolving literature has led to various stand-alone PVTs and, increasingly, use of standard neuropsychological tests as 'embedded' PVT / effort measures. These tests are generally referred to as performance validity tests (PVTs), as distinct from self-report symptom validity tests (SVT), with the latter being less well developed. However, there are good data to show an association between PVT performance and symptoms reporting (Lippa, Lange, French & Iverson, 2019), with patients that fail PVTs, reporting more implausible symptoms, hence showing that PVT performance is related to self-reported symptom validity.

The percentage of patients who feign or exaggerate, or fabricate cognitive deficits during neuropsychological evaluation is significant, and although difficult to study with precision, estimates vary from less than 10% in medical populations without external incentives (Wodushek & Domen, 2018) to up to 40 per cent in personal injury and disability evaluations (Larrabee, Millis, & Meyers, 2009; Mittenberg, Patton, Canyock, & Condit, 2002; Ruff, Klopfer, & Blank, 2016) and up to 50 per cent or higher in criminal justice, penal, and military settings (Ardolf, Denney, & Houston, 2007; Jones, 2016). Greve, Binder, & Bianchini, 2009 estimate rate of up to 50 per cent in pain clinics.

The purpose of performance / symptom validity tests is to detect non-credible or invalid responding and prevent Type 1 interpretation errors or false positive conclusions of brain damage, which could do iatrogenic harm by creating or strengthening erroneous beliefs about brain damage and impairment, trigger unnecessary referrals and wasting healthcare resources. There are criteria to guide clinicians in determining levels of invalid responding / 'malingering' (Slick, Sherman & Iverson, 1999) and just recently revised for a second time (Sherman, Slick & Iverson, 2020). These new proposed criteria simplify diagnostic categories of non-credible symptoms, expand and clarify external incentives, more clearly define the role of compelling inconsistencies, address issues concerning performance and

symptom validation tests (i.e., number administered and false positives), and importantly, clearly define exclusionary criteria based on the last two decades of research on malingering in neuropsychology.

Whilst the original and revised guidelines are well-reasoned and clinically useful, the diagnosis of 'malingering' remains problematic. The position of the present authors is that non-credible and biased responding can be identified on PVTs / SVTs, demonstrated mathematically, has clinical and behavioural correlates, and the degree to which, behaviours are volitional is ordinarily identified by excluding plausible explanations for non-volitional behaviours. For example, inconsistencies and variability that exceeds what is typical in functional disorders or in other clinical populations. However, malingering is scientifically and epistemologically problematic as intentions cannot be measured, only inferred. In a medicolegal context, short of a person confessing, this inference is a matter for the Court to decide having considered expert clinical findings and opinions on the history and broad clinical picture. Sherman et al (2020) discuss whether the term 'malingering' is outdated and pejorative, but adopt the view that alternatives labels which do not imply intent, such as 'misrepresentation' or 'disability exaggeration' lack clear meaning.

As the PVT / SVT literature has developed and neuropsychologists have become more experienced in their use, the importance of using tests of different sensitivity, measuring performance in different cognitive domains (i.e., so-called chaining) and interspersing tests throughout the examination has become recognised (Lippa, 2018)

We present cases F and G as examples of non-credible clinical presentations and draw attention to some important considerations in the analysis of the neuropsychological test data in differential diagnosis of feigning / exaggeration. Much of the PVT / effort test literature sets a cut-off score on a test to have acceptable sensitivity and specificity, generally around 90 per cent (Boon, 2007; Morgan & Sweet, 2009). Sensitivity and specificity are psychometric properties and do not change. However, the meaning of these psychometric properties will depend on the prevalence or base rate in the population (the base rate of response bias in this case) and clinicians need to consider not just sensivitity and specificity but positive predictive power (PPP) and negative predictive power (NPP).

$$\text{PPP} = \frac{\text{Sensivitity x base rate}}{\text{(sensitivity x base rate + ((1-specificity) x (1 – base rate))}}$$

$$\text{NPP} = \frac{\text{Sensivitity x (1-base rate)}}{\text{((1 – base rate) x specificity) + (base rate x (1-sensitivity))}}$$

For example, the base rate of feigning in a memory clinic will be lower, and may be very low, relative to a medico-legal clinic, which will increase the likelihood of a failed effort test result in the memory clinic group being a false positive because the PPP is reduced. In a medico-legal or forensic population with a higher base rate of response bias, the PPP will be higher and the likelihood that the effort test failure is a false positive will be reduced. Bunnage (2017) emphasises the importance of considering base rates and PPP and NPP in test interpretation. As well as clinicians being aware of the association between base rates and predictive values in test interpretation, in severe brain damage populations such as established dementia or severe brain trauma, patients could fail effort tests, many of which are memory tests, due to bona

fide cognitive impairment. In other words, there will be a level of cognitive impairment where effort tests measure impairment and tell us little or nothing about test effort or engagement per se. In such populations, effort tests that take a hierarchical approach and go beyond a pass / fail approach are useful (McGuire, Crawford & Evans, 2019). Tests such as the Word Memory Test (WMT) and the shorter verbal and non-verbal versions (MSVT) allow 'profile analysis' as a technique to differentiate a 'genuine impairment fail' from a 'low effort fail' to assist clinicians in reducing false positive errors when identifying non-credible / invalid test performance (Green, 2008; Hampson, Kemp, Coughlan, Moulin & Bhakta, 2014; Alverson, O'Rourke & Soble, 2019). We illustrate this technique with Patient F.

Significantly below-chance findings on forced-choice tests are the strongest evidence of deliberate under-performance and would be consistent with a conclusion of 'malingering', as it provides evidence that responses are highly unlikely to be explained by 'unlucky guessing'. If the result is significantly below-chance at the .05 level, then it is probable that the patient intentionally generated incorrect answers (Binder, 1990; Slick et al 1999; Sherman et al, 2020). McWhirter et al (2020) do not appear to have considered the diagnostic relevance of this criterion of feigning before concluding that PVTs / effort testing would not differentiate between individuals with feigned and 'real' cognitive problems. In our clinical experience, it is not uncommon for patients to perform at or below-chance yet perform reasonably well on standard memory tests, which constitutes a 'reverse gradient' and a discrepancy in the cognitive profile requiring interpretation. Given the clinical and medico-legal importance of statistically significant below-chance performance, it is important that clinicians are familiar with a test method. Of the methods available, the easiest is probably binomial theorem and the on-line binomial probability calculator at the Vassarstats website: http://vasserstats.net/textbook/ch5apx.html. This method is outlined in Patient F and Binder & Chafetz (2018) provides a helpful discussion of the significance levels (i.e., .05 to .20) one-tailed versus two-tailed tests and combining scores on significance testing.

We propose that the type of discrepancies, which go beyond the type of degree of internal inconsistency seen in FCD / FND and neuropsychological test findings seen in Cases F and G, cannot comfortably be attributed to any other diagnostic category and can reasonable to labelled as feigning / exaggeration. We set out these features relative to health anxiety and FCD in Table 2. In Table 3, we set out the clinical features more consistent with feigning / exaggeration, and we propose that this guidance as particularly relevant for consideration differential diagnosis between credible and non-credible presentations short of under-chance PVT findings. There is a small literature on the performance on FCD / FND patients on PVTs / effort tests. Kemp et al (2008) examined the base rate of PVT failure in an FND sample relative to two groups of simulators. The FND group generally performed well on PVTs and whilst this was a non-litigation sample, some patients were claiming state benefits and this was thought to be a potential external incentive in the 11 per cent of FND patients that failed. Most studies suggest only a minority of FCD patients perform poorly on PVTs (Cragar et al, 2006; van Beilen et al, 2009; Binder, Salinsky & Smith, 1994). Although some studies have shown worse performance than controls (Teodoro et al, 2018), on balance, inability to complete PVTs / effort tests, which are relatively simple cognitive tasks, does not appear to be explicable by the

psychopathology of functional disorders and alternative explanations need to be sought.

Table 3: clinical neuropsychological features more consistent with feigning and non-credible presentations

Neuropsychological and clinical features more consistent with feigning than FCD
PVT / effort test failure, with failure of multiple tests and particularly under-chance performance as determined by binomial probability theory providing strong evidence.
Multiple implausible symptoms on an SVT and / or a clinical presentation that do not have psychological coherence in terms of a biopsychosocial understanding of illness.
Marked and compelling discrepancies that exceed what is seen clinically in FCD / FND patients: for example, a patient arriving alone at a clinic, but asking for a map to leave the building / a patient stating that they use a wheelchair, but observed playing sport.
Marked and compelling discrepancies between neuropsychological test performance and daily functioning: for example, performing worse than patients with severe brain injury or dementia, but passing college courses and living independently.
Marked and compelling discrepancies between symptom complaints and known neuropsychological and psychosocial responses to brain injury and physical injury.
Marked and compelling discrepancies between self-reported symptoms / history and the medical records.
Marked and compelling discrepancies between self-reported symptoms / daily functioning and the other sources of information, which could be observations or social media.
Presence of external incentive.

Conclusions

There is increasing recognition of a cognitive subtype of functional neurological disorder (functional cognitive disorder: FCD) with some recognised clinical features of what is probably an under-diagnosed condition without specific evidence-based treatments. However, the existing literature provides only limited neuropsychological characterisation of FCD, in particular with regard to the differentiation from feigned cognitive presentations. There is an expanding body of neuropsychological knowledge on performance validation tests (PVTs) / symptom validation tests (SVTs) to assist with the detection of feigning / malingering and updated guidance for clinicians (Sherman et al, 2020; Sweet et al, 2021) that consider these specialist neuropsychological findings and the bigger clinical picture, including multiple sources of information. Utilising seven anonymised case histories, we position FCD between focal health anxiety and feigning / malingering, setting out clinical and neuropsychological features of all three disorders, and a statistical method to assist clinicians in detecting feigning. We present these categories as relatively distinct in

symptomology and neurocognitive profile. With detailed clinical assessment and neuropsychological testing, we propose that most patients will be classifiable, albeit with overlapping features.

This work adds to a body of recent literature aiming to improve the recognition of FCD as distinct from other mental health presentation and distinct from feigning / malingering, as a basis for developing treatment protocols and improve care pathways.

Limitations of this work are that whilst we draw on the recent literature and current neuropsychological approaches in particular, our clinical characterisations are in part based on our collective and multidisciplinary expertise and case material, hence there is a need for empirical validation of our proposed framework (Table 1). Also, we recognise that PVT / SVT results falling in the borderline range, just below cut-off, generally and in different clinical, demographic and cultural groups, but well above-chance level presents particular interpretation challenges and is an area for on-going study.

The purpose of this paper is to further contextualise FCD relative to how it is defined in the recent literature, with particular focus on the clinical and neuropsychological features that differentiate it from other conditions along the spectrum that we set out throughout this paper, and specifically from mental health disorders impacting on cognition and from feigning / malingering. We propose that FCD is a clinically useful diagnosis conforming to the same or similar pathogenesis and treatment challenges as other functional disorders. Whilst we see FCD as essentially separate from feigning and do not anticipate that will change much in the future, the nosological status of FCD as distinct from other mental health disorders impacting on memory and cognition could change and diagnostic boundaries could well shift. In the recent literature, focal or situational anxiety about memory / cognition (category 3 in Table 1) is regarded as a functional presentation. We argue that patients with this presentation show a profile of complaints and neuropsychological performance that is clinically distinguishable from other phenotypes, and we see FCD as a more complex disorder involving more elaborate and diverse illness behaviours and conforming more strongly to a biopsychosocial framework of understanding, albeit with phenotypic overlap. We hope that this paper adds to the on-going debate on FCD and the development of nosological classification, clinical understanding and treatment planning in this important field.

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