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AUTHOR'S REPLY

HYPNOSIS AND MADNESS: IDENTIFYING THE ISSUES

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On first consideration, perhaps the most condemnatory and controversial comments regarding the views expressed in my article (Wagstaff, 2000) come from Bentall (2000). Bentall variously argues that the experts for both the prosecution and defence in the *McKenna* vs *Gates* case misinformed their lawyers by using the COWDUNG approach to schizophrenia, thus perpetrating an 'unintentional con' resulting in a 'muddled debate that was incapable of shedding light on an issue of importance'.

This is strong rhetoric, however. Although Bentall's views about schizophrenia may be considered interesting in their own right, I would argue that they do not bear directly on the central issues involved in this case. Indeed, none of the arguments I presented in my article assumes or requires an allegiance to a particular view of schizophrenia.

Does schizophrenia exist, and does it matter anyway?

For example, the first substantial point that Bentall makes is that there is no clear dividing line between madness and sanity. Hence, psychotic symptoms such as hallucinations and delusions exist in a sizeable minority of individuals who do not seek psychiatric help. Bentall then quotes Venables (1990) regarding the view that schizophrenia should be construed as a dimension rather than as a typology. This is an important point, but it has no relevance to the present case. Neisser puts the matter succinctly:

> The fact that a distinction is sometimes vague does not make it invalid, as long as it is clear much of the time. S.S. Stevens has often remarked that we do not deny the difference between day and night just because they are hard to distinguish at twilight (1967: 121).

For example, according to Eysenck (1970), extraversion is a dimension, and most people possess some extraverted characteristics; however, Eysenck did not assume that it is meaningless to label those people at one extreme of this dimension 'extraverts', and those at the opposite end 'introverts', or to speculate as to the causes of 'extraversion'. Consequently, the quote from Venables that schizophrenia lies on a continuum does nothing to question the validity of schizophrenia; indeed, it simply affirms Venables' view that it is meaningful to talk of a person with an aggregate of symptoms at the extreme of this dimension as 'severely schizophrenic'. In the same way, Bentall himself seems to reject the view that we can never know whether someone is mad or not, for in the second part of his commentary he accepts that we can label some individuals as 'psychotic' and 'mentally ill'.

The second substantive point that Bentall makes is that the symptoms ascribed to schizophrenia are not discrete and can be found in other diagnostic groupings;

accordingly, psychopathologists should concentrate on explanatory models for specific psychotic symptoms, such as hearing voices, paranoid beliefs or incoherent speech. He goes on to argue that as schizophrenia has no particular symptoms, no particular outcome and no particular cause, there is little to be gained by comparing the properties of schizophrenia with hypnosis (hence the 'unintentional con' perpetrated by the experts in this case).

An initial response to this might be that, if schizophrenia has no particular symptoms, it obviously makes no sense to argue, as does Venables, that it can vary in severity along a dimension with severe schizophrenia at one end and sanity at the other. However, once again, this preoccupation with the usefulness of schizophrenia as a classificatory label deflects from the main purpose of my article and the central issues in the case.

The plaintiff, according to the psychiatric reports, presented with a number of symptoms; these included paranoia, auditory hallucinations, thought disorder, lack of insight and lack of volition. Whether these symptoms are assumed to represent a collective syndrome we can label 'schizophrenia', or are more vague and disparate, does not detract from the facts that his symptoms were sufficiently severe for him to warrant treatment on the grounds of psychiatric illness, and that arguably most authorities, including Bentall it seems, would describe at least some of these symptoms as 'psychotic'.

However, this leads to an obvious problem with Bentall's argument. According to Bentall, notwithstanding the fact that there is no clear dividing line between sanity and madness, and that there is no common cause for the symptoms alleged to constitute schizophrenia, we *can* label some individuals as suffering from 'severe mental illness' and categorize certain symptoms as 'psychotic'; moreover, we can even make overall assertions about their causes. In particular, Bentall tells us that psychotic episodes are associated with life event trauma, including childhood sexual abuse and experience of warfare. But most of the behaviours associated with schizophrenia are generally labelled 'psychotic'; if the behaviours associated with schizophrenia are so disparate that we cannot isolate any common causal element, it is difficult to see how we can then proffer a general causal element, traumatic life events, for the even more general category of 'psychosis'. Not surprisingly, the standard arguments against the proposition that life event trauma is the main determinant of psychotic episodes are exactly the same as those put forward against such trauma as the main determinant of schizophrenia. Simply, large sections of the general population seem able to endure the most extreme trauma, including surviving concentration camps, murder of loved ones, warfare, and childhood sexual and physical abuse, without experiencing severe schizophrenic or psychotic episodes. Moreover, the fact that the vast majority of patients displaying psychotic behaviours have experience of traumatic episodes in the past does not rule out the possibility that psychotics, by nature of some biological predisposition, are vulnerable to trauma, invite trauma, or live in traumatic environments created by families who share the same predispositions.

It should be emphasized, however, that a dichotomy between the social and the 'biological' might be an oversimplification. Social events, in as much as they are processed by the brain, affect brain activity. Thus there is no reason, for example, why an acceptance of studies showing that certain psychotic symptoms may be associated with frontal lobe function, should, by itself, be taken as a commitment to 'neoKraepelinian biology'. Regardless of whether we accept schizophrenia as a unitary entity, it is undeniable that associations *have* been reported between psychotic

symptoms and brain activity, no matter how weak and inconsistent the data may be (see, for example, Banich, 1997). There is, therefore, a case to answer; which is why I addressed this point in my article. In this regard, I would consider Heap's point (Heap, 2000) more pertinent – that is, if the prosecution's argument was that Gates had some induced abnormality in brain function related to hypnosis, its case would have carried considerably more weight if it had been able to demonstrate this.

But even if it were the case that psychotic or schizophrenic symptoms are solely the result of traumatic life events, have no recognizable physiological correlates, and that the stage hypnosis act could, in principle, have triggered Gates' symptoms, this still misses the point. There was no denial by any party in this case that social and environmental factors can play a key role in the manifestation of psychotic symptoms; indeed, this was a critical feature of the prosecution's case. As Heap points out in his commentary, in the original pleadings the trauma of participating in the stage show was offered as the main explanation for Gates' illness. But the charge was one of *negligence*: this requires the prosecution to establish that the defendant should have predicted such a connection. Hence, Mr Justice Toulson's final decision was based not on the proposition that it was *impossible* that the defendant's stage act could have triggered the plaintiff's symptoms, only that it was unlikely, and thus not reasonably predictable. I quote:

> I conclude that it is highly improbable that the onset of the Plaintiff's schizophrenia had anything to do with his participation in the hypnotism show in which he took part. The only conceivable way in which there could be a causative link would be by classifying the experience as a life event and postulating that the stress which it generated was responsible for triggering the disease. Research in this area is incomplete, but the absence of any previously recorded case makes it unlikely (Toulson, 1998: 34).

The judge then goes on to say:

> Even if there were a connection, the strong possibility must be that the Plaintiff was on the point of manifesting the illness in any event and that the most that the incident could have done was to affect its timing (1998: 34).

Toulson's main argument in the latter respect is that, given that 'an event involving not a particularly high level of stress' was involved, then if it had triggered the onset of the plaintiff's symptoms, then:

> commonsense and reason would suggest that his condition must have reached the stage at which it could easily have been triggered and therefore would either have manifested itself without any triggering event or would have been triggered by any other life event involving comparable stress (1998: 35).

In other words, although it is possible that McKenna's stage show could have triggered Gates' symptoms, this is unlikely given that there are no other similar recorded cases, despite the thousands of people participating in stage hypnosis shows every year; and even if the show did trigger the symptoms, they could just as easily have been triggered by any other comparably stressful life event, and the defendant could not possibly have predicted this. To reiterate, and as Heap also points out, to demonstrate negligence it is necessary to establish that the defendant could reasonably have foreseen and forestalled the harm that allegedly resulted, not simply that a connection between the defendant's actions and the harm to the plaintiff was possible in principle.

Given these considerations, I cannot really see how adopting Bentall's position on schizophrenia would have clarified the debate. I doubt whether relabelling the plaintiff's symptoms under the more general heading of 'psychotic' would have made any difference, and the point that psychotic behaviours have been statistically associated with traumatic life events was readily acknowledged and covered in detail in the trial. If, however, Bentall has some fresh ideas as to the possible causes of the symptoms under consideration that we could usefully discuss in the light of what we know about hypnosis, it would be interesting to hear about them.

Assumptions about and expectations regarding hypnosis

The remaining commentaries speak more directly to central issues in the debate. Heap provides an excellent, informed overview and discussion of some of the assumptions that reoccur in claims that individuals have been damaged by hypnosis. Certainly, all of these occurred in the pleadings relating to the present case. The ideas that the plaintiff was placed in a deep trance, was of high suggestibility, was inadequately dehypnotized, and that somehow these factors were important in producing his symptoms, were important features of the prosecution's argument. Heap provides a number of good reasons for rejecting this type of argument. However, Heap also suggests that, although the notion of a hypnotic trance is unnecessary to account for the behaviour of participants in stage hypnosis (hence a number of performers manage to conduct their acts without any attempt to induce a 'state of trance' in their participants), the fact that a stage situation is defined as 'hypnotic' may have important consequences for the attributions that people may make for their (and others') behaviour, particularly if the participant is already suffering from a psychological disorder. This raises an important issue.

Even if we allow that the procedures that we usually accept as defining a context as hypnosis do not place subjects in some kind of vulnerable brain state; indeed, even if we abandon the whole traditional notion of a unique 'hypnotic state', it is possible, nevertheless, to construct an argument that hypnotic procedures *could* pose unique problems for certain vulnerable individuals because of the unique expectancies that surround hypnosis (for example, loss of volition). Ultimately, this is an empirical issue; and here the commentary by Lynn, Myer and Mackillop (2000) comes in.

In their incisive and scholarly commentary, Lynn et al. describe a number of important empirical studies that go beyond the early work of those such as Coe and Ryken (1979). Briefly, Lynn et al. found that, compared with other activities, including a class examination, a 'body sensation' experiment, and sitting quietly, routine hypnosis procedures produce no more negative effects. Of particular interest in Lynn et al.'s commentary are the findings regarding the effects of recounting early memories. Because of the possible trauma relating to 'abreaction' effects, the Home Office guidelines argue that age regression should not be part of the stage hypnotist's repertoire. However, this recommendation, no matter how sensible it might seem, is based on supposition and anecdotes rather than empirical studies. The studies by Sivec and Lynn reported in Lynn et al.'s commentary are, to the best of my knowledge, the first to look specifically at the effects of recalling events from childhood in a non-clinical context. Their findings do not support the view that hypnotic age regression is dangerous; indeed, in the case of simple recall of childhood memories, the results indicate the opposite; there was an increase in pleasant experiences and a decrease in unpleasant experiences, perhaps because of a cathartic effect.

This is not to deny, of course, that abreactions can and do occur occasionally during hypnotic age regression in therapeutic situations, but the evidence (as far as it goes) gives us no grounds to assume that hypnotic age regression is any more likely to produce abreactions than a variety of other situations that might remind people of their pasts. Indeed, hypnotic age regression is a routine suggestion in the Stanford Hypnotic Susceptibility Scale: Form C (SHSS:C), but 'abreactions' are not cited as a common complication of this measure.

It seems, therefore, that, regardless of the expectations that subjects may have about hypnosis in general, there is nothing in the findings of controlled empirical studies to support the view that expectancies about hypnosis are likely to have more negative effects than a variety of other fairly neutral contexts. Furthermore, Lynn et al. report that negative post-hypnotic reactions are unrelated to suggestibility; but they do correlate with negative experiences reported before hypnotic induction. Taken together, these results suggest that, when increases in negative responses occur after hypnosis procedures, they occur primarily as a result of more general variables such as 'participation in an experiment', or 'evaluation apprehension', rather than anything to do with receptiveness to the induction procedure or responsiveness to the other suggestions given.

However, as Lynn et al. point out, there is no available systematic research that has specifically looked at the long-term and short-term effects of stage hypnosis. What little data there are, however, do not suggest any particular dangers associated with stage hypnosis. For example, Echterling and Emmerling (1987) conducted detailed interviews with 18 participants in a stage hypnosis show, all of whom had, in their words, 'been in a trance' at the time. On the basis of their research, Echterling and Emmerling estimate that 'about half of the individuals who have a trance experience during a stage hypnosis program will generally enjoy it and will have some positive aftereffects ... About one-fifth of its trance subjects will have both a negative experience during the trance and some negative aftereffects' (1987: 152). If at all accurate, however, these results compare favourably with the 31% of subjects who reported negative effects following an administration of the SHSS:C (Hilgard, 1974).

Of course, it could be that the severity of symptoms is greater following stage hypnosis, but, as I noted in my article, even then the problem would be one of determining how much the symptoms resulted from the fact that the subjects were performing on a stage, rather than participating in hypnosis (on a stage). As Lynn et al. also note, it does not even follow that the attributions that people may make about their symptoms actually relate to the causes of their symptoms. For example, as I also point out in my article, some subjects report 'a feeling of lack of control' as a negative effect of stage hypnosis (and sometimes routine hypnosis). However, this does not necessarily mean that hypnosis rituals per se, because of the nature of the phenomena they produce, or the expectancies they generate, especially give rise to negative experiences of 'lack of control'. If they did, one might expect such experiences to relate to suggestibility. The experience of 'lack of control' could arise in any situation in which one feels socially obliged to conform to the dictates of another, or the expectations of an audience.

For instance, in a study of the impact of stage fright on student actors, Steptoe, Malik, Pay and Pearson (1995) identified two factors associated with stage fright in actors: concerns about physical collapse, and thoughts centred on *panic and loss of control*. Hence, although, because of expectations regarding hypnosis, certain experiences may be attributed to hypnosis, it does not follow that expectancies regarding

hypnosis were actually responsible for generating the effects. I have personally witnessed a student collapsing during a class presentation, and amateur actors being physically sick while offstage during plays. Had these behaviours occurred during performances of stage hypnosis the temptation might have been to attribute the effects to some feature of 'hypnosis'.

This brings us to another point, this time raised in the commentary by Vingoe (2000). As well as providing a concise and lucid account of the main issues in this debate, Vingoe stresses the importance of looking for alternative 'suspects' when negative effects occur after hypnosis. Because of beliefs surrounding the special properties of hypnosis, when problems occur following hypnosis it is tempting to follow *post hoc ergo propter hoc* theorizing – that is, because A follows B, A must have caused B. Certainly, in many cases I have come across, hypnosis has indeed turned out to be the 'usual suspect', as Vingoe puts it. However, as Vingoe also emphasizes, in many instances it is important to recognize that even if hypnosis is excluded as the key causal variable, this should not stop us from examining alternative causes. In cases of sexual assault using hypnosis, for example, once hypnosis as a state of automatism is ruled out as a viable explanation of the victim's compliance, all too often the conclusion drawn is that there is no case to answer. But one does not need to postulate hypnotic automatism to account for compliance with unwanted sexual advances in therapeutic situations; an unscrupulous hypnotist can use a variety of factors to induce submission, including trust, rapport, relaxation, eye closure, fear of embarrassment, ambiguity, and physical manipulation. However, any therapist or doctor can use similar ruses, and manifestly some have used them. With regard to the present case, Vingoe's point is pertinent. Even if we accept that the fact that the defendant's act was construed as 'hypnosis' was not responsible for the plaintiff's symptoms in any reasonably predictable way, this should not stop us from examining other features of the act. And, indeed, in the case it was important for the defence to establish that there was nothing especially lurid, embarrassing or potentially physically or psychologically damaging about what the participants were asked to do.

Returning to the more specific issue of whether hypnosis can cause madness, Vingoe also points out that there is a distinction to be made between the idea that a hypnotist may intentionally cause madness and the idea that there is something particular about hypnosis that may unintentionally result in madness. Vingoe concludes that hypnosis is only one of many ways that one might induce madness in someone who was psychologically disturbed. But this perhaps brings us back to Heap's 'question to ponder' (Heap, 2000): that is, is it advisable for a person with a psychotic predisposition to take part in stage hypnosis?

The problem of forestalling madness

As Lynn et al. comment, we lack data from controlled studies on this issue. However, although one's 'gut feeling' might be that it is not advisable for a person with a psychotic predisposition to participate in stage hypnosis (because hypnosis might feed delusions and so on), one of the great difficulties involved in assessing the impact of stage hypnosis on psychiatric illness is that we lack base rate information regarding the incidence of psychiatric illness following other single life events. For example, Zax and Cowen (1976) report the case of a 58-year-old widow who developed psychotic symptoms following minor gynaecological surgery (a stressful event). Presumably, most authorities would argue that, given that most people who undergo

minor surgery do not become psychotic, then anyone who develops psychotic illness after minor surgery must have had some kind of predisposition. But, assuming that their physical problem is not life-threatening, does this mean that we should discourage patients with psychotic symptoms from undergoing surgery? Take another case described by Zax and Cowen:

> Irene L., a 24 year old graduate student of psychology was voluntarily admitted for the first time to a private mental hospital in the southwest on the referral of the psychiatrist at the university where she was studying.... The onset of her disorder occurred three weeks before her hospitalization after a very frank discussion between Irene and a group of fellow graduates following a lecture concerning Freud's theories of psychosexual development ... shortly thereafter she became uneasy and avoided as many of these fellow students as she could. About two weeks before her hospitalization she came home for a weekend, during which she was extremely sensitive and overemotional. When it was time to return to school she was profusely grateful to her parents for having provided such a marvelous weekend of activities. Her anxiety persisted and two days before she entered the hospital she awoke and told her roommate that she could see both physical objects and concepts more clearly than ever before. She claimed that she was experiencing a nirvana like feeling and spent the entire day in the university chapel. That night she told her roommate that she felt as though she has experienced rebirth. When she awoke in the middle of the night and stated her intention to return to the chapel, her roommate became alarmed and phoned the dean of students.... The next day she protested that everything was perfect and that she had now been transformed into the Virgin Mary (1976: 154–5).

Subsequently, she displayed a variety of symptoms including autistic and disorganized speech, and inappropriate affect (laughing and crying for no apparent reason). She was diagnosed as suffering from 'acute schizophrenia' and discharged from hospital after several weeks. Again a direct link between the onset of her symptoms and the lecture and discussion about Freud's views on sex is not entirely implausible – a person with sexual problems might find these distressing, and the stress could trigger symptoms in someone predisposed; but does this mean we should discourage those with a psychotic predisposition from attending lectures and discussions on Freud (or any other sex-related topic)? The question is not necessarily rhetorical, but it does perhaps generate an appropriate context for the consideration of similar questions regarding hypnosis.

At the moment, there is only one case in the literature in which stage hypnosis has allegedly been associated with the diagnosis of schizophrenia, and that is the *McKenna* vs *Gates*. Given this, one might suggest that you are no more likely to suffer from the symptoms associated with schizophrenia after stage hypnosis than after a lecture and discussion on Freud. However, this perhaps leaves us with another point to ponder. Supposing Irene L. had experienced the transformation into the Virgin Mary following a performance of stage hypnosis; would we be as willing to reject the idea that there was a direct and foreseeable connection between the events?

A final thought

As Heap and Lynn et al. comment, there are a number of good reasons why those involved in the serious use of hypnosis might object to the use of hypnosis for entertainment purposes. It is also undeniable that some participants in stage hypnosis do experience negative, and sometimes distressing, effects, and that stage hypnosis is not

a context conducive to the prevention of such problems. However, the idea that there is 'something' about hypnosis that, when used on stage, especially predisposes individuals to develop serious psychiatric symptoms, is a serious charge that may have far wider implications for the hypnosis community. If hypnosis is really as potentially dangerous as this, we may find that it is not only stage hypnotists who end up with their activities curtailed.

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