Dissociation and functional (‘medically unexplained’) symptoms

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What are functional symptoms?

<table>
<thead>
<tr>
<th>Total</th>
<th>Subjects with MUS (total)</th>
<th>Percent cases of MUS (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dental</td>
<td>26 (71)</td>
<td>37 (25–48)</td>
</tr>
<tr>
<td>Chest</td>
<td>24 (59)</td>
<td>41 (28–53)</td>
</tr>
<tr>
<td>Rheumatology</td>
<td>41 (91)</td>
<td>45 (35–55)</td>
</tr>
<tr>
<td>Cardiology</td>
<td>49 (92)</td>
<td>53 (43–63)</td>
</tr>
<tr>
<td>Gastroenterology</td>
<td>30 (52)</td>
<td>58 (44–71)</td>
</tr>
<tr>
<td>Neurology</td>
<td>64 (103)</td>
<td>62 (52–72)</td>
</tr>
<tr>
<td>Gynecology</td>
<td>54 (82)</td>
<td>66 (56–76)</td>
</tr>
<tr>
<td>Total</td>
<td>288 (550)</td>
<td>52 (48–57)</td>
</tr>
</tbody>
</table>

- 5th most costly group of medical complaints in the Netherlands (Van der Maas et al., 1998, Brit Med J, 317; 111)

- $256 billion annual medical cost in the USA (Barsky et al, 2005; Arch Gen Psychiatry, 62, 903)

- In the UK:
  - 10% of NHS budget
  - 42 million work days lost each year

What should we say to patients with symptoms unexplained by disease? The “number needed to offend”

Jon Stone, Wojtek Wojcik, Daniel Durrance, Alan Carson, Steff Lewis, Lesley MacKenzie, Charles P Warlow, Michael Sharpe

“If you had leg weakness, your tests were normal, and a doctor said you had ‘X’ would he be suggesting that you were Y (or had Y).” Percentage responses among 86 new neurology outpatients, offence score, and “number needed to offend”—that is, number of patients who would have to be given this diagnostic label before one patient is “offended”

<table>
<thead>
<tr>
<th>Diagnoses (X)</th>
<th>Putting it on (yes)</th>
<th>Mad (yes)</th>
<th>Imagining symptoms (yes)</th>
<th>Medical condition (no)</th>
<th>Good reason to be off sick from work (no)</th>
<th>Offence score (%)</th>
<th>Number needed to offend (95% CI)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms all in the mind</td>
<td>71 (83)</td>
<td>27 (31)</td>
<td>75 (87)</td>
<td>57 (68)</td>
<td>36 (42)</td>
<td>35</td>
<td>2 (2 to 2)</td>
</tr>
<tr>
<td>Hysterical weakness</td>
<td>39 (45)</td>
<td>21 (24)</td>
<td>39 (45)</td>
<td>28 (33)</td>
<td>36 (42)</td>
<td>52</td>
<td>2 (2 to 3)</td>
</tr>
<tr>
<td>Psychiatric weakness</td>
<td>21 (24)</td>
<td>10 (12)</td>
<td>16 (19)</td>
<td>24 (29)</td>
<td>15</td>
<td>82</td>
<td>3 (2 to 4)</td>
</tr>
<tr>
<td>Medically unexplained weakness</td>
<td>21 (24)</td>
<td>10 (12)</td>
<td>27 (31)</td>
<td>32 (37)</td>
<td>35 (41)</td>
<td>35</td>
<td>3 (3 to 5)</td>
</tr>
<tr>
<td>Depression associated weakness</td>
<td>16 (21)</td>
<td>6 (7)</td>
<td>17 (20)</td>
<td>13 (15)</td>
<td>24 (26)</td>
<td>33</td>
<td>4 (3 to 5)</td>
</tr>
<tr>
<td>Stress related weakness</td>
<td>8 (9)</td>
<td>3 (6)</td>
<td>12 (14)</td>
<td>14 (16)</td>
<td>20 (23)</td>
<td>20</td>
<td>6 (4 to 9)</td>
</tr>
<tr>
<td>Chronic fatigue</td>
<td>0 (0)</td>
<td>1 (2)</td>
<td>9 (10)</td>
<td>16 (19)</td>
<td>12 (14)</td>
<td>15</td>
<td>7 (5 to 13)</td>
</tr>
<tr>
<td>Functional weakness</td>
<td>6 (7)</td>
<td>2 (2)</td>
<td>7 (8)</td>
<td>7 (8)</td>
<td>17 (20)</td>
<td>12</td>
<td>9 (5 to 21)</td>
</tr>
<tr>
<td>Stroke</td>
<td>2 (2)</td>
<td>4 (5)</td>
<td>4 (5)</td>
<td>5 (6)</td>
<td>10 (12)</td>
<td>12</td>
<td>9 (5 to 21)</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>0 (0)</td>
<td>1 (1)</td>
<td>3 (3)</td>
<td>3 (3)</td>
<td>7 (8)</td>
<td>5</td>
<td>22 (9 to =)</td>
</tr>
</tbody>
</table>

*Proportion of patients who responded “yes” to one or more of “putting it on,” “mad,” or “imagining symptoms.”
†Calculated according to the offence score.
Functional neurological symptoms

- 'Unexplained' neurological symptoms
  - seizures; gait disturbance; sensory loss; tremor; paralysis etc.
  - 30% new neurology outpatients (Stone et al, 2010; Clin Neurol Neurosurg, 112, 747)
  - Low misdiagnosis rate (< 5%; Stone et al, 2009; Brain, 132, 2878)

- High co-morbidity with depression, anxiety and other functional symptoms
  - FNS most characteristic of patients with MUS in multiple systems
    (e.g., Gara et al., 1998, Psychiatry Res, 81, 77)

- Variable estimates regarding prognosis

- No properly evidence-based treatments although some promising evidence (e.g., Goldstein et al., 2010; Neurology, 74, 1986)

from Bryant & McConkey (1989), *J Abnorm Psychol*, 98, 326
What is dissociation?

• “[dissociative symptoms] are characterized by (a) a loss of continuity in subjective experience with accompanying involuntary and unwanted intrusions into awareness and behavior (so-called positive dissociation); and/or (b) an inability to access information or control mental functions, manifested as symptoms such as gaps in awareness, memory, or self-identification, that are normally amenable to such access/control (so-called negative dissociation); and/or (c) a sense of experiential disconnectedness that may include perceptual distortions about the self or the environment”

• (Cardeña & Carlson, 2011; pp. 251-252)
**Dissociative Experiences Scale** (DES; Bernstein & Putnam, 1986)

- Designed to quantify “trait” dissociation

- **Sample items**
  - “Some people have the experience of feeling that other people, objects, and the world around them are not real”
  - “Some people find that they have no memory for some important events in their lives (e.g. wedding, graduation)”
  - “Some people find that when they are watching television or a movie they become so absorbed in the story that they are unaware of other events happening around them”
  - “Some people sometimes find that they hear voices inside their head that tell them to do things or comment on things that they are doing”

- DES taxon (i.e. sub-set of items measuring “pathological” dissociation) are often used instead of total score
Total DES scores across disorders (adapted from van Ijzendoorn & Schuengel, 1996)
<table>
<thead>
<tr>
<th>Study</th>
<th>Method quality</th>
<th>PNES: Mean DES score (sample size)</th>
<th>Epilepsy: Mean DES score (sample size)</th>
<th>Effect size (d)</th>
<th>Significant?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alper et al (1997)</td>
<td>High</td>
<td>15.0 (132)</td>
<td>12.7 (169)</td>
<td>0.19</td>
<td>x</td>
</tr>
<tr>
<td>Bowman and Coons (2000)</td>
<td>Low</td>
<td>20.2 (15)</td>
<td>10.7 (15)</td>
<td>0.63</td>
<td>✓</td>
</tr>
<tr>
<td>Fleisher et al (2002)</td>
<td>Medium</td>
<td>22.7 (31)</td>
<td>15.1 (32)</td>
<td>0.45</td>
<td>x</td>
</tr>
<tr>
<td>Dikel et al (2003)</td>
<td>Low</td>
<td>22.8 (17)</td>
<td>14.1 (34)</td>
<td>0.76</td>
<td>✓</td>
</tr>
<tr>
<td>Reuber et al (2003)</td>
<td>Medium</td>
<td>17.2 (98)</td>
<td>8.8 (63)</td>
<td>0.73</td>
<td>✓</td>
</tr>
<tr>
<td>Akyuz et al (2004)</td>
<td>Medium</td>
<td>29.8 (33)</td>
<td>17.6 (30)</td>
<td>0.68</td>
<td>✓</td>
</tr>
<tr>
<td>Goldstein &amp; Mellers (2006)</td>
<td>Low</td>
<td>24.9 (25)</td>
<td>14.5 (19)</td>
<td>0.64</td>
<td>✓</td>
</tr>
<tr>
<td>Lawton et al (2008)</td>
<td>Low</td>
<td>20.2 (32)</td>
<td>11.8 (37)</td>
<td>0.39</td>
<td>x</td>
</tr>
<tr>
<td>Mazza et al (2009)</td>
<td>Medium</td>
<td>17.6 (30)</td>
<td>6.4 (30)</td>
<td>1.49</td>
<td>✓</td>
</tr>
<tr>
<td>Proença et al (2011)</td>
<td>Low</td>
<td>53.4 (20)</td>
<td>22.0 (20)</td>
<td>1.56</td>
<td>✓</td>
</tr>
<tr>
<td>Medians across studies</td>
<td></td>
<td>21.5</td>
<td>13.4</td>
<td>0.66</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from Brown & Reuber (2016a)
Somatoform dissociation (e.g., Nijenhuis, 2004)

- Earliest psychological theories of dissociation (e.g., Janet, 1889) emphasised physical (‘somatoform’) as much as psychological (‘psychoform’) symptoms
- Classified together until they were separated into the somatoform and dissociative disorders in DSM-III
- Somatoform Dissociation Questionnaire 20 (Nijenhuis et al, 1996)
  - trouble urinating
  - having an attack that resembles an epileptic seizure
  - my body feels numb
  - I cannot hear for a while
  - I am paralysed for a while
DISSOCIATION

Identity disturbance
Defence mechanism
Amnesia
Divided attention
Absorption
Hypnosis
Possession states
Hypnosis
Depersonalization
Derealization
Reduced awareness
Flashbacks
"Made" actions
Intrusive thoughts/feelings
Pseudohallucinations
Hypothetical dissociative continuum

Absorbed states

Transient depersonalization

Hypnotic phenomena

Depersonalization disorder

Dissociative amnesia

Somatization disorder

Dissociative identity disorder

Increasing dissociation
Two qualitatively different types of “dissociation”  
(Holmes, Brown, Mansell et al, 2005)

**Detachment**
An altered state of consciousness characterized by a sense of separation (or “detachment”) from aspects of everyday experience

**Compartmentalization**
A reversible deficit in the ability to deliberately control processes or actions that would normally be amenable to such control
Dissociation in non-epileptic attack disorder
Panic like features

Motor behaviour

Trauma common but not inevitable

Comparatively low explicit anxiety

Perceived involuntariness

Trait dissociation often but not always high

Other functional symptoms

Alteration/loss of consciousness

NON-EPILEPTIC ATTACKS

Brown & Reuber (2016a,b)
Reduced integrative capacity

"Me"

Mental fragmentation

"Not me"

Intrusion of trauma fragments

In the Freudian conversion model, fragmentation is primarily defensive

Structural dissociation (compartmentalisation) model
Panic-like features

Trauma common but not inevitable

Motor behaviour

Comparatively low explicit anxiety

Perceived involuntariness

Other functional symptoms

Loss of consciousness

Trait dissociation often but not always high

Brown & Reuber (2016a,b)
Panic without panic (detachment) model

(Chalder, 1996; Goldstein & Mellers, 2006)

PREDISPOSING FACTORS
- Beliefs about emotion
- Experience with epilepsy
- Emotional suppression

TRAUMA, STRESS, LIFE EVENTS

THREAT SENSITIVITY

SURGE IN AUTONOMIC AROUSAL

TRIGGERS

PREDISPONING FACTORS

THREAT APPRAISAL

NON-EPILEPTIC SEIZURE

HYPERVIGILANCE, AVOIDANCE & OTHER ILLNESS BEHAVIOURS

SHAKING, SWEATY, LIGHTHEADED, FAINT, DIZZY

“panic without panic”
Panic like features

Motor behaviour

Trauma common but not inevitable

Other functional symptoms

Loss of consciousness

Perceived involuntariness

Comparatively low explicit anxiety

Trait dissociation often but not always high

Brown & Reuber (2016a,b)
Neodissociation theories (e.g., Hilgard, 1977; Woody & Bowers, 1994; Woody & Sadler, 2008)

- Most processing managed outside of awareness by low level control systems
- Awareness, attention and volition (i.e., “executive ego”) only needed for initial selection of lower systems
- Most processing is performed in a “dissociated” fashion
If behaviour is instigated by the executive (i.e. “on purpose”) but is inconsistent with our goals then we experience it as having *made a mistake*

BUT only if we *know* we did it on purpose!

If we don’t realise this (e.g. because we forget, we weren’t paying attention, or we don’t represent it in this way) then we experience it as *happening to* us.
If behaviour is instigated by the executive (i.e. “on purpose”) but is inconsistent with our goals then we experience it as having made a mistake BUT only if we know we did it on purpose!

If we don’t realise this (e.g. because we forget, we weren’t paying attention, or we don’t represent it in this way) then we experience it as happening to us.
Inhibition of the executive (e.g. by intense emotion) reduces control over low level systems, making the system vulnerable to stimulus-driven activation of unintended behaviours.

FND similar to everyday action slips and utilisation behaviour in some patients with frontal lobe damage.
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FND similar to everyday action slips and utilisation behaviour in some patients with frontal lobe damage.
Integrative cognitive model of NES (Brown & Reuber, 2016b)

NES are a transient loss of cognitive/behavioural control driven by the activation of a seizure routine (“seizure scaffold”) in memory, in the context of dysfunctional inhibitory processing.

Three stages in many NES

1. Acute increase in sympathetic arousal
2. Execution of the seizure program
3. Interruption of sympathetic arousal

Of these, only stage 2 is necessary and sufficient.
NON-EPILEPTIC SEIZURE THREAT INHIBITORY PROCESSING DYSFUNCTION

TRAUMA, CHRONIC STRESS, LIFE EVENTS ETC.

INHIBITORY PROCESSING DYSFUNCTION

ACTIVATION OF SEIZURE SCAFFOLD

DEVELOPMENT OF SEIZURE SCAFFOLD

SCAFFOLD SHAPING FACTORS

SURGE IN AUTONOMIC AROUSAL

Integrative cognitive model of NES (Brown & Reuber, 2016b)
Clinical implications

• Provides an explanation of FNS that emphasises disruption of normal psychological processes
  ➔ normalising, non-blaming and face valid
  ➔ suggests possible tools for socialisation (e.g. placebo effect, ball trick, hypnotic phenomena)

• Suggests that most effective interventions will be those that target the relative precision of somatic signals and symptom priors
  ➔ choice of intervention(s) depends on formulation
  ➔ approach is likely to reflect, at least in part, whether (and how) emotional avoidance is a likely driver of symptoms
Clinical implications

- It may be possible to adapt/develop new interventions to address relevant maintaining factors
  - techniques to activate more adaptive expectations in memory (e.g. imagery, hypnotic/non-hypnotic suggestion)
  - attention training/mindfulness for inhibitory dysfunction and disengagement difficulties
  - body scan and perceptual differentiation training to improve precision of sensory signals
References


