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Functional neurological disorders in personal injury

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THE EXPERT REPORT

The structure of the expert report and the role of the expert witness are beyond the scope of this article. However, certain points are relevant when considering a claimant's functional symptoms. In the history section, it is useful to describe a typical day and the range of activities undertaken on both 'good' and 'bad' days; this is important in interpretation of video surveillance. It helps to include a brief vignette of the claimant's life, including any childhood adversity, if relevant—they may be more at risk of developing functional neurological disorder (FND), for example.[1] It is worth asking the claimant what they feel about the accident; if they believe there is permanent damage done, they are less likely to improve[2] and if they harbour grievance towards the 'perpetrator', they are more likely to develop post-traumatic symptoms.[3 4]

Reliability of the claimant

In legal claims, and in clinical experience, claimants/patients with FND, and indeed with any neurological condition, may have a degree of volitional symptom control. This may be produced in order to gain relief from responsibilities, for example, or a more subconscious exaggeration to convince others of the patient's/claimant's suffering. At the other end of the spectrum is factitious disorder, a psychiatric condition, where patients willfully fabricate symptoms; and malingering, whereby symptoms are consciously fabricated for (usually material) gain. Given that, in FND, it will superficially appear the claimant's symptoms and signs are under voluntary control, it is not possible to be sure if that claimant is fabricating or not. However, there may be suggestions that the claimant is an unreliable witness; for example, *prominent* mismatch between reported and actual function (observed by covert surveillance, for example), markedly different histories given to different professionals (although physical examination findings may vary), or a microbiology report suggesting a wound may have been tampered with. Ultimately, reliability of a claimant is for the court, not the expert, to decide.

Causation

FND is commonly triggered by, often minor, accidents and injuries. However, claimants who develop functional symptoms post-trauma may be predisposed to developing such symptoms anyway, and the effect of the accident needs to be addressed. It can be helpful to construct a table of general practitioner visits before and after the accident; if they are roughly the same, with similar reports, then the accident may not be responsible for ongoing symptoms. If a symptom does seem to be temporally related to the accident, consideration should be given to how severe and salient the accident was, and thus how plausible it was for triggering current symptoms. It must be borne in mind that many factors give rise to such symptoms, and in predisposed individuals, such symptoms may have occurred spontaneously—it can be helpful to put a figure on this likelihood. It is also worth considering that many of these (painful) conditions overlap, and vulnerability to one often increases vulnerability for another.[5]

FUNCTIONAL NEUROLOGICAL DISORDER

Synonyms of FND include psychosomatic, dissociative, nonorganic, conversion disorder, psychogenic. FND can comprise, for example, nonepileptic attacks, movement disorders and motor/sensory loss. FND is pertinent to medicolegal practise because it is common, can be confused with malingering, is often 'overlaid' onto other disorders and often occurs after physical insults. Functional disorders can occur in all medical specialties and include chronic fatigue, fibromyalgia, irritable bladder, irritable bowel and noncardiac chest pain.

FND is defined in DSM 5:

A. The patient has ≥1 symptoms of altered voluntary motor or sensory function.

B. Clinical findings provide evidence of incompatibility between the symptom and recognised neurological or medical conditions.
C. The symptom or deficit is not better explained by another medical or mental disorder.
D. The symptom or deficit causes clinically significant distress or impairment in social, occupational or other important areas of functioning or warrants medical evaluation.

Note that (in contrast to old definitions, and with the recognition that FND can occur in patients with normal mental health) there is no requirement to demonstrate a psychological trigger.

Patients with FND have neurological symptoms, but with no structural correlate. The deficit appears voluntary but is produced subconsciously.[6 7] This is distinct from factitious disorder or malingering, whereby symptoms are consciously feigned. FND is thought to arise from increased attention to (a 'rogue representation of') the body,[7 8] abnormal predictions about the body (informed by expectations from society/media/prior beliefs and so on) and altered agency (the brain misperceives internal sensations as external symptoms). This model explains why FND is often triggered by physical injury[9]—because the body 'feels different', especially in circumstances of heightened vigilance and salience (like an accident). Those with chronic stress, childhood adversity and certain personality factors may be more prone to developing FND[1 10] (although they can occur in people with no prior adversity or personality factors). This model of FND also explains the persistence of, say, functional cognitive complaints—the brain 'expects' to have symptoms that reflect 'brain damage'. In the case of, say, complex regional pain syndrome (CRPS), the patient/claimant can 'see evidence' of an ongoing physical process, and it can be difficult for them to understand the brain's role in the development of this disorder. It can also help to explain trends in litigation and post-traumatic syndromes, for example, whiplash, repetitive strain injury, 'railway spine'. Thus, society 'suggests' a certain outcome from a particular injury, which is incorporated into the collective lay belief system and the brain 'predicts' such an outcome at an individual level.

The diagnosis of FND is made by detection of specific signs on examination[11] (see criterion B). Thus, it is not a diagnosis of exclusion, as is often thought. Most signs are based on distraction (eg, the patient's examination normalises when their brain is not focusing on the symptom) or lay beliefs about illness (eg, dragging of the foot of a weak leg). That signs improve on distraction, or when the claimant thinks they are not being observed, should not, therefore, be taken as evidence that they are feigning. FND is very common,[12] and the misdiagnosis rate is very low. A study published in 1965[13] that reported a high misdiagnosis rate has been 'revisited'[14] and the methods and interpretation called into question[15]; the low misdiagnosis rate has been supported by subsequent studies.[16]

It is not possible to know for sure, in an individual person, whether their symptoms and signs are feigned or are functional (ie, with little or no conscious awareness that the signs are produced internally), aside from, for example, video surveillance evidence of a marked discrepancy in reported and actual function. Symptoms can be feigned in the context of factitious disorder or malingering. Factitious disorder is a mental health condition[17] and involves feigning symptoms for personal gain. There may be features that are more likely to occur in patients with factitious disorder that help distinguish from patients with FND.[18] Malingering is the feigning of symptoms for a specific purpose, that is, material gain (like litigation) or relief from responsibilities. It should be emphasised again that if a claimant has functional symptoms or signs, this is not evidence of feigning.

Patients can have an enduring tendency to suffer functional disorders and develop several/ sequential (not only neurological) symptoms such as fibromyalgia and irritable bowel syndrome; a condition defined in DSM5 as persistent (if >6 months' duration) somatic symptom disorder (see online supplemental file 1). It is important to recognise this because symptoms may appear to be related to an accident, but actually they may have occurred anyway.

Treatment for FND includes:

1. Understanding: a good consultation can be therapeutic[19]; website resources can be useful (eg, www.neurosymptoms.org, www.headinjurysymptoms.org), as can patient groups (FND Hope/FND Action/FND Dimensions/FND Friends).
2. Neurophysiotherapy[20] using techniques that reduce focus on the abnormal body part.
3. Cognitive therapies and/or psychiatric: a psychology or psychiatric opinion may need to be sought. Neuropsychology is often helpful in cases of persistent cognitive deficits; however, patients with FND can score very poorly on cognitive testing and this should not be mistaken for having a dementing illness or persistent 'brain damage'.
4. Reducing maintaining factors, which are typically low mood, poor sleep, maladaptive illness beliefs, side effects of medication (especially opiates), comorbidities such as migraine and other pain syndromes, and adverse social circumstances, which may include litigation. Such factors are very common after accidents and injuries but may also predate the index accident.

A systematic review of prognosis in FND showed that the range of prognosis is very wide (10%–90%), with a mean of 39% being the same or worse at a mean follow-up of 7.4 years.[21] Complete remission rate is estimated at 20% (for functional motor disorders).[22] It is very difficult to estimate the prognosis for an individual claimant, and one must consider premorbid factors, 'maintaining' factors (some of which can be ameliorated) and duration of symptoms.

MILD TRAUMATIC BRAIN INJURY

There are several definitions of mild traumatic brain injury (mTBI).[23 24] Unfortunately, some symptoms within some classifications are somewhat vague—'daze', for example. Dissociation (especially derealisation—the sense of feeling detached from one's surrounding,[25]), caused by the stress and anxiety of the accident, may be one explanation for 'loss of consciousness' (and amnesia, confusion, 'dizziness', 'daze' and even seizures). It is, therefore, important to record contemporaneous objective findings (from paramedic and hospital records), rather than relying solely on the claimant's retrospective recall. In many ways, whether a claimant fulfils criteria for mTBI or not is not particularly relevant; persistent symptoms are likely to be functional, whether there has been an insult directly to the head or not.

POSTCONCUSSION SYNDROME

This term describes a constellation of symptoms that can occur after 'concussion' (usually taken to mean mTBI), such as poor memory, pain, headache, dizziness, fatigue and psychiatric symptoms. The terminology can be confusing and misleading, and some have suggested a change in terminology, for example, to 'post-traumatic syndrome'.[23 26 27] The term postconcussion syndrome (PCS) is unhelpful, and it is clearer to describe each symptom in turn and consider the pathophysiological basis for each.

While it is certainly possible an isolated mTBI can produce temporary damage to the brain,[28] cognitive (and other) symptoms that persist beyond the expected weeks to months are likely to have a functional basis.[29] The symptoms of PCS are nonspecific and also occur in non-head injury trauma controls,[30 31] healthy volunteers[32] and personal injury litigants without head injury.[33] Many more examples (mental health problems, chronic pain, post-traumatic stress disorder, whiplash, sleep deprivation, intercurrent illness, substance abuse, medication side effects, personality disorder and even the way symptoms are elicited by the interviewer) are discussed by Kaufman and colleagues in their thoughtful review.[23] Premorbid factors predict outcome after traumatic brain injuries,[34] and several meta-analyses have shown that mTBI itself does not lead to persistent deficits—see Larrabee and Rohling,[31] Kaufman *et al*[23] and references therein.

The 'memory' problems described after mTBI (part of the PCS) are often deficits of attention and concentration, that is, working, as opposed to autobiographical, memory. Attention and concentration are often affected by anxiety, depression, fatigue, sleep deprivation, medication, pain and so on—all common after mTBI. Lower cognitive function and alcohol use[35] may be a risk factor for mTBI, potentially explaining some post-accident deficits.[36]

By definition, patients with (uncomplicated) mTBI have normal (standard) structural imaging. However, it has been argued that abnormalities in diffusion tensor imaging (DTI) reflect axonal damage, and abnormalities in DTI have been described after mTBI. Changes in DTI are not the same as 'diffuse axonal injury' (DAI), a term used to denote pathological changes after moderate to severe TBI. Abnormalities in DTI found in patients post-mTBI do not, however, provide evidence that PCS is caused by axonal injury at the time of the head injury. Many studies have examined DTI changes well after the hyperacute phase of injury, which may reflect post-traumatic stress disorder, for example. DTI changes are not specific to TBI and have been found in patients with depression, borderline personality disorder, ageing, opiate addiction and in healthy volunteers. It is difficult to predict neuropsychological outcome from DTI changes, and results have been inconsistent. If PCS was caused by axonal damage at the onset of head injury, it might be expected that cognition would improve over time, as the injuries recovered; however, in many patients with PCS, cognition deteriorates over time. This might be expected to occur in patients with untreated functional disorders, as opposed to direct injuries. It should be noted that there is no good evidence that an isolated mTBI causes dementia. Whatever the interpretation of DTI changes, the technique should neither used as a proxy for DAI nor to provide evidence that (micro)structural brain injury is responsible for PCS, and more research on this subject is required. The issue of DTI and PCS is discussed elsewhere.[26 31]

It is important to transmit to the claimant/patient that their 'PCS' is likely to have a functional basis, as opposed to being secondary to persistent 'brain damage'; if the patient has a prior belief that their cognitive symptoms are due to irreparable brain damage, or some medical 'syndrome' (ie, PCS), they are likely to worry further, thus producing more cognitive symptoms and so on. It is known that such illness beliefs are a key predictor of outcome.[2 37 38] The website www.headinjurysymptoms.com is an excellent resource for patients and their family and explains this well.

DIZZINESS

Persistent or recurrent vertigo post-trauma can occur due to direct damage to the vestibular apparatus, vestibular migraine or through functional mechanisms. It is important to distinguish vertigo (a sensation of movement), from other forms of dizziness, such as light headedness. Rarely, dizziness is due to posterior circulation stroke (or brain stem trauma), and bedside tests can be used in the emergency setting to distinguish between central and peripheral causes of vertigo.[39]

The symptoms and signs of benign paroxysmal positional vertigo (BPPV) are characteristic: vertigo after a brief latency, provoked by head movement and of a duration usually less than a minute, diagnosed by the presence of symptoms and rotatory nystagmus on provocation tests (side lie test or Dix-Hallpike) and cured by particle repositioning manoeuvres (Semont or Epley, respectively). Post-trauma, however, BPPV may involve uncommon or

multiple canals,[40] making it more difficult to diagnose and treat.

Vestibular migraine is a common cause of vertigo and is defined by the International Headache Society (IHS): https://ichd-3.org/appendix/a1-migraine/a1-6-episodic-syndromes-that-may-be-associated-with-migraine/a1-6-6-vestibular-migraine/. Episodes of vertigo related to vestibular migraine tend to be of a longer duration and associated with other migrainous features (such as nausea and photophobia). While, like BPPV, it can be provoked by head movement (migraine is associated with motion sensitivity), there are often other triggers, such as glare, and vivid patterns.

'Persistent postural-perceptual dizziness' (PPPD) (see online supplemental file 1)[39 41 42] is essentially a failure of the brain's adaptation to a vestibular insult (or dizziness from acute anxiety and so on), often in predisposed individuals, leading to chronic dizziness and maladaptive behaviours (such as avoidance, a shift in favour of visual or sensory inputs over vestibular and cocontraction of leg muscles, leading to unsteadiness). PPPD is essentially a type of functional disorder.

Different forms of vertigo can coexist; for example, PPPD can be triggered by a vestibular insult, such as BPPV precipitated by head injury, which then resolves, leaving only the PPPD, or it can be accompanied by (and triggered by) vestibular migraine.

HEADACHE POST-TRAUMA

Headache post-trauma is recognised by the IHS (although this does not prove a direct causal link). Chronic headache post-trauma is often associated with 'PCS' and medication overuse. Chronic migraine should be treated in the usual way, and simple analgesia is limited to <10 days a month.

RISK OF EPILEPSY POST-MTBI

Functional seizures are often precipitated by acute stress and trauma and may be confused with epileptic seizures, particularly post-head injury. However, there is no clear evidence for a risk of epilepsy post-mTBI. Several studies, including a large study in 1998[43] suggested a small increased seizure risk, but this cannot be taken as evidence for an increased risk of epilepsy post-mTBI. There are a number of reasons why this study (and other similar studies) does not provide evidence for an increased risk of epilepsy post-mTBI. First, non-head-injury trauma controls should be used because there may be factors that predispose to injury in orthopaedic controls, as opposed to 'normal controls' (such as depression, medication use, alcohol intake, sleep disturbance and so on…).[35 44–47] Second, 'seizures' are not the same as 'epilepsy' (a tendency to recurrent seizures); antiepileptic medication use (for epilepsy, as opposed to mental health disorders or pain) may be a surrogate marker for epilepsy, and this figure should be recorded specifically for patients with mTBI. Third, 'seizures' are not necessarily epileptic seizures and may be syncope, migraine,[46] or, indeed, functional seizures.

COMPLEX REGIONAL PAIN SYNDROME

CRPS is defined by the Budapest criteria (see online supplemental file 1).[48] The role of psychosomatic factors, and overlap with functional disorders,[49] in CRPS is another area of controversy. Such controversy often arises because there is an implicit suggestion that 'psychosomatic' or 'functional' in some way implies willful exaggeration or fabrication and ignores the brain's role in 'peripheral' abnormalities (autonomic and inflammatory changes in a limb, for example). Furthermore, it can also imply a psychological trigger or vulnerability, which is not always present in a person with CRPS (or, indeed, FND). Patients with FND (and other functional conditions), however, are not willfully exaggerating or fabricating their symptoms; 'central'/top down mechanisms can influence 'peripheral' changes and vice versa. I have not discussed CRPS further because the case for phenomenological and pathophysiological overlap between CRPS and FND is made eloquently by Popkirov and colleagues,[50] and potential harm from such a diagnosis is discussed by others.[51]

CONCLUSION

The constellation of symptoms that follow (minor) injury often has a functional basis. This should not be taken to mean that the symptoms are not real or fabricated. Conversely, it is difficult to demonstrate whether a claimant is willfully exaggerating their symptoms, but it is possible in some cases to suggest they may be an unreliable witness, although that is for the court to decide. Functional symptoms should be taken seriously and addressed directly with the patient/claimant/defendant—if they think their symptoms are due to 'nerve/brain damage', they are less likely to improve. FND is treatable, and the prognosis, although variable, can be good. Nestled among functional symptoms may also be other (also treatable) conditions, both potentially related to an accident (eg, BPPV, migraine) or not (eg, obstructive sleep apnoea). It is important, in a medicolegal setting, to make a judgement as to whether such symptoms would likely have occurred despite the accident and the objective previous medical history is crucial.

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