Correspondence

Edited by Kiriakos Xenitidis and Colin Campbell

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Time to start taking an internet history?

Young people are turning increasingly to the internet to meet their educational, entertainment and social networking needs, and in times of emotional or psychological difficulty, they may likewise seek information online. The scope for anonymity, information, interaction, and sometimes fantasy, without fear of repercussion, makes the web an obvious choice for young people who are reluctant to disclose their difficulties to parents or professionals.

In the aftermath of the Bridgend suicides, recognition of the dangers of life online was recently highlighted by a government pledge to strengthen the Suicide Act 1961 by specifying the illegality of any internet activity which encourages suicidal behaviour. The move comes in response to public concern over internet safety, particularly for young and vulnerable groups, who may access websites or online communities that promote suicide or self-harm. In this context, and with approximately 170 000 adolescent self-harm hospital presentations per year, the revised legislation is an important and timely step.

A new diagnosis of ‘internet addiction’ has been proposed and much of the current research into this problem comes from Asia, where cardiopulmonary-related deaths and even gamede related murders in internet cafes are now regarded as serious public health issues. In the West, most psychiatrists share the view that use of the internet is not a mental disorder in itself, but may contribute to the development of psychiatric symptoms; the key is for a careful and sensitive internet history as part of routine psychiatric history taking.

Eliciting a careful and sensitive internet history as part of routine psychiatric history taking may prove invaluable in assessing young people at risk of self-harm and suicide and in uncovering other aspects of psychopathology associated with excessive or unhelpful internet use. Above all, further research into the relationship between online activity and mental health among adolescents and the general population is crucial if we are to manage risks associated with internet use and also take advantage of potential benefits of new technologies.


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Homicide due to mental disorder

The article by Large et al describes the rise and fall in homicides attributed to mental disorders in England and Wales over the past 50 years. Since 2000, the rate of homicide due to a mental disorder in England and Wales has been 0.07 per 100 000 or lower. Encouraged by the authors, we examined the rate of homicides due to a mental disorder in The Netherlands. Dutch law considers responsibility for crimes to be diminished if there is a causal relationship between a mental disorder and the crime committed. Five degrees of responsibility are defined (i.e. complete responsibility, slightly diminished, diminished, considerably diminished, and total absence of responsibility). A severe psychiatric disorder, usually of a psychotic nature, is a necessary condition for a ‘total absence of responsibility’ finding.

From 1212 cases of homicide between 1 January 2000 and 31 December 2006, 1020 (84.2%) defendants were psychiatrically assessed pre-trial. Of these, 58 (5.7%) were considered to have total absence of responsibility. Furthermore, 63 (6.2%) were found to have considerably diminished responsibility, 239 (23.4%) diminished responsibility, 309 (30.3%) slightly diminished responsibility, and 259 (25.4%) complete responsibility. A psychotic disorder was diagnosed in 115 (11.3%) people, which is in line with earlier studies. The rate of homicide due to mental disorder would be 0.11 per 100 000 when individuals with a total absence of or strongly diminished responsibility are included. If individuals with a diminished responsibility are also included, this would be 0.32 per 100 000.

The difference between England and Wales and The Netherlands may be explained by a different view on the issues of a diminished responsibility. This may also explain the rise and fall of homicides due to mental disorders in England and Wales over the past 50 years.


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A conclusion in the abstract of Large et al is illogical. If the same sociological factors causing increase in ‘other’ homicides up until the 1970s had caused the increase among the mentally ill, then they should have continued to have exerted this effect, with a continuing rise corresponding to ‘other’ homicides instead of a fall. Similarly, if the subsequent decline in homicides among the mentally ill were due to improvements in psychiatric treatment and service organisation as the authors suggest, then the rise in their rates prior to that period must have been due to the converse: a deterioration in quality of treatment and service organisation. The obvious explanation (which is now politically incorrect) is the closure of mental hospitals and rehabilitation at that time because of almost non-existent community care.

In reality, it is highly unlikely that there has been a true rise and fall in homicide among mentally ill people in England and Wales over the past 50 years. These figures are entirely based on statistics which reflect the workings of the Criminal Justice system (a charge to which I plead guilty). They merely reflect changes in processing defendants by the courts. The probable culprit for declining diminished responsibility was declining enthusiasm for treating personality disordered and sexually deviant killers under the Mental Health Act legal category ‘Psychopathic Disorder’. The authors did not provide statistics on other forms of manslaughter. These have increased in recent years, suggesting that defence lawyers have become more successful in putting forward alternative defences to murder than diminished responsibility.

I agree with the authors that sociological and legal factors (mainly the latter) have effects on rates of homicide due to mental disorder. But it is the overall base rate of homicide in the population that matters and with which these figures must be compared. This differs markedly between different countries. In those where it is very high, such as South America and Sub-Saharan Africa, mental disorder is almost irrelevant as an epidemiological risk factor. The authors refer to a small number of studies suggesting a correlation between rates of homicide among the mentally ill and rates among the rest of the population. It may well be that the ‘laws’ they refer to are too rigid. For example, it makes sense that a country that allows handgun ownership is more likely to have killers with schizophrenia who use a handgun, and at a rate higher than in countries where hand guns are banned, although the evidence for this remains thin on the ground. But from the public health perspective does it matter? Handguns are the key risk factor, not schizophrenia.

England and Wales have a low but steadily rising rate of homicide. It is unrealistic to propose mental health services as a public health intervention, but will be popular with politicians. Social geographers have demonstrated that social exclusion and growing social inequalities are the strongest correlates with this phenomenon affecting young men in England and Wales.3


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Antipsychotics and risk of diabetes in schizophrenia

Smith et al state that there is increasing concern among clinicians about the association between second-generation antipsychotics and diabetes.1

It is interesting then that while commenting on the lack of systematic reviews and meta-analyses that support this concern, the authors go on to investigate not the relationship between starting antipsychotics and developing diabetes, but the relative risk of developing diabetes between groups of patients commenced on first-generation and second-generation antipsychotics. It is questionable whether this meta-analysis addresses, in any clinically meaningful way, the risk of developing diabetes after starting an antipsychotic, whether second or first generation. This would appear to be more usefully addressed by looking at the absolute risk.

The authors report on the difficulties in finding high-quality trials to include in their study. This is illustrated by the inclusion of only 11 trials out of an identified 1974. Smith et al then go on to outline their own criteria for a study to be considered of ‘high quality’. These criteria include a prospective design and at least 1 year of follow-up recorded. It is of note then that of the 11 studies eventually included in the analysis, only 3 were prospective. Furthermore, of these 3 prospective trials, none was longer than 3 months. All trials included in the review could, therefore, be classified as low quality. The test for heterogeneity between studies, applied by the authors, further illustrates the highly significant methodological heterogeneity between studies.
We would suggest that given the overall poor quality of studies found in the review there seems to be no rationale for going on to conduct a meta-analysis. One common pitfall of any meta-analysis is that if you put only poor-quality data in, you will get poor-quality data out. Consequently, this meta-analysis would seem to add little to the current evidence base with regard to antipsychotics and diabetes, except, perhaps, the confirmation that the studies on this subject are heterogeneous and generally of poor quality.

If one does want to consider whether a significant relationship exists between antipsychotic use and diabetes, or a metabolic syndrome, then the CATIE study2 would seem to provide reasonably robust evidence that such a relationship does exist. This large, randomised, prospective study, carried out over a period of 18 months, has data collected at baseline and following the introduction of antipsychotic, and demonstrates clinically and statistically significant adverse changes in blood glucose, weight and cholesterol. This is particularly the case for those patients commenced on olanzapine.

Declaration of interest
R.P. has received speakers’ honoraria from Janssen-Cilag, Eli Lilly and Wyeth Pharmaceuticals.


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Authors’ reply: We acknowledge Smith & Porter’s interest in the reasons for why we did not focus on the relationship between merely starting any antipsychotic and developing diabetes, but instead reviewed the evidence for an association between diabetes and type of antipsychotic medication. There has been increasing concern that second-generation antipsychotics may be more diabetogenic than first-generation antipsychotics in patients with schizophrenia. Despite this concern, there is a lack of good evidence to support this apparent phenomenon and so it was essential to carry out our systematic review prior to developing guidelines for diabetes screening and management.

We agree with Smith & Porter that our paper has found strong heterogeneity between studies which is clearly an important finding from our study. It is only by undertaking systematic reviews that one can determine that heterogeneity exists. Therefore, without our systematic review this would not have been clear. Our meta-analysis uses random effects methodology, which means we have analysed the average effect over the studies. This is a meaningful concept in the presence of heterogeneity. As for looking at absolute risks, the heterogeneity between studies is so great as to make even random effects pooling absurd. This is why pooled analyses virtually always pool relative risks rather than risk differences.

Smith & Porter have highlighted our conclusions that methodological limitations were found in most studies. As current evidence is poor, it should not be used alone in making clinical decisions concerning diabetes screening and management for patients with schizophrenia. Regardless of whether first- or second-generation antipsychotics are prescribed, routine screening for diabetes in all patients with schizophrenia should be undertaken.

Pharmacology and human morality
Maybe I am missing something but what is new in the proposition Spence has outlined?1 When a Yanomani tribesman snorts a powerful concoction of hallucinogens he does so as part of a ritual that includes the shamanistic healing of others in the tribe and maintaining tribal cohesion through tradition. When a footballer plays on despite injury, with pain relieved by analgesia, he does this in part for his team and fans. When a Peruvian highlander chews coca leaves so that he can work longer hours he does so to keep his family fed; and the same applies to the kratom user in the Far East. When millions of soldiers took amphetamines to enable them to fight for longer hours, thereby exposing themselves to ever greater dangers, they did so to win what they believed to be just wars. When a mother solicits fertility treatment so as to produce a child that will not only add to the family, but also potentially save the life of another sibling, the use of these potentially dangerous drugs is largely driven by the mother’s need to save the other child. When groups of men gather every afternoon in the Yemen and chew qat, this is a social activity enhanced by the use of qat. In the Middle East, coffee shops have always served this purpose, providing socially stimulating conversation, and do so in Europe to this day. Tobacco has had a similar use in many countries and alcohol has done much the same, despite the harm associated with the use of both of these substances. Psychiatrists, on a small scale, have started to use what some term empathogens (i.e. MDMA) so that they can better understand and help their patients (although the less charitable question their motives).

I think we would be splitting hairs to argue that taking a drug to achieve a moral end is fundamentally different from achieving a moral end through use of a drug; they exist on a continuum. Drugs simply allow us to explore and alter our behaviour and thoughts. How we use this allowance is up to us.


In a recent editorial, Spence stated that the pharmacological interventions currently available in psychiatry also improve moral behaviour.1 He subsequently argued that there is no fundamental difference with moral enhancement therapy, medication specifically developed to increase moral behaviour. Spence gave the example of a patient who continues to take antipsychotic medication because he knows he can be violent when unwell and he wants to prevent risks to others.

Spence asserted that whether an intervention assists in ‘moral enhancement’ or not crucially depends upon the goals of the

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Duration of untreated psychosis in LAMI countries

I have some reservations regarding the conclusions drawn by Large et al1 in their study on duration of untreated psychosis in low- and middle-income (LAMI) countries. This is because the samples are not really representative of the occurrence of psychosis. It seems, people with untreated psychosis who have recovered or remitted without antipsychotic or medical treatment are excluded from this study. There is enough evidence that in LAMI countries, a substantial proportion of patients with psychosis seek treatment from traditional healers,2 use indigenous methods based on their non-biomedical beliefs3 or pathways to care.4,5 Perhaps, many of those who fail to respond to these methods seek psychiatric help. Thus, the sample which reaches psychiatric services is a biased one. In clinical practice, we do encounter patients who have had previous episodes of psychosis which remitted spontaneously or by indigenous methods. Studies on duration of untreated psychosis should be community or general population based to overcome the confounding effects of non-psychiatric treatments and biased sampling. This is true more so for LAMI countries where such non-medical services are popular, in contrast to high-income countries6 with well-organised health services, where any patient with psychosis is likely to reach psychiatric services without the pathway to care through non-psychiatric methods. This limitation needs a mention by the authors.1


patient concerned, i.e. the ‘ends’ he or she is pursuing. However, ‘the goals of the patient concerned’ can be problematic in the cognitive enhancement debate and this formulation can conceal important ethical issues.

Spence mentioned the concept of meta-responsibility, the fact that somebody can be responsible for becoming irresponsible, in the case of the example that somebody can be responsible for deciding not to take medication.2 In a somewhat similar way as Mitchell, Frankfurt3 discussed the difference between first- and second-order desires. One can have a desire for smoking, which is a first-order desire. One can also have a second-order desire, namely the desire not to have the desire for smoking.

One could argue that in the future pharmacological interventions might be able to interfere with second-order desires. Second-order desires according to Frankfurt are the core aspect of personhood. Even if one does not want to go as far as Frankfurt in stating that the second-order desires determine personhood, moral enhancement treatment can be problematic if it could change second-order desires. In that case, people’s goals would alter. Contrary to Spence’s view, moral enhancement pharma-
terapy can be quite controversial if it interferes with second-

order desires.


Author’s reply: The varied correspondence precipitated by my editorial has invoked a great many issues. However, the sole aim of my original piece was to examine whether a current concern with the putative cognitive-enhancing effects of certain medications might be redirected towards the possible enhancement of other human attributes such as moral behaviour.1 Should this be of interest to psychiatrists? Well, I believe that there is something worth scrutinising within the medical consultation when a patient (a moral agent) considers the likely impact of their future conduct upon others, and the various means via which such conduct might be modulated. Drugs are not the only means by which such modulation might occur but they do provide an interesting example. Nevertheless, as I acknowledged in the editorial, such a juxtaposition of pharmacology with morality risks provoking reflexive responses: strong opinions unencumbered by reflection.

Clearly, the situation in the consulting room with an antisocial or aggressive man is rather different from that outlined by Al-Adwani. We are not talking about the social consumption of stimulants and intoxicants or the enforced ingestion of medicines by combatants in order for them to fight for longer. We are talking about what individual patients might choose to do about their own future behaviours, sometimes under very difficult circum-

stances; indeed, an antisocial male may not even enjoy a

community of peers with whom to consume coca, kratom or qat. I apologise if this was not sufficiently obvious.

With respect to Frankfurt’s conjecture that we might all harbour first- and second-order desires, Hubbeling’s point is well taken: that if we posit such a hierarchy of desiring processes, then an individual’s second-order (pro-social) desire to control an aberrant first-order desire (to react aggressively, to assault someone) might utilise a pharmaceutical agent only, to discover (later on) that the latter had modulated not only the first-order construct but the second-order one as well. The questions arising, here, are: (a) whether such first-order and second-order desires enjoy any empirical demonstration of their existence; and (b) whether, if second-order desires really exist, we are currently managing to avoid affecting them when we prescribe psychotropic medications or engage in any form of dynamic psychotherapy. To my mind, this makes the central question of even greater interest and one deserving of further empirical exploration.


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Authors’ reply: Professor Chaturvedi raises the possibility that our systematic review of the length of untreated psychosis in LAMI countries was confounded by a definition of treatment that was based on presentation to psychiatric services and did not account for presentations to traditional healers.

We acknowledge that a minority of the studies included in our review were based on population-based surveys of psychosis and that most of the studies did not include patients who only presented to traditional healers or did not receive any psychiatric treatment.

However, poor outcome in schizophrenia is known to be associated with delay in commencing treatment with antipsychotic medication, whereas little is known about the effects of a delay in non-pharmacological treatment. Furthermore, in a literature review (submitted for publication: details available on request) we confirmed the findings of Marshall et al. that the adverse effects of delaying antipsychotic treatment are similar in high-income and LAMI countries. Hence, we believe that non-psychiatric treatment for psychosis is best thought of as a potential cause of prolonged duration of untreated psychosis, rather than a confounding factor in the definition of duration of untreated psychosis.

Psychoses with acute onset and short duration that might remit without treatment may be more common in LAMI countries. Patients with a short-lived psychosis might not always present to psychiatric services in LAMI countries, although in high-income countries acute psychosis is associated with a shorter duration of untreated psychosis. We do not know whether the exclusion of patients with a potentially short duration of untreated psychosis and those who only present to traditional healers would increase or decrease the mean period of non-treatment. In our review, population-based studies tended to report much longer mean periods of non-treatment than studies based on presentation to psychiatric services, although it is also possible that the lower mean duration of untreated psychosis found in upper-middle income countries was due to more individuals with an acute onset presenting for treatment early in their illness.

We agree that the pathways to care through non-psychiatric treatments warrant further investigation, but these studies should be conducted as part of an effort to reduce the unacceptably long duration of untreated psychosis in many LAMI countries.

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To prescribe or not to prescribe?

Despite the possible heterogeneity among some of the studies included in Tsapakis et al.’s study,1 the results, if accepted by the psychiatric fraternity, could lead to further reduction in the use of antidepressants in the child and adolescent population. The use of antidepressants in this group has already decreased by 33% since the Committee on Safety of Medicine’s (CSM’s) warning against the use of most antidepressants in children and adolescents.2 Although the National Institute for Health and Clinical Excellence guidelines on the treatment of depression among children and adolescents states that medication should only be used in conjunction with psychological interventions,3 the provision of psychological therapies remain thin on the ground in most parts of the country, which means that medication is often the only option available to clinicians for treatment of severe depression.

Although purely pharmacological treatment would be the least desirable option in depression and research evidence on the efficacy of antidepressants for those with depression in all age groups is either mixed or at best shaky, depending on which side of the debate one is on,4 most clinicians would agree that many patients with significant depression do improve on antidepressants. Although it is too early to judge whether reduction in antidepressant prescribing resulting from the CSM warning has resulted in an increase in depressive morbidity among children and adolescents in the UK, disturbing evidence is already emerging from the USA, Canada and the Netherlands5 on an increase in completed suicide among children and adolescents, which seems to coincide with the reduction in antidepressant prescribing following warnings by regulatory agencies. In a retrospective study done in Canada, a significant reduction in antidepressant prescribing, accompanied by a statistically significant increase in suicide among children and adolescents (relative risk=1.25, 95% CI 1.08–1.44; annual rate per 1000=0.04 before and 0.15 after the warning) was noted in the 2 years following issuance of the warning.6

Given the well-established link between depression and suicide, one can only conclude that clinicians may be under-treating depression in children and adolescents since the emergence of concerns in relation to antidepressants. I feel clinicians should use their own clinical judgement and take into account local resources before making decisions on the course of treatment in juvenile depression. This would help one maintain the right balance between evidence-based practice and what’s best for individual patients, especially in an area of practice where research evidence is often ambiguous and contradictory.

families is hard to find. However, the proposition that antidepressants may have similar effects at all ages is inconsistent with our findings of quite limited, and perhaps inversely age-dependent, efficacy of antidepressants, as a class, as well as a lack of statistically significant differences between older and modern agents (especially of tricyclics vs. serotonin reuptake inhibitors), and the powerful influence of study size on conclusions about ‘significance’ of separation of antidepressants from placebos.1

A timely and pressing question is whether antidepressant treatment alters suicidal risks. Depression and suicide are strongly associated, but prediction of suicidal behaviour, even in individuals with depression, is very difficult, and evidence concerning relationships of antidepressant treatment to suicidal behaviour, although consistent in randomised clinical trials, remains controversial.2,3 Whether or not youth suicide rates will consistently increase or decrease, remains to be seen, and to be sorted out from high international variation in yearly suicide rates and poor documentation of attempts.2

For now, it seems an inescapable conclusion that clinicians are left to their own clinical judgement about using antidepressants for young individuals diagnosed with major depressive disorder. Furthermore, disbelief that modern antidepressants show relatively modest effects compared with placebos and fail to separate statistically from older agents,1 paired with the repeated and the poorly documented assertion that some modern antidepressants work well in clinical practice, seems to avoid the issues. We considered various ways in which even randomised controlled trials may be misleading, including selection of atypical or mildly ill out-patients or use of inadequate doses of antidepressants,1 as well as current controversy about how to diagnose and quantify changes in affective disorders in children and adolescents. Nevertheless, it is difficult to simply dismiss and ignore the findings of the research that has been done to test the efficacy of antidepressants in juvenile depression.1


After the earthquake, the number of suicides surged in Nantou,1,2,3 The general patterns of suicide in both regions are similar,4,5 what happened in Nantou after the earthquake should inform suicide prevention in Sichuan.

Table 1 shows the suicide rates in Nantou before and after 1999. The female suicide rate rose more than doubled immediately – from 6.1 in 1998 to 14.2 in 1999, whereas a very small increase was observed in males. However, the male suicide rate showed substantial increases in both 2000 and 2001, indicating a delayed effect. On the whole, the rate of increase in Nantou was higher than that in other parts of Taiwan (81% v. 25%).

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<th>Table 1 Suicide rate for Nantou County and Taiwan Region before and after the earthquake, 1999</th>
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<td><strong>Male</strong></td>
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<td><strong>Nantou Taiwan</strong></td>
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<td>Suicide rate (per 100,000)</td>
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The death of one’s spouse may trigger suicidal thoughts, especially when compounded with the loss of the major income source. As men are more likely to be the ‘bread winner’ in rural areas, widows might suffer from a profound feeling of hopelessness immediately after a natural disaster. In the case of Sichuan, it is further aggravated by the loss of many children in the collapsed schools, many of them from one-child families (owing to the State’s family policy). In contrast, unemployment carries major risk for male suicides; men are likely to be of high risk when the earthquake’s impact on the local economy is fully manifested. This can explain the gender difference in the timing of heightened suicide risk in Nantou. It also suggests that the restoration efforts in Sichuan should devote resources to preventing suicide attempts among women in the short term, while devising strategies to prevent further causality for male suicides before the local economy fully recovers.

Acknowledgements
The author would like to pay tribute to those who have worked tirelessly to save the survivors.

Increasing awareness of eGFR monitoring

We are grateful to the Journal for highlighting the important issue of epidermal growth factor receptor (eGFR) monitoring in psychiatric patients prescribed lithium. We recently carried out an audit of renal function monitoring across Camden and Islington NHS Foundation Trust. The aim was to assess whether renal investigations for grade 3 chronic kidney disease and referrals for specialist advice are being documented according to the 2006 Royal Colleges of Physicians and General Practitioners and Renal Association guidance in the notes of those psychiatric in-patients currently prescribed lithium across the Trust. These guidelines were recommended to psychiatrists in October 2007 (www.rcpsych.ac.uk/members/rcpsychnews/october2007.aspx). Currently, eGFR is not part of the Trust-wide lithium blood-monitoring documentation.

A total of 303 sets of in-patients’ notes were reviewed from across the Trust. An audit tool was designed to record patient information relating to the lithium regime and serial recording of eGFR. It was also recorded whether investigative parameters were documented to have been carried out in the presence of abnormal eGFR results. Requests for specialist medical opinions were also noted. Electronic pathology results were used where there was no written record in patient notes.

Of 18 in-patients prescribed lithium:

(a) 3 (16.7%) patients had one-off abnormal results with other values recorded within the normal range;
(b) 1 (5.6%) patient had one documented eGFR (54) that was abnormal. Owing to their African–Caribbean ethnicity, this may not have been significant;
(c) 3 (16.7%) patients had no eGFR results recorded;
(d) no patients had eGFR documented in their notes.

When eGFR was abnormal, no further investigations were documented or specialist opinions sought.

This audit demonstrates that eGFR is not routinely monitored or documented in patients in our Trust who are on lithium, despite the guidance.

No outcome was documented on abnormal values. When values were abnormal, further investigations were not documented to have been performed. This supports Morriss & Benjamin’s view that psychiatrists require education about recent developments in renal monitoring in patients on lithium.

The audit results were presented at a local educational meeting and various local recommendations were made with the Pharmacy Department to improve awareness and practice.

There was agreement that the current lithium documentation charts should be modified to include eGFR as a routine part of the lithium work up and ongoing monitoring process. In conjunction with the Pharmacy Department, a brief, tabular form of the College guidance was developed to be incorporated onto Trust lithium-monitoring forms and in-patient pathology result forms. It was suggested that the Medical Education Department would add the guidance to the pharmacy section of the junior doctors’ induction training package. The importance of documenting patients’ ethnicity, age and gender on blood request forms in order that eGFR can be accurately calculated was also highlighted.

It is hoped that these recommendations may be helpful in assisting other mental health organisations both in monitoring their own practice, and in raising awareness among clinicians and other staff of the importance of eGFR monitoring in patients prescribed lithium therapy in hospital.


Corrections

* Cover picture: Norris Embry 1921–1981. BJP, 193, A21. Text was written by Dr Alexandra Pitman; edited by Allan Beveridge.

* Neuropsychiatric systemic lupus erythematosus associated with neuroleptic malignant syndrome. BJP, 193, 507–508. Authorship should read: Philippe Verdoot, MD, Eric L. Constant, MD, PhD and Arlette Seghers, MD, Université Catholique de Louvain, Cliniques Universitaires Saint Luc, Adult Psychiatric Unit, Belgium. Email: philippe.verdoot@uclouvain.be.

* The online Journal has been corrected post-publication in deviation from print and in accordance with these corrections.