the columns

correspondence

Liaison services – collaborative working

We read with interest Kewley & Bolton's survey on liaison psychiatry (*Psychiatric Bulletin*, July 2006, **30**, 260–263) and the related correspondence of Pitman & Catalán (*Psychiatric Bulletin*, January 2007, **31**, 33). In the wake of threats to close or merge liaison service with crisis resolution teams, it is imperative not to compromise patient care. The liaison psychiatry service in Birmingham Heartlands Hospital has developed a way of working to adhere to the time targets in accident and emergency (A & E) departments which neither compromises psychosocial assessment (National Institute for Clinical Excellence, 2004) nor overburdens the existing psychiatric services.

The protocol for psychiatric assessment is based on the SAD PERSONS scale (Juhnke, 1994) and has been devised in consultation with the A & E department. The A & E department is responsible for initiating the psychosocial assessment and classifying patients either as high or low priority, based on needs and risks. The majority of psychiatric patients attending A & E departments out of hours are needing assessment and treatment for self-harm. The patients who are deemed high priority are referred to the local crisis resolution teams for emergency assessment. Low-priority patients are referred after medical assessment to the psychiatry clinic in the A & E department on the next working day. This efficient collaboration reduces the number of 'did not wait' patients and possibly avoids breaching A & E waiting time targets.

In a 6-month period, 46% of psychiatric patients attending the A & E department out of hours have been referred to the clinic. If this way of collaborative working can be adapted to meet local hospital needs, it might address some of the concerns raised by Kewley & Bolton.

JUHNKE, G. (1994) SAD PERSONS Scale review. Measurement and evaluation. *Counselling and Development*, **27**, 325–327.

NATIONAL INSTITUTE FOR CLINICAL EXCELLENCE (2004) *Self-Harm. The Short-Term Physical and Psychological Management and Secondary Prevention of Self-Harm in Primary and Secondary Care*. NICE. http://www.nice.org.uk/pdf/CG016NICEguideline.pdf

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Clozapine-induced speech dysfluency: further cases

Lyall *et al* (*Psychiatric Bulletin*, January 2007, **31**, 16–18) presented two cases of clozapine-induced speech dysfluency and suggest that there are only four cases in the British and American literature. However, we do not think that their literature search was comprehensive. Begum (2005) reported stuttering, facial tics and myoclonic seizures, which developed a few days after initiation of clozapine for treatment-resistant schizophrenia. Furthermore, Bär *et al* (2004) examined the hospital records of about 6000 German patients receiving antipsychotic treatment over 3 years for evidence of stuttering as a possible side-effect. They described seven patients with stuttering induced by the atypical antipsychotics olanzapine (six cases), and clozapine (one case).

We also observed a man in his early 40s who developed stuttering when his clozapine was increased from 400 mg/day to 450 mg/day. This was also associated with a marked increase in seizure activity which necessitated reducing and stopping clozapine.

We suggest that future case reports in the *Psychiatric Bulletin* should describe a systematic search of standard databases for other case reports and the time period covered by such a search. This would be beneficial to the *Psychiatric Bulletin* and the wider readership.

BÄR, K. J., HÄGER, F. & SAUER, H. (2004) Olanzapine- and clozapine-induced stuttering: a case series. *Pharmacopsychiatry*, **37**, 131–134.

BEGUM, M. (2005) Clozapine-induced stuttering, facial tics and myoclonic seizures: a case report. *Australian and New Zealand Journal of Psychiatry*, **39**, 202.

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We read with interest the report by Lyall *et al* (*Psychiatric Bulletin*, January 2007, **31**, 16–18) of speech dysfluency associated with clozapine and would like to report our experience in a patient we are treating. Our patient is currently 44 and experienced his initial episode of psychosis when he was 23. Aged 27 he was diagnosed with schizophrenia and maintenance typical antipsychotic medication was prescribed, with initial good effect. However, he continued to have low-grade positive symptoms and the negative syndrome also became apparent. Over the subsequent 10 years he had many changes of medication with little positive effect. At the age of 38 he was commenced on clozapine and his positive symptoms rapidly receded. At a dose of 200 mg he developed a stutter (he had not had this problem as a child), but the dose was increased to 350 mg daily because of its overall positive effect. However, the stutter was so disabling that clozapine optimisation strategies were employed and the clozapine dose was gradually reduced. Owing to a lack of local speech therapy services our patient was referred to a neurologist, who confirmed our findings and supported our medication strategy. Amisulpride and low-dose benzodiazepines were added and the dose of clozapine was reduced. The stutter reduced with these changes and disappeared when the clozapine was stopped. His illness is currently well controlled and his current prescription is amisulpride 400 mg twice daily with clonazepam 0.5 mg twice daily.

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Medication side-effects – informing the MHRA

Lyall *et al* (*Psychiatric Bulletin*, January 2007, **31**, 16–18) described how two