

HONE, M. P., LOWE, M. R. & BATCHELOR, D. H. (1985) Reported neurotoxicity with the lithium/haloperidol combination – A literature review. World Psychiatric Association Regional Symposium, Athens, Greece. *Abstracts* – p 108.

JEFFERSON, J. W. & GREIST, J. H. (1980) Haloperidol and lithium: Their combined use and the issue of their compatibility. In *Haloperidol Update: 1958–1980* 7, 73–82 (ed. F. J. Ayd) Baltimore: Ayd Medical Communications.

#### Anticholinergic Anti-Parkinson Medication for Neuroleptic-induced Extrapyramidal Side Effects

Sir: We have recently received a piece of promotional literature entitled *Disipal in the Neuroleptic Syndrome*. The content of this document conveys a general impression that orphenadrine is a safe drug to use for the treatment of drug-induced extrapyramidal symptoms both in acute situations and for long term maintenance. We find the document biased and against all current scientific opinion on the subject and would like to counter some of the arguments presented in it.

Extrapyramidal side-effects frequently accompany neuroleptic treatment. In fact, anticholinergic agents used to reduce these symptoms are the commonest form of concurrent medication with neuroleptic therapy. There is, however, much debate about the adverse effects of combined therapy with these agents. It has been claimed that their use can result in exacerbation of psychosis, a delay in symptom improvement in patients with acute schizophrenia, and a predisposition to the development and/or deterioration of tardive dyskinesia. Many studies have reported that co-administration of anticholinergic drugs resulted in lower blood levels of neuroleptics. In others, although no differences were observed, combined therapy did have clinical implications (Bamrah *et al*, 1986). Clinically stable patients on neuroleptics do not normally show accompanying extrapyramidal symptoms except when dosages are increased or if extraneous factors lead to unpredictable increases in blood levels. As clinicians, therefore, we do not see the need for long-term medication especially if this is going to be associated with other interactions.

The document alleges that orphenadrine withdrawal may precipitate “a pronounced depressive state” in patients on combined haloperidol-orphenadrine therapy (Altamura *et al*, 1983). Whereas the relationship of depression with haloperidol is controversial, it is not unreasonable to assume that stopping a “mood elevating” drug could cause a rebound precipitation of dysphoric mood. One does not have to invoke “pharmacological interaction between orphenadrine and haloperidol leading to increased bioavailability of the latter” to explain this phenomenon. It seems to us irresponsible to elevate their “mood lightening” properties to

a position of clinical relevance since these are the very properties which may give them their drug-dependence potential.

These drugs have a powerful anticholinergic action which can summate with the anticholinergic action of neuroleptics to produce severe constipation or ileus, urinary hesitancy or retention, and intolerable dry mouth and blurring of vision (Lader, 1980). Besides, anticholinergics are themselves known to produce toxic psychoses (Shader & Greenblatt, 1971). Therefore, their use should be tempered with caution. Finally, if prescribed with abandon they would only provide the depressed patient with another means of self extermination.

We accept that the anticholinergic anti-Parkinsonian agents are valuable in clinical settings where it is necessary to relieve acute extrapyramidal symptoms following neuroleptic therapy, but we do not feel that the majority of patients require this treatment for more than three to four months. A literature review suggests that only a third of patients on maintenance treatment have any real need for this additional medication to control extrapyramidal symptoms, with the majority of surveys suggesting a substantially smaller proportion (Johnson, 1985).

SOM D. SONI

*Prestwich Hospital,  
Manchester M25 8BL*

D. A. W. JOHNSON

*Withington Hospital,  
Manchester*

#### References

- ALTAMURA, A. C. *et al*, (1983) Italian Society of Biological Psychiatry. In *Disipal in the Neuroleptic Syndrome*, West Byfleet, Surrey: Brocades (Great Britain).
- BAMRAH, J. S., KUMAR, V., KRASKA, J. & SONI, S. D. (1986) Interaction between procyclidine and neuroleptic drugs: some pharmacological and clinical aspects. *British Journal of Psychiatry* (in press).
- JOHNSON, D. A. W. (1985) Antipsychotic medication: Clinical guidelines for maintenance therapy. *Journal of Clinical Psychiatry* 5, 6–15.
- LADER, M. (1980) *Introduction to Psychopharmacology*. A ‘Scope’ Publication. Kalamazoo, Michigan: Upjohn Company.
- SHADER, R. I. GREENBLATT, D. J. (1971) Uses and toxicity of belladonna alkaloids and synthetic anticholinergics. *Seminars in Psychiatry* 3, 449–476.

#### Lithium Induced Hypothyroidism Presenting with Carpal Tunnel Syndrome

Sir: We describe a patient presenting with a recognised but unusual symptom of hypothyroidism which to our knowledge has not previously been reported in association with lithium.

*Case Report:* The patient was a 43 year old Kenyan Asian woman with a long-standing history of manic-depressive psychosis who had been treated with lithium for 10 years.

At the out-patient clinic she gave a three month history of paraesthesiae in the hands, the right worse than the left, which woke her at night and were relieved by hanging her arms over the edge of the bed. There were no other symptoms of thyroid disorder. On physical examination pinprick and light touch were reduced over the median nerve distribution. There was a slight delay in ankle jerk relaxation and a smooth thyroid was easily palpable. Laboratory tests of thyroid function (which had been normal eighteen months previously) revealed total T4-12 nmol/L (NR 50-150), free T4-1 pmol/L (NR 10-28), TSH < 50 mU/l (NR 0-7 mU/l). Thyroid antibody screen was negative. Thyroxine replacement was instituted (100 ug daily) and after two to three months of treatment the symptoms of carpal tunnel syndrome resolved completely. Lithium was continued throughout.

Carpal tunnel syndrome may result from lithium therapy because of fluid retention caused by the drug. In this woman's case, however, the primary

cause must have been hypothyroidism because the symptoms resolved with thyroxine while lithium was continued. Several points are raised by this case. First, lithium induced hypothyroidism may present in unusual ways without typical symptoms such as lethargy, weight gain, voice change and cold intolerance. Secondly, hypothyroidism may present late - in this instance after 10 years therapy during which thyroid function had been normal on several occasions. Thirdly, the need for regular monitoring of thyroid function in patients taking lithium is clear (although a symptomatic presentation may still occur).

KENNETH A. WOOD  
ROBIN J. JACOBY

*The Bethlem Royal Hospital,  
Monks Orchard Road,  
Beckenham BR3 3BX*

## A HUNDRED YEARS AGO

### Asylum Reports

"If such is the case, and we are bound to believe the statement, we do not hesitate to say that Holloway's is a great Charity wasted, for Mr Holloway's original intention or plan was to provide for private patients just above the pauper class - *none* over 21s. a week. *Now* the class he intended to benefit is expressly excluded. Endless structural alterations are or have been required, and a great charity is nullified. 'But yet

the pity of it, Iago!' What unspeakable good *might* have been done with that money on the lines of the first intention! We are almost tempted to say that the taste of the pill though silvered is scarcely disguised, and that there are not a few 'dead flies which cause the ointment of the worthy donor to send forth a stinking savour'. We do not refer to the imperfect drainage only."

### Reference

Journal of Mental Science (1887) 33, 319.

*Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey.*