part of a biopsychosocial approach rather than a treatment of last resort.

Finally, I am glad to hear about the Royal College of General Practitioners' involvement with the Royal College of Psychiatrists in coming up with a collaborative framework. I welcome the Bailey *et al* article and the joint collaboration and would hope more joint work is carried out in the future between primary and secondary care teams.

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2 British Medical Association, NHS Employers. *Quality and Outcomes Framework for 2012/13: Guidance for PCOs and Practices*. NHS Employers, 2012.

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4 Gubbins A, Lally J, McDonald C. Metabolic syndrome in patients attending psychiatric day centres: prevalence and associations. *Psychiatrist* 2012; **36**: 326–31.

5 de Haan L, Hinszen HD, Lenior ME, de Win ED, Gorsira R. Duration of untreated psychosis and outcome of schizophrenia: delay in intensive psychosocial treatment versus delay in treatment with antipsychotic medication. *Schizophr Bull* 2003; **29**: 341–8.

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Authors' response: Dr Chaparala asks if we would have been better mentioning how duration of untreated psychosis affects long-term outcomes. Is not a 20-year mortality gap for men, and 15 years for women, a significant long-term outcome and an impact of untreated cardiometabolic risk deserving of some earlier intervention?

Notwithstanding incontrovertible evidence that antipsychotics cause problematic weight gain, we do not suggest antipsychotics are the sole explanation of increased cardiovascular disease, but do highlight how antecedent risks can become established in the critical early treatment phase. This is further supported by another recent systematic review observing cardiometabolic changes only after antipsychotic initiation.[1] The subsequent trajectory of weight gain, increasing metabolic disturbance and sustained heavy smoking provides a compelling link between schizophrenia and cardiovascular disease,[2] the single most important cause of premature death in this population.

Furthermore, the National Institute for Health and Clinical Excellence (NICE) are clear in their recommendations that these adverse cardiovascular risks should be identified at the earliest opportunity and managed using the appropriate NICE guidance for prevention of these conditions (the 2009 updated guidance for schizophrenia, CG82; recommendation 10.4.1.3). And yet when the recent Royal College of Psychiatrists' National Audit of Schizophrenia (NAS) examined the implementation of NICE recommendations in community settings (NAS report 2012; www.rcpsych.ac.uk/quality/NAS), it found that only 29% of people with schizophrenia across England and Wales had received an adequate assessment of cardiometabolic risk within the previous 12 months; 44% had not even been weighed.

Does this apparent lack of concern about adverse cardiometabolic consequences revealed by the NAS matter? After all, Dr Reed is reassured about antipsychotic safety by the FIN11 study of Tilhonen *et al*. However, authorities De Hert *et al*[3] have challenged this study's conclusions, listing methodological weaknesses which include

> 'incomplete reporting of data, questionable selection of drug groups and comparisons, important unmeasured risk factors, inadequate control for potentially confounding variables, exclusion of deaths occurring during hospitalization leading to exclusion of 64% of deaths on current antipsychotics from the analysis, and survivorship bias due to strong and systematic dierences in illness duration across the treatment groups.'

Dr Reed raises the issue of switching antipsychotics and how this may destabilise control of psychosis but may have missed the point of Weiden's editorial that he refers to. While indeed not advocating switching antipsychotics in someone established on treatment, Weiden highlights how two randomised studies demonstrated the positive value of switching antipsychotics to counteract rapid weight gain and metabolic change, concluding: 'Practice guidelines and public policy should recommend that clinicians consider the value of switching antipsychotics in patients with elevated metabolic risk.'[4]

Dr Chaparala suggests we are abandoning antipsychotics. No, but we are in good company in questioning the dominance of psychopharmacology.[5] Moreover, excessive reliance on antipsychotic treatment is suggested by the NAS finding of wide variation in the availability of psychological treatments across England and Wales: even in those patients whose response to antipsychotics had been unsatisfactory, 34% were not offered any form of psychological treatment despite NICE recommendations that these should be considered.

What we urge is responsible prescribing, particularly in the critical early phase of illness and sensitivity by us as doctors to how these young people may feel about the effects of our treatments. Perhaps the final word should go to the closing comment of Dr Tagore's letter: 'We must never be economical with the truth about the drugs we are all too happy to dish out.'

Declaration of interest

D.S. is current member of two Guideline Development Groups (GDG) for NICE: NICE guidance for children and young people affected by psychosis and schizophrenia, and NICE guidance for adults with psychosis and schizophrenia. The views expressed are not those of GDG, NCCMH or NICE. (The declaration applies to this letter and to the original article.)

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3 De Hert M, Correll CU, Cohen D. Do antipsychotic medications reduce or increase mortality in schizophrenia? A critical appraisal of the FIN-11 study. *Schizophr Res* 2010; **117**: 68–74.

4 Weiden PJ. Switching antipsychotic medications: not enough, too often, or just right? *Am J Psychiatry* 2011; **168**: 882–4.

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A case of clozapine-induced diabetic ketoacidosis

A 29-year-old male of Yemeni descent detained in a medium secure unit was commenced on clozapine; after 4 weeks of treatment he was taking a total of 275 mg in divided doses. He developed nausea and vomiting which progressed over 36 hours to a point where he needed to be urgently transferred to the local accident and emergency unit. At assessment he was experiencing breathing problems, vomiting and he was incontinent of urine; he had a Glasgow Coma Scale score of five. He was immediately transferred to the intensive care unit. The differential diagnoses included drug overdose, alcohol intoxication and clozapine-induced hyperglycaemia. His blood chemistry showed evidence of diabetic ketoacidosis; his blood glucose level was grossly elevated. The clozapine was stopped and the patient was given appropriate treatment with glycaemic agents.

In summary, the patient had become seriously unwell over a period of 36 hours. Apart from having a slightly raised body mass index, he was fit and well and had no family history of diabetes. His pre-treatment blood glucose had been normal.

Diabetic ketoacidosis is over ten times more common in patients treated with atypical antipsychotics than in the general population,[1] although the evidence is largely restricted to case reports and series.[2] Clozapine has a higher risk of ketoacidosis than other oral antipsychotics[3] and it tends to develop after a shorter duration of treatment, with a high proportion of patients developing it within 3–6 months. Low doses, being a young male and having a negative family history seem to be significant risk factors.[4] There is also significant mortality.[5] The unusual aspect of this case (although not unknown) was the occurrence of diabetic ketoacidosis during the titration phase of treatment.

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'Deaf-mute': time to abandon stigmatisation of the deaf community

I was dismayed to read Akintomide *et al*'s reference to the subject of their case review as a person who was 'profoundly deaf-mute'.[1]

'Deaf-mute' is an outdated term originating in the 18th/19th century. It carries very derogatory connotations, and is no longer used in reference to individuals with profound deafness. The term 'mute' implies a lack of ability to make noise. Such a label is technically inaccurate when applied to deaf individuals, since they generally have functioning vocal chords and therefore retain the ability to make vocalisations (http://wfdeaf.org). Those who are profoundly deaf from early life struggle to develop an oral language, given that hearing is required to facilitate a modulation of one's voice into speech. Many will therefore employ non-verbal communication in the form of sign language instead. This is a complex combination of hand signals, with its own regional dialects and international differences.

Over 75 000 people in Britain currently use British Sign Language (BSL) as their first or preferred language. The majority of these sign language users consider themselves as members of a distinct cultural community with a strong social identity.[2]

To this day the social image of deafness remains impaired on an international scale. This manifests itself in the form of a deeply rooted pathological stigma, negative stereotypes and prejudiced attitudes towards the deaf.[3] It would seem that such ignorance also persists among health professionals. Ralston *et al*[4] surveyed the attitudes of 165 physicians and identified a significant difference in attitudes towards hearing patients compared with deaf patients. Munoz-Baell & Ruiz[3] suggest that much of the stigma relating to the deaf community arises from an extensive social lack of appreciation of both their communication mechanisms and their culture. Unfortunately, in spite of more recent advances in healthcare legislation,[5] it would appear that there is still some way to go before members of the deaf community achieve the equality of health and social standing to which they are entitled.

The summary for Akintomide *et al*'s paper states that it is the first published case report of catatonia in someone who is profoundly deaf. It is a shame therefore that, rather than taking the opportunity to present a positive reflection of managing patients with profound deafness, the authors have merely succeeded in perpetuating existing negative stereotypes about this sector of the population.

Nb. Deaf is used in reference to those born deaf whose first language is BSL. It is used as a generic term, and for those with acquired deafness whose primary form of communication is oral.

1 Akintomide GS, Williams Porter S, Pierce A. Catatonia in a woman who is profoundly deaf-mute: case report. *Psychiatrist* 2012; **36**: 418–21.