Correspondence

EDITED BY KHALIDA ISMAIL

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Value of early intervention in psychosis

The energy invested in debates about the benefits of early intervention sometimes generates more heat than light, especially when the issue is seen as related to the allocation of resources. The practice of medicine in Canada, while unique, has some parallels to the UK system and so the recent debate (Pelosi/Birchwood, 2003) is of considerable interest to us. Although we find ourselves in agreement with Dr Pelosi’s concerns about intervention in putative prodromal phases of psychotic illness, the potential value of prompt intervention once psychotic illness has been established seems quite defensible on the grounds of both reducing ongoing suffering and possibly improving long-term outcome. Although the evidence for prompt treatment improving the long-term outcome for psychosis is not irrefutable, there is substantial evidence that such a relation may well exist (Norman & Malla, 2001; Malla et al., 2002).

Dr Pelosi implies that first-episode psychosis programmes are elitist and excluded from mainstream psychiatry. Enthusiasm for the early intervention approach need not be to the detriment of other aspects of the mental health system. Our experience is that such programmes increase the public recognition of the need for, and influence the political will to provide, a higher standard of care for people with psychotic disorders. However, we must continue to evaluate whether early intervention with phase-specific pharmacological and psychosocial interventions reduces the overall burden of chronicity or residual symptoms in these patients.

We have become concerned that the focus on prompt intervention will deflect attention from the need for delivering appropriate interventions – timing is certainly not everything! Early intervention programmes should, in time, also be able to provide information to better identify those likely to have a ‘prolonged recovery’ (Edwards et al., 1998) or be treatment refractory. This is not the time to turn back, but to move forward and support controlled trials to assess the efficacy of early intervention.

Hopefully, the development of early intervention programmes will result in better linkages between child and adult psychiatry services and also with those involved in long-term care to ensure treatment of psychosis throughout the life cycle and not just for the first 2–3 years. Early intervention programmes are the first steps towards achieving these goals.


R. Manchanda University of Western Ontario, London Health Sciences Centre, Victoria Campus, 375 South Street, London, Ontario, Canada N6A 4G3

R. M. Norman University of Western Ontario, Prevention & Early Intervention Program for Psychoses (PEPP), London, Ontario, Canada

A. Malla McGill University, Montreal, Quebec, Canada

Author’s reply: I find myself in agreement with many of the observations of Manchanda and colleagues. There are many examples in medicine (for example surgery) where the need to advance clinical care, to keep up with new approaches and to develop research, has led to greater specialisation within a discipline. Dr Pelosi’s charge of elitism seems to me a professional one; in early psychosis, in line with the predictions of Manchanda and colleagues, I think this focus in the UK is directly responsible for the increase in public recognition of the underinvestment in these services (Rethink, 2002) and for the development of the political will for reform. The longitudinal studies have shown clearly that long-term disability and course trajectories are in place within 3 years, yet all resources are downstream (assertive community treatment, rehabilitation); thus, this new investment has been warmly welcomed by consumer groups (Rethink, 2002).

This service structure now provides an unparalleled opportunity for further research and service innovation. Important research questions now come into focus. What kind of intervention will bring the early cycle of relapse under control, and will young people find it acceptable? What strategies are effective in encouraging help-seeking to reduce duration of untreated psychosis, and what is its impact? I think it is important to emphasise that early intervention services can only provide vehicles for intervention and are not an intervention in themselves; the litmus test of a service is its ability to engage (a major problem in early psychosis) and to fix existing service problems. For example, as Dr Manchanda illustrates, the early intervention focus enables us to think creatively about how to improve continuity of care between child and adolescent mental health services and adult services and to infuse the concepts so familiar to child and adolescent services into the adult arena and vice versa (Birchwood, 2003). I agree with Dr Manchanda that continuity can work forward in time, too; however, there is a risk that early intervention, like existing services, could trap people unnecessarily in long-term services. Preparing for exit and developing community support strategies and identifying cases of ‘prolonged recovery’ are also important.

Dr Manchanda comments about Dr Pelosi’s concerns about the ethics of ‘prodromal intervention’. I too share these, but this continues to be a research issue and does not form part of the vision for early intervention services. However, the cases thrown up by the ‘ultra high risk’ or prodromal research involve people suffering from distressing psychotic experience that has not reached the ICD threshold; these people are all seeking help and the majority are already receiving care from
Secondary services. All clinicians will be familiar with such individuals, who present a therapeutic challenge where equipoise is acknowledged. One benefit of this research, therefore, is its potential to inform a non-pharmacological protocol of treatment, capitalising on the efficacy of cognitive–behavioural therapy in psychosis and emotional disorders.


The International Early Psychosis Association would like to contribute to the debate on early intervention (Pelosi/Birchwood, 2003).

First, the international network promoting reform in early psychosis is led by clinicians and academics who have a record of commitment to evidence-based medicine and leadership in scientific research. The attempt to discredit this network as mere evangelism does not bear scrutiny. However, successful reform in health care is always a blend of logic, evidence and advocacy. The latter is not only a legitimate but an essential element.

“We should be active and loud advocates of the mentally ill and be in the forefront of their battle to realise their rights. This might require that we relinquish some of our professional role and add some political activism to our daily chores – a sometimes difficult but now ever more necessary reorientation for doctors in general and psychiatrists in particular” (Sartorius, 1998).

Second, Dr Pelosi seriously underestimates the weakness of existing generic models of care for early psychosis patients and their families (Garety & Rigg, 2001). Access to and quality of initial care for psychosis onwards and do not specifically include the prodromal phase, which remains a research issue. There are genuine issues involved in sub-threshold detection of a low-incidence disorder and these remain to be solved. However, the caution required in extending intervention to potentially prodromal patients cannot be used as an argument for delaying intervention to people with clearly diagnosable first-episode psychosis.

Far from being wishful thinking, this reform process is already leading to improved short-term outcomes for young people with psychotic illness in many centres around the world (Edwards & McGorry, 2002). The reform is delicately poised in the UK and there may well be secondary effects on mainstream systems, but these should not be seen as fatal flaws, rather as problems to be solved. In the UK setting, it is to be hoped that psychiatrists will play a leadership role in this vital endeavour, which should ultimately lead to a strengthening of the specialist mental health system. In other parts of the world we are looking to you to make a success of this important task and hope your pioneering reforms will help to guide our own efforts.


P.D. McGorry On behalf of the International Advisory Board, International Early Psychosis Association (IEPA), Locked Bag 10, Parkville, Victoria, Australia 3052. E-mail: mcmgorry@ariel.ucs.unimelb.edu.au

Author’s reply: The advocacy and political activism of the International Early Psychosis Association has clearly been successful in the UK since teams for their narrow sub-specialty have been introduced despite widespread shortages of trained mental health professionals. General psychiatrists also consider themselves to be advocates for people with mental illness. They may not have the public relations skills of the International Early Psychosis Association, but they believe that clinical experience and knowledge of epidemiology and health economics should be more important in determining health policy.

The most ambitious aim of the early intervention specialists has been to identify and treat people during a pre-psychotic phase of illness. There now seems to be unanimous agreement that any such attempts to prevent the onset of, for example, schizophrenia could only lead to more harm than good. The International Early Psychosis Association should return to users, carers, policy makers and members of the public whom they have influenced (Goode, 1999) and explain the epidemiological and clinical errors behind their previous dreams of primary prevention.

There should also be unanimous agreement with your earlier correspondent that provision of care to young people who have recently developed a psychotic illness is not ‘rocket science’ (Owen, 2003). I have read and re-read accounts of the clinical methods
of the early intervention practitioners. They describe straightforward psychosocial and pharmacological therapies that should be used by all multidisciplinary teams. The only distinguishing feature is the sub-specialists’ touching faith in the effectiveness of antipsychotic medicines, which presumably arises from lack of prolonged experience with individual patients.

This inadequate experience of chronic illness is certain to lead to tragedies in the UK. The chosen remit of early intervention practitioners is to assist patients during the first 3 years of illness (Birchwood et al., 1998) – unless case-loads are high, when the ‘critical period’ can be reduced to 18 months (McGorry et al., 1999). When relapses occur, ordinary in-patient and community teams will, of course, have to pick up the pieces and I am in no doubt that they will be criticised for not being as attentive and caring as previous keyworkers.

Community mental health teams do not ‘inevitably focus on the needs of “prevalent” rather than “incident” cases’. Those who are definitely – or probably, or possibly – in the early stages of psychosis are high in their list of priorities. Unlike Manchanda et al., they do not require ‘controlled trials to assess the efficacy of early intervention’. These patients are unwell and they all require prompt and appropriate treatment. One of the most important tasks of consultant psychiatrists is to prioritise according to clinical need and it is frustrating when diversion of resources to highly protected teams makes difficult decisions even more painful. Your correspondents are shirking their responsibilities in depending on central planning to protect their case-loads (Milner, 2003; Owen, 2003). Valuable work has been done in this area (Kennedy & Griffiths, 2001) and training would be available for any sub-specialist who returns to mainstream practice.

The introduction of early intervention teams in the UK should now be halted. This will provide an opportunity for proper scientific evaluation by comparing the processes and outcomes of care in areas where these teams have and have not been established. It will also free up some financial and human resources for serious hospital and community psychiatry.


A. J. Pelosi Hairmyres Hospital, East Kilbride G75 8RG, UK. E-mail: anthony pelosii@compuserve.com

Testosterone and psychosis

Increased testosterone may be the cause of the finding of Sundquist et al (2004) that ‘A high level of urbanisation is associated with increased risk of psychosis and depression for both women and men’. Two hypotheses are required to explain this.

It is my hypothesis (Howard, 2001a) that human evolution is driven by testosterone. Based on this, I suggest the ’secular trend’, the increase in size and early puberty of children, is actually an increase in the percentage of individuals of higher testosterone. The trend may actually be a change in percentage of individuals within our populations and their ‘characteristics’ may also be increasing. This phenomenon occurs when a ‘feed and breed’ environment occurs. In these situations, individuals of higher testosterone, both men and women, will increase more rapidly than those of lower testosterone over time. Individuals of higher testosterone are more aggressive and sexual.) Urban areas are ‘feed and breed’ centres; I suggest urban centres are areas of higher testosterone.

I hypothesise that dehydroepiandrosterone (DHEA) is directly involved in growth and development, and subsequent maintenance, of all tissues, especially the brain. (The large brain of mammals may have resulted from an evolutionary increase in DHEA; Howard, 2001b.) Numerous reports of beneficial effects of DHEA on neurons and tissue-level structures of the brain exist in the literature. I have suggested in the past that depression and schizophrenia, among other mental disorders, result from low DHEA during growth and development, subsequently exposed by adverse circumstances during maintenance.

In depression and schizophrenia DHEA is low. Two other hormones may adversely affect the function of availability of DHEA: cortisol and testosterone. Over the past few years a connection with low DHEA, along with increased cortisol, has been demonstrated regarding depression. It is known that schizophrenia is often characterised as resulting from a non-causal, but significant, stressful event (cortisol) usually beginning in the late teens or early twenties (testosterone of puberty, in men and women, along with the natural decline of DHEA which begins at around age 20). In individuals of low DHEA, increased cortisol and testosterone may expose underlying, silent pathology.

Therefore, I suggest that increased rates of psychoses and depression in urban areas may be the product of increased stress and testosterone in both men and women. As suggested above, the secular trend may be due to increasing numbers of individuals of higher testosterone. This increase in these individuals of higher testosterone, along with increasing stress of urbanisation, may account for the findings of Sundquist et al, as well as reports of recent increases in these mental disorders.


J. M. Howard 1037 North Woolsey Avenue, Fayetteville, Arkansas 72701-2046, USA. E-mail: jmhoward@anthropogeny.com

Neurosurgery for mental disorder

Dr Persaud provides an ardent but ultimately flawed argument in favour of allowing neurosurgery for mental disorder (NMD) to die out (Persaud/Crossley & Freeman, 2003).

Patients who are considered for NMD are among the most severely ill and disabled who come into contact with any branch of the medical profession, and such presentations merit conceptualisation as rather more than having ‘psychological problems’.

It is also disingenuous to argue that ‘psychosurgery’ (sic) tries to locate complex psychiatric disorders in ‘one so-called “abnormal” brain region’. Such hangovers...
from Cartesian dualism fail to advance clinical neuroscience or the practice of psychiatry. Dr Persaud will, of course, be aware of the compelling evidence for changes in brain function and structure in both depression and obsessive-compulsive disorder, the main indications for NMD (Drevets, 1998; Szeszko et al., 1999).

The argument that there is a lack of randomised controlled trial (RCT) data to support NMD applies equally to a range of ‘cutting edge’ medical and surgical procedures. The proportions of medical and surgical treatments based on RCT data are 53% and 24%, respectively (Ellis et al., 1995; Howes et al., 1997). In such situations, prospective clinical audit becomes the tool of choice. If Dr Persaud demands that NMD cease because of the absence of robust RCT support, then he must surely demand the same rigour from other interventions such as heart transplantation or dynamic psychotherapy.

With respect to the issue of consent, in Scotland NMD does not take place unless the patient provides informed consent and the Mental Welfare Commission for Scotland agrees both that it is an appropriate treatment and that consent is valid. Regrettably, Dr Persaud continues to trade on the outdated image of patients receiving NMD. Indeed, he implies that chronic intractable mental illness robs patients of their capacity to provide informed consent. It is demeaning to assert that individuals are incapable of evaluating the risks and benefits of a treatment simply because they have a mental illness. Perhaps it is the failure to appreciate this perspective that leads to excessive concern for the ‘stigmatised profession of psychiatry? Believing ourselves to be persecuted perpetuates outdated views of psychiatry, and does nothing to reduce the stigma of mental illness.

**Declaration of interest**

K.M. has received payment for lectures on the management of depression from various pharmaceutical companies. K.M. and M.S.E. run the Dundee Neurosurgery for Mental Disorders Service.

**Author’s reply:** My necessarily abbreviated arguments against the continued practice of NMD are intended to be within the spirit of the debate section of the _Journal_. A debate necessarily requires two sides. Given that the title of the debate I was given included the term ‘mental disorder’ I am confused that an objection should be raised to my nod towards the well-recognised controversy over the modern phenomenological localisation of psychiatric disorder. But I am perhaps mostly perplexed by the failure to see that the use of an irreversible surgical treatment directly applied to the brain necessarily demands much higher standards of certainty over its benefits than something like dynamic psychotherapy, particularly given the political context of a profession with obvious public image difficulties. Any-one aware of the widespread coverage that our debate received in the Scottish newspapers would be immediately impressed by this public relations context, which is precisely the area the coverage focused on.

R. Persaud The Maudsley Hospital and Institute of Psychiatry Croydon Mental Health Services, 49 St James’ Road, West Croydon CR9 2RR, UK

**Stigma and somatisation**

In their exhaustive review of the impact of globalisation and culture on depression, Bhugra & Mastrogianni (2004) highlight the role of somatisation in many parts of the world, where it often accounts for ‘common presenting features of depression’ (p. 16). Emphasising both the ubiquity and cultural aspects of somatisation, they cite an earlier characterisation of common mental disorders that refers to the ‘black box of somatisation’ (Bhui, 1999). In doing so, however, they miss an important explanatory feature of this process with substantial practical and clinical significance – that is, the role of stigma. Despite increasing availability of effective treatments, many people with depression (perhaps even a majority) do not seek professional help because of the stigma associated with the illness. Efforts to clarify the impact of stigma are crucial for explaining cultural aspects of illness-related experience and meaning, and highly relevant for planning interventions that are culturally appropriate and locally effective.

As one effort towards elucidating the experience of depression, in a study in Bangalore, India, we examined the role of self-perceived stigma (Raguram et al., 1996). We found that greater severity of depression was associated with higher stigma scores, but more somatisation was associated with less stigma. Through qualitative analysis of patients’ narratives, we also demonstrated that patients viewed depressive, but not somatic, symptoms as socially disadvantageous. Somatic symptoms were considered to be less stigmatising since they resembled illness experiences that most people could expect to have from time to time. Consequently, studying the work of culture clarifies the nature of somatisation. From a Western vantage point, somatisation may appear enigmatic, but attention to stigma helps to illuminate the internal structure of the black box.

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**References**


R. Raguram National Institute of Mental Health & Neurosciences, Bangalore 560 029, India

M. Weiss Department of Public Health & Epidemiology, Swiss Tropical Institute, Socistrasse 49, Basel, Switzerland

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**Notes**

is more complex than that. In search of making sense of symptoms by the health professionals, we believe that the first step is by understanding the symptoms and the distress experienced by the individuals themselves through their identification that something has gone wrong; then their search for a possible explanation for their distress will lead to identifying possible sources of help and then finding a way to seek relief. However, in this process of help-seeking there are numerous culturally determined barriers. Stigma will indeed be a potential barrier but it is also likely that other factors may help modify the idioms of distress. In an earlier study of middle-aged Punjabi women, we found that they were able to identify symptoms of depression, and life events causing it, but they also felt that these symptoms were part of life’s ups and downs and not a medical condition; hence, they preferred to seek solace in religious places (Bhugra et al., 1997). They identified both psychic and somatic symptoms but were also clear in their discussion that sources of help were not medical. Similar observations were made in Dubai (Sulaiman et al., 2001). Our conjecture is that globalisation will influence the way individuals see their distress because media influences may affect their cognitive schema. Cognitive schema determine the meanings we impart to ongoing experience and give an expectation of the future and the present, and thus determine barriers. Stigma will indeed be a potential barrier but it is also likely that other factors may help modify the idioms of distress.

First, their multivariate analysis of variance compared the degree of disability of persons with PTSD with that of people with other mental health problems. From their results they could only conclude that PTSD caused no additional disability compared with other mental health problems. Moreover, from a statistical point of view, the sample size is not sufficiently large, especially when one tries to find differences between groups given the significance level used ($P=0.01$). In addition, the authors do not give insight in the multicollinearity between the independent variables of the multiple regression analysis; the expected high intercorrelations may have influenced the results.

Second, is it not strange to question disability in people with PTSD, major depressive disorder or alcohol dependence, while disability in social or professional functioning or in other important areas is a requirement for all DSM–IV diagnoses? Also, the authors took subjective judgement of disability as their main outcome measure and not objective measures of disability, such as the number of days not at work.

Third, previous studies found contrasting results. Brown et al (1996) and Lydiard (1991) report that major depressive disorder comorbid with anxiety disorders (i.e. PTSD) is more severe than major depressive disorder alone in terms of depressive symptoms, course of illness and treatment response. Finally, even if PTSD does not cause additional disability above major depression, the diagnosis is still relevant for the correct choice of treatment.

In defence of complainants

It is interesting that the complaints involved within the study by Lester et al (2004) were not subject to independent legal scrutiny. The reader therefore has no idea of their merits.

Anyone who has experienced the difficulties of authorities and courts will realise that bureaucracy and confusion pervade each institution. Anyone who has attended one of our supreme courts will know that the service is slow, correspondence often goes missing, checks are required to ensure that the correct folders and paperwork are presented, and often uncomfortable questions are ignored. These are characteristics


**D. Bhugra** Section of Cultural Psychiatry, Institute of Psychiatry, PO25, De Crespigny Park, Denmark Hill, London SE5 8AF, UK

**Disability and post-traumatic stress**

Neal et al (2004) recently found no association between post-traumatic stress and judgement of disability. Therefore, they concluded that the clinical importance of post-traumatic stress disorder (PTSD) and its symptoms may be questionable. However, in our opinion their conclusions need additional consideration.

First, their multivariate analysis of variance compared the degree of disability of persons with PTSD with that of people with other mental health problems. From their results they could only conclude that PTSD caused no additional disability compared with other mental health problems. Moreover, from a statistical point of view, the sample size is not sufficiently large, especially when one tries to find differences between groups given the significance level used ($P=0.01$). In addition, the authors do not give insight in the multicollinearity between the independent variables of the multiple regression analysis; the expected high intercorrelations may have influenced the results.

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**J. Roorda** Regional Health Authority Twente, Enschede Firework Disaster Health Monitoring Project, Postbus I400, 7500 BK Enschede, The Netherlands

**M. L. Meewisse, G.-J. de Vries** Specialty Programme for Psychotrauma, Academic Medical Centre of the University of Amsterdam, The Netherlands

**Author’s reply:** The multivariate analysis of variance demonstrated no significant difference between the group with DSM–IV PTSD and the group without DSM–IV PTSD in terms of the severity of disability. This finding is unrelated to the other mental health problems measured in the study, as shown by the analysis of covariance. The power of the study was 0.85 (assuming a detectable difference of 3 out of 30 on the Sheehan Disability Scale and $\alpha=0.01$). This is acceptable for limiting the chances of type II error. Multicollinearity is only of importance when trying to draw inferences about the relative contribution of more than one predictor variable to the success of the model. In this study the Beck Depression Inventory (BDI) (or its variant the M–BDI) was the only variable retained in the regression models and so multicollinearity is not an issue. Disability is not an absolute requirement in DSM–IV. The utility of objective measures of disability $v$. subjective measures was discussed in the paper. However, the subjective experience of the patient is probably of most value in clinical terms. Other studies have found contrasting results, as discussed in the paper’s introduction. However, most have methodological limitations. The treatment of PTSD, as opposed to depression, may be relevant to the DSM–IV diagnostic criteria but may not be relevant to the patient.

**L. A. Neal** King’s College London, and Bristol Priory Hospital, Heath House Lane, Stapleton, Bristol BS16 1EQ, UK
of the average day of a normal and rational human being attempting to protect his or her civil liberties. These are practical problems faced by the average person.

With the advent of the Human Rights Act 1998 civil liberties have come to the forefront. It is an Act that cannot be ignored. Indeed, with increasing litigation, authorities have by nature become defensive. Part of the method of making life impossible for complainants is to increase the bureaucracy.

The number of letters, phone calls, etc. reported by Lester et al (2004) may be part of ‘normal’ human behaviour and reaction to bureaucracy. In a democratic country, we all have a right to protect our civil liberties. Often litigants lack knowledge, have no idea of procedures, and are misled by authorities who have a vested interest in protecting themselves. To label this behaviour as an ‘abnormality’ or something that requires psychiatric intervention is ludicrous. Indeed, I note the Royal College of Psychiatrists runs a very successful anti-stigma campaign to stamp out discrimination against those with mental illness. The diagnosis of querulous paranoia runs the risk of misuse by those who wish to use psychiatry as a manner of silencing criticism. The behaviour exhibited in the study is indeed a normal reaction to the circumstances faced. ‘Normal’ of course depends on many variables such as response time of the complaint officers, failure to address questions, replies to phone calls, etc. These factors have not been addressed.

It stands to reason that psychiatrists are not judges. Indeed, the merits of the complaint will be subjectively assessed by each psychiatrist based on his or her prejudices. This is hardly independent.

Querulous paranoia is a diagnosis best left within the darkened past of psychiatry – perhaps pre-war Russia where Stalin often used ‘madness’ to silence his critics. Genetically, we are all ‘different’ by nature and react in various ways to injustices. It is essential to maintain the civil right to seek a remedy without interference from psychiatry. Interference from psychiatry will only increase the stigma associated with it for so many years.

It is often the case that different personas, atypical to the perceived norm, are subjected to psychiatric analysis. There is a minority of serial complainants but the difference is to ascertain whether their complaints have merits or not. A psychiatrist cannot assess this fairly. Without an independent legal assessment, any person who attempts to fight or campaign for their civil liberties runs the risk of being labelled with a psychiatric illness. Their credibility will often be substantially affected. This, indeed, may be a rather convenient way of silencing uncomfortable critics of negligent authorities. This was not what psychiatry was meant for and neither should it risk going down that route, given the good work done by the College’s anti-stigma campaign on raising awareness of discrimination in mental health.

Author’s reply: Judging from Dr Pal’s letter we failed totally to communicate adequately the purpose, the methodology or the conclusions of our paper on unusually persistent complainants. Dr Pal’s letter comes, therefore, as a welcome opportunity to clarify our views.

We scrupulously avoided the term querulous paranoia. The unusually persistent complainants and their controls were selected by professionals working within the ombudsman’s offices, many of whom are legally trained. We are studying not courts and bureaucracies, but organisations whose mission is to assist complainants find a satisfactory resolution to their grievances. The organisational responses to the complaint, far from being ignored, were examined as the most likely precipitant of unusual persistence. Dr Pal’s passionate defence of civil liberties and attack on ‘misleading’ bureaucracies set on ‘silencing criticism’ seems misplaced as a criticism of a paper aimed at understanding and assisting those currently damaged by engagement within systems of complaints resolutions. Dr Pal clearly has a generous view of ‘normal reactions’, which incorporates behaviours involving a total fixation on a grievance to the point where individuals consume all their time, resources and energies in a futile pursuit that lays waste their own, and their families’, lives. Dr Pal also presumably encompasses within the notion of normal overt and covert threats against complaint officers and their families.

Having our approach compared to Stalin, even a Stalin who Dr Pal seems to believe improved his behaviour post-war, might be considered intertemporal, directed as it is at the authors of a paper which attempted to broaden the sympathies and concerns of mental health professionals for a distressed and disturbed group within our communities.

R Mullen Victorian Institute of Forensic Mental Health, Thomas Embling Hospital, Yarra Bend Road, Fairfield, Victoria 3078, Australia

GHB and date rape

I read with interest the important editorial by Rodgers et al (2004) on γ-hydroxybutyrate (GHB, liquid ecstasy) and the new threat it poses to young adults. It is worth adding the growing threat of the use of GHB as a ‘date/acquaintance rape’ drug; GHB is cited in this regard along with psychoactive substances such as flunitrazepam and ketamine (Smith, 1999).

GHB is a typical ‘date rape’ agent (O’Connell et al, 2000) as it is relatively easy to obtain, and it causes a rapid relaxing and disinhibitory effect. Moreover, since it is colourless and odour-free, it is easily added to the potential victim’s drink without arousing any suspicion. These characteristics make it easy and less risky to perpetrate the crime. Additionally, GHB frequently causes the victim to be regarded as unreliable in the eyes of law-enforcement authorities because of changes in consciousness, perception, and antero-grade amnesia, and at times hallucinations during and following the act.

Since GHB is difficult to identify in the urine as it is quickly eliminated from the body, it is rarely collected as evidence of the crime. This drug is not routinely checked for in urine toxicology screening kits and is therefore likely to be missed at the emergency room. Doctors and other professionals working with sexual assault victims should be aware of the possibility of GHB intoxication, more often than not, of an unknowing victim.


Creative debate misses the point
The debate rages between Schlesinger (2004) and Wills (2004) over the evidence for a link between mental illness and creativity, but I believe that their focus is wrong.

Most studies to date have either focused on anecdotal (biographical) evidence or have been methodologically flawed retrospective cohort studies, and all would rate low on the hierarchy of evidence. Whatever the outcome of Schlesinger’s and Wills’ arguments, the question will remain unanswered until better controlled, masked, prospective and replicable randomised studies are carried out.

What is not in question is that mental illness is at least as prevalent in the creative community as in the general population and there are even examples of how some artists, including Dali and Munch, have used their mental illness to feed into the creative process (Saloman, 1996; Rothenberg, 2001). Given the hefty side-effect profiles of most psychiatric treatments, surely the emphasis should be on how best to treat such exceptional patients – indeed all patients – in a way that minimises their symptoms without rendering them incapable of practising their trade. That is, after all (at the risk of sounding naive), what we are here for.


Comedians: fun and dysfunctionality
The astonishing levels of drug- and alcohol-related morbidity in the history of jazz and popular music is well described by Wills (2003). After reading his paper I reflected on another group of my heroes, comedians, about whom popular biographies also abound. As I thought of a list of comedy greats, the well-published problems of many – indeed, almost all – of them was striking. Here follows an unresearched short list of some of my favourite great comedians, who manifest a range of neuroses, affective disorders, psychoses and substance problems: Caroline Aherne, Woody Allen, Lenny Bruce, Graham Chapman, John Cleese, Peter Cook, Tommy Cooper, Tony Hancock, Spike Milligan, Dudley Moore, Richard Pryor, Victoria Wood.

The thought of a 2-minute after-dinner speech, let alone three shows per night at the Glasgow Empire, illustrates how unusual any group of comedians must be. There may be a need for somewhat hypnotic thinking to improve comedy. There is possibly some mileage in the ‘bullied at school’ manic defence explanation for becoming a clown. Such factors suggest the possible preselection of high-risk people to enter the comedy field. Once selected, the factors suggested by Plant (1981) to explain why some occupations have a high risk of drinking, and by extension drug use, all seem applicable: availability; social pressure to use; separation from normal social or sexual relationships; freedom from supervision; very high or very low income; collusion by colleagues; and strains, stresses and hazards.

The popular ‘myth’ that, beneath the motley, clowns are distressed, may account for some over-reporting of comedians’ problems, but perhaps some truisms are just that.


A. J. McBride Specialist Community Addictions Service, The Rectory Centre, Rectory Road, Oxford OX4 1LU, UK.
E-mail: andrew.mcbride@oxmhc-tr.nhs.uk
as long as they continue to take thyroid. I have observed that the simultaneous exhibition of syrupus ferri iodidi appears to assist its action. We see this class of patients described by such terms as dull, listless, apathetic, taking a long time to comprehend and to answer questions, of sluggish ideation, of sluggish mentation, demented, depressed, moping, lethargic, suspicious, of impaired memory, sleepy, torpid, contented, and irritable. I suggest that this proves on careful inquiry to be but part of the truth and that the majority of these cases are sent to an asylum in error and could be as well treated outside one.

**REFERENCE**

*Lancet*, 23 April 1904, 1117.

Researchers by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Epsom, Surrey

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**Corrigenda**

Computer-aided self-help for phobia/panic via internet at home: a pilot study. *BJP*, 184, 448–449. The authorship for this paper should read: Mark Kenwright, Isaac Marks, Lina Gega and David Mataix-Cols. The online version of this paper has been corrected accordingly.

Global burden of depressive disorders in the year 2000. *BJP*, 184, 386–392. The third sentence under ‘Comparison of GBD 1990 and GBD 2000’ (p. 390), col. 2) should read: The first was that the epidemiological data used as input for the original GBD study to calculate the burden due to depressive disorders remain debatable: episode incidence was modelled as 2927 per 100 000 per year for women, and 1676 per 100 000 per year for men. The tenth sentence (p. 390, col. 3) should read: In the GBD 2000 the incidence estimates used were higher (4930 per 100 000 per year for women and 3199 per 100 000 per year for men) and with incident cases of depressive episodes appearing at younger ages than in the GBD 1990.

Global burden of depressive disorders: the issue of duration. *BJP*, 181, 181–183. The penultimate sentence of the fourth paragraph under ‘Scientific studies of duration’ (p. 181, col. 3) should read: The GBD 1996 results were generally accepted, but calculations for depressive disorders remained debatable: episode incidence was modelled for women as 0.29% and for men 0.16%; episode average age at onset was 37.1 years, with an average episode duration of 6 months (Murray & Lopez, 1996: pp. 601–606).
In defence of complainants
R. Pal
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