COLUMNS

Correspondence

President’s response to editorial by John Cox & Alison Gray

I have been asked to comment on the editorial in this edition of the Psychiatric Bulletin.1

Beyond politics, beyond factions. Just try a little intelligent kindness — after all this is about putting patients first. To put patients first, professionals themselves have to be valued and supported.

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To view a sample chapter from Intelligent Kindness: Reforming the Culture of Healthcare by J. Ballatt and P. Campling, visit the College website: www.rcpsych.ac.uk/files/samplechapter/IntelligentKindnessSC.pdf

Overselling risk assessment

I need to congratulate Roychowdhury & Adshead1 on a thought-provoking critique. Their arguments struck a chord in exposing the flaws in risk assessment tools and their unjust application in preventative detention; however, I was disappointed that they did not go further. All of these tools, structured clinical judgement included, apply population-derived data to individuals, thus painting them with the behaviour of their peers. The central flaw of risk assessment lies in presuming causality from association. The premise in these tools that symptom severity invariably correlates with risk is demonstrably fallacious, as any psychiatrist could counter-cite cases where treating the mental illness improves functional ability in patients who choose pro-criminal lifestyles.

The second problem, as previously highlighted by Szmukler,2 is their inherent determinism by casting the subject (participant) as a hapless automaton. Society is rightly critical of the boorish youth who binge drinks and gets into fights, yet exculpates the capacitous non-adherent person with schizophrenia — and holds their psychiatrist vicariously liable for their violence.

Risk assessment attempts to sanitise an unpalatable fact that violence is part of the human condition, which exists independently of mental illness. Milgram3 and Zimbardo4 infamously illustrated this. Nonetheless, even when convicted, the offender without a mental disorder rarely faces the sanction of possible indefinite detention. Indeed, it was implicit in the debate around dangerous and severe personality disorder and the 2007 revisions to the Mental Health Act that psychiatry could be manipulated into preventingly detaining risky individuals in society without the bothersome need for a trial.5

The truth is that risk assessment has become an industry. Those devising the next ‘marginally-better-than-chance’ tool can live off the proceeds of the copyright, training seminars and subsequent release of version 2.0. It is also politically expedient in reverse-engineering a scapegoat and providing glib platitudes that ‘lessons are learnt’, and ‘something is done’ in a world increasingly tilting at the reality of rare unpleasant events.

I believe that expectation regarding the prescience of risk assessment has far outstripped the reality of what it can achieve. The evidence base for risk assessment, by the authors’ own conclusion, would not support its use as a diagnostic instrument; yet in clinical practice it is insidiously taking over as a priority. Criminal justice operates on the principle that it is better to let ten guilty men go free than convict one innocent. If the original question was one of ethics, surely for an exception to be made for those with a mental illness is frankly discriminatory.

Furthermore, the question around the ethical principle of beneficence remains unanswered: if risk assessment is a priority activity, what is the evidence that it improves outcomes over and above quality standard care? I cannot offer an alternative other than to lament the fact that the Richardson Committee’s report in 1999 on transforming mental health legislation from risk- to capacity-based was never realised. We need to refocus this debate clinically by emphasising ‘needs assessment’ over ‘risk assessment’. Risks are unavoidable; but good-quality evidence-based care should not be usurped by the latest fashionable risk assessment tool.


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GMC guidance needed

Roychowdhury & Adshead should be thanked for raising the issue of the ethics of the use of actuarial risk assessment in psychiatry.1 These ethics might at first appear obvious: medical practitioners must have an overriding duty to protect the public from serious crime. It follows that they must do everything possible to accurately assess the risk of such crime, including the use of these assessment instruments. However, as Roychowdhury & Adshead point out, these instruments will produce misleading results if the prevalence of the serious crime being considered in the relevant population is low or unknown. Indeed, they point out: ‘A key challenge in psychiatry is that base rates [of the prevalence of serious crime] are often
not known, are low and vary for different types of violence. So if doctors use these assessments they risk wrongly identifying their patient as at high risk of committing a serious crime, and then act in a way that is not in the best interests of that patient. Such an act would of course be inconsistent with the duties of a doctor as set out by the General Medical Council (GMC) in Good Medical Practice. It follows that while the prevalence of particular serious crimes in various patient populations is unknown or is known to be low, the use of these actuarial risk assessments will remain unethical. As Roychowdhury & Adshead conclude: [structured professional judgement] tools used as checklists of risk factors without construction of risk scenarios or a risk management plan remains harmful and unethical practice. In my opinion psychiatrists would value guidance on this issue from the GMC.

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Risk assessment and evidence-based medicine

The article by Roychowdhury & Adshead starts to place violence risk assessment in the context of medical care. Although this is welcome, their partial defence of risk assessment in general, and of structured professional judgement in particular, is based on some significant distortions.

The first distortion is the gross overestimation of the power of risk assessment to discriminate between low-risk and high-risk people. The authors present a contingency table that they imagine shows the ‘potential’ outcomes of a violence risk assessment (Table 2). Using their tabulated data, a diagnostic odds ratio for risk assessment can be calculated to be 81, indicating that the risk of violence in the high-risk group (50%) is hugely higher than in the low-risk group (1.2%). These figures are totally unrealistic. In fact, the diagnostic odds ratio of violence risk assessment in replication studies was recently estimated by meta-analysis to be 3. Roychowdhury & Adshead overestimate the discriminating power of risk assessment by 27 times. Moreover, even an unrealistically powerful risk assessment with diagnostic odds of 16 is of little or no value because of failure to detect potential violence in the low-risk group and the large proportion of false positives in the high-risk group.

The second distortion relates to the underestimation of the precision of medical tests. In fact, the authors seem to have had difficulty finding any medical test with diagnostic odds that they could compare to a violence risk assessment. Instead they chose to compare two medical treatments. They argue that the high number-needed-to-treat as a result of a violence risk assessment is acceptable in psychiatry because in cardiology the number of bypass grafts needed to prevent one fatal outcome has been calculated to be 53. However, the meta-analysis they derived this figure from compared coronary bypass surgery to angioplasty – both of which are highly efficacious treatments for angina. In reality, medical tests that are used to diagnose conditions with serious implications for the patient are very accurate – biopsy is an excellent indicator of cancer and an angiogram a good indicator of coronary heart disease.

Despite these limitations, I support the authors’ general idea of viewing risk assessment as a medical procedure. I would go further: surely violence risk assessment should be judged by the standards of evidence-based medicine. The real questions then become: (1) are there any rational interventions that can be justified in terms of cost and benefit that might reduce violence among high-risk patients (many of whom will not be violent) and yet should not be offered to low-risk patients (who commit as many or even the majority of acts of violence); and (2) is there evidence that shifting treatment resources from low-risk to high-risk people can, in any way, reduce overall levels of harm?

The answer to both these questions is no. There is no doubt that medical diagnostic tests serve as a good basis for medical treatment and that medical and surgical treatment can save lives. It is simply disingenuous to suggest that the same can be said of violence risk assessment.

Declaration of interest: M.L. has provided expert evidence in matters relating to risk assessment.

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Author response: We thank Dr Matthew Large for his helpful comments. We wished to respond only by clarifying that the figures in Table 2 were from a hypothetical population, based on a hypothetical risk assessment tool with certain sensitivity and specificity values. The purpose was to illustrate that, even in risk assessments with unrealistic accuracy levels, the positive predictive value (PPV) was still low, as it was greatly influenced by the base rate. Any misleading odds ratios arising from the table was not intentional and arose (perhaps ironically) by chance.

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OCTET Study: flawed by type 2 error

The OCTET study overcame many legal and ethical difficulties in setting up a randomised controlled trial (RCT) of community treatment orders (CTOs). We welcome the acknowledgment

COLUMNS
Correspondence

196
of some of the limitations of the trial, but are surprised that claims are still being made that the study demonstrates that CTOs do not achieve their principle purpose of reducing relapse and readmission.\(^2\)

Imagine a hypothetical RCT comparing medication with placebo. The trial would be powered based on estimated effect size and its duration would be based on expected time for response. If, in this scenario, 25% of those in the placebo arm had inadvertently been given the active drug, and if the duration of the study had been only a third of that planned, it would be inconceivable that the investigators would claim a negative result proved the drug ineffective. Yet this is analogous to what has taken place with OCTET.

In OCTET, median length of compulsion in the community was 183 days in the CTO group v. 8 days in the Section 17 group. Although this seems to indicate that it was a trial of people who were largely either subject to long periods of community compulsion (CTO group) or only a few days of compulsion (Section 17 group), a more detailed examination brings this into question. Almost 25% of the Section 17 group were still subject to compulsion by the end of the study, and the mean length of compulsion in this group was 46 days. In the CTO group, only 50% were subject to compulsion by the end of the study, with a mean length under compulsion of 170 days. This has two main implications.

First, the difference in mean length of compulsion between the CTO group and the Section 17 group was only 125 days, or a little over 4 months. It is questionable whether this is sufficient time for any benefits of CTOs to become apparent, and presumably the initial intention had been to compare 12 months in each arm.

Second, in effect, a quarter of the control group were receiving the same type of intervention as the CTO group throughout the course of the study. Any possible benefit in the CTO group would have been offset by the same effects in a large number of control subjects, leading to a large reduction in the power of the study and to type 2 error. The sensitivity analysis does nothing to address this loss of power. We contend that given these problems, in conjunction with the broader issues of recruitment and selection,\(^3\) it is not possible to claim that OCTET demonstrates CTOs to be ineffective.

The OCTET trial, community treatment orders and evidence-based practice

Based on the findings of the OCTET study,\(^1\) Burns & Molodynski reject observations of consultants who reported directly observable benefits from community treatment orders (CTOs). They argue that it is not possible to ‘see with one’s own eyes’ a probabilistic outcome that takes months to manifest itself.

This is a false analogy. In a subgroup of patients, CTOs result in a striking improvement in treatment adherence: if the CTO is lifted, patients discontinue treatment; re-implement the CTO (following relapse and re-hospitalisation) and treatment adherence is achieved again. In such cases, clinicians are able to ‘see’ the effect of CTOs on treatment adherence and reasonably expect improved clinical outcomes in the longer term. With such a dramatic response (treatment adherence) to the intervention (CTO), it would be scientifically unnecessary,\(^2\) and ethically unacceptable, to refer patients to a randomised controlled trial (RCT).

A number of previous reports have highlighted the potentially detrimental flaws in the methodology of the OCTET,\(^3,4\) which could explain the apparent paradox between the naturalistic observational studies that have shown significant benefit from CTOs,\(^5\) and the negative findings of the OCTET.

Take the scenario of a young man with chronic schizophrenia, who attends the psychiatric out-patient department escorted by his carer. He has a long history of non-adherence to treatment, as well as multiple formal admissions. The patient is known to discontinue treatment immediately after discharge from hospital, invariably leading to rapid relapse and hospitalisation. Since discharge from hospital on CTO 3 months earlier, his mental stability has been maintained and he has been accepting his fortnightly antipsychotic depot injections. His positive psychotic symptoms are minimal. He has become more sociable and has applied for a part-time college course. The psychiatrist tells the patient and his carer that he is going to lift the CTO. To his dismay, the carer asks the psychiatrist ‘Have you not seen with your own eyes that the CTO works?’ The psychiatrist replies, ‘Yes I have, but an RCT says this could not have been possible’. Would this be evidence-based practice?


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Author reply: Evidence matters (hopefully). Dr Owen (like Dr Curtis\(^1\) whom he cites) fails to distinguish between intervention and outcome in the OCTET trial. The intervention is the imposition of a community treatment order (CTO). The time under initial compulsion (183 v. 8 days on Section 17) demonstrates a clear and unequivocal difference. Where his figure of only 50% of CTO patients experiencing compulsion comes from baffles us. The difference in the total time under compulsion during the 12-month follow-up that he cites
includes the difference between the two outcomes (which includes in-patient compulsion from readmissions in both groups). There is no evidence that recruitment and selection were biased in any way and again we fail to understand on what Drs Owen and Curtis base this criticism. We adhered to the highest research standards throughout and the study has been extensively and rigorously peer reviewed.

Dr Mustafa in his letter advances no scientific critique of our work but does articulate the common response of many clinicians – ‘I have seen it work’. We have sympathy with this – we both entered this study expecting to find improved outcomes from CTOs. However, they do not deliver them and we were as disappointed as Dr Mustafa. Psychiatry has a long history of clinicians clinging to ineffective treatments convinced that they work. This is not surprising given the variation in outcomes in psychiatry and the fluctuating natural history of psychoses. Naturalistic observational studies do not prove otherwise – they have produced contradictory results, some for, some against. That is why we need rigorous randomised controlled trials. OCTET is such a rigorous trial and its findings, however unpalatable to some, are robust. It is also worth remembering that the only two other trials found the same. A profession that aspires to evidence-based practice should take these results seriously.


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Insulin coma therapy

Anyone working in an insulin unit in the 1950s would not recognise Dr Pimm’s account of the results of their treatment, or details of what it involved. The patients received daily and increasing doses of insulin, rising to many hundreds of units, for a 6-week period. The depth of the resulting hypoglycaemic coma was determined by the patient demonstrating a Babinski response over a period of 15 min. They were then revived by ingesting glucose.

I worked in the insulin unit at Newcastle General Hospital from 1956 to 1959, when I was senior registrar to Sir Martin Roth. Insulin treatment was reserved for people experiencing their first attack of schizophrenia, and from memory I would say half made a complete remission and another 25% improved. Nobody thought that we were effecting a cure, but remissions lasted about 2 years. One woman relapsed 9 years after her treatment. Of course there were dangers, but in those days the alternative was incarceration in a locked ward in a Victorian asylum, with little hope of rehabilitation or discharge.

Martin Roth was an intellectual giant, but also a man who was perspicacious and compassionate, and who would not have contemplated using such a treatment if he did not think it effective. The depth of the coma seemed to me to be critical in terms of remission. A few patients did not regain consciousness when given glucose, but usually ‘came out of it’ after some hours, although there was the occasional death. Very occasionally, a patient who was clearly psychotic who had an ‘irreversible coma’ on recovery was greatly mentally improved. These days, people find this difficult to believe, but I witnessed it on one occasion. I find it inconceivable that a multitude of psychiatrists, working in Europe and North America over 25 years, would not have noticed that the treatment they were giving was having no effect, when it clearly was, if only for a limited period. The real question was not whether insulin worked but how did insulin work.

I have no wish to minimise the success of Dr Bourne’s crusade, but what made insulin units redundant was the realisation that the new antipsychotic drugs actually worked, and at last, we had an effective and cheap and at last method of managing a seemingly incurable disease. This was generally accepted by 1960.


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Hope and hopelessness in carers of a relative with schizophrenia

In her editorial, Rebecca McGuire-Snieckus warns clinicians against promoting optimism in their clients, since this can lead to unmet expectations and negative reactions when such expectations are not realised. In his commentary on the editorial, Femi Oyebode criticises Martin Seligman for exaggerating the importance of happiness at all costs as a goal of existence, and quotes Aristotle as stating that it is the mark of a courageous man to face things that are terrible to a human being. I wish to illustrate this in the context of family carers of relatives with schizophrenia. In particular, I focus on the overinvolved carer who is unable to relinquish her/his hopes and expectations for the affected relative. They are readily recognised by habitually referring to their relative in the past tense, for example, ‘she was such a beautiful girl’ or ‘he was such a good student’. This form of speech reveals the fact that the carer is living in the past and has not come to terms with the reality of their relative’s illness. This is particularly hard on the patient, who then feels driven to attempt to satisfy the carer’s need for their success, and fails again and again. The remedy is to offer the carer grief work to mourn their losses and to accept the reality of their relative’s disability and release both parties from this impasse, enabling them to develop a more realistic view. The patient will also benefit from grief work, administered separately from the carer.


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