ARTICLE

Explaining the unexplained

Richard J. Brown, winner of the Society's Division of Clinical Psychology May Davidson Award, proposes a model of 'medically unexplained' symptoms

Many of the patients encountered within medical settings present with symptoms that seem to defy medical explanation. Such 'medically unexplained' symptoms (MUS) can be extremely distressing and debilitating but are often difficult to treat. They are also an increasing priority for the NHS, which spends a substantial proportion of its budget on people with MUS.

Early psychological theorising emphasised the role of dissociation in MUS and their apparent overlap with hypnotic phenomena, whereas subsequent theoretical developments focused on the psychodynamic and cognitive-behavioural aspects of these conditions. Although these approaches have proved clinically useful, none is able to explain how some of the more dramatic MUS, like seizures or paralyses, can occur in the absence of medical pathology.

questions

What can MUS tell us about medically explained symptoms?

Does a psychological theory of MUS necessarily imply Cartesian dualism?

resources

www.neurosymptoms.org.uk

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I first encountered medically unexplained symptoms (MUS) as a Research Fellow at the National Hospital for Neurology and Neurosurgery, Queen Square, in the late 1990s. All of the patients I tested had 'neurological' symptoms: seizures, paralyses, gait disturbances, sensory problems, and so forth. In each case, extensive investigation had failed to identify an organic cause for the symptoms in question: they were 'unexplained' from a medical perspective. As a tertiary centre, the National Hospital treats some of the most complex and severe cases of MUS: on average, patients in our study had a history of over 30 different unexplained symptoms across several bodily systems, including pain, gastrointestinal and sexual-reproductive symptoms alongside their neurological problems (Brown et al., 2005). Their suffering was evident: most were highly distressed and disabled by their symptoms, and many had either never worked or had retired on medical grounds.

Although such severe problems are relatively rare, they are simply one end of a spectrum, with MUS themselves being extraordinarily common. Epidemiological studies suggest that just over half of all hospital outpatients have at least one unexplained symptom (Nimnuan et al., 2001), while approximately 8 per cent of general practice patients have three or more currently debilitating symptoms and a long history (≥ 2 years) of MUS (Kroenke et al., 1997). As these patients present within medical settings, many psychologists are unfamiliar with MUS, but they are an increasing priority for the NHS. On top of the distress and disability that MUS can cause, they carry a tremendous societal cost. One estimate (Bermingham et al., 2010) suggests that the NHS spends £3 billion a year on MUS-related healthcare costs for working-age adults alone (approximately 10 per cent of total NHS expenditure at that time), and that 42 million work days are lost annually due to these complaints.

These figures make more sense when one considers that every 'medically explained' symptom (i.e. those with a documented organic cause) has an unexplained counterpart, and every medical specialty has its own unexplained syndromes (see Brown, 2007). Well-known conditions such as irritable bowel syndrome, fibromyalgia, chronic fatigue syndrome (i.e. myalgic encephalomyelitis), multiple chemical sensitivity, temporo-mandibular joint disorder, tinnitus and gulf war syndrome all defy medical explanation, for example. Whether these syndromes should be included in the MUS category is extremely controversial, however; many patients argue that the failure to identify a cause for their symptoms reflects the inadequacy of medical science, rather than the actual absence of disease. While this possibility remains, for unexplained neurological symptoms at least, the misdiagnosis rate is low (≤ 5 per cent; comparable to 'explained' neurological conditions (Stone et al., 2009) and has remained stable for decades despite the advent of new diagnostic techniques such as brain imaging (Stone et al., 2005). What is beyond dispute is that these symptoms can be as real and debilitating as any associated with organic disease.

Also controversial is the MUS label itself, with its (misleading) implication that these symptoms defy explanation altogether. The label also obscures the fact that MUS are a heterogeneous group of conditions. Research suggests that we should distinguish between three main types (Kirmayer & Robbins, 1991):

- symptoms of an overt anxiety or

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affective disorder that is presented as a somatic problem (i.e. a 'somatised' mental disorder); for example, someone who has a panic attack but focuses mainly on their palpitations, breathlessness and dizziness rather than their fear;
- normal sensations/minor ailments that are mistakenly attributed to serious physical illness, which are often seen in people with high levels of health anxiety; and
- multiple unexplained symptoms that are not obviously attributable to anxiety, depression or another psychiatric condition, so-called 'functional' symptoms.

The third category of complaints accounts for about half of all MUS patients seen in primary care (Kirmayer & Robbins, 1991), who are often seen as poorly understood and difficult to treat. My work has mainly focused on trying to understand the psychological mechanisms of these conditions in particular.

Hypnosis and dissociation

Unbeknownst to me, my efforts to unravel the mysteries of MUS began before I worked at the National Hospital, when I was learning to elicit and experience hypnotic phenomena – 'involuntary' behaviours, paralyses, sensory changes, amnesia, and so forth – as part of my PhD on hypnosis and suggestion at University College London. The similarity between some of these phenomena and unexplained neurological symptoms is striking: in both cases, there is an alteration in perceived sensory, cognitive or motor function that suggests a neurological problem, but where a disease explanation can be ruled out. At that time, I also encountered theories of hypnosis based on the concept of dissociation, which originated in Pierre Janet's seminal work on hysteria (a now obsolete term for unexplained neurological symptoms and related complaints) in the late 19th century (Janet, 1889). The basic concept in these theories is that hypnotic and hysterical phenomena reflect the activation of semi-autonomous parts of the mind, which have become separated (or 'dissociated') from mental functioning more generally. In Janet's original model, this separation was said to occur as a result of psychological trauma, with hysterical symptoms reflecting the activation of dissociated memory fragments related to the events in question. Dissociation theories of hypnosis, in contrast, argue that the mind is naturally fragmented, and that hypnotic procedures simply capitalise on and amplify this. Either way, many theorists have argued that hypnotic phenomena involve similar processes to dissociative symptoms, and that hypnotic suggestibility is a risk factor for developing the latter.

Research concerning dissociation and MUS is mixed. There is some evidence for elevated hypnotic suggestibility in patients with unexplained neurological symptoms, for example, but not for high hypnotisability *per se* (Roelofs et al., 2002). Most other studies have simply administered questionnaire measures of dissociation to people with unexplained neurological symptoms, or physical symptom measures to people with dissociative disorders. Many studies have found elevated dissociation scores in MUS patients, but by no means all of them; similarly, evidence suggests that MUS are more common in patients with some dissociative disorders (e.g. dissociative identity disorder) but not others (e.g. depersonalisation disorder (Brown, 2005). Our own research suggests that amnesia is more common in somatisation disorder patients but not depersonalisation, derealisation or identity disturbance (Brown et al., 2005), despite all being 'dissociative' problems.

These apparently contradictory findings seem to stem from problems with

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how dissociation is defined. The modal model – which is implicit in many questionnaire measures – suggests that dissociation is a unitary construct, and that depersonalisation, derealisation, amnesia, unexplained symptoms, identity alteration, and so on, differ only in the 'amount' of dissociation involved. In contrast, we have argued that the available evidence is more consistent with a model that assumes two qualitatively distinct forms of dissociation: detachment and compartmentalisation (Holmes et al., 2005). We defined detachment as an altered state of consciousness characterised by a sense of separation (or 'detachment') from aspects of the world (as in derealisation) or the self (as in depersonalisation and emotional numbing). Such states are characteristic of so-called peri-traumatic dissociation (i.e. dissociation occurring during a traumatic event) but also occur at other times, and may or may not be pathological. Although many people are referring to detachment when they speak of someone 'dissociating', it was not part of Janet's original dissociation concept and arguably has little relevance to most MUS.

Our second type of dissociation – compartmentalisation – is much closer to Janet's original account and is nicely illustrated in a study comparing amnesia in patients with epileptic (i.e. medically explained) and non-epileptic (i.e. medically unexplained) seizures (Kuyk et al., 1999). All of the patients in this study were hypnotised and given the suggestion that they would be able to remember everything about their last seizure, before being taken through a procedure designed to facilitate recall of the relevant material. As expected, none of the epilepsy patients was able to recall new material during this procedure, suggesting an encoding deficit. In contrast, 17 of the 20 non-epileptic seizure patients were able to retrieve new, independently corroborated, information. Clearly, these patients had encoded information about their seizures, but were unable to retrieve that information under normal circumstances; in other words, it was 'compartmentalised' within their brain for some reason. Such compartmentalisation is also apparent in some patients with medically unexplained sensory loss (e.g. blindness, deafness, anaesthesia) when asked to discriminate between stimuli presented to the affected modality. Here the patient may perform significantly worse on the task than would be expected by chance alone (i.e. if they were guessing), suggesting that they are processing the stimuli at some level despite being subjectively unaware of them. Performing significantly below (rather than above) chance also suggests that their performance is influenced by implicit ideas about how a person with sensory loss would behave on these tests (e.g. 'a blind person will do badly on a visual processing test').

Janet's model was subsequently appropriated by Breuer and Freud (Breuer & Freud, 1893–1895/1955), who argued that dissociation protects the individual by allowing traumatic memories and feelings, particularly those related to childhood abuse, to be converted into physical symptoms (hence the term 'conversion disorder', which encompasses unexplained neurological symptoms in current psychiatric taxonomies). Later psychodynamic theory extended this idea to incorporate problematic relationships with early caregivers more generally, as well as other psychological functions for MUS, such as eliciting care from attachment figures. Consistent with this, we found a very strong correlation (r = .70) between number of MUS and early exposure to emotional abuse, with the most disabled patients having been exposed to regular shouting, severe criticism and/or extremely insulting remarks for long periods, typically from more than one family member (Brown et al., 2005).

> "There are numerous examples of how consciousness distorts reality"

Unsurprisingly, these severe MUS patients described a lack of cohesion in their childhood families and higher levels of conflict compared to medical controls. Our sample also reported harsher childhood physical abuse, although sexual abuse was not more common in this group.

The available evidence suggests that psychodynamic therapy can be an effective treatment for MUS (Abbass et al., 2009), particularly for patients with a history of childhood adversity (Creed et al., 2005). Many MUS patients, however, deny having experienced adverse events in their childhood. More than once I have encountered the suggestion that these patients must be repressing their adverse experiences, and that the MUS themselves are evidence of this. Even if one ignores such circular reasoning, the question remains: how are the symptoms created in the first place? Moreover, recent cognitive-behavioural models have shown that it is possible to explain MUS without referring to childhood adversity. According to this approach, MUS arise when benign bodily sensations, such as those resulting from normal variation, emotional disturbance or minor pathology, are selectively attended to and misinterpreted as evidence of illness (e.g. Deary et al., 2007); the resulting changes in behaviour (e.g. reduced activity) and physiology (e.g. increased autonomic arousal) then create a vicious cycle that maintains symptom reporting. Consistent with this, we have shown that high symptom reporters focus more on the body shortly after exposure to body threat stimuli than control participants (Brown et al., 2007). Many other cognitive and behavioural factors have also been implicated in cross-sectional research on MUS, and there is good evidence that CBT can be an effective treatment for these complaints (Kroenke & Swindle, 2000).

The integrative cognitive model

Although the CBT model can account for many MUS, there have been few attempts to apply it to unexplained neurological symptoms. Indeed, it is difficult to

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envisage how some of the more dramatic symptoms in this category, such as seizures or blindness, could result from the misinterpretation of benign bodily sensations. While the psychodynamic and CBT models can account for different aspects of the data on MUS, and are therefore clinically useful, neither provides a comprehensive account of how a compelling 'neurological' symptom can exist in the absence of medical pathology. Dissociation theory identifies psychological compartmentalisation as relevant here, but this is more a description of the symptoms than an explanation of how they come about.

I have attempted to address this question by developing an account of functional MUS that integrates the main elements of the dissociation, psychodynamic and CBT models within a unifying framework: the integrative cognitive model (ICM: Brown, 2004, 2006). My experience with hypnosis provided one of the organising principles for this approach: that functional symptoms can be experienced by people with little or no obvious psychopathology. As such, the model seeks to explain functional MUS without relying on the concepts of early adversity or psychiatric disturbance, whilst still accommodating their apparent importance in many cases.

A key concept in the ICM is that functional MUS can be thought of as involving either a distortion in consciousness or a disturbance of voluntary control. There are numerous examples of how consciousness distorts reality, often due to prior expectancies about what is happening in the world. A classic example is the placebo effect, where an inert substance (e.g. a sugar pill) brings about a significant reduction in pain or other symptoms when presented as having restorative properties. Suggesting that the substance has adverse effects can also have a corresponding effect on symptoms (the so-called nocebo effect). Other examples of the effect of expectancy include hypnotic phenomena, some illusions and magic tricks (see e.g. www.gustavkuhn.com) and 'phantom vibration syndrome' (i.e. the experience of one's mobile phone vibrating when it is not; so-called ringxiety). Strong expectations can also give rise to errors in our behaviour, such as when we dial a familiar but out-of-date telephone number.

Why are some people more likely to develop symptoms than others?

These examples demonstrate that information in memory (broadly speaking, prior expectations) plays a critical role both in what we experience and what we do. Drawing on mainstream cognitive research and theory, the ICM assumes that sense data automatically activate several predictions or hypotheses about that input within memory systems. Sensory information is then combined with the most active hypothesis to produce a working representation of the environment. This 'best-guess' interpretation of the world corresponds to the contents of conscious awareness; it also provides the basis for further processing and action, via a hierarchical network of behavioural programs that are triggered automatically when a threshold activation level is reached. According to the model, deliberate reflection on action is only required when the behavioural programs in memory are inadequate for the current task, or a habitual response needs to be overridden (e.g. inhibiting a bad habit).

This system enables us to interpret events and respond quickly and effectively to them by capitalising on prior experience. In most cases, sensation and experience match because our pre-conscious predictions about the world tend to be accurate. Compelling distortions in awareness can arise, however, if a hypothesis in memory is disproportionately active (e.g. because of a strong expectation or motivation) and therefore inappropriately selected as the most likely interpretation of sensory input. The nature of this distortion depends on what material (or 'rogue representation') is overactive in memory. The central thesis of the model is that functional MUS arise when rogue representations relating to illness or other distressing alterations in function are involved.

There are many different possible sources of these representations, both direct (e.g. previous experiences of illness or physical sensations in ourselves) and indirect (e.g. encountering symptoms in other people or through the media), meaning that we all have the potential to develop MUS through this process. The question is why some people are more likely to develop symptoms than others. One of the strengths of the model is that it provides a unifying account of the various psychodynamic, interpersonal, cognitive and behavioural risk factors that have been identified for MUS, which can be thought of exerting their effects via the activation and selection of rogue representations. In most cases, attentional processes are implicated in this. For example, focusing on one's physical symptoms as a way of

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avoiding emotional or interpersonal conflict (as in the psychodynamic model) serves to increase the activation of symptom representations in memory, as do mentally 'checking' on symptoms, worrying what they mean and seeking reassurance about them (as in the cognitive-behavioural model). These processes then increase the frequency and saliency of symptoms, prompting further reactions and creating a vicious cycle. It is likely that certain intrinsic factors may also make some people more likely to experience symptoms than others. For example, highly suggestible individuals may be more prone to the automatic activation of mental representations by incoming information (Brown & Oakley, 2004).

As well as being able to accommodate existing research and theory (see Brown, 2004), the model gives rise to a number of novel predictions about MUS. Like CBT theory, for example, the model predicts that people who are 'hypervigilant' for bodily sensations will be more likely to experience MUS than those who are not. The model also predicts, however, that problems disengaging attention from those sensations will contribute to symptom experience by increasing the activation of their representations in memory. Furthermore, the model suggests that symptom experience will be moderated by an increased reliance on 'top-down' (i.e. knowledge, beliefs) compared to 'bottom-up' (i.e. stimulus-driven) factors in somatic perception.

We investigated these hypotheses by comparing high and low symptom reporters on a tactile cueing paradigm that measured how quickly their attention was drawn to, and then disengaged, from a bodily stimulus (Brown, Danquah et al., 2010). As predicted, high symptom reporters were slower at disengaging their attention from the body under normal circumstances. Following a film designed to induce negative affect, however, high symptom reporters showed reduced attentional capture, suggesting avoidance of body stimuli in this condition. Such avoidance is likely to foster increased reliance on top-down factors during somatic perception, consistent with the model.

Top-down versus bottom-up processing bias can also be investigated using the so-called rubber hand paradigm, a striking illusion in which participants experience touch to the back of their (hidden) hand as originating in a fake hand that is being stroked in front of them. The illusion is thought to arise because visual input (i.e. the sight of the rubber hand being touched) often overrides tactile input (i.e. stroking on the actual hand) in perception. We found that high symptom reporters were less responsive to this illusion than low symptom reporters, consistent with a decreased emphasis on bottom-up visual input in this group (Miles et al., 2011).

Intrinsic factors may make some people more likely to experience symptoms

Another prediction from the integrative model is that the tendency to experience MUS will be associated with a propensity for somatosensory distortion more generally. In order to measure this, we developed a novel paradigm (the somatic signal detection task: SSDT) in which participants have to decide whether a weak vibration has been delivered to their fingertip. We have shown that many participants will, on 10–20 per cent of trials, report the presence of the vibration when no stimulation has been delivered (Lloyd et al., 2008). Such 'illusory touch' experiences on the SSDT – which are strikingly similar to MUS – are associated with activation in brain regions responsible for top-down processing (Lloyd et al., 2011); they are also highly reliable over time (McKenzie et al., 2010), suggesting that they can be used to measure individual differences in somatosensory distortion. We have shown in several studies that illusory touch on the SSDT correlates with physical symptom reporting (Brown, Brunt et al., 2010; Brown et al., 2012), as predicted.

In addition to making novel predictions, the model has a number of important clinical implications. Of these, probably the most important is that it enables clinicians to explain their patients' symptoms without relying on traditional psychodynamic assumptions (e.g. that the client has unresolved issues from childhood, or that they are actually anxious or depressed but unaware of it), or the cognitive-behavioural idea that they are simply worrying about normal sensations. This can be particularly useful when the patient is experiencing symptoms that are so obviously outside the normal realm of experience, such as seizures. Examples of how the brain often makes mistakes about what is going on in the body (the placebo effect, ringxiety, hypnotic phenomena, illusions and magic tricks, etc.) can be used to socialise patients to the model, and demonstrate how psychological processes can give rise to physical symptoms without suggesting that they are either imaginary or done 'on purpose'.

In terms of interventions, the model suggests that treatment should target those factors that are contributing to the activation of the mental representations underlying symptoms, with the choice of techniques depending on the case formulation. Although the model is described in cognitive terms and provides a rationale for existing CBT approaches, it is also compatible with the idea that psychodynamic factors (needs, defences, conflicts, relationship problems, etc.) can contribute to MUS and justifies the use of psychodynamic therapy in appropriate cases (see Brown, 2013). There could also be mileage in adopting/developing new interventions to address relevant maintaining factors, such as attention training or mindfulness to address attentional disengagement difficulties and top-down processing bias, and perceptual training for people who are prone to experiencing somatosensory distortions. We are currently investigating some of these clinical possibilities, whilst continuing to refine and test the model itself.

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