Functional cognitive disorders: clinical presentations and treatment approaches

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ABSTRACT

Functional cognitive disorders (FCDs) are a common cause of subjective and mild cognitive impairment. Isolated FCDs commonly present to the cognitive clinic, but examination of the nature of the symptoms suggests that they can also be understood as a transdiagnostic feature of many other conditions. This article examines methods of formulating the cognitive difficulties in order to identify treatment targets in people with FCDs.

INTRODUCTION

All neurologists will have encountered patients with severe and distressing cognitive difficulties but who have no structural or degenerative disease of the brain. Better recognition of functional cognitive disorders (FCDs) in recent years has stimulated better clinical description and specificity. Here, we aim to frame these developments within a 'bedside' clinical approach to the assessment, diagnosis, and treatment of FCDs.

WHERE MIGHT WE SEE FCD?

FCDs are epidemiologically important, and yet have been neglected in neurodegenerative disease research. Over the last 10–20 years, a focus on identifying biomarkers of neurodegenerative disease has led to an emphasis on identifying disease, but arguably, at the cost of losing curiosity and understanding of cognitive syndromes that are not the result of disease.

Research models, which have influenced practice in memory clinics, stratify patients into a theoretical continuum, based on the history and cognitive examination. This continuum starts with subjective cognitive decline, then mild cognitive impairment, and finally a dementia syndrome. With wider access to investigations, this clinical risk stratification is increasingly helped by the availability of biomarker status. And yet biomarker profiles—even in combination—are not yet adequately specific to enable diagnosis of impending dementia in an individual with subjective symptoms or mild impairment; and the risk of false positives is potentially catastrophic. We are familiar with cases in which adults have made life-changing decisions based on an expectation of terminal decline and death following misdiagnosis of Alzheimer's disease in mid-life, and in whom diagnosis was later revised to that of an FCD.

A loss of interest in the wider clinical picture in favour of advanced imaging and cerebrospinal fluid (CSF) biomarkers has brought a situation where the detection of neurodegenerative disease has been the priority and where non-disease causes of cognitive symptoms are less well understood.

An historical loss of interest in non-disease cognitive symptoms can also be seen in the psychiatric literature. Early descriptions of a variety of 'pseudodementia' presentations[1] were superseded by use of the term 'pseudodementia' to refer exclusively to a state of profound depression with the appearance of advanced dementia. Perhaps in consequence, the view from psychiatry has often been that all non-dementia cognitive symptoms are caused by depression; whereas detailed inquiry into the phenomenology of functional cognitive impairment suggests that this is only sometimes the case.

One priority of recent FCD research has been to identify positive diagnostic features of the condition, allowing a diagnosis based on positive signs and not simply following exclusion of disease. As figure 1 illustrates, use of positive diagnostic features and clinical profiles is crucially important in interpreting biomarker profiles and therefore more accurately communicating clinical risk. Those people presenting to cognitive clinics with subjective decline and mild cognitive impairment are at risk of 'false positive' prediction of dementia risk; if the clinical profile supports a diagnosis of

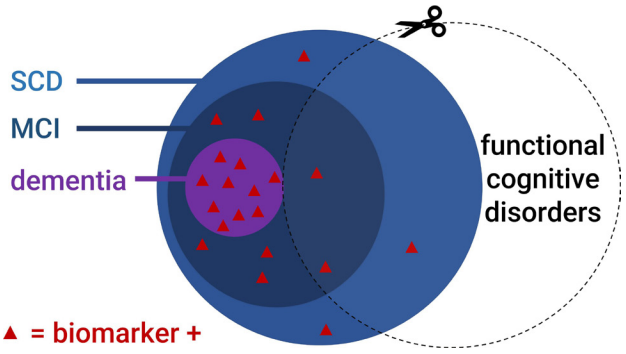

Figure 1 This circle describes a group of 'all-comers' to a memory clinic, stratified on the basis of clinical history and cognitive testing into those with SCD (subjective cognitive decline), MCI (mild cognitive impairment) and dementia. Red triangles represent biomarkers suggesting presence of neurodegenerative disease. By positively identifying individuals with FCD, based on positive clinical features (not by exclusion of disease), we might refine our view of future dementia risk and—crucially—offer different treatment and advice to this group of individuals with potentially reversible symptoms. FCD, functional cognitive disorder.

functional cognitive impairment, the risk may be reduced and most importantly, they might be offered treatment and support with the aim of improvement in their cognitive symptoms.

The memory clinic is not the only, or even the most frequent, clinical setting to encounter FCD. Functional cognitive symptoms are almost always present in patients with functional neurological disorders and are commonly present (in some cases alongside other impacts on cognition, such as sedative medications) in pain and fatigue syndromes. FCDs may often form part of the wider clinical picture in the small proportion of individuals who develop distressing and disabling neurological symptoms after mild traumatic brain injury (as part of a 'postconcussion syndrome').[2] Indeed, we are familiar with presentations of FCD as part of postinjury and postillness syndromes more broadly, including in some people with long COVID.[3] In fact, the mechanisms of FCD are likely relevant to cognitive symptoms across a range of disorders in which a symptom burden impacts on available attentional resources (boxes 1–4).

Improved recognition and understanding of these disorders is likely to have broad benefits in terms of our academic understanding of cognitive symptoms, and in turn on our clinical practice. FCDs prompt us to move away from a reciprocal disease↔deficit model of cognitive performance and towards a model in which various

Box 1 De novo functional cognitive disorder

A 62-year-old man presents to the memory clinic reporting forgetfulness over the previous 6 years, following early retirement from technical manual work. He gives a detailed description of four recent events in which he was transiently unable to recall quite specific details of a previously watched television series and of an ex-colleague's name. He also reports having left his wallet in a specific shop and having had to return to collect it later the same day when he realised that it was missing. More generally, he notes that he has struggled to find routine and meaning since his retirement, even though he had looked forward to it very much. His wife has no concern about his memory and describes him as responsible for all of the household bills and administrative tasks.

MR scan of brain shows some white matter hyperintensities of uncertain significance. He scores in expected-for-age ranges on cognitive tests. He is signposted to community resources with a view to becoming more physically and socially active, and at 6-month follow-up reports feeling 'a lot better' and is no longer particularly concerned about his memory.

Box 2 FCD following panic and dissociation, with obsessional symptoms

A 45-year-old accountant has a 2-month history of poor memory. The symptoms started suddenly while giving an important presentation at work: he felt suddenly dizzy, breathless and shaky, then developed a 'spaced out' feeling. He left work that day, was signed off for 1 week ('labyrinthitis') but since returning has struggled to keep up with his work. He finds himself repeatedly checking his work in case he has made a mistake, staying late every day and feeling too fatigued to engage with usual activities such as going to the gym. He often lies awake worrying about the following day's work. He reports that his manager has not noticed any problem with his work but he feels it is 'only a matter of time' and is sure he will lose his job. He acknowledges a tendency to perfectionistic high standards, which has previously been to his advantage. His grandmother died in her 80s of Alzheimer's disease, and he is very concerned that he is developing the same illness.

Neurological examination is normal and he achieves high scores on cognitive screening tests. He gives an extremely detailed account of his difficulties. His mood is euthymic.

He is given a diagnosis of FCD and followed up twice. He is supported to try behavioural experiments reducing the amount of checking and preparation he undertakes, to no effect. He is encouraged to resume enjoyable activities. He starts SSRI medication. At 6 weeks follow-up, he reports reduced anxiety, of which he is better aware. He feels more comfortable with his work performance, which he acknowledges is more than satisfactory. He remains concerned about future dementia. He agrees to follow-up after 6 months.

FCD, functional cognitive disorder.

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Box 3 FCD after COVID-19 and in the context of multiple somatic symptoms

A 52-year-old woman in part-time secretarial employment has a 15-year history of widespread pain for which she has received a diagnosis of fibromyalgia, and longstanding migraine since teenage years. Nine months after a mild respiratory illness with COVID-19, managed in the community, she experienced marked fatigue, worse after activity, and an increased frequency in migraine, with now daily headaches for which she takes tramadol. She attends complaining of forgetfulness and poor concentration. She is concerned that her cognitive symptoms are worsening and may be the result of damage and continuing inflammation caused by COVID-19.

On assessment, she gives a full and detailed account of her difficulties, both longstanding and recent, and of previous frustrating interactions with health professionals. On cognitive examination, she becomes tearful and distressed and loses points for verbal fluency and delayed recall but manages tests of praxis and construction quickly.

She is offered a diagnosis of FCD, with advice about management of fatigue and a switch from tramadol to more appropriate migraine prophylaxis. At 6-week follow-up, she reports continuing episodes of poor concentration but reduced concern about her memory, and has started a graded return to work, having also addressed some stressful personal and domestic issues.

FCD, functional cognitive disorder.

dynamic behavioural, psychological and social factors hold sway over how individuals can access and effectively use the cognitive abilities available to them.

Box 4 FCD after mild traumatic brain injury/concussion

A 24-year-old postgraduate student is involved in a 'clash of heads' in a university rugby game. She is briefly knocked unconscious (less than 2 min) and feels groggy and dizzy the next day. She has no post-traumatic amnesia and can remember 'coming round' on the pitch. She takes a week of sickness absence from her university course and lies in dimmed light in her flat. On returning to university, she feels immediately panicky and overwhelmed, cannot tolerate the lights and noise, and so takes leave of absence for the following term; she does not return to football training or to her part time retail job. Over the following months, she develops increasingly frequent headaches for which she takes daily paracetamol and codeine. Her primary concern is about her poor memory and concentration: she fears that she will be unable to return to study or work due to her brain injury and that her symptoms will get worse.

FCD, functional cognitive disorder.

HOW TO MAKE A DIAGNOSIS

Proposed diagnostic criteria require one or more symptoms of impaired cognitive function, with clinical evidence of internal inconsistency, which are not better explained by another medical or psychiatric disorder, and which cause clinically significant distress or impairment.[4]

Imaging and extensive cognitive testing have limited value in the diagnosis of FCD. Although there are certainly circumstances in which a scan is appropriate, bear in mind that the most anxious patients may not be reassured by a normal scan but may be made more anxious by an incidental finding. Our experience is that brief cognitive tests can help in terms of assessing the patient's approach, behaviour, and emotional response to a cognitive challenge, but that the raw scores can be difficult to interpret: some patients with FCD score highly on tests, others perform poorly.

This is not a diagnosis of exclusion, and as such the key to diagnosis is in positive clinical evidence of internal inconsistency. Importantly, the key 'internal inconsistency' does not refer to the sort of fluctuations in function over time that may occur in delirium, or dementia with Lewy bodies. Rather, functions that remain easy and automatic become difficult when attention is focused towards them. For example, in a patient unable to register a name and address in clinic, but who is managing in cognitively difficult employment without issue; or a patient who reports extremely poor recall but who provides detailed examples of a range of specific forgetting events over previous weeks and months. Table 1 lists clinical features of FCDs as distinct from presentations of neurodegenerative disease.

It is important to recognise the potential impact of medical and psychiatric comorbidities and of both prescribed and non-prescribed medications. It may sometimes be difficult to disentangle where the influence of unhelpful psychotropic medications, or of depression, ends and where the FCD begins. We suggest that patients welcome honesty about these sorts of uncertainty. Collaborative formulation, as described below, can often help to identify multiple 'targets' for treatment or improvement in each patient.

FORMULATING SYMPTOMS IN INDIVIDUAL PATIENTS

Taking a detailed history from the patient with FCD is not only necessary to make a diagnosis, but also helps to formulate how and why these difficulties might have arisen, at this time, in this individual.

Current understandings of FCD emphasise the roles of attention and metacognition. In our experience and understanding (figure 2) a heterogeneous range of internal, external and behavioural factors feed into a common final process, in which cognitive task failures are perceived as evidence of global failure. This leads to altered behaviour and reduced attention available for tasks (therefore causing more cognitive failures).

Table 1 Clinical features of functional cognitive disorders compared with neurodegenerative disease

	Functional cognitive disorder	Neurodegenerative disease
Interaction in clinic	Attends alone having travelled independently to clinic.	Attends with another; required assistance to travel to clinic.
Speech and language	Full and detailed account. Speaks for >1 min in response to opening question. Communicates clearly. Recounts many examples of symptoms and lapses.	Brief answers to open questions. Answers lacking in detail and complexity. Repetitions without awareness.
Nature of complaints	Episodes of memory failure typically associated with failure to encode. Increased frequency of inattentive lapses which can occur in normal life. May report 'gaps' during which functioned normally, for example, a car journey. Blocks for overlearned material for example, PIN and passwords In acute onset (eg, after minor traumatic brain injury) may report loss of autobiographical memories	May be unaware of memory problems. Dismisses or normalises memory failures suggested by others – 'Just my age', 'Could happen to anyone'.
Collateral report	Less concerned than the patient about the cognitive symptoms. May emphasise other symptoms as more important—for example, pain, anxiety	More concerned about symptoms than the patient. May report forgetfulness, repetitiveness or changes in social/emotional behaviour that the patient seems unaware of.
Cognitive screening test performance	May score in high/normal range against expected norms; or may score below expected norms, particularly if anxious/tearful during cognitive testing. Often anxious and may become tearful and distressed during cognitive testing. Evidence of continuous self-evaluation during testing 'I'm not doing very well here'. May refuse to attempt some questions or stop trying—'can't do it'. May score inconsistently in same domain across multiple tests. Starts well and shows signs of mind going 'blank'. Signs of sudden fall-off in performance after a small error.	Less likely to become tearful and distressed during cognitive testing. Scores below norms and in patterns expected of neurodegenerative disease.
Performance validity test performance (pass/fail via standard cut-offs)	May pass, may score within 'dementia' range, or may fail performance validity tests.	May pass, may score within 'dementia' range or may fail performance validity tests.

We know that attention is important, because people with FCD report symptoms that are primarily inattentive. For example, the experience of going into a room and forgetting what you have gone in there for; a feeling of drifting off during a conversation; perceiving a 'gap' in memory for a common journey or routine event; missing an appointment or forgetting to take medication. Even those symptoms experienced as forgetting or memory failure can generally be attributed to a failure of encoding the information in the first place. As noted in relation to diagnostic features, a detailed recall of a forgetting event is reassuring in terms of memory function; but it may well demonstrate an attentional lapse. Although described in terms of significant distress and disability, when we stand back we might recognise these sort of lapses as those occurring commonly in everyday life.

In FCDs, there is most often a striking and catastrophic interpretation of this experience of cognitive failure. Frequently, patients report an unusually perfect and exceptional memory before the symptom onset.[5] In contrast, the description of current memory function is depicted in terms of absolute failure and disability. Whereas the patient with incipient Alzheimer's dementia might report a 'not too bad' memory both before and after onset of symptoms, the patient with FCD feels that they were excellent and now are failing, with little evidence of experiencing or tolerating an 'in between' state.

The observation of metacognitive failure has led to several interesting experimental studies comparing metacognitive accuracy in FCD compared with healthy controls.[6–8] These studies have reproduced a slightly unexpected finding. That is, they have shown that patients with FCD (certainly, those performing in the healthy range on cognitive tests) are accurate on minute-to-minute task-focussed measures of metacognitive accuracy—that is, they can accurately assess how well they have performed; but despite this evidence their global metacognitive accuracy is poor. So people with FCD perceive that they are failing cognitively even when their own assessment of their reasonable cognitive function is accurate.

A tendency to rigid high standards and poor tolerance of occasional failure in many patients with FCD leads in some cases to an overlap with obsessive compulsive

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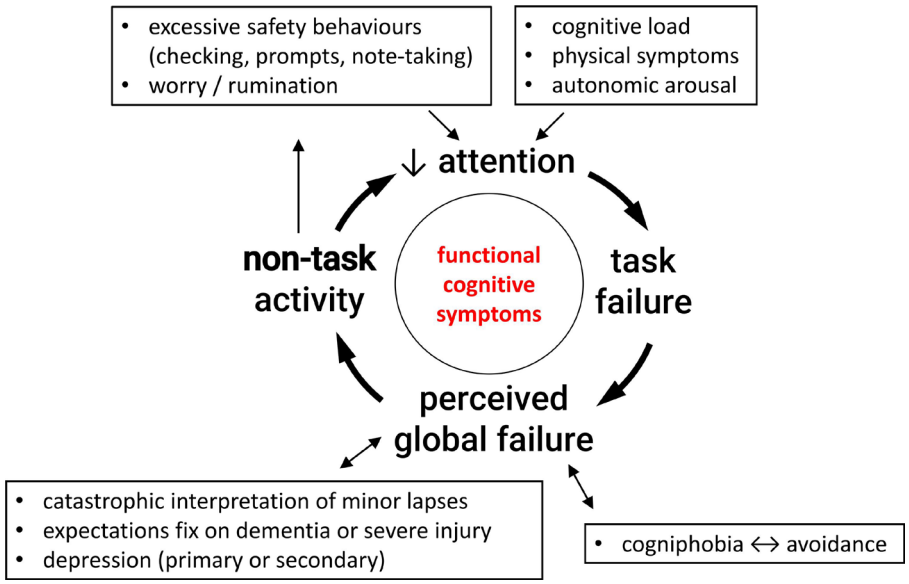

Figure 2 Mechanism and formulation. Current understandings of functional cognitive disorder mechanisms emphasis the roles of attention and metacognition. Environmental or social demands, physical symptoms, self-initiated behaviours, and perseverative cognitions may all contribute to depleted attentional resources, increasing the likelihood of an inattentive cognitive lapse. People with FCD appear able to evaluate their minute-to-minute cognitive performance accurately but erroneously interpret their global cognitive function as failing even where performance is satisfactory. Finally, autonomic arousal and the misperception that cognitive effort is harmful can lead to cogniphobic avoidance of cognitive challenge, increasing disability and reducing opportunities for cognitive success. This figure created with reference to Teodoro *et al*.[12] FCD, functional cognitive disorder.

disorder. Excessive attention to detail, to avoid error, historically described as 'obsessive slowness' is probably contributes to cognitive inefficiency and fatigue in some people with FCD. Clinical experience as yet unsupported by observational data suggests that obsessional and perfectionistic personality styles—which may in some cases have supported high academic and employment achievements—are a predisposing factor for developing a FCD after a triggering event.

Depression is well known to cause of cognitive difficulty. Severe depression in the elderly, leading to a dementia-like syndrome of functional impairment—which is however reversible—has historically been called 'pseudodementia'. This presentation may overlap with FCD but has a poorer prognosis and is more likely a harbinger of dementia even where there is an initial mood and cognitive recovery.[9] More often, mild-to-moderate depressive symptoms, characterised by anhedonia, loss of drive and fatigue, presents as a part of the picture in FCD, such that it is difficult to tell whether the depression came first or resulted from the cognitive concerns and withdrawal from activities. And where obsessive–compulsive disorder and obsessive–compulsive personality styles both predispose to depression, note that obsessional symptoms can arise as a secondary feature of depression. It is worthwhile and important to treat the depression, whether or not it came 'first' or 'after'.

Anxiety symptoms are also common in those with FCDs, although it can again be difficult to disentangle where they are a primary part of the mechanism in any individual or rather a perpetuating factor. The connotations of 'anxiety' can be challenging for patients who have previously perceived that they have been flippantly reassured about the nature of their symptoms. Therefore, it can be helpful to introduce the broader (and more pathophysiologically meaningful) concept of central nervous system arousal, or, depending on level of understanding, 'fight or flight' states.

Clinicians should make the effort to investigate the cognitions and symptomatology behind any activities that the individual has stopped doing. Just as we are familiar with 'kinesiophobia'—fear and avoidance of movement—in functional motor disorder, and in conditions such as complex regional pain syndrome; and phobic anxiety about falls and freezing in patients with Parkinson's disease; so we often see a phobic anxiety about cognitive tasks in people with FCD. This may be particularly problematic after a mild traumatic brain injury/concussion, where lack of clarity or understanding can lead to a belief that symptoms (which in fact can often be boiled down to symptoms of autonomic arousal, or anxiety, or of migraine or benign paroxysmal positional vertigo) are evidence of actively occurring brain damage. People therefore, avoid cognitively challenging tasks, but as expected the symptoms occur at lower and lower levels of stimulation leading to progressive avoidance and withdrawal, and so to severe disability. Without unpicking these situations in clinic and challenging the underlying harmful understanding it is difficult to make progress in other areas of treatment.

TAILORING TREATMENT TO INDIVIDUAL FORMULATION

Research into treatments has lagged behind other areas of functional neurological disorder, and in FCD there is little evidence on which to base treatment decisions. That said, clinical experience supports the use of methods that have been helpful (and which do have a stronger evidence base) in functional motor disorders, dissociative seizures, as well as in the treatment of neuropsychiatric symptoms including fatigue, pain, depression and anxiety across a range of other neurological conditions.

Indeed, our experience in clinics would suggest that cognitive symptoms, alongside fatigue and pain, are transdiagnostic features of neurological diseases with much in common and responding to similar treatment approaches. Research in postconcussion symptoms is particularly relevant.[2 10]

The next 5–10 years are likely to yield a range of new evidence-based treatments for FCD. While awaiting results of trials, our clinical experience supports an individualised approach to treatment of functional cognitive symptoms, based on each patient's specific psychopathological formulation, circumstances and comorbidities.

Importantly, even in a routine outpatient appointment, it is possible to give a short explanation of the way in which functional cognitive symptoms might arise that can give the patient confidence in the diagnosis and help them to make sense of their difficulties. Along with the suggestions below, it is often possible to identify 'easy wins' in terms of optimising management of a comorbid condition (such as migraine) or reducing an unhelpful medication. Patients can be directed to resources such as the neurosymptoms website (www.neurosymptoms.org), which includes a helpful printable leaflet about FCDs. For patients who ultimately require more intense therapeutic input, clinical neuropsychologists can help in providing treatment, which might take a cognitive–behavioural therapy, compassion-focused therapy or acceptance and commitment therapy approach. We suggest that referral for treatment, rather than detailed neuropsychological testing, is the most helpful use of (often limited) neuropsychologist time in this group of patients.

Using brief 'bedside' formulations like the above (figure 1) in clinic can help both physician and patient to identify in-roads for treatment, even when the situation feels 'stuck'. Importantly, the patient need not have 100% 'buy in' to the model to gain benefit, and early improvements in symptoms as a result of low-cost behavioural experiments can aid therapeutic alliance for continuing treatment.

The following broad approaches might be helpful, with emphasis and order tailored to the individual patient:

EXPLANATION TO CONTEXTUALISE SYMPTOMS

- Most lay people have a limited understanding of how memory works. Thus, it is helpful to explain the importance of attention as a requirement to learning new information, and therefore, being able to recall that information.
- Similarly, 'autopilot' experiences can be explained as a generally helpful and efficient way of the brain minimising use of attentional resources.
- Help patients to identify areas in which cognition is working well to illustrate inconsistent performance. For example, many patients dismiss the significant cognitive skills used in everyday domestic tasks, personal and family organisation, and travel.
- Use evidence of 'base rates' of cognitive lapses in healthy adults; contextualising lapses as something that may also happen frequently in health.[11]
- Give a personalised explanation of why the clinical features and/or investigation findings are not consistent with neurodegenerative or structural disease, while also outlining patient-specific risk factors.
- For patients concerned about particular potential risk exposures, it can be helpful to discuss candidly the relevant contribution of various risk factors on dementia and mortality rates. For example, the relative contribution of cerebrovascular disease risk factors vs minor head injury to personal risk profile.
- If relevant to a specific patient, explain the effects of central nervous system arousal (eg, 'fight or flight') on cognitive performance. Explain that the cognitive component of a 'fight or flight' response prefers to remain vigilant to the whole surrounding environment for danger and does not want us to focus in on a detailed task.

IDENTIFYING DEMANDS ON ATTENTION

Having established the role of attention as the 'gatekeeper' of an effective working memory, it can be helpful to support the patient to identify unhelpful drains on their attention. These might be:

- Somatic—Identify poorly controlled symptoms, for example, pain, breathlessness or fatigue. Explain likely contribution to depleted attentional focus. Patients can often notice a relationship between severity of other symptoms and their cognitive symptoms
- Environmental—Identify unhelpful environmental factors contributing to symptoms, for example, working from a busy home, environmental noise, open-plan office working, misuse of multiple screens (to do multiple tasks, rather than to aid function on a single task), excessive smart-phone push notifications or checking.
- Social—Encourage better sharing of occupational, family and domestic responsibilities. Encourage confident boundary-setting with colleagues and employers regarding workload, especially where there has been 'role creep', in which an effective employee is asked to take on more and more tasks beyond their intended role.

OPTIMISING COGNITIVE FUNCTION BY ADDRESSING IMPAIRING FACTORS

- Collaboratively explore the list of medications and identify drugs with unhelpful psychoactive effects.

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- Identify and treat depression, anxiety disorders, hyperventilation or sleep disorders.
- Optimise management of comorbidities. For example, hyperglycaemia in poorly controlled diabetes is associated with fluctuating subjective and objective cognitive difficulty.

EXAMINE AND ADJUST COGNITIVE BEHAVIOURS

- Some patients with FCDs use excessively time-demanding and attention-demanding cognitive strategies. For example, taking excessive verbatim notes during meetings, using excessively frequent reminders and alarms, relying on diaries and calendars for 'low-stakes' activities (such as eating meals, going to the toilet) so that 'higher stake' appointments become lost in the noise. Patients can be encouraged to try to reduce these so as to use 'just enough' safety strategies and no more.
- Support patients to challenge and find alternatives to catastrophic thoughts about the perceived 'worst that can happen' in the event of a cognitive lapse.
- Encourage patients to normalise 'good enough' performance with occasional errors. Patients who are sceptical about the frequency of cognitive errors in healthy people can be encouraged to ask friends and family, or to make a point of noticing other people's cognitive lapses.
- Address avoidance of perceived cognitively challenging tasks. Explain that if we do not do anything cognitively challenging there will be no way to know if things are improving.

CONCLUSION

FCD is best understood as a heterogeneous set of conditions with a common final pathway, characterised by cognitive tasks becoming excessively effortful and with a global perception that one is cognitively failing—generally despite evidence to the contrary.

The troublesome symptoms may reflect disordered allocation of attention, and often cognitive symptoms occur alongside others, including fatigue, widespread pain, sleep disorders, anxiety and depression. Anecdotally, perfectionism is common, with reports of exceptional premorbid memory, and inability to tolerate errors that are demonstrably common in healthy people.

Broader experience in the neurology clinic supports an idea that functional cognitive symptoms, like fatigue, might also be understood as transdiagnostic features of symptomatic illnesses; as a 'side effect' of increased interoceptive demand via pain and other symptoms.

Imaging and cognitive tests have limited value in the diagnosis of FCD. Rather, clinical observation, careful examination of the psychopathological phenomena, and—in some cases—longitudinal follow-up, are key to making an accurate diagnosis.

Patients with FCDs may present with symptom profiles and severity that seem overwhelming. Neurologists can look forward to a developing body of evidence for specific treatments over the next few years. In the meantime, making a simple formulation to identify contributing comorbidities, cognitions and behaviours, can often reveal targets for treatment, helping to demonstrate the dynamic nature of the symptoms and therefore restoring healthy metacognitive attitudes.

Key points

- Functional cognitive disorders are a common cause of subjective and mild cognitive impairment; they can also be understood as a transdiagnostic feature of other medical conditions with significant symptom burdens.
- Key diagnostic features include a detailed accurate account of symptoms and events, with detailed recall of cognitive lapses.
- Collaborative formulation can help to identify specific targets for treatment.

Further reading

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