remission in 10–30% of patients. In the controlled trial performed by Tamminga *et al*, patients that received clozapine not only had a significant improvement in their TD, when compared to both their own baseline and to those subjects receiving haloperidol, but maintained this improvement for the 12-month duration of the trial. Clozapine doses in all of these studies were similar to those typically used to treat psychosis.

In summary, there exist considerable data, from a wide variety of centres, and including double-blind evidence, all consistent with the hypothesis that clozapine is effective in the treatment of tardive dyskinesia. While clozapine's mechanism of action in TD may remain unclear (Young *et al*, 1997), its efficacy in this chronic, severe, and often disabling condition appears to be beyond doubt.

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Sir: Duncan and colleagues have produced an informative review of a complex subject (*Psychiatric Bulletin*, July 1997, **21**, 422–425). The article mentions tardive dystonia only briefly and, understandably, the discussion is weighted towards the management of classic tardive dyskinesia. We wish to comment on the role of botulinum toxin in this area. It is conceded that dystonia occurring as the sole or predominant abnormality of movement among patients with tardive dyskinesia is relatively uncommon. Yassa *et al* (1992) documented a prevalence ratio for tardive dystonia of 2% in a prospective study of elderly patients receiving neuroleptic drugs. In comparison orobuccal-lingual stereotypies or stereotypies elsewhere were present in approximately 30% of the same group. However, in the series of 100 consecutive patients with tardive dyskinesia examined by Stacy *et al* (1993) stereotypies were observed in 78 individuals and dystonic movements in 75. In only 22 of these cases was there dystonia in isolation and clearly in many cases the movements were coexistent. Furthermore both these authors and others have commented that distinction between dystonic movements and stereotypies may at times be very difficult.

Duncan and colleagues state that botulinum toxin is a useful therapy only in cases of tardive dystonia. This statement deserves qualification. First, there may be a dystonic component within a complex dyskinesia amenable to treatment with botulinum toxin. Second, our clinical experience in Middlesbrough and Newcastle would suggest that the stereotypies of classic tardive dyskinesia can also be ameliorated using botulinum toxin (P. D. and M. H.). Tardive dystonia is, however, more frequently a cause of morbidity than stereotypy and our experience of treating dystonia is greater. We would therefore encourage psychiatrists faced with intractable and symptomatic stereotypies, rarer tardive phenomena such as myoclonus and tremor or complex dyskinesias to consider referral to a botulinum toxin clinic even in the absence of a purely dystonic syndrome. Only with such referrals will the true value of botulinum toxin in the treatment of tardive dyskinesias became apparent.

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Tarasoff and the duty to warn third parties

Sir: We were interested to read the editorial by Turner & Kennedy (*Psychiatric Bulletin*, August 1997, **21**, 465–466). They suggest that psychiatric practice in the UK, has altered in recent years towards an increased likelihood of warning third parties, who may be at risk. They relate this to post-Tarasoff discussions and recent UK enquiries (Ritchie *et al*, 1994).

In 1995 we conducted a postal survey of local Glasgow psychiatrists in which we asked them to indicate their likely response to vignettes which posed issues of confidentiality. We received 53 responses (59% of total), 19 respondents were consultants (35.8%). The first vignette described a 17-year-old out-patient living with her parents and younger sisters who discloses sexual abuse by her father. The patient requests confidentiality. In response