Correspondence

EDITED BY MATTHEW HOTOPF

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Concerns over confidentiality

We recently received our copy of Good Psychiatric Practice 2000 (Royal College of Psychiatrists, 2000). On reading it there was little one could disagree with and much to commend it. However, there was one matter regarding confidentiality which raised our concern: “the psychiatrist will . . . respect the confidentiality of sensitive third-party information and only divulge such information either to the patient or others with the consent of that party” (p. 19).

At face value this sounds reasonable, but it is questionable whether this advice is always justifiable and legal. This is particularly the case when third-party information involves an accusation about the patient or his or her behaviour.

We have recently been involved in a case where sensitive third-party information was given about a patient who was detained under section 37/41 of the Mental Health Act 1983. The information was thought to be believable and related to prior actions of the patient unrelated to factors involved in their current hospitalisation. If the information were believed, then this would profoundly affect issues around risk management and thus the likely future care of the patient. The informant refused to inform the police of the allegation despite encouragement and refused to give us permission to disclose it to the patient. This placed us in a difficult position. It was unclear how we could take note of the informant’s opinion if it was not fully investigated. There was also the question about the right of a patient to be aware of a factual matter which was taken into consideration when decisions were made about his/her care and discharge. In view of this we took legal advice, which would appear to contradict the advice given by the Royal College of Psychiatrists (2000).

There are three points which seem worth mentioning. First, the European Court of Human Rights (Convention for the Protection of Human Rights and Fundamental Freedoms, article 6: http://conventions.coe.int/treaty/EN/Treaties/html/003.htm) states that any person who is charged with a criminal offence is entitled to a fair hearing by a tribunal, and has the right to be informed promptly of accusations against them. This may well have implications for detained patients who appeal for a mental health review tribunal where all allegations regarding their behaviour or mental state are ‘accusations against them’. Second, doctors have a clear and overriding duty to their patients. Psychiatrists have a duty to act in good faith and in the patient’s best interests. This involves informing them of any information which will affect clinical decisions and is likely to include any information discussed with the Home Office in the case of a restricted patient. In short, our duty to the patient and the public interest outweigh any duty to the informant. Third, if an allegation involves sexual abuse, it raises our responsibilities with regard to child protection legislation and the public interest. Enacting this may lead to investigation and hence to the patient being aware that information has been given and being able to identify the informant.

We would suggest that the College reviews its recommendations over third-party information, and recommend that any advice take account of the fact that, in certain circumstances, the rights of a patient may outweigh the rights of an informant to confidentiality.


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Whose consent is it anyway?

We were interested to read the editorial by Finner (2000). In particular, the significant advocacy role we have traditionally accepted of relatives, which may conflict with the views of elderly people in general. It has become accepted good practice in clinical situations and research to seek and be influenced by the opinion of carers and relatives acting in this role.

The issue of consent among mentally incapacitated adults is a complex problem. We have been studying the views of elderly patients with mental health problems towards cardiopulmonary resuscitation (CPR). The study was confined to an acute in-patient population. Part of this enquiry required us to ask patients with a debilitating illness “If your heart was to stop now, would you want us to bring you back to life?” and “If you were suffering from an incurable illness, would your answer be the same?” Other than completing a severity rating scale and depression inventory nothing else was required of the patient.

Relatives were asked for permission to approach patients. Considerable effort was made to recruit support by lengthy discussions and written material but to no avail. Eleven consecutive relatives refused, saying, in all cases, that the question would upset the patient too much.

There is evidence that relatives’ proxy consent does not necessarily reflect the wishes of individuals and where divulging the diagnosis of dementia is concerned relatives wish this information to be withheld from the patient when they would expect to be told if they were affected (Maguire et al, 1996). This double standard also seems to affect psychiatrists (Hospital Doctor, 16 July, 1997). Denial has been reported as a means of coping by Alzheimer’s patients (Bahro et al, 1995); is it possible that carers’ decisions are influenced more by processes of denial and emotional self-protection than the needs of the patient?

Although there have been concerns that discussions about CPR with elderly patients might be distressing, the evidence indicates that elderly people are grateful for the opportunity to discuss this subject, which they consider important and upon which they wish to make their views known (Morgan & King, 1994). It is also clear that decisions and policies about CPR are usually absent or unclear and decisions are frequently left to junior staff in an emergency. Moreover,
nursing and medical records are frequently contradictory and do not necessarily coincide with patients’ expressed wishes (Aarons & Beeching, 1991; Morgan & King, 1994).

Unless we are to make blanket decisions of policy or rely heavily on proxy consent that may not represent patients’ wishes, how are these matters to be decided? At best, the present position hinders research and at worst does patients the injustice of excluding them from important decisions about their life or death.


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Evolution and psychiatry

Dr Abed (2000) states that “ancestral females, of course, were never uncertain about the genetic relatedness of their offspring”. This is a very confident statement – much more confident than most of Charles Darwin’s. Could he provide the evidence that makes him so certain?


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The editorial by Abed (2000) demands further comment. The author writes as if unaware that ‘evolutionary psychology’ is only the latest in a line of contentious theories of biological determinism that includes 19th-century ‘eugenics’ and 20th-century ‘sociobiology’ (Rose & Rose, 2000). Moreover, the version of evolution put forward by the author has been rejected as grossly oversimplistic by many modern evolutionary biologists ( Lewontin, 2000). The author appears to believe that natural selection is the sole mechanism of evolution, which has, over aeons, honed every detail of all life forms into states of exquisitely adaptation. Our minds and brains, accordingly are viewed as perfectly adapted to the hunter–gatherer way of life prevalent on the savannah of half a million years ago.

In fact, there are many elements in the living world which have evolved through processes other than natural selection and which are neutral or even negative with respect to adaptation (Sober & Wilson, 1998). The mind and brain are therefore much more complex than is suggested by theories which reduce ‘natural’ human behaviour to that of a particular imagined past.

Evolutionary psychologists reveal their lack of balance and antipathy to complexity most clearly when they ‘discuss’ the social sciences. No references are cited by Abed (2000) when he implies, disingenuously, that all social scientists believe the mind starts as a tabula rasa. And his statement, “social and cultural factors cannot be considered as separate and independent causative agents acting independently on individual minds” exemplifies both the bias and the flawed logic of his position. Social, cultural, economic and historical explanations need in no sense imply the irrelevance of intrapsychic factors. They differ from psychological explanations as a biomechanical explanation of muscle contraction differs from a biochemical one. So while some, such as Abed, find conceptual pluralism unsatisfactory, it is probably the only realistic approach to a true understanding of the complexities and unpredictabilities of human behaviour.

Finally, there is no mention of the fact that, historically, ‘biology-as-destiny’ models have been used to legitimate a range of shameful practices, including sterilisation of people with disabilities and vicious racism. Although it is an open question whether such theories inevitably lead in that direction, evolutionary psychologists should at least acknowledge their discipline’s own ‘evolutionary history’.


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Abed’s (2000) enthusiastic advocacy of evolutionary psychology contains much that is sensible but its central hypothesis that psychiatry is weak because of its conceptual pluralism is unsatisfactory. Conceptual pluralism may be a sign of weakness but it is hardly unique to psychiatry. Abed’s example of a physicist not violating the Newtonian law of gravity is particularly unfortunate. Einstein’s general theory falsified the Newtonian theory of gravity nearly 100 years ago, but physicists still use the Newtonian theory when it is useful. Indeed modern physics abounds with mutually incompatible theories, and the mutually incompatible corpuscular and wave theories of light have been jostling side by side for a couple of centuries. If physics, the fundamental science, tolerates conceptual pluralism, then the other sciences, which are based on the laws of physics, cannot be criticised too severely for also being pluralistic.

This has led some philosophers of science to suggest that it is unrealistic for science to aim at the truth; rather, the purpose of scientific hypotheses is to provide a theoretical framework to help us overcome problems that we encounter in nature – the instrumentalist view (van Fraassen 1980; Churchland & Hooker, 1985). This instrumentalist view of science is less ambitious, but given the history of science seems more practical and persuasive. We should not, therefore, be too embarrassed by the conceptual pluralism of psychiatry – we are in good company.


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<th>Author's reply</th>
<th>Psychotropic drugs and sudden death</th>
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<td>I did not mean to suggest that a mother is always certain of the pater-</td>
<td>In their editorial, Appleby et al (2000) indicated that the mechanism of sudden death</td>
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<td>nity of her offspring – merely that she is certain that her offspring per-</td>
<td>among patients taking antipsychotic medications might be ventricular arrhythmias</td>
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<td>petuate her genes, while a father can never be similarly sure that his</td>
<td>and that QTc prolongation might be a particularly important harbinger of these</td>
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<td>genes are perpetuated in the offspring of his sexual partners. Hence, it</td>
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<td>is suggested that each gender faced distinct reproductive problems that</td>
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<td>required different adaptive solutions.</td>
<td>with sudden death from ventricular fibrillation is the Brugada sign (i.e. right bundle</td>
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<td>I am indeed aware of the long-time opponents of evolutionary psychology</td>
<td>branch block and elevation of the ST segment; Brugada &amp; Brugada, 1992). Buckley</td>
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<td>that Lucas refers to. Most (e.g. Rose &amp; Rose and Lewontin) are evolutionary</td>
<td>&amp; Sanders (2000) have commented that although no specific antipsychotic has been</td>
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<td>biologists who are prepared to accept that the organs (e.g. the eye or the</td>
<td>directly associated with the Brugada sign (unlike the tricyclic antidepressants),</td>
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<td>hand) have been designed by selection but draw the line at the human psyche</td>
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<td>or mind. Their antipathy to any suggestion that the human mind may have</td>
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<td>any architecture whatsoever that could have been shaped by the evolutionary</td>
<td>In addition to the risk factors mentioned, drug–drug interaction is an important</td>
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<td>Moreover, Lucas is quite mistaken in suggesting that evolutionary psychology</td>
<td>bined with an antipsychotic medication. Inhibition of the cytochrome P450 enzymes</td>
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<td>is biologically deterministic. Biological determinism is simply wrong and you</td>
<td>involved in the metabolism of psychotropic drugs leads to increased blood levels,</td>
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Cognitive–behavioural techniques in practice

The randomised controlled trial reported by Turkington & Kingdon (2000) demonstrated therapeutic benefit from a general psychiatrist using cognitive–behavioural techniques with patients experiencing psychotic symptoms. However, the conclusion that “general psychiatrists could help their patients with schizophrenia by using cognitive–behavioural techniques” (p. 101) may not fully reflect the fact that the sole therapist in this study is an international expert in cognitive–behavioural therapy (CBT) with psychosis, who has co-written one of the seminal texts (Kingdon & Turkington, 1994).

Lack of time is highlighted as the main difficulty for psychiatrists wanting to use CBT, but other limiting factors may include the knowledge, skills and attitudes of the psychiatrist. Experience as a clinical psychologist supervising trainee psychiatrists suggests that the knowledge base is not generally problematic–medical training equips practitioners with the ability to assimilate new information rapidly.

Skills development in CBT is more difficult. The difference between the Turkington & Kingdon approach and cognitive therapy is that a formulation is not used, but it may be that an experienced CBT practitioner (as in their study) employs an implicit individualised formulation, whereas a more typical general psychiatrist would not. Cognitive–behavioural techniques can be powerful, and using these techniques without a formulation can be clinically dangerous. For example, undertaking attributional change in a patient with paranoia without regard for the link between persecutory delusions and self-esteem (Lyon et al, 1994) may trigger depression. Cognitive–behavioural skills development requires clinical supervision.

Attitudinal change is most difficult. Consider the response of a psychiatrist to a patient’s question, “Should I take my medication?”. Valid responses might include “Yes” from a biological psychiatrist, and “Here’s the evidence of effectiveness . . .” from a social psychiatrist. However, a response such as “How will you decide?” is a more appropriate cognitive–behavioural response. Psychiatrists can develop these alternative attitudes, but a ‘psychiatric’ mind-set often re-emerges in therapy.

Improved access for patients to psychological therapies is imperative. However, although it may serve the profession of psychiatry well to indicate that the use of CBT is easily within the grasp of all its practitioners, it is not clear that this is empirically demonstrated.


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Authors’ reply: We thank Dr Slade for his interest in our paper and are grateful for the chance to clarify these crucial points. At the time this study was carried out D.K. was a general psychiatrist working full-time in the community. He did have a general cognitive therapy training and applied the techniques that he had learned for the treatment of depression and anxiety in an adapted form to his patients with schizophrenia (Kingdon & Turkington, 1991). We then designed this study to test the efficacy of these techniques as against a befriending control (Kingdon et al, 1989). The study was then carried out in the course of D.K.’s clinical work.

We accept that a shared formulation is a fundamental component of cognitive therapy with schizophrenia (Fowler et al, 1995), which not only helps to direct the process of therapy but which can also help to predict the emergence of depression as a relapse recedes. The identification of key maladaptive core beliefs is part of the process of formulation and helps when there are blocks in progress and in the prevention of relapse. Psychiatrists do not normally identify such core beliefs in their case formulations and may not share all of the formulation with the patient. However, the formulations of psychiatrists do contain an aetiological component comprising such issues as genetic predisposition, birth traumas, early losses and personality. The precipitation of the psychosis in relation to any pertinent life events is included, as are maintaining factors such as isolation, poor adherence or high expressed emotion within the family (Gelder et al, 1983). Formulation is so central to psychiatric practice that it is a key component of the Royal College of Psychiatrists’ membership examination. With such formulations psychiatrists can safely use cognitive–behavioural techniques as long as they remain aware of the risk of emerging depression or increased suicidal ideation linked to improved insight. Such phenomena are widely recognised in psychiatry when certain types of symptoms (e.g. grandiose or systematised delusions) respond to antipsychotic medication.

The purpose of our paper was to attempt to shift attitudes in psychiatry in order that we can be better engaged with our patients who have psychoses by working more directly with their symptoms rather than simply monitoring them and titrating antipsychotic medication. If general psychiatrists can make this shift in attitude, training workshops and supervision will be necessary. We expect that this shift could well facilitate the delivery of formal cognitive therapy by other community mental health teams trained in this approach and help with the implementation of other psychosocial interventions in this patient population.


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Assessing quality of life in schizophrenia

Reading Wilkinson et al (2000), I felt that their new questionnaire tends to measure symptoms rather than quality of life. Obviously the symptoms and side-effects scale measures symptoms but a number of items in the other two scales measure symptoms as well, for example, “I lack the energy to do things”. The relationship between symptoms and quality of life is complex but issues such as the availability of money or quality of accommodation must have something to do with it and neither is covered in their questionnaire.

The authors state that in measuring quality of life the measure has to be subjective, which makes sense, but whether it has to be self-reported is questionable. The authors suggested greater honesty might be outweighed by the disadvantage that no help is available if there is a confusion regarding an item. It can see no advantages in using this new tool over existing tools, such as the Manchester Short Assessment of Quality of Life (Priebe et al, 1999), which is similarly short and useful in clinical practice.


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Authors’ reply: The Schizophrenia Quality of Life Scale (SQLS) is a measure of quality of life based on statements made by people with schizophrenia. We asked participants to state how their quality of life was affected by their mental health and the result was a mixture of symptoms, side-effects and psychosocial issues. Naturally, there is an overlap in the use of these terms, but our method elicits responses related to quality of life from a patient’s perspective.

We believe that nobody can know their quality of life better than the person him- or herself. We have no reason to believe, on the basis of our findings, that people with schizophrenia are incapable of accurately representing their quality of life using the SQLS. It is of interest that we found that the people we interviewed voiced a variety of concerns, albeit not about possible financial and accommodation problems, which are, in any event, objective issues.

The measurement of quality of life is in the scientific domain and different methods, whether by questionnaire or interview, need to demonstrate reliability and validity. Thereafter, the choice of instrument requires a trade-off, representing a decision about the best instrument for a particular purpose. Factors such as ease of use, acceptability and cost must be considered.

In contrast to measures such as the Manchester Short Assessment of Quality of Life (MANSA), the SQLS has been specifically developed from interviews with people with schizophrenia, as opposed to being based on other measures. It was also developed as a measure for use in clinical trials and other research studies, as opposed to the assessment of community programmes, which is the case with the Lancashire Quality of Life Profile (Oliver et al, 1996), the measure from which the MANSA was derived. We would emphasise that the administration procedures contained in The User Manual for the SQLS (available from Oxford Outcomes) recommend, among other things, that the SQLS is completed in the presence of the researcher or is administered as an interview if the patient has difficulty with self-completion.

The SQLS has been very well received by colleagues both in the UK and internationally and the development of the instrument is continuing. It is clear to us from the response so far that the SQLS is recognised to fulfil a significant clinical and research need in relation to the assessment of quality of life in people with schizophrenia.


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G. Wilkinson University Department of Psychiatry, Royal Liverpool University Hospital, Liverpool

Is couple therapy better than antidepressant drugs?

The conclusion that couple therapy “is much more acceptable than antidepressant drugs” is not based on the findings of this study (Leff et al, 2000). To generalise results of a trial on desipramine, a tricyclic antidepressant (followed by trazodone and fluvoxamine), to antidepressant drugs as a whole is misleading. Other antidepressant drugs may be more acceptable than the trial drugs and the conclusions should have been limited to the drugs used.

Intention-to-treat analysis can sometimes be taken to the extreme. In this study, the majority of subjects in the antidepressant group (56%) did not receive the antidepressant as randomised and therefore it is not surprising that the antidepressant group did poorly. It would have been interesting to see, in addition, the results of an explanatory analysis that would have shown the outcomes for those who actually received antidepressant drugs compared with those who actually received couple therapy. Although the study would, technically, no longer be a randomised trial, this would not be a problem because the baseline comparison shows that the two groups are comparable, the essence of randomisation. It is not always the case that intention-to-treat analysis is better than explanatory analysis.

Although comparatively new, the number needed to treat is now widely understood as a simple and meaningful analysis of trials. What is the improvement rate attributable to couple therapy in this trial and how many patients will need to be treated for one more patient to gain improvement with couple therapy over desipramine?

Finally, the effect of an important confounding variable has not been discussed. The results may have been confounded by marital discord that will respond better to couple therapy than to antidepressants.

These must be considered before recommending couple therapy over antidepressant drugs in people with depression living with partners.


L. O. Ogundipe Lyme Brook Mental Health Centre, North Staffordshire Combined NHS Trust, Bradwell Hospital, Tatke Road, Newcastle-under-Lyme, Staffordshire ST5 7TL

Authors’ reply: Dr Ogundipe is, of course, quite right in pointing out that our conclusion about the greater acceptability of couple therapy compared with antidepressant drugs can only apply to the particular
Citalopram-induced bruxism

There have been several reported incidents of iatrogenic bruxism (involuntary clenching or grinding of the teeth). These have involved diurnal bruxism (Micheli et al., 1993), felt to be associated with dopaminergic blockade, and nocturnal bruxism. Nocturnal bruxism has been reported with venlafaxine, a serotonin/noradrenaline reuptake inhibitor, which responded to gabapentin (Brown & Hong, 1999), as well as three selective serotonin reuptake inhibitors (SSRIs), paroxetine (Romanelli et al., 1996), fluoxetine and setraline (Ellison & Stanzi- ni, 1993). In both reports the SSRI-associated bruxism was treated with buspirone.

I report two cases of nocturnal bruxism secondary to the SSRI citalopram, a previously unreported adverse effect. One patient was started on citalopram 20 mg/day. After 6 weeks the dose was increased to 40 mg. Ten days later nocturnal bruxism developed to such an extent that extraction of a molar was required. Buspirone was started and the bruxism ceased.

Another patient with panic disorder and moderate depression with somatic symptoms was referred to the clinic. The existing medication was a tricyclic and buspirone. Subsequent to non-response, medication was changed to citalopram, eventually reaching 40 mg/day. After an improvement in mood a behavioural programme was used to treat his anxiety symptoms. Four months into the programme the buspirone was reduced from 10 mg twice daily to none. Three weeks later he reported nocturnal bruxism. This ceased after reducing the citalopram to 20 mg/day. Thus, in this case, occult nocturnal bruxism was revealed by the reduction of a treatment agent.

These cases highlight that nocturnal bruxism can occur in response to any of the SSRIs, and that induction may be dose-dependent. They add to the literature suggesting that nocturnal bruxism can be treated with buspirone.

Traditional treatment is by necessity a multi-layered approach using both psychological and environmental techniques.

Psychological debriefing – does it never work?

Mayou et al. (2000) conclude in their 3-year follow-up study of road traffic accident victims that psychological debriefing is ineffective and has, in fact, adverse long-term effects. The intervention group reported significantly worse outcome at 3 years in terms of more severe psychiatric symptoms, impact of event symptoms, anxiety, depression, obsessive-compulsive problems and hostility, pain, major chronic health problems and financial problems. The findings support the suggestion that routine use of psychological debriefing among trauma victims should be discontinued (Bisson et al., 1997).

However, this conclusion is premature. A most serious problem in previous research is that the term psychological debriefing has been used for different types of interventions, for example, in terms of number of sessions and individual or group debriefing. Mayou et al. offered individual one-session intervention, without any follow-up. This kind of intervention is contrary to most clinical thinking: first, assess the trauma; second, offer treatment accordingly. Nobody would recommend that all victims of traffic accidents should be given a standard surgical procedure of 15 minutes in the operating room. For patients with major traumas, the results may be worse than having no operation. The conclusion based on such an approach might easily be that surgery after traffic accidents should not be performed.

A flexible and individual approach is a much more reasonable and appropriate strategy (Rose et al., 1999). Future studies of psychological debriefing should use an individualised design including screening of psychopathology before intervention, if any, is offered. To assess the effect of one session of debriefing, only subjects who are likely to benefit from such a limited intervention should be included (i.e. those who are at greatest risk for post-traumatic stress disorder should be excluded).

The Impact of Event Scale scores for patients with high initial scores was 25.9.
11.8 in the control group (Mayou et al., 2000). This may indicate that the trauma of meeting a debriefer for 1 hour was comparable to the trauma of the traffic accident itself.

If the findings of Mayou et al are valid, it shows a tremendous potential for psychological intervention. However, we are still not convinced that a 1-hour psychological intervention unintentionally can do so much harm.


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Diazepam in the treatment of GHB dependence

We read with great interest the recent case reported by Price (2000) concerning the utility of diazepam in suppressing gamma-hydroxybutyrate (GHB) dependence and related withdrawal symptoms. Before describing the case Price states, “I believe this to be the first reported case of in-patient detoxification”. However, the first report on the utility of the same doses of diazepam in treating GHB withdrawal syndrome was published a year earlier by our group in a patient taking about 181 g/day GHB for 4 months, for its euphoric and anxiolytic effects (Addolorato et al., 1999). On discontinuation of GHB, the patient showed a withdrawal syndrome consisting of high anxiety levels, tremor, sweating, tachycardia and nausea. Complete disappearance of drug withdrawal symptoms was achieved within 2 hours in the first day of treatment with diazepam 20 mg orally administered, and the patient was treated with the same dose of diazepam for another 6 days. After suspension of the diazepam, the symptoms did not recur. Also in this case, the detoxification programme was safe. We are very pleased to know that our findings are in perfect agreement with that of Price; since several cases of GHB misuse and dependence have been described in recent years (see Addolorato et al., 2000), we think that these reported experiences, as well as the recent case described by Hutto et al (2000) about the utility of chloral hydrate, could be of clinical relevance, particularly considering the difficult management of these patients.


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Seasonality of suicides

We read with interest Yip et al’s (2000) report on the decrease or disappearance in seasonal variation of suicides in the 1980s and 1990s in England and Wales. The authors have suggested a similar trend in other countries (Ho et al., 1997; Yip et al., 1998). This paper inspired us to explore seasonality of suicide in Japan, where national data on monthly suicides are collected and published every year by the Ministry of Welfare. We examined monthly data by gender for the 15-year period 1982–1996. A daily mean suicide incidence was calculated for each month. Harmonic analysis (Pocock, 1974) was applied to identify seasonal components in the variation of monthly suicides.

A total of 332 651 suicides (215 686 male, 116 965 female) were identified for the period (mean suicide rate 25.8 and 13.2 per 100 000 for men and women, respectively). During the entire period, the age distribution of suicide rates, which generally increases with age, and popular methods of suicides (approximately 60% violent, nearly 35% non-violent, remainder unclassifiable or method not known) were generally unchanged. Regardless of gender, mean daily suicide incidence by month demonstrated a clear bimodal distribution, with a largest peak in April and a smaller peak in September. Harmonic analyses revealed that over 65% (66.7% for men and 66.2% for women) of the total variance in suicides was explained by the seasonal component, where one-cycle and two-cycle components illustrate the majority of total seasonal harmonics (one-cycle: 64.3% for men and 69.1% for women; two-cycle: 32.9% for men and 28.7% for women). No gender difference was found in the results from the harmonic analyses.

Unfortunately, no data on monthly suicides by age, by method or by place are available from the Japanese Government, which complicates further analyses regarding the backgrounds of such a marked seasonality in suicides. It may be noted that the school and business year begins in April in Japan, which may affect the seasonality of suicides, at least the largest peak of suicides in April. Overall, the results of our analyses indicate limitations in generalising from Yip et al’s conclusions and suggest a wide variability in seasonality of suicide in different areas.


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Reflecting on the diminished seasonal variation in suicides reported by Yip et al (2000), I was mindful of the recent extreme weather conditions – gales, floods, tornadoes – and the absence of ‘November’ – trees retaining their leaves, moderate temperatures, the absence of frost. Additionally in recent years we have been spared cold winters and lamented the loss of any recognisable British summertime. Therefore, one might wonder whether the disappearance of seasonal variation in suicide mirrors the
disappearance of familiar markers of the seasons. In the absence of hard data, perhaps global warming rather than global communication (e-mail, telephone, etc.) underlies the observed trend. In future maybe other changes in traditionally seasonally linked illness patterns may be seen (e.g. in seasonal affective disorder) and this may also account for the current excess of patients with mania on our local admission ward – they seem somehow to have missed the expected springtime peak.

Reduced suicide rates among the divorced and widowed may be linked to societal changes such as the greater acceptability of single status.

We may expect human cycles (illness, behaviour) to change in line with adaptation of society and the eco- and planetary system.

But for now let us hope that there is no direct causal link between psychiatric morbidity and rainfall (Ohl & Tapsell, 2000).


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Remember the depressed vegetarian

A recent experience has raised the issue of whether prescribers consider the suitability of medication for their vegetarian patients. A 30-year-old woman was prescribed an antidepressant for a depressive disorder. She later informed us that she could not take them as they contained gelatin, which is not suitable for vegetarians. This experience raises many questions. Which antidepressants contain gelatin, are prescribers aware of this information and do prescribers routinely enquire whether their patients are vegetarians prior to prescribing antidepressant medication?

We researched commonly used antidepressants by contacting the pharmaceutical companies regarding the origin of the excipients in their antidepressants. Those investigated included fluoxetine, citalopram, paroxetine, sertraline, reboxetine, venlafaxine, amitriptyline, dothiepin, imipramine, lofepramine, trazodone and nefazodone. Of these, five antidepressants are definitely suitable for vegetarians. These are sertraline tablets, venlafaxine tablets (but not capsules), fluoxetine liquid (but not capsules or tablets), amitriptyline mixture (but not capsules or tablets) and imipramine mixture (but not tablets).

Assuming the lifetime risk of developing a depressive disorder warranting treatment is 10% and there are about 4 million vegetarians in the UK (The Vegetarian Society, Altrincham, personal communication, 2000), 400 000 vegetarians may need antidepressant medication.

Hence, vegetarians are commonly likely to be prescribed antidepressants by their general practitioners or psychiatrists. It is important that enquires be made routinely regarding whether an individual is a vegetarian prior to prescribing antidepressants.

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One hundred years ago

London County Asylum, Claybury, Essex (Annual Report for 1899)

... Of insanity associated with senile decay only 10 per cent. were over 60 years of age, against 18 per cent. during the previous year. General paralysis has been found in 11 per cent. of the male admissions and in nearly 4 per cent. of the female. “There has been a high percentage of insanity from alcohol, and more than double the number of women than men have been admitted suffering from mania a poti.” It is also ascertained that women relapse into insanity from alcohol and are re-admitted with far greater frequency than men. Their weakened inhibition appears to be unable to withstand the slightest temptation, and Dr. Jones points out that the best treatment for such cases is that of long detention in inebriate homes, which naturally cannot apply to asylums from which patients are discharged when mentally fit. Previous attacks and hereditary influences were ascertained to be the most probable cause of insanity in 34 per cent. of the admissions. Several patients who were admitted had delusions that they were “hounded by Kruger’s relatives” and that “Spion Kop” was hissed into their ears.

REFERENCE

Lancet, 22 December 1900, 1831.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Epsom, Surrey
Citalopram-induced bruxism
M. E. J. Wise
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References
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