

SUICIDE AND CANCER

DEAR SIR,

Dr Barraclough (*Journal*, September 1978, 133, 287), appears unduly concerned lest my paper (*Journal*, March 1978, 132, 269-74) be quoted in support of conclusions with which he disagrees. I can only hope that the following answers to his criticisms will help to allay his anxieties.

I do not agree with him that road accident victims are unrepresentative of the general population. In Australia the number of motor vehicles per head of population is one and a half times that in Britain. Thus most of the adult population will own or have access to a vehicle. Bearing in mind that only about half the cases of malignancy had been diagnosed before death and that most of the cancer cases and suicide victims were ambulant up to the time of their deaths, the argument that they would have been in bed or in hospital at the time lacks supporting evidence. In any case, many of the accident victims were passengers or pedestrians and not drivers. Dr Barraclough's criticism comes a little strangely, as in his own paper on suicide and old age: (In *Recent Developments in Psychogeriatrics*, Ed. Kay and Walk, 1971) he used precisely the same control group with which to compare his suicide cases; a procedure which apparently did not incur his censure at the time.

There is no reason for assuming that the victims of suicide were examined more meticulously than the road death cases by my colleagues, the Government pathologists. When these autopsies were carried out the question of the data being used by me some years later simply had not arisen. I can assure Dr Barraclough that these post-mortem records are of the highest quality, a standard which has been maintained for many decades. Hence, if tumours of any kind had been present this would have been recorded, regardless of the cause of death.

While I agree that malignant disease rates are third or fourth in the list of causes of death of persons aged less than 50, the actual rate is very low. It quadruples between the ages of 45 and 55 and continues to rise steeply thereafter. The sole reason for taking 50 as the lower limit of the age-range was to avoid scrutinizing a large number of autopsy records in which the likelihood of finding cancer would be very small. In any case, my setting an age limit does not, *per se*, invalidate the conclusions reached for the age group I investigated.

Dr Barraclough implies that the controls and cases were not matched for age. If he reads my paper again he will find that they were matched for age and sex. It hardly seemed necessary to spell this out in

detail, as I had assumed that most readers would take the statement to mean what it said. In fact, for each case a control was matched exactly or within three years of the age of the case. The age-ranges and standard deviations of the two groups did not differ.

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SWEAT LITHIUM IN MANIC-DEPRESSION

DEAR SIR,

There is surprisingly little recorded on sweat gland secretion of lithium in manic-depression (Amdisen, 1977). If lithium is excreted in sweat, prolonged loss by this route could theoretically assume sizeable proportions under certain conditions, e.g. in the summer or in the tropics. We therefore measured the concentration of lithium in pilocarpine-stimulated sweat in thirteen consenting and compliant manic-depressive patients who had been on daily doses of 750-1500 (mean 1000) mg of lithium carbonate for periods of from 10 to 63 (mean 33) months. There was no biochemical evidence of renal disturbance in these patients whose renal clearance of lithium (mean 20.6 ml/min/1.73M², SD 5.7) fell within reported values (Thomsen, 1978).

The patients omitted the morning's dose of lithium and attended the clinic at 0830 hours, when blood was collected for a baseline serum lithium concentration. Lithium carbonate 500 mg (= 7.46 mmol) was given orally, and blood was collected for further serum lithium concentrations at ½, 2, 4, and 6 hours. Forearm sweating was promoted by prostigmine iontophoresis (Landauer, 1963) and sweat was collected for one hour between the 4th and 5th hours of the study. The sweat was collected on to a pre-weighed gauze swab held tightly to the forearm by a square of plastic and non-porous adhesive dressing. The damp swab was quickly transferred to a sealed plastic container and weighed; the increase in weight represented the amount of sweat. The lithium in the sweat was eluted from the swab in 10.0 ml of potassium diluent solution made up in de-ionized water. The lithium concentration was measured with an Instrumentation Laboratory (IL) Lexington, Mass., U.S.A., model No. 343 flame photometer and the sweat lithium concentration calculated.

Lithium was found to be secreted in concentrations of 0.96-4.78 (mean 2.08, SD 0.93) mmol/l in the pilocarpine-stimulated forearm sweat of these thirteen controlled manic-depressives. These sweat lithium concentrations were 1.2-4.6 (mean 2.3)

times the corresponding average serum lithium concentrations (measured from the appropriate area under the blood concentration versus time curve). They were thus very similar to previously described concentrations of lithium in mixed saliva, which were 1.6–4.5 times those of plasma (Groth, Prellwitz, and Jähnchen, 1974).

Allowing for the very marked effects of acclimatization and for the fact that the lithium concentration of heat-stimulated sweat may differ from that of pilocarpine-stimulated sweat, we would still suggest that prolonged sweat losses of lithium should be taken into consideration, especially if a manic-depressive patient previously well controlled on lithium goes out of control in hot weather. Whether drugs which promote sweat secretion, e.g. phenothiazines, if given concomitantly with lithium therapy could lead to increased loss of lithium in sweat may be another point worth considering.

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References

- AMDISEN, A. (1977) Serum level monitoring and clinical pharmacokinetics of lithium. *Clinical Pharmacokinetics*, **2**, 73–92.
- GROTH, U., PRELLWITZ, W. & JÄHNCHEN, E. (1974) Estimation of pharmacokinetic parameters of lithium from saliva and urine. *Clinical Pharmacology and Therapeutics*, **16**, 490–8.
- LANDAUER, K. S. (1963) *Guide to Diagnosis and Management of Cystic Fibrosis*, p. 72. National Cystic Fibrosis Research Foundation, New York.
- THOMSEN, K. (1978) Renal handling of lithium at non-toxic and toxic serum lithium levels. A review. *Danish Medical Bulletin*, **25**, 106–15.

LITHIUM AND MEMORY LOSS

DEAR SIR,

Memory loss as a complication of lithium therapy has not received extensive attention. Recently, Kusumo and Vaughan (*Journal*, November 1977, **131**, 453–7) discussed 'Effects of lithium salts on memory' and concluded that 'there was some indication that patients on lithium may show an impairment of short-term memory at fifteen-second delay intervals . . .'.

Recently G. F. Bajor and D. Preodor *et al* noted memory loss in lithium maintenance therapy, 'an inability to recall details that interfere with daily functioning'.

We have recently seen a patient with a severe affective disorder whose mood changes were well controlled with lithium but who developed a severe memory defect within a few days of starting lithium.

A 22-year-old woman was admitted in a profound psychotic depression with paranoid features. Despite management with haloperidol 20 mgm daily and imipramine 200 mgm daily, she became so profoundly suicidal that ECT was instituted in combination with imipramine. After the sixth treatment she suddenly became hypomanic. Imipramine was stopped. Four days later she was again psychotically depressed and suicidal. With one ECT she promptly became hypomanic. Lithium carbonate (900 mgm daily) was started, with consequent rapid remission of hypomania and stabilization of mood. She began to complain, however, of a severe memory disturbance. She was correctly orientated, and showed no impairment of reasoning, thought process, calculating ability or long term memory. Although she had had a typical mild retrograde amnesia after ECT, she complained that this memory impairment was different, inhibiting normal routines of life.

On close mental examination it was evident that, while both immediate and long-term recall were intact, she was unable to process new information. A delay of only a few minutes left her incapable of recalling digits, sentences, objects or daily routines.

She was so distressed that the lithium was discontinued, and there was immediate clearing of memory functions as shown by digit retention, the Babcock sentence, and recall of daily routine. Unfortunately she relapsed almost at once into psychotic depression. Because of the severity of her illness we concluded that the inconvenience of memory loss was less threatening than the suicidal-hypomanic alternations in her mood. Accordingly, lithium carbonate was cautiously re-introduced.

As her blood levels approached a therapeutic level (0.6 mEq/l) she again became anxious as her memory deteriorated. Diazepam (8 mg daily) controlled anxiety sufficiently so that despite mild memory dysfunction she was able to engage in normal social routines.

In this case memory impairment with lithium therapy was sufficiently disabling to pose a hazard to the effective management of the disorder.

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