Furthermore, however one interprets the lack of effect of tryptamine reported by Coppen *et al.* (4), evidence that peripheral or exogenous tryptamine alters mood in man is tenuous. Thus, the therapeutic action of methysergide upon mania taken by Dr. Dewhurst (5) to substantiate his tryptamine hypothesis was not apparent in a double-blind trial (6), and could have been equally interpretable in terms of 5-hydroxytryptamine antagonism.

The 'hypomanic patient (secreting) large amounts of tryptophan' is patient number 1 of reference (7). Interpretation is difficult because he was also secreting large amounts of 5-hydroxytryptophan and because it was thought impossible to distinguish between biochemical and psychiatric factors in his mood swings between depression and elation (Ashcroft, personal communication).

The suggestion that mood is simply a function of net brain level or turnover of any single amine would be naïve, and was not made in my paper. Dr. Dewhurst states correctly that low CSF 5-hydroxyindole-acetic acid (5HIAA) has been found in both depression and mania. However, in a recent confirmation of this (8) it was also found that there was a negative correlation between 5HIAA and degree of depression on the MMPI scale, but a positive correlation between 5HIAA and degree of mania (approaching 5 per cent significance).

2. One cannot reasonably argue with Dr. Dewhurst's use of 'primary' to indicate that mood changes are principal symptoms in depression. I used 'primary' and 'secondary' to indicate a temporal order of biochemical changes, and did not, I hope, imply that secondary changes were necessarily of unique or dominating importance.

3. In thyrotoxicosis, MAO activity is low and tryptamine excretion high (9). While phasic excretion of amines may well occur, this was not investigated in the paper to which Dr. Dewhurst refers, albeit indirectly (9). Apart from this, his third point usefully comments upon various factors which might influence amine metabolism.

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PSYCHIATRIC SEQUELAE OF CHILDHOOD BEREAVEMENT

Dear Sir,

Might I be permitted to respond to some of the points raised by Professor Munro in reply to my recently published letter.

It *is* important to differentiate between parent death and parent loss from other causes, and quite unhelpful to deliberately confound them as Munro claims he and Griffiths did (1969). We are at present in no position to say whether the two forms of loss are identical in their effects, and, Munro himself contends, though without justification, that the one is a more potent cause of psychopathology. Hill's (1969) remark that loss due to cause other than death may denote a higher index of psychiatric disturbance in this group of parents is not a 'theory' but a serious methodological consideration. Parent death is a valuable experimental opportunity, for it is as close as one can get in the human situation to lowering a hand into the cage and removing the parent animal. Loss from other causes may be preceded and succeeded by discord between the parents and within their families, which may be of greater aetiological significance than the separation experience itself.

This confusion between the two forms of loss is related to the next point. To explain a high incidence of parental bereavement in depressed patients on the grounds of contamination by cases of personality disorder, psychopathy, delinquency and attempted suicide is unreasonable. As it happens, an interesting point in itself, the majority of studies of depression

have been concerned only with parent death, whereas studies of these contaminating conditions have tended to be concerned with a variety of forms of loss, and show (i) that the incidence of parent death is certainly no higher than it is in depressed patients, and (ii) significant relationships are more likely to be found with parent loss due to causes other than death. This is only to be expected, for patients with character disorders are likely to have parents with such disorders who are more likely to separate or get divorced.

It is no longer clear what the constitution of Professor Munro's 1966 depressed series was. Though he claims to have excluded all cases of neurotic depression, he states in his paper, 'Originally an attempt was made to distinguish between endogenous and neurotic depression, but since all cases were severe enough to warrant hospital admission this really only differentiated severe from moderately severe depression . . . terms such as 'endogenous', 'psychotic', 'reactive' and 'neurotic' depression are too vague to be satisfactory.' How then did he identify cases of neurotic depression to exclude them, and how is this compatible with his current assertion that 'most psychiatrists agree that there is a clinical entity which is usually known as endogenous depression'? It would be important to know what proportion of these hospitalized depressives was eliminated.

In my own series all hospitalized depressives were included but were divided according to the severity of depressive symptoms. Though the term is difficult to define, I would imagine that neurotic depressives accounted for quite a high proportion of the moderately depressed group. As patients needed to be practically psychotic to be included in the smaller, severely depressed group, it is probable that all of these would be categorized as cases of endogenous depression. The incidence of early bereavement was significantly higher in the severely depressed compared with the moderately depressed group. A similar finding was reported by Beck, Sethi and Tuthill (1963). There is a complete refutation of the claims by Munro and Griffiths (1969) that 'the nearer the diagnosis is to "endogenous" or "manic depressive" depression the less important is parental deprivation in the actual aetiology'.

My point regarding groups of mixed diagnosis is this: one can make no special claim for an association between endogenous depression and early bereavement unless one has compared such cases with other depressives, appropriately matched (approximately was a printer's error), depressives with non depressives and psychiatric patients with the general population. Mental illness is not a meaningless term, and there are good reasons why psychiatric patients as a whole may have experienced more bereavement than other people. Hill and Price (1967) point out that 'apart from any effect in causing illness, it would be natural for an orphan to seek help from a psychiatrist where another patient, equally distressed, might turn to a parent'. Or, as the work of Barry, Barry and Lindemann (1965) suggests, orphaned individuals may grow up seeking help from all and sundry and tend to develop clinging, dependent personalities.

The objections I have raised are not so much semantic as methodological. Psychiatric research is still largely a jungle. Progress is slow, but will only be made by mapping one's course meticulously and proceeding by careful steps.

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DEAR SIR,

All these statistical articles about parental deprivation and mental illness are doubtless very interesting (and possibly even meaningful) to statisticians. To those of us who are more used to dealing with people than figures they give the impression of emanating from the same synthetic country (in this case, Cloud-cuckoo-land might be the most appropriate name for it) as do those curious census reports that indicate that the average family consists of 2·2 adults and 2·6 children, and has 1·7 pets. All these surveys seem to be based on the convenient if fallacious belief that all families are nice neat little nuclear units each bombinating in an emotional vacuum. This may be the pattern for the future, but it is not true yet, thank God—as I discovered by bitter practical experience in my early days as a psychiatric S.H.O., when, having spent a large part of the interview taking a nice orthodox family