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Conversion disorder and the trouble with trauma

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There has been much debate around whether conversion disorder should still be considered psychogenic, in nomenclature and in conceptualisation. There have been a number of articles (Stone and Edwards, 2011; Stone *et al.*, 2011; Edwards *et al.*, 2013) questioning the 'psychogenic model' and calling for its replacement, with arguably the greatest shift coming in the removal of the psychogenic criterion ('preceding trauma that could explain the illness') from DSM-5's *Conversion Disorder* (*Functional Neurological Symptom Disorder*) (APA, 2013). At a time when the field is making great progress, with large-scale clinical trials finally underway, and significant patient groups appearing for the first time, the trauma history has become perhaps *the* critical issue in the field. Some, particularly neurologists, see evidence of it in few enough cases to make it seem, at most, a risk factor, with patients damned by suspicion of a trauma concealed; while others, particularly therapists, see it as key to understanding the whole disorder, with ignoring it as political expediency at best. In the following, we shall explore why getting the evidence on this issue is so difficult, how those difficulties might be overcome, and why it is important that we do.

The untimely death of Sigmund Freud

With the advent of DSM-5 it became clear that Freud was finally dead. When DSM-III (APA, 1980) moved to aetiological neutrality, Freud was systematically purged from its analytically-inspired pages. The one disorder where his influence explicitly remained was Conversion Disorder (CD), where the name still evoked his doctrine of conversion, and the diagnostic criterion of understanding the disorder in terms of preceding trauma was retained – until DSM-5, over 30 years later.

So why is his death untimely? Because without this 'Freudian' criterion, the DSM-5 criteria appear to be for an as-yet unexplained neurological disorder, not a psychiatric one (Kanaan, 2016). There will be some who think that is as it should be. Those who have appealed for a reframing of conversion disorder have pointed to patient preference for a less stigmatising classification, as well as to the limited evidence for a psychological cause (Stone and Edwards, 2011; Stone *et al.*, 2011; Edwards *et al.*, 2013) –indeed, much of the progress in the field can be attributed to the move away from a rigid orthodoxy that alienated many patients and scientists alike. Hysteria, as CD was formerly known, has only been thought psychiatric for a fraction of its long history (Veith, 1993): prior to that it was thought to be neurological, and when the problems with that view became apparent, the alternative for most neurologists seems to have been to consider it malingering (Kanaan and Wessely, 2010). The idea that it should be a psychiatric disorder, for which Freud must take most of the credit (Bogousslavsky, 2011), is a relatively novel, perhaps passing idea, and one that requires justification. The failure to find a neuropathological explanation clearly isn't sufficient, since that would apply to many other disorders considered neurological (Sykes, 2010). It derives some support from the high rates of co-morbid depression, anxiety and personality disorder (Nicholson *et al.*, 2011), and from the impressive placebo response, but these are also true of many neurological disorders (Rickards, 2005; Goetz *et al.*, 2008; Rai *et al.*, 2012; Schmidt *et al.*, 2013). Making it psychiatric needs Freud, or someone like him.

For the 'Freudian' criterion in DSM-IV and in ICD-10 made clear that conversion disorder was, in the broadest sense, psychogenic – that it arose in the context of life problems. Other, obviously organic, conditions can be provoked by life problems, of course (Nicholson *et al.*, 2011), but by making it a necessary condition for conversion disorder the relationship with stressors was made clearly different. With a defined aetiology, CD stands to gain the relatively protected status that post-traumatic stress disorder (PTSD) has, as originating 'outside' the patient – saving it not only for psychiatry, but from much of the stigma of implicit responsibility. Without it, CD may be just unexplained neurology: and since most neurologists insist CD can't have a neuropathological basis (Kanaan *et al.*, 2011) then it risks being labelled mere feigning again – which would undoubtedly be a disaster, making it even *more* stigmatised. Does it have to be Freud? No, but it has to be someone who can give an account – a model – of the psychogenesis. For as we shall discuss later, it may be far from obvious

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what the stressors or other aetiological factors are in conversion disorder – they may be very unlike the traumas relevant to other disorders, and they may operate in a very different way.

A post-traumatic stress disorder?

The two major psychogenic models of conversion disorder in use today – dissociation and conversion (Gottlieb, 2003) – were developed in the 19th century by Pierre Janet and Sigmund Freud, respectively. Though they disagreed on the mechanism, they agreed on the aetiological process: an event would convey an idea that would operate on the predisposed patient to produce symptoms. According to Janet, the suggestion of their illness would become a 'fixed' idea, dissociated from the rest of their consciousness: 'the hysterical phenomena… are the result of the very idea the patient has of his accident'(Janet, 1920); according to Freud, the idea of the event was painful, so deliberately avoided ('repressed') which would give rise to symptoms ('converted') by some somatic tendency, or symbolic association with earlier experiences (Kanaan, 2016).

For Janet, these potent events were those that could suggest the idea of illness. For Freud, they were those that were sufficient discomforting, but which the patient could not face. Though there were many differences between them, on this they can be seen to agree: that conversion disorder is caused (Freud, 1953) by an experience, albeit one which may not appear objectively traumatic. If not a post traumatic-stress disorder, it is at least a post-stressor disorder, captured reasonably well by DSM-IV's criterion B: 'the initiation or exacerbation of the symptom … is preceded by conflicts or other stressors'(APA, 2000).

The difficulties with a psychogenic criterion

The two big problems with using this as a psychogenic criterion as far as DSM-III was concerned were that these stressors were unobservable and aetiological. Though the same was true of PTSD, whose inclusion was resisted on the same grounds (Young, 1995), the 'post-stressor' criterion would seem to create additional problems of validity and reliability.

Sometimes the presence of a causal relationship is clear. In a case we published (Kanaan *et al.*, 2007), for example, the patient developed her symptoms at the very moment her partner announced he was leaving her – not days, or even minutes, afterwards. Given that temporal proximity, a relationship of some kind between event and symptoms seems indisputable, even if the nature of the causal relationship or mechanism is wholly unspecified. Such cases are not typical, of course, but reveal that sometimes the criterion holds. The next question is then, more generally, *when* does it hold and *how* do we know?

As DSM-IV's criterion was so broadly drawn it risks vacuity, as psychological factors of some kind (distress, for example) would commonly be associated with *any* symptom or deficit; but it appears to be saved from it by the specification that this is because the symptom has a *preceding* stressor or conflict. This does not specify nearly enough, however, as everyone has had stressors preceding every symptom or deficit of any kind (their birth, to take an example *ad absurdum*, is a stressful event which precedes every subsequent ailment). How do we define 'preceding', what counts as a 'stressor', and how do we know they are 'associated'? In short, if we are to address the criterion in a non-vacuous way, we need a psychogenic theory – a description of the objects of the theory (the stressors, the symptoms) and the relationship(s) between them. But there are three broad problems in specifying this: the identification of stressors, the evaluation of stressors, and the verification of the relationships.

These are difficult problems for a psychogenic theory of conversion disorder – much more difficult than for the canonical post-traumatic condition, PTSD. In PTSD, they are all obvious, at least in typical cases. It is obvious what the stressors are because they are major, usually public, events – disasters, battles, assaults; it is obvious that they are important because the patient tells you so, and suffers in a way that directly relates to the event – re-experiencing, avoiding, ruminating; it is obvious that they are the right stressors because the mechanism by which they might cause symptoms is also obvious – a frightening event remains frightening.

The contrast with CD is stark. In CD, the range of possible stressors is vast, almost unbounded – Freud's cases included rejections, frustrations, disappointments – how could these be systematically enquired about and identified? The evaluation of these potential stressors is equally problematic because the patient is, by hypothesis, *not* complaining about them, but is downplaying them, repressing them, dissociating from them – how can an event be judged disappointing if the patient doesn't say so? The verification of these stressors is most difficult of all. Even if a psychiatrist were confident enough to judge that a disappointment, for example, must have been important, despite what the patient says, how can they know they're right, and that it wasn't any of the other myriad rejections, frustrations and disappointments of that week – or all, or none of them – that was responsible for their becoming ill? There is currently no plausible, verifiable mechanism connecting a particular event and a sudden paralysis of the left side of the body, for example – at least not one that makes sense to most people.

Given these difficulties, it is not surprising that demonstrating psychogenesis has been challenging. Those who think the trauma story overblown can point to a recent study (Kranick *et al.*, 2011), which found that only 13% of conversion disorder patients reported a traumatic event before symptom onset using the Distressing Event Questionnaire. But this study typifies the difficulties in identifying appropriate events, for it maximised reliability at the expense of validity. It used a questionnaire designed to identify the kind of life-threatening events that lead to PTSD (the questionnaire they used has recently been renamed *the PTSD screening and diagnostic scale*), and which no one thinks are the relevant events to most conversion disorder. Which is not to say that such events can't cause conversion disorder – they manifestly did so in the 'shell-shock' of the First World War – only that they are not the typical peace-time causes postulated by Freud, or anyone else. Even our prototypical case (Kanaan *et al.*, 2007) did not suffer from a trauma that would qualify as 'life-threatening'. By contrast, studies which rely on the medical interview to identify events typically find them in the majority of cases, and twice as often as questionnaires do (Ludwig *et al.*, 2018), but interviews are inherently harder to quantify: one may take comfort in the validity of an experienced practitioner's careful assessment of what is relevant to the patient becoming ill, but little comment can be made on its reliability.

Saving conversion disorder

This tension between reproducible questionnaires, which cannot hope to capture such diversity of events, and meaningful interviews, which cannot evade their subjectivity, is longstanding. A

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determined effort to resolve it in the 1970s led to the Life Events and Difficulties Schedule (Brown and Harris, 1978). It used an exhaustive semi-structured interview to reduce recall bias and identify a broad range of events, but, critically, the evaluation of those events was not made by the interviewer, who might be influenced by the subject or their reaction to them, but by a blinded panel, who made an assessment of how a typical person would evaluate that event *if they were in the same circumstances* as the subject. This approach has been applied to many conditions with success, including functional voice disorders (House and Andrews, 1988; Baker *et al*., 2013), and it was the one we adopted in our study of motor conversion (Nicholson *et al*., 2016). It goes some way to overcoming the difficulties in identifying and evaluating events – in our study, plausible preceding events were identified in over 90% of patients (Nicholson *et al*., 2016), albeit in a relatively chronic, psychiatric sample.

But what of verification, showing that these really are causative? Sometimes this may be obvious if the recollection of events systematically provokes the symptoms (Ejarah Dar and Kanaan, 2017), but that is unusual. Freud was convinced by the recovery of patients after their memories were explored using his cathartic 'exposure therapy', but that has not been systematically studied – except in cases where a preceding trauma was clear (those with co-morbid PTSD), where it appears to be successful (Myers *et al*., 2017). In general, a psychogenic theory is required to specify relevant events from the incidental events which will inevitably occur more frequently in those with significant psychiatric co-morbidity. We employed the theoretical construct of 'Escape' – the quality some events have that allows their consequences to be ameliorated by becoming ill (hence, illness allows 'escape' from the stressor (Raskin *et al*., 1966)) – though other theoretical approaches are equally possible (House and Andrews, 1988; Baker *et al*., 2013). This allowed us to show that events with this quality were not only much more common preceding the onset of conversion symptoms than in healthy controls, but also, more impressively, than preceding depression (an odds ratio of 7:1 in the preceding month (Nicholson *et al*., 2016)), where adverse events in general are frequent. Moreover, the events were not spread evenly throughout the preceding epoch, but clustered in the hours and days before symptom onset. These don't prove that the events are causally important, but they certainly help.

It goes some way towards demonstrating a causal relationship between life stressors and conversion disorder. Of the Bradford-Hill criteria for causation, we can argue it shows an appropriate *Temporal Relationship* (the events were all before the onset of symptoms), *Strength of Association* and *Specificity*. But the remaining criteria would be even more challenging. Most tendentious would be showing *Biological Plausibility* – though fMRI studies of the recall of these events (Aybek *et al*., 2014) may go some way. Above all, however, what would be needed is replication, to meet the *Consistency* criterion. Inevitably, different approaches will yield different results, but the difference between these results may be substantial (Ludwig *et al*., 2018), and even meta-analysis is ultimately little more than the averaging of the good and the bad. What is needed is more of the good: high-quality studies that can make a claim to both validity and reliability.

Why does it matter?

Without them, the debate on this issue is arid. It devolves to a contest of clinical anecdotes and political priorities. Those who find critical traumas in their patients after painstaking confidence-building will be bound to wonder whether other cases would yield the same given enough time (Kanaan *et al*., 2016); those, like most of us, whose necessarily briefer clinical assessments reveal such traumas only rarely can only guess what fraction of the true proportion they represent (Nicholson *et al*., 2016).

But what if the studies are done, and the numbers are in: what then? We can be sure the proportion will not be 0%, and it will not be 100%. How will we be any further forward if all we can hope to say is 'it's a proportion'? Well, the exact size of that proportion may yet be vital, and the aetiologies that are implicated may be critical in redefining the disorder (Kanaan *et al*., 2017), and answering the key question: is Conversion Disorder an essentially post-traumatic disorder, like PTSD? If the proportion we found, of over 90%, is convincingly replicated, that becomes plausible; if it is 60%, far less so. Realistically, Conversion Disorder is no different than any other disease attempting to move beyond its syndromic description to one based on aetiology, and the attempts with other diseases have varied: some have split into distinct diseases (think dementia); some have coalesced into a canonical disease, plus its atypical variants (think Parkinson's); others yet the disease and its mimics (think stroke). That proportion, depending on what it is, and what it does, could revitalise the conceptualisation, return trauma to its heart, and save conversion disorder – or damn it to historical obscurity.

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