cent) subjects who had no recorded history of antipsychotic medication, abnormal movements were present in 120 (31.7 per cent). The Chi square test (Kirkwood, 1981), indicated that the difference in prevalence of abnormal movements between subjects who had received antipsychotics and those who had never been exposed to these compounds, was statistically significant ($P < 0.001$), thus confirming the association of dyskinetic movements with antipsychotic medication.

The presence of spontaneous dyskinesia in 120 (31.7 per cent) of the 378 subjects who had never received antipsychotic medication confirms an earlier study in which 38 (18 per cent) of 211 residents who had never been treated with an antipsychotic drug showed dyskinetic movements (Bourgeois *et al*, 1980).

The Task Force of the American Psychiatric Association (1980) reporting on late neurological effects of antipsychotic drugs suggested that the ageing brain may have an increased likelihood of antipsychotic related dyskinesias, especially of the oral region, and also drew attention to the fact that in the elderly, studies had shown that the prevalence of spontaneous buccolinguomasticatory movement abnormalities, is close to that found in antipsychotic treated geriatric patients.

Our own study has shown that in a group of elderly subjects, age range 59–102 years (mean 82.7 years) there is a considerable prevalence of spontaneous dyskinesias, and that antipsychotic drugs do seem to increase the risk of developing dyskinesias during old age.

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HYPOALGESIA IN DEPRESSIVE ILLNESS

DEAR SIR,

In his comment on Ben-Tovim and Schwartz's paper (*Journal*, January 1981, **138**, 37–9) Professor Whitlock makes a didactic statement that "tricyclic and other types of antidepressant medication have pronounced analgesic properties" (*Journal*, May 1981, **138**, 437–8). Without qualification this statement could be quite misleading, as is evident from a careful look at the papers to which he makes reference.

Ward and his colleagues (1979) describe a study in patients who were depressed and who were also found to have chronic pain complaints. Turkington (1980) studied patients with leg pain secondary to diabetic neuropathy who were all found to have 'substantial degrees of depression'. In both studies treatment with tricyclic antidepressants produced improvement in both the pain symptoms and the depression. The obvious implication of these reports is that when chronic pain symptoms are associated with pathological depression, antidepressants are effective in relieving the pain at the same time as the depression. Indeed this is the conclusion reached by the authors: they do not suggest that antidepressants are 'analgesic'.

Antidepressants are now widely used in patients with chronic pain though it is not clear whether they have a specific therapeutic effect in such patients, or if they do, how they are working. When benefit is obtained there are three likely explanations: that it is purely secondary to their antidepressant activity; that it represents some sort of intrinsic 'analgesic' action as suggested by Whitlock; or that it is a reflection of their sedative effects whereby they modify the central perception of pain at the cortical level, or the psychological reaction to painful stimuli (Hanks, 1981). There is insufficient evidence at present to be confident which pharmacodynamic effect is most important.

In animal pharmacological models neither the tricyclics (Spencer, 1976) nor the more recent drugs such as mianserin, nomifensine or trazodone, exhibit any analgesic effects. A difficulty here is that these models are only predictive for acute pain and are generally not sufficiently sensitive to identify non-narcotic analgesics. No-one has suggested that antidepressants are effective in the treatment of acute pain, but their possible effects in chronic pain cannot be

investigated in animal models. In man the difficulties of extrapolating from experimental pain in the laboratory to chronic pain patients is succinctly put in the critique (Loeser, 1979) of an investigation of doxepin in acute pain (Chapman and Butler, 1978). It is therefore necessary to look at clinical data.

In assessing reports of antidepressants in chronic pain it is clearly important to have information on the mood state of the patients because the presence of significant depression will influence the response of the pain symptoms to this treatment. It is partly because insufficient attention has been paid to the presence or absence of depression in patients with chronic pain that so many questions still surround the use of antidepressant drugs in such patients.

The report by Ben-Tovim and Schwartz illustrates very clearly that pain has both an affective and a cognitive component, and may be greatly influenced by the general level of emotional response. The hypoalgesia in their patients was to acute painful stimuli and one would be wary of speculating on the relevance of these findings to patients with chronic pain. Since acute and chronic pain are such different clinical entities these results are not necessarily incompatible with the common association of chronic pain and depression.

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OESTROGENS, DOPAMINE AND MOOD

DEAR SIR,

The letter of Ms Skutsch (July 1981, **139**, 80) has interestingly drawn attention to evidence from animal experiments that oestrogens interact with neural mechanisms that mediate the effects of dopamine. The reference she gives (Euvraard *et al*, 1980) documents such interactions, but does not support her contention that oestrogens act on dopaminergic neurones *per se*, or alter the turnover of dopamine. Moreover the example she gives—the rise in plasma prolactin caused by oestrogens—is thought not to be mediated by a reduction in output of dopamine but by changes in the lactotroph cells. Thus Piercy and Shin (1980) showed in the rat that while oestrogens can increase the capacity of the pituitary to synthesise and secrete prolactin by a factor of ten, the relative role of dopamine is not changed. Also Dufy *et al* (1979) showed that oestrogen causes a change in the electrical properties of prolactin-secreting cells, increasing the frequency of calcium-dependent action potentials, and perhaps also changes the receptors. The evidence from Euvraard *et al* is also that oestrogen affects the post-synaptic (cholinergic) cells in the striatum, and not dopamine turnover. Thus in both the striatum and the pituitary the recognized effects of oestrogen are on cells that are normally inhibited by dopamine.

The increase in prolactin secretion caused by oestrogen might be expected to increase rather than reduce dopamine turnover in the hypothalamus (Eikenburg *et al*, 1977).

Finally, in a neurochemical hypothesis of mental illness might it not be more physiological to refer to activity in specified neurotransmitter-pathways rather than to levels of the putative transmitter? The available evidence is compatible with the hypothesis that a dopaminergic pathway is overactive in mania (Silverstone, 1979; Post *et al*, 1980).

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