accounted for. The excess of men, given that TD is commoner in women (Jeste & Caligiuri, 1993), and the somewhat high prevalence of trunk and limb TD among those aged 20–39 years, further suggest that the group studied were not 'typical' schizophrenic patients. Finally, it would be reassuring to know that steps were taken to exclude neurological co-morbidity.

Tardive dyskinesia is poorly defined, and similar movement disorders have been described in the pre-antipsychotic era, and in contemporary antipsychotic-naive schizophrenic patients (Owens *et al*, 1982). However, most authorities consider that antipsychotic exposure is necessary to allow a diagnosis of TD, and some diagnostic criteria (Schooler & Kane, 1982) require treatment for three months. By these criteria the 13 patients receiving no antipsychotics, and perhaps any patients receiving less than 100 mg chlorpromazine equivalents per day, should be excluded from the analysis. Antipsychotics suppress TD symptoms and, given the impressive maximum daily antipsychotic dosage of 4380 mg chlorpromazine equivalents, this may be a further confounding factor in the present study.

Finally, Liddle *et al*'s conclusions that "in the case of orofacial dyskinesia, the prevalence increased significantly with increasing age" and that "the prevalence of trunk and limb dyskinesia did not increase significantly with age" must be treated with caution. If their younger group of subjects had a more usual prevalence of trunk and limb TD, the latter conclusion would not have been reached, while the former conclusion must be tempered by the reservations already discussed. Furthermore, we are not provided with sex ratios for the age bands used; the apparent excess of TD in older patients may be because post-menopausal women were over-represented.

Only longitudinal studies which pay attention to diagnosis, sex, organic illness, hospitalisation, and type (i.e. typical or atypical), dosage and duration of treatment with antipsychotic drugs, will unravel the links, if any, between schizophrenia, its symptoms and treatments, and TD.

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SIR: Poverty of speech and flat affect will not differentiate between primary negative symptoms of the disease process and those secondary to drug-induced Parkinsonism (DIP), a potential confounding variable (Brown & White, 1991). Perhaps this is not important, since the hypothesis Liddle *et al* tested did not concern the association between negative symptoms and TD *per se*, but rather that the presence of negative symptoms brought forward the onset of TD. However, if secondary negative symptoms had a substantive confounding effect, then the differences between patients with and without negative symptoms may have been most evident among the older patients, in whom DIP is more common (Ayd, 1961).

In the three age groups (20–39 years, 40–59 years, and 60–89 years) (Table 2) there is said to be an increasing proportion with negative symptoms (56%, 64% and 69%, respectively). This increase is most evident in the group with orofacial dyskinesia, 60% of those less than 40 years old and 73% of those older than 40 years having negative symptoms. Respective figures for patients without orofacial dyskinesia are 55% and 52%. An alternative hypothesis is that orofacial dyskinesia may bring forward the onset of negative symptoms, and this is supported (although less convincingly). Thus, in all three age groups the proportion of the TD group with negative symptoms (60%, 76%, and 71%, respectively) was greater than in the non-TD group (55%, 50%, and 58%, respectively). This excess was most marked in the middle age group, reaching statistical significance ($\chi^2=5.4$, d.f.$=1$, $P<0.05$). In view of the importance of antipsychotic medication in the development of TD, it could be argued that these medications may also facilitate the development of the type II syndrome. This would not be surprising if similar processes were responsible for both TD and negative symptoms. Indeed, it has been suggested that such common pathology need not act on anatomically distinct sites (Brown & White, 1992). Although we are familiar with the many side-effects of dopamine-blocking drugs, including secondary negative symptoms, the notion that they may interact with the disease process and age to bring forward the onset of substantive negative symptoms must be cause for concern.

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AUTHORS' REPLY: Wright & Taylor question our use of the term 'tardive dyskinesia' on the grounds that a small minority of cases were not receiving antipsychotic medication at the time of assessment. In fact, all patients had received antipsychotic medication at some time. In the text of the paper we referred explicitly to either orofacial dyskinesia or to trunk and limb dyskinesia to minimise ambiguity, but nonetheless consider that our use of 'tardive dyskinesia' in the title is reasonable. Wright & Taylor also suggest that our conclusion is unwarranted because our patients were atypical. As we reported, our patients were either undergoing rehabilitation or were long-stay patients, and hence represent a seriously disabled group. The negative symptoms of our patients might reasonably be described as symptoms of the defect state. Our study supports the hypothesis that these persistent negative symptoms are associated with earlier onset of orofacial dyskinesia. The study was not designed to determine whether other negative symptoms, such as the transient negative symptoms that sometimes accompany acute exacerbations of illness, are associated with vulnerability to dyskinesia.

With regard to the issue of institutionalisation, there was no significant difference in the duration of the current hospital admission between those with and those without orofacial dyskinesia, within each age band. With regard to sex differences, orofacial dyskinesia increased with age within both sexes.

Wright & Taylor imply that had we recruited a sample more representative of young schizophrenic patients in general, we might have concluded that trunk and limb dyskinesia increases with age. If we had done so, we would have been in danger of drawing a spurious conclusion, because it is virtually inevitable that elderly patients, with whom the young are compared, will have suffered sustained illness. For the purpose of our study, it was desirable to recruit in a setting that minimised the risk that young patients would represent a less severely ill group. Our finding that the prevalence of trunk and limb dyskinesia was independent of age is not only consistent with the other studies we reviewed in our paper, but also increases our confidence that the observed increase in prevalence of orofacial dyskinesia with age cannot simply be attributed to a selection bias, such that the younger patients had intrinsically less persistent illness associated with a lesser amount of non-specific neurological dysfunction. Clearly, it would be preferable to study patients longitudinally, but bias due to loss of contact with recovered cases during a five-decade study might still be a problem.

Simon Taylor raises the question of whether or not drug-induced Parkinsonism might have influenced our results. In a subsample of 105 cases in whom Parkinsonism was assessed, tremor and limb tone were not significantly worse in patients with orofacial dyskinesia than in those without, and hence it is unlikely that a global Parkinsonian syndrome affecting limb and facial muscles confounded our results. However, it is not possible to exclude influence from drug-induced diminution of facial expression. It is possible that the diminution of facial expression due to drugs and that intrinsic to the illness both reflect dopaminergic underactivity. We agree that it is also possible that dopamine-blocking drugs might contribute to negative symptoms in schizophrenic patients, although the evidence for the existence of negative symptoms before the development of antipsychotic drugs implies that factors intrinsic to schizophrenia play a substantial role.

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Low serum cholesterol and suicide attempts

SIR: We read with interest the article (*BJP*, June 1993, **162**, 818–825) commenting on the association between low serum cholesterol and suicide attempts among psychiatric patients. Although findings have been inconsistent (Pekkanen *et al*, 1989; Davey Smith *et al*, 1990), a meta-analysis (Muldoon *et al*, 1990) and recent cohort studies (Lindberg *et al*, 1992; Schuit *et al*, 1993) have found an increase in deaths from external causes, including suicide, to be associated with low serum cholesterol. However, Goble & Worcester (1992) have argued that this observation could be due to a confounder – depressed patients (at risk of suicide) with decreased