Correspondence

EDITED BY STANLEY ZAMMIT

Contents

■ Explanatory models in psychiatry ■ Ethnic differences in prisoners: describing trauma and stress ■ Specialist care for prisoners? ■ Consent and treatment in prisons ■ Amisulpride-induced mania in a patient with schizophrenia ■ Changing use of ECT ■ Inappropriate use of psychostimulants ■ Stigma as a cause of suicide ■ Social capital and mental health v. objective measures of health in

Explanatory models in psychiatry

Dein (2002) comments on our editorial on explanatory models (Bhui & Bhugra, 2002), but fails to apprehend the conceptual flaws in his assertions, promotes a complacent attitude to the challenges of cultural psychiatry, and is threatened by a patient’s explanatory model that differs from his own. Dein agrees with us that explanatory models are not stable, and are dynamic, complex, shifting entities, making more research necessary for any consistent theory about their role in routine clinical practice. None the less, their role in improving understanding of patients’ cultural world views has not previously been in dispute (American Psychiatric Association, 2002). Although Dein gives greater weight to behavioural expressions of explanatory models, he does not question whether explanatory models can or should be considered as a psychological construct of the individual, or as a group or social–behavioural phenomenon, or both. Each of these conceptualisations is certainly distorted by theorising more concrete, but more easily understood, expressions of explanatory models. Contrary to the historical anthropological paradigm, it is not useful to psychiatric practice if valuable anthropological critiques simply ignore psychological and non-behavioural data. More worryingly, Dein assigns a patriarchal role to the psychiatrist, a role that cannot lead to a collaborative therapeutic relationship. It seems Dr Dein is not prepared to accept that a patient may pursue his or her own explanatory model and associated interventions, alongside those recommended by the psychiatrist. A fuller discussion of these alongside the psychiatrist’s own models allows for a shared vision of treatment and recovery.

Why is an exorcism problematic for the psychiatrist? It is not in the realms of psychiatric knowledge or skills, and if helpful for recovery from illness, rather than disease, it should not be hindered. Dein appears to show contempt for a territory in which psychiatrists are not expert (possession and exorcism: see Pereira et al., 1995), and certainly does not show the respect for cultural beliefs that is part and parcel of a scientific or anthropological study of healing, let alone clinical practice. His approach smacks of a patriarchal conviction that the diagnosis is more than a theory, and that psychiatric interventions are not to be questioned. To diagnose is to classify and to predict a course and treatment based on the vagaries of statistics and experience: it is to take what can be a serious risk (Romanucci-Ross et al., 1991).

Although he cites a single example, it is not the case that the evidence base of traditional healing approaches are researched to the levels of esoteric knowledge found in biomedicine, except for, perhaps, acupuncture and Ayurveda where there is a growing literature. People will always be keen to try anything that helps them, biomedicine or culturally sanctioned traditional therapies. Surely he does not mean that we as psychiatrists have nothing to learn about treating illness from the traditional and complementary sector. Our view is we have plenty to learn and research. Eliciting explanatory models is a beginning of the process in consultations and offers an easily understandable method of learning about a patient’s culture. However, Dein’s view appears to be that we know enough, and need not discover more. We are surprised at this view and cannot agree.


K. Bhui, D. Bhugra Department of Psychiatry, Barts and the London, Medical Sciences Building, Mile End Road, London E1 4NS, UK

Ethnic differences in prisoners: describing trauma and stress

I read with interest the two articles by Coid et al. (2002a,b) but was puzzled by the use of the term ‘post-traumatic stress’ to describe the psychiatric response of prisoners who had experienced adverse or negative life events. The authors use the term post-traumatic stress without specifying whether they are referring to the specific diagnosis of post-traumatic stress disorder (PTSD), a recognised psychiatric condition in the DMS–IV (American Psychiatric Association, 1994) or simply a vague amalgam of neurotic symptoms which the authors infer are a consequence of the various stresses the prisoners experienced in their lifetimes. The confused terminology in this respect, which is present in both papers, is unhelpful in assessing what precisely is psychiatrically wrong with these prisoners. A Criterion A trauma, that is a trauma that may precipitate PTSD in some individuals, is specifically defined and described in the DSM–IV as an event in which the person experiences, witnesses or is confronted with an event or events that involves actual or threatened death or serious injury to the self or to others, and to which the individual responds with intense fear, helplessness or horror. The experiences that were screened for in the original study by Singleton et al. (1998) would not normally be considered to represent a Criterion A event, but merely negative or adverse life events which have no specific aetiological links with any distinct clinical diagnosis. The experiences included by the authors in their screening include bullying and marital separation, which do not constitute Criterion A events for the purpose of making a PTSD diagnosis. Similarly, many other traumatic experiences were not apparently screened for by the authors, such as rape or adult sexual assault, combat, being assaulted in the street (although violence in the home or at work are included). Thus, the selection, definition and description of these events as traumatic is misleading, while the inclusion of negative life events that are clearly more traumatic
makes the interpretation of any resulting phenomenology extremely difficult.

It is also clear from the original publication by Singleton et al that no specific assessment for PTSD was carried out, although validated and reliable instruments for this exist (e.g. the Clinical Assessment for PTSD, Blake et al (1995) or the Posttraumatic Stress Symptoms Interview (PSSI) or Posttraumatic Stress Symptoms Self-Report (PSS-SR), Foa et al (1993)). The authors did a partial screen for a few recognised PTSD symptoms, such as re-experiencing and avoidance, but there was no systematic assessment of the condition that would have allowed them to diagnose the full disorder. It should be recognised that PTSD is a major psychiatric disorder that constitutes a serious burden for the individual and for society (Kessler, 2000). A diagnosis of PTSD has implications in terms of assessing the individual’s risk and in terms of treatment recommendations. It is important that the term post-traumatic stress should not be confused or conflated with the term ‘post-traumatic stress disorder’. The description of post-traumatic stress made by Coid et al cannot be evaluated without deconstructing more precisely what this means. As there are now well-recognised instruments to assess PTSD and lifetime experience of traumatic events in a range of settings, without these being used then terms such as post-traumatic stress should be avoided.

Specialist care for prisoners?

In his recent editorial on mental health in prisons Dr Reed (2003) urges, understandably and in most cases correctly, that the quicker that patients with psychosis are transferred to specialist psychiatric care, the better.

However, there are prisoners with schizophrenia, willing to take medication, who survive reasonably comfortably in the prison milieu. Their great fear is that they will be transferred to a special psychiatric hospital; ‘nutted off’ in prison speak. They have reason to fear a transfer, for it effectively exchanges a finite sentence for an indefinite one. In the case of those serving a life sentence, it means their fate is in the hands of a mental health review tribunal rather than the Parole Board, the latter, they believe, being less cautious in recommending discharge. As an ex-member of both organisations, I would agree with them.

So, while prison is obviously bad for people with mental illness, hospital is sometimes worse.


Consent and treatment in prisons

I read the article by Earthowel et al (2003) with interest. The issue of providing treatment to prisoners, who are frequently incapable of consenting, will not be unfamiliar to psychiatrists providing mental health care in these establishments. Although the authors correctly state that there is no legislative framework for providing treatment for mental disorders in prisons, they may be slightly disingenuous. The current legislative framework that provides for the treatment of mental disorders, namely the Mental Health Act 1983, is clear that prison health-care wings are not hospitals. It follows that any treatment that is administered forcibly must be consistent with common law. Separate legislation is therefore unnecessary.

They also appear to have overlooked recent guidance on this matter. The Department of Health (2002) in collaboration with the Prison Service has set out, in detail, good practice guidelines for providing care to both competent and incapacitated adult prisoners. These outline circumstances in which prisoners who lack capacity can receive treatment. We have found this very helpful in developing protocols for treatment in the prisons we visit.

The development of policies and protocols will assist in establishing who, when and in which circumstances incapacitated prisoners may be treated and allow us to be more confident when making these difficult decisions.


I. Qurashi Guild Lodge, Whittingham Lane, Preston PR3 2AZ, UK

Authors’ reply: We fail to understand Dr Qurashi’s comment that ‘separate legislation is therefore unnecessary’. Our paper sets out a policy for providing treatment to people with mental disorder based on common law (Earthowel et al, 2003). We are not proposing separate legislation.

Dr Qurashi also mentions that we appear to have overlooked recent guidance from the Department of Health (2002). The Department of Health guidelines were produced in July 2002, after our paper was accepted for publication.

These guidelines provide guidance on establishing capacity but, in our opinion, they do not tackle the practical issues relating to the management of prisoners with mental disorder in any great detail, they do not deal with the ethical issues surrounding the provision of an equivalent service in prisons adequately and detailed guidance on making a concerted effort to obtain treatment under the Mental Health Act in hospital before proceeding with treatment under common law is lacking. In our view, these are serious omissions.

Declaration of interest

J.O. is a member of the Department of Health Prison Expert Group.
Amisulpride-induced mania in a patient with schizophrenia

Numerous case reports of atypical antipsychotics inducing hypomanic/manic symptoms have been published; most concern the use of risperidone and olanzapine (Aubry et al., 2000), but quetiapine (Benazzi, 2001) and ziprasidone (Lu et al., 2002) have also been implicated. A literature search using Medline and PubMed revealed no such reports associated with amisulpride. Although the manufacturer has accumulated a small number of reports of manic symptoms developing during amisulpride treatment, a recent internal review concluded that no causality could be established (Sanofi-Synthelabo, personal communication, 2002). I report a case of amisulpride-induced mania.

A 17-year-old female with a 4-year history of schizophrenia was commenced on amisulpride for persistent negative symptoms. It was cross-titrated with olanzapine, over a 4-week period, to 400 mg. She continued taking citalopram 20 mg, which had been started 6 months previously on the basis that her negative symptoms could be secondary to a masked depression. On commencement of amisulpride her negative symptoms, as rated on the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1982), rapidly and linearly improved. Her mood, however, continued to rise and by 3 months she had developed a manic episode without psychotic features. She exhibited insomnia, hyperactivity, distractibility, disinhibition and an abnormally and persistently elevated mood that continued despite the immediate cessation of citalopram. There was no evidence of substance misuse or akathisia. These features improved after halving the amisulpride to 200 mg and reintroducing olanzapine 15 mg. They fully remitted within days of stopping the amisulpride.

No other concomitant medication was used. The delay in development of overt manic symptoms may reflect having to overcome a baseline SANS score of 68.

The mechanism of action of mood changes induced by atypical antipsychotics is unknown, with speculation centering exclusively on a 5-HT2A and D2 economy. Lane et al. (1998) argue that a higher ratio will increase frontal dopamine release, whereas others point to the combined blockade enhancing the ability of 5-HT1A to release frontal dopamine (Ichikawa et al., 2001). These theories do not explain the manicogenic effects of amisulpride, which has no serotonin affinity. I propose that the ability of low doses of amisulpride to differentially block presynaptic D2 and D3 autoreceptors enhances dopamine transmission in the frontal cortex and can lead to the development of manic symptoms in susceptible subjects. Presumably this mechanism contributes to its antidepressant efficacy, for which it is used in many countries. The theory implies induction of manic features at low doses only.

Declaration of interest

B.P.M. works for ORYGEN, which has received an unrestricted educational grant from Sanofi-Synthelabo.


B. P. Murphy Early Psychosis Prevention and Intervention Centre, ORYGEN Youth Health, Locked Bay 10 (35 Poplar Road), Parkville, Victoria, Australia 3052.

Changing use of ECT

I would like to point out a couple of facts about the decline in electroconvulsive therapy (ECT) use not mentioned by Eranti & McLoughlin (2003) in their recent editorial.

The use of ECT without consent has not declined at all since 1985. There were 3362 people given ECT without their consent under section 58 of the Mental Health Act 1983 in England and Wales in the 2-year period 1985–87, 4454 in 1987–89 and 4463 in 1999–2001, with little change in the years between (Mental Health Act Commission, 1988–2002).

It was the 1970s that saw the greatest decline in ECT use, from an estimated 60,000 courses in Great Britain in 1972 to 30,000 in 1979 (Pippard & Ellam, 1981).

The decline in ECT use over the past 20 years or so has been marked by regional variations. While in England ECT use fell fairly steadily during the 1980s, in Scotland it remained fairly constant during the 1980s and early 1990s and then fell by about a half in the mid-1990s (Freeman et al., 2000). In the East Anglian region ECT use actually increased during the 1980s (Pippard, 1992).

I think it is hard to reconcile these facts with the authors’ suggestion that new drugs, improvements in patient care and better appreciation of the indications for ECT are responsible for the decline in ECT; although this would be the most respectable explanation for the decline in use of a treatment which is still described as safe, effective and life-saving – especially since the textbook indications for its use have changed little over the past two or three decades. Is it really the case that fewer people need ECT nowadays – or was it given needlessly to large numbers of people in the recent past? Since no research into the reasons for the decline in the use of ECT has been done, it remains impossible to answer this question with any certainty.


S. Kemsley Address supplied; correspondence to the British Journal of Psychiatry, 17 Belgrave Square, London SW1X 8PG, UK.
Authors' reply: Sue Kemsley has raised some important issues regarding ECT. The use of ECT without consent has not declined in absolute numbers since 1985 but, as discussed in our editorial (Eranti & McLoughlin, 2003), the total number of patients receiving ECT has substantially fallen during this period. Little research has been directed at understanding this change in the pattern of ECT use. One possibility is that there exists a core group of patients with severe depressive illness and possible psychosis that requires treatment with ECT, while the decline in use predominantly occurs in people with less severe illness. So why has the use of ECT declined in this latter group?

As we have already suggested, we believe that this is due to historical changes in general psychiatry, especially pharmacology. One has to bear in mind that, following its introduction in 1938, ECT was one of the first truly effective treatments for severe debilitating psychiatric disorders and thus its use rapidly became widespread (Fink, 2001). We are currently investigating trends in ECT practice over the past 50 years in the Maudsley and Bethlem Royal Hospitals in south London. Its use peaked in 1956 when 34% of admissions were treated with ECT. This rate fell steadily thereafter to 30% in 1959, 21% in 1968 and 5% in 1987. It is interesting to note here that imipramine was introduced in 1958, coinciding with the beginning of this decline in use of ECT. Similarly, ECT use further declined after the introduction of fluoxetine, the first of the selective serotonin reuptake inhibitors, in 1988, such that by 1991 2% of admissions received ECT. Currently, less than 1% of admissions are treated with ECT and nearly 90% of these have a diagnosis of major depressive disorder, which is well-established as being the main indication for contemporary ECT (Carney et al., 2003).


S. V. Eranti, D. M. McLoughlin Institute of Psychiatry, Section of Old Age Psychiatry, Box P070, De Crespiigny Park, Denmark Hill, London SE5 8AF, UK

Inappropriate use of psychostimulants

Rey & Sawyer (2003) ask ‘Are psychostimulant drugs being used appropriately to treat child and adolescent disorders?’ – the answer is no. Like most articles on psychostimulants, they avoid discussion of the fundamental question that needs tackling for their conclusions to have any meaning – is attention-deficit hyperactivity disorder (ADHD) a valid medical disorder? The answer is no (see Timimi, 2002). This disorder is best understood as a cultural creation. Rey & Sawyer illustrate how deeply practice in this area is influenced by cultural dynamics. They show how there are large variations in the way diagnostic criteria are used both between countries (not surprisingly, they only mention Western ones) and within them. They show that there are also large variations in the way psychostimulants are used.

Children are already the losers here. There are reports of some primary schools where nearly 40% of the students were taking psychostimulants (Runnheim, 1996). Rates of diagnosis of ADHD and subsequent medication use continue to rise alarmingly in most Western countries. This is a massive, dangerous and scandalous experiment in which millions of children are being exposed to highly toxic, addictive and brain-disabling drugs whose medium- and long-term efficacy and safety have not been established (Breggin, 2002). The only winner is the profit margin of the pharmaceutical industry.

I realise this is emotive language, but then the business of what values we hold when it comes to children is too important to allow us to hide behind dry, detached, academic pretence. We live in a culture that has a deep intolerance for children. This is at the heart of why we are labelling physically healthy children with fictional medical disorders. Doctors becomes a symptom of this intolerance, not part of the solution.

This is all so unnecessary. For years I have been working with these children and their families using diverse perspectives based on a more humanitarian value system (Timimi, 2002). Not only are my clients grateful for this, they often recommend others to come and see me.


S. Timimi Child and Adolescent Mental Health Services, Ash Villa, Willoughby Road, Seaford, East Sussex BN23 8QA, UK

Stigma as a cause of suicide

We read with great interest the article by Eagles et al. (2003) in which, among the various interventions discussed to prevent suicide, it was suggested that according to patients’ opinions there should be a decrease in the stigma attached to psychiatric illness. We share that opinion and suggest that another goal of suicide prevention is the reduction of the stigma attached to suicide.

The term stigma refers to a mark that denotes a shameful quality in the individual so marked. Mental illness is widely considered to be such a quality, an assumption supported by a number of beliefs such as the association between mental illness and irrational and unpredictable violence as portrayed by the media and the notion that mental illness is not a ‘true’ illness like organic disease. And yet, people do fear mental illness and do not know how to avoid it by following the types of precautions and guidelines available for so many organic disorders.

Not only does the stigmatisation of mental illness prevent people from seeking treatment, which in turn exposes them to a greater risk of suicide, but also suicide can appear to be the best solution for a stigmatised individual. A number of environments can be traced where this process takes place. In the family, the family members’ relationship to the patient may affect the extent to which the patient’s stigma is transferred to the family members, as in the case of schizophrenia (Phelan et al., 1998). In such extreme cases, difficulties in dealing with a chronic disease, which often results in relapses, hospitalisations and social impairment, leads family members to stigmatise the patients. They behave in a way that may lead the patient to assume that suicide might be a solution for their situation. Family members may also unconsciously believe that suicide might be a solution. In the hospital, staff’s
attitudes towards patients who are at risk of suicide deserve consideration. Acceptance of a patient's suicide as a solution to problems, wishes that a patient would commit suicide as a solution to his or her problem, fear of the patient and difficulties in dealing with suicidal individuals are some of the most important sources of stigma in mental health environments. Also, following an attempt many individuals feel isolated or ignored by health professionals (McGaughy et al., 1995). In the military environment, stigma towards mental illness is very strong and military personnel tend to deny any form of mental disorder unless they are hoping to get another job. This exposes such a population to the risk of suicide.

Yet suicide is, itself, a source of stigma as anyone with suicidal ideation is considered weak, shameful, sinful and selfish, which prevents these individuals from seeking treatment early in the suicidal process. These judgements are often shared by active churchgoers (Sawyer & Sobal, 1987), teachers and parents. Also, parents and widows of victims of suicide are stigmatised, which makes recovery from this type of loss particularly difficult (Smith et al., 1995).

Destigmatisation should be addressed to mental illness as well as suicide. Increasing the stigma associated with having suicidal feelings will increase the suicide rate. Interventions among families, mental health professionals, military personnel and church activists aimed at decreasing the stigma associated with mental illness and suicide may contribute to the reduction of deaths by suicide.

**Social capital and mental health v. objective measures of health in The Netherlands**

McKenzie et al. (2002) reported that social capital in the neighbourhood may be beneficial for health and mental health in adults. We have reported associations between neighbourhood social capital and mental health service use in children (Van der Linden et al., 2003). We wished to investigate whether such effects on mental health were accompanied by similar effects on physical development, and investigated sensitive, cumulative objective measures of child health, height and weight at different ages, in relation to the neighbourhood environment.

We recorded all height and weight data registered regularly in the Municipal Youth Health Care Centre from birth up to the baseline measurement of our cohort study of 1009 children aged approximately 11 years living in the 36 neighbourhoods of a Dutch city (response rate of both child and one parent of 54%) (Drukker et al., 2003). This study on the effects of neighbourhood variables also included family-level and child-level measures, such as family socioeconomic status. In addition, social capital dimensions of (a) informal social control and (b) social cohesion and trust were measured in a community survey and aggregated to neighbourhood level.

Data were part of a three-level structure with height and weight measurements at different ages nested within children, and children nested within neighbourhoods. Growth curves were estimated using a multi-level random-effects regression model (including age and age²). The outcome measures were height, weight, and body mass index (weight/height²), and all variables except for age were considered fixed factors. When neighbourhood variables and individual level confounders were added to the models, results showed that none of the social capital measures was associated with any of the outcomes.

Therefore, we conclude that neighbourhood measures play a role in mental health, but that effects are more readily expressed in the psychological rather than the physical domain, in children living in The Netherlands.

**One hundred years ago**

**Epileptic colony, Ewell, Surrey**

On Wednesday, July 1st, the first rate-supported epileptic colony in this country, founded by the London County Council for the epileptic insane of the metropolis, was opened by H.R.H. the Duchess of Fife and the Duke of Fife, K.T., Lord Lieutenant of the County of London.

Situated on the north-eastern corner of the Horton Estate (facing the Epsom Downs), purchased in 1896 for asylum purposes, and on which the Manor Asylum (for 700 female lunatics) and the Horton Asylum (for 2,000 lunatics) have already been erected, it has a demesne of 112 acres, to be devoted to colony purposes, separated from the rest of the estate by a public road.
Corrigendum

Early intervention service for non-abusing parents of victims of child sexual abuse. Pilot study. *BJP*, 183, 66–72. Table 1 (p. 69), published norms (col. 3) for the Child Behavior Checklist should read: total score: referred=52.1, non-referred=23.1; internalising sub-scale: referred=14.6, non-referred=6.3; externalising sub-scale: referred=17.5, non-referred=8.2.
Epileptic colony, Ewell, Surrey
Henry Rollin
BJP 2003, 183:174-175.
Access the most recent version at DOI: 10.1192/bjp.183.2.174-a

References
This article cites 0 articles, 0 of which you can access for free at:
http://bjp.rcpsych.org/content/183/2/174.2#BIBL

Reprints/permissions
To obtain reprints or permission to reproduce material from this paper, please write to permissions@rcpsych.ac.uk

You can respond to this article at
/letters/submit/bjprcpsych;183/2/174-a

Downloaded from
http://bjp.rcpsych.org/ on July 8, 2017
Published by The Royal College of Psychiatrists