and attempted suicide is not supported by the evidence. Studies which claim to have shown an association between depression and early 'deprivation' (Brown, 1961, Dennehy, 1966, Hill and Price, 1967) have all been studies of early bereavement. There are no studies which claim a relationship between early bereavement and personality disorder or delinquency, though there are a number which show other early deprivation experiences to be related to such conditions. Similarly, of the many studies of attempted and successful suicide only those of Greer (1966) and Greer *et al.* (1966) show a relationship between early bereavement and suicide attempt. It is more likely that the relationship between suicide and parent death is secondary to that between depression and parent death. In a further study (1970c) I have shown that a very much higher proportion of a group of attempted suicides was severely depressed than of a matched control group, though the proportion of moderately depressed was similar in the two groups.

It is unreasonable to criticize Hill and Price, and Gay and Tonge, for not making comparison between depressed patients and the general population. The only value of general population comparisons is to ascertain whether early deprivation is commoner in the mentally ill than in healthy controls and a sample of mixed diagnoses is preferable for this purpose. Studies such as those of Brown and Munro, which have compared depressives with the general population, reveal nothing about depression as such, but show only a relationship between bereavement and mental illness in general. Information about the relationship between deprivation and specific clinical syndromes can only be obtained by comparing clinical groups, approximately matched, one with another. Which leads to a final criticism of Munro and Griffiths: in none of their comparisons is there matching for age. There is a definite relationship between early parent death and age. Diagnostic groups differ in age-distribution, as do in-patients and out-patients. In fact, the just significant excess of early bereavement in in-patient and out-patient depressives may well be due to the fact that the in-patients were older, though it is more probably because they were more severely depressed and contained more psychotics.

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DEAR SIR,

Dr. Birtchnell has started so many hares in his letter that it would be impossible to answer him adequately without writing another article. I have spent time wrestling with the subtleties of his thought, but I confess that I am still not sure that I have mastered fully the import of a number of his statements.

To take first the question of the type of deprivation experience: Dr. Griffiths and I deliberately failed to distinguish between parental death and parental loss from other causes in our review of the literature, since we know of no good evidence that death of a parent is of greater or different significance in predisposing to psychiatric illness than other types of parental deprivation. Death has a mystic significance to many people and it is a readily measurable phenomenon, but, as I point out elsewhere (Munro 1969), its quality as a deprivation experience differs according to the circumstances and to the individual child. I am convinced that it is very often a less potent cause of psychopathology than other, less permanent, forms of parental absence. Hill's theory, as quoted by Dr. Birtchnell, is interesting and worthy of study but completely speculative at present.

As regards the diagnosis of endogenous depression,

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Dr. Birtchnell excludes all but one of my criteria for distinguishing between severe and moderate depressive illness. Since cases of 'neurotic' depression were specifically excluded from my series (Munro, 1966) it is irrelevant and misleading to say that the criteria I used distinguish between 'endogenous-manic-depressive' and 'neurotic' forms of depression. His findings on the frequency of parental death in severe and moderate depressives and in the general population appear remarkably similar to mine (Birtchnell, 1970; Munro, 1965; Munro, 1966; Munro and Griffiths, 1968), but he makes no mention of this.

The remark that there is no evidence for a relationship between deprivation by parental death and personality disorder and delinquency is simply not true (*vide* Brown and Epps, 1966). My original depressive cases were carefully selected, and I shall continue to put forward the theory that I found a lower frequency of parental death in my series than others found in theirs because I excluded a number of contaminating conditions which may artificially raise this frequency. In a recent article (1969b) I provide the following figures (Tables I and II) relating to a group of 187 'endogenous' depressives in order to demonstrate how rates for parental death and absence may be affected by increasingly 'refining' the diagnosis of depression. Until we are more sure of what we mean when we talk about depressive illness (or any other psychiatric illness) it is a little facile to pretend that we can define stable and meaningful rates of bereavement or deprivation in relation to a particular diagnosis.

I am afraid that I cannot be sure what Dr. Birtchnell means in his remark about samples of mixed diagnoses. Most psychiatrists agree that there is a clinical entity which is usually known as endogenous depression, and it may be of some value to know that parental bereavement is (or, in my opinion, is not) of significance in predisposing to this condition. 'Mental illness' is a relatively meaningless term, and I think we shall learn precious little about aetiology if we try to stick to mixed samples.

TABLE I

*Death of a parent before the patient's 16th Birthday*

| | Total nos. | A. Death of mother: No. whose mother died | A. Death of mother: per cent | B. Death of father: No. whose father died | B. Death of father: per cent |
|---|---|---|---|---|---|
| Total series .. .. .. .. .. | 187 | 19 | 10·2 | 25 | 13·4 |
| *Less* 34 with personality disorder .. .. | 153 | 15 | 9·8 | 20 | 13·1 |
| *Less* 31 with attempted suicide .. .. | 122 | 13 | 10·7 | 14 | 11·5 |
| *Less* 30 with agitated depression .. .. | 92 | 12 | 13·0 | 12 | 13·0 |
| *Less* 12 with bipolar depression .. .. | 80 | 10 | 12·5 | 11 | 13·8 |
| *Less* 24 with moderate depression .. .. | 56 | 9 | 16·0 | 9 | 16·0 |

TABLE II

*Absence, for any reason, of a parent before the patient's 16th birthday*

| | Total Nos. | A. Absence of mother: No. whose mother was absent | A. Absence of mother: per cent | B. Absence of father: No. whose father was absent | B. Absence of father: per cent |
|---|---|---|---|---|---|
| Total series .. .. .. .. .. | 187 | 29 | 20·9 | 83 | 44·4 |
| *Less* 34 with personality disorder .. .. | 153 | 29 | 18·9 | 69 | 45·1 |
| *Less* 31 with attempted suicide .. .. | 122 | 26 | 20·9 | 51 | 41·8 |
| *Less* 30 with agitated depression .. .. | 92 | 23 | 25·0 | 36 | 39·1 |
| *Less* 12 with bipolar depression .. .. | 80 | 20 | 25·0 | 31 | 38·8 |
| *Less* 24 with moderate depression .. .. | 56 | 15 | 26·8 | 20 | 35·7 |

I take the last point concerning the age-distribution of the patient groups in the study by Dr. Griffiths and myself. Gregory (1958) pointed out that because of improved mortality rates younger individuals nowadays are much less liable to experience the death of a parent. In our series there may be a slight tendency to underestimate the significance of parent-loss among the schizophrenics, who are probably younger on average than the control individuals. This would not apply to the affective disorders, in which the age distribution would be relatively similar to that of the controls.

This field is bedevilled by conflicting results, failure to make adequate definitions, and a tendency to rush into hasty conclusions, of which we are all guilty. Many of our difficulties are semantic, and I regret that, in my opinion, Dr. Birtchnell's letter has increased rather than decreased such difficulties.

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## UNILATERAL AND BILATERAL ECT

DEAR SIR,

My apologies to Drs. Sutherland *et al.*, for my inexcusable error in reading their paper (*Journal*, September 1969, pages 1059 to 1064). Unfortunately their letter (*Journal*, January 1970, p. 126) does not answer the points which I raised. Perhaps I could elaborate upon these.

1. One cannot be satisfied that they were in a position to make any statements about the relief of depression, since this was not assessed in their trial. The number of ECTs given is surely not a reliable indication of response to treatment, particularly as several different psychiatrists were involved in deciding what this would be for any particular patient. We all differ in our ways of deciding when a patient has had enough ECT and what constitutes 'an adequate course of treatment'. A therapeutic trial should attempt to minimize this personal and idiosyncratic judgement.

2. They do not tell us how double-blind assessments of such variables as 'time taken to breathe spontaneously' were made. I take this to mean that the observer was not in the room at the time when the shock was given, and that he was informed of the exact time when this had occurred. Since the time intervals involved were relatively short, fairly elaborate arrangements would be needed to avoid any bias on the part of the person administering treatment. One can think of various ways in which this could be done, but the paper does not describe the method adopted. It is also extremely difficult to make a very definite decision about the beginning of spontaneous respiration, since many patients start off with small and almost imperceptible inspirations.

3. I wonder what led the writers to conclude that the EEG assessor was able to guess correctly the method of treatment any more frequently than would be accounted for by chance? Table III shows that the allocation was correct in only 10 of 19 bilateral cases and 11 out of 18 unilateral non-dominant cases. Admittedly the assessor did rather better on the dominant cases (14 out of 22), but I find it difficult to see how these figures could yield a value of $p = \cdot 00003$. Could the writer enlighten us on the statistical procedure employed?

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## AMPHETAMINE TAKING AMONG YOUNG OFFENDERS

DEAR SIR,

We were interested to read Drs. Cockett and Marks' article (*Journal*, October 1969, pp. 1203–4). Our interest in this subject was also aroused by Scott and Wilcox' study (1965), and for the past twelve months we have been screening the urine of all boys aged 14–16 admitted to Rose Hill Remand Home, Manchester. Rose Hill receives boys mainly from the Cities and County Boroughs in Lancashire, including Manchester, Salford, Bury, Bolton, Blackburn, Oldham, Preston and Warrington. Many of these places have the sort of clubs which are associated with drug-taking.

*Method.* Urine was collected from each boy as soon as possible after admission to the remand home. Younger boys in whom drug taking was suspected were also tested. Samples were screened by the method of Mellon and Stiven (1967). Those showing spots in the area Rf 0·70–0·95 were further investigated, in duplicate, by the method of Beckett *et al.* (1967), one extract being run in butanol/acetic acid/water (5 : 4 : 1), the other in isopropanol/5 per cent ammonia (10 : 1). Spots were developed with 0·5 per cent methanolic bromo-cresol green. Coincidence of spots on each system with those of control urines con-

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