not simply to suggest a connection between lithium and remission of psoriasis, but to invite comment on this interesting case with its many paradoxical aspects.

Chaput *et al* (1985) showed psoriasis to be more common among a sample drinking more than an average of 50 g alcohol a day compared with those consuming less than that amount. They point out that this is not synonymous with a causative relation – high alcohol intake may precede psoriasis or psoriasis may enhance the onset of alcoholism. In the three cases reported by Vincenti & Blunden (1987) there was rapid remission of psoriasis during detoxification intimately related to reduction in alcohol intake. In the case we reported, following initial referral the patient became abstinent with only minor early relapses, and has remained so since. It was not until two years later that lithium treatment was started and improvement in his psoriasis was noted. Prior to this his psoriasis had remained active, and had been present for many years before his drink problem. In this case, reduction in alcohol consumption did not relate to improvement in psoriasis.

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General Practice Patients on Long-term Psychotropic Drugs

SIR: I would like to make several points about the study by Catalan *et al* (*Journal*, March 1988, **152**, 399–405). Firstly, the authors did not explicitly blame the GPs for the poor prescribing practices described but that was the general impression that I gained from the paper. This impression may or may not be valid. As their data was obtained from FP10 prescriptions, I wonder how many of these prescriptions were initiated, maintained, or advised by psychiatrists.

The authors state that 24% of index patients had received psychiatric out-patient treatment, and 10% had received in-patient treatment, at any time before the 12-month period of the study. Even assuming no overlap of these two groups, we would be left with 24% of the patients on long-term psychotropic medication who had been seen by a psychiatrist in the past. It may have been pertinent to ascertain how many of the index group had been assessed by a psychiatrist in the recent past, for example in the 12 months before the study period. It would then be interesting to clarify the contribution (if any) the psychiatrists had made to reviewing these prescriptions.

Secondly, the authors suggest that practices which have links with visiting psychiatrists could review patients on long-term psychotropic medication. The number of patients (318 out of a practice of 8842) would represent a major undertaking for a psychiatrist working in a 'traditional' hospital-based service. This study I feel highlights the need for psychiatrists to work at least partly in primary health care settings as described by Mitchell (1985).

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MITCHELL, A. R. K. (1985) Psychiatrists in primary health care settings. *British Journal of Psychiatry*, **147**, 371–379.

SIR: As Dr Donnelly points out, we did not explicitly blame the GPs for the prolonged prescribing of psychotropic drugs. We would like to stress that we certainly had no intention of blaming them implicitly. When the patients in our study were started and maintained on their drugs, the climate of opinion among doctors and people in general was in favour of such prescribing. Recently, this climate of opinion has changed with the increasing concern about the efficacy and side-effects of many psychotropic drugs prescribed in general practice.

Our findings suggested that psychiatrists had played a small part in initiating prescribing for the patients: 3% of those on anxiolytics, 5% of those on antidepressants, and 9% of those on major tranquillisers. The GP had initiated prescribing for most of the patients: 50% of those on anxiolytics, 60% of those on antidepressants, and 42% of those on major tranquillisers. Among the remaining patients, prescribing had been initiated by a hospital physician or the patient's former general practitioner. Although nearly a quarter of the patients in our study had a history of past psychiatric consultation, no patient had consulted a psychiatrist in the 12 months before the study period.

As mentioned in our paper, we agree with Dr Donnelly about the need for members of the mental health team to work closely with the GP in the management of such patients. However, this collaboration can take various forms, which have yet to be evaluated.

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P3 and CT Scan in Patients with Chronic Schizophrenia

SIR: We read with interest the comments of Ebmeier *et al* (*Journal*, February 1988, **152**, 290–291). It is suggested that the increase in P3 latency reported in some studies of schizophrenic patients may be related to treatment with anticholinergics. In our study (Barrett *et al*, *Journal*, March 1986, **148**, 414–420) we did not find a difference in P3 latency between schizophrenics and normals, but employed a four-way tone discrimination paradigm with a longer inter-stimulus interval than is usual (1.8 s). We compared patients on anticholinergics with a group on neuroleptics alone and found no significant difference in P3 latency. However, N1 latency was significantly longer in the anticholinergic group.

We would agree that it is a selected group of patients who agree and are able to participate in this type of psychophysiological study. The more emotionally blunted or thought disordered patient would not, in our experience, be able, willing, or interested in performing the tasks involved. Hostile and suspicious patients steer clear. Our group was predominantly 'paranoid' (on RDC subtyping; $n = 16/20$). The small non-paranoid group did in fact have significantly longer P3 latency than the paranoid group (P3 latency at PZ in ERPs to infrequent 'target' stimuli: paranoid group $= 353 \pm 41$; non-paranoid group $= 379 \pm 41$). It would be interesting to know if P3 latency in schizophrenics bears any relationship to the cerebral atrophy in this group.

Finally, there is good evidence that the cognitive variables that influence the various ERP components do not do so by raising or lowering a single 'wave', but have rather more prolonged effects which overlie one or more components. We found that a mean amplitude measure from 276 to 426 ms was more discriminating between normals and schizophrenics than measures of amplitude or latency. These differences were more marked in ERPs to frequent 'non-target' stimuli than in ERPs to infrequent 'target' stimuli. Subsequent work has indicated that prominent late positivity to non-targets only occurs where longer interstimulus intervals and more complex tasks are involved.

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Unilateral auditory hallucinations

SIR: Khan *et al* (*Journal*, February 1988, **152**, 297–298) report unilateral auditory hallucinations arising from left otitis media in a chronic schizophrenic patient. They did not locate any of the many similar reports or my extensive but not exhaustive review (Gordon, 1987). I will list some of its conclusions in the light of more papers I have since unearthed.

(a) Hallucinations in various sense modalities can arise from the ear or labyrinth (Ireland, 1893), although only auditory ones will be considered here.

(b) Otopathic hallucinations arise more from otitis media (often serous) than from cochlear deafness. Robinson (1927) found middle ear deafness in 61% of hallucinating mental patients, compared with 21% of the non-hallucinating, whereas the inner ear figures were 22% and 23%.

(c) Ear disease is very common in the insane. Robinson found normal ears and hearing in only 14% of the hallucinating and 56% of the non-hallucinating patients, and that was without audiometry or tympanometry. At the turn of the century Fraser found chronic otitis in 5 out of 22 hallucinating patients (Henderson *et al*, 1913), but abandoned further attempts to establish a pathological basis for tinnitus since they were untestable.

(d) The question of lateral bias is intriguing. In this Journal in 1901 Robertson reported a marked sinistral bias (Gordon, 1987), but Robinson (1927) found a dextral preponderance of ear disease (10 right, 2 left).

(e) Tinnitus is probably a necessary condition for production of hallucinations. Over half with definite hallucinations complained of tinnitus, and most of the rest had ear conditions favouring its occurrence (Robinson, 1927).

(f) The crucial question is whether tinnitus is also a sufficient condition. Robinson thought not. However, Bjeljakow (Ireland, 1893) thought ear infections could lead to insanity and even secondary dementia (i.e. schizophrenia). Peripheral irritation