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Correspondence

Letters for publication in the Correspondence columns should be addressed to:
The Editor, British Journal of Psychiatry, Chandos House, 2 Queen Anne Street, London, W1M 9LE

ANTICIPATORY GRIEF AND WIDOWHOOD

DEAR SIR,

In the paper by Clayton *et al.* entitled 'Anticipatory grief in widowhood' and in the companion paper by Bornstein *et al.* (*Journal*, January 1973, **122**, 47–51; May 1973, **122**, 561–6), the authors conclude that in their sample of unselected widows of mean age 61 years 'duration of illness [of deceased spouse] was unrelated to the prevalence of symptoms [in the survivor]'.

A recent study by Parkes, Glick, Weiss and Napier (in preparation) provides comparable information about a younger sample of American widows and widowers which conflicts with these findings and suggests that, whatever the validity of the concept of 'anticipatory grief', there are some bereaved persons who, given the opportunity, are able to prepare themselves for bereavement.

This study was confined to the under 45 age group because previous work had suggested that age at bereavement was so important a determinant of outcome that failure to control for this factor would make it impossible to identify other determinants. Twenty-four Bostonian widows and widowers who had had less than two weeks warning of probable death *and/or* less than three days warning that death was imminent were compared with 46 who had had a longer preparation for bereavement. Data were obtained at interviews conducted three weeks and 13 months after bereavement, and have now been re-analysed, using criteria which were as close as possible to the criteria adopted by Clayton *et al.* to identify the 'Depressive Symptom-Complex'.

The Table shows that 52 per cent of respondents had a 'Depressive Symptom-Complex' 13 months after bereavement, a figure considerably in excess of the 20 per cent reported by Clayton *et al.* and conforming with Maddison and Walker's discovery of a high incidence of disturbance in this age group of Boston widows and widowers (1967). It also showed that 'Depressive Symptom-Complex' was very much more common in the 'short preparation' group (χ^2 with Yates' correction 5·0; 1 d.f.; $p < ·05$). This group also showed significantly more anxiety and self-reproach, and their 'overall outcome' ratings were less good than those with a longer preparation for bereavement. Differences were just as pronounced at follow-up 2–4 years after bereavement.

TABLE
Proportions in short and long preparation groups showing depressive symptom complex

	Depressive Symptom Complex	No Depressive Symptom Complex
Short preparation group ..	17 (74%)	6 (26%)
Long preparation group ..	19 (42%)	26 (58%)
Both groups	35 (52%)	32 (48%)

These findings seem to suggest that sudden or unexpected losses are more traumatic in the younger age group we studied than in the predominantly older sample studied by Clayton and Bornstein. It might be postulated that in the 60-year-old no conjugal bereavement is entirely unexpected, and that the process of 'disengagement' has already started, whereas younger persons may benefit from an adequate warning of bereavement. The implications of this conclusion for members of the medical and nursing professions, who are often in a position to give such warnings, are obvious.

I am indebted to Dr. Paula Clayton for this opportunity to comment on the second paper concurrently with its publication.

C. M. PARKES.
School of Family Psychiatry and Community Mental Health,
Tavistock Centre,
Belsize Lane,
London, NW3 5BA.

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DEPRESSIVE ILLNESSES IN LATE LIFE

DEAR SIR,

On different occasions both of us have calculated the distribution of scores on the Newcastle diagnotic

scale obtained by series of patients with depressive illnesses, and have failed to find the unambiguously bimodal distribution that Carney and his colleagues obtained in their original study (Carney *et al.*, 1965; Kendell, 1968; Post, 1972). In fact, we both obtained score distributions that did not depart significantly from normality; and, partly for this reason, we have remained sceptical of the Newcastle group's claim to have demonstrated the existence of distinct endogenous and neurotic forms of depression. Dr. Garside has now combined the scores of our two series and demonstrated that the overall distribution for all 222 patients shows a point of rarity at +5 and is significantly non-normal (Garside, 1973). On this basis he claims that the 'unimodal hypothesis' is conclusively disproved, and by implication that his bimodal hypothesis is supported.

As Kendell's 130 patients were derived from two separate series, one of 53 patients rated by himself and another of 77 rated by Hemsi and McClure, and as we have data from a further series of 49 geriatric patients rated by Post, Cawley and Whitehead, there are now four separate series of scores available for scrutiny, two derived from patients below the age of 60 and two from patients above that age. The distribution of scores on the Newcastle scale for all four series is shown in the attached Table. None of the four on its own departs significantly from normality, nor do the 130 patients under 60 or the 141 above that age when considered separately. Only if all four are combined is the distribution of scores significantly non-normal ($\chi^2 = 36{\cdot}04$; d.f. = 14; $p < {\cdot}01$). However, if this distribution is plotted as a histogram it will be seen to be trimodal rather than bimodal, with a cleft at +1 and +2 at least as impressive as that at +5. (The same is true of the score distribution of the 222 patients Garside was concerned with, although he ignored the fact.)

TABLE

Scores of Maudsley cases on the Newcastle Scale

Score on Newcastle Scale	Kendell	Hemsi and McClure	Post	Post, Cawley and Whitehead	Total
+13	0	0	1	0	1
+12	0	1	0	0	1
+11	1	0	0	0	1
+10	2	1	3	1	7
+ 9	0	1	2	3	6
+ 8	1	9	6	4	20
+ 7	7	7	9	3	26
+ 6	7	7	13	4	31
+ 5	2	8	6	6	22
+ 4	6	12	8	5	31
+ 3	5	9	10	7	31
+ 2	6	2	9	5	22
+ 1	7	4	6	3	20
0	4	6	11	7	28
− 1	4	5	6	1	16
− 2	1	4	1	0	6
− 3	0	1	1	0	2
Total ..	53	77	92	49	271

A bimodal distribution of scores on a discriminant function is evidence for the presence of two distinct populations, and a unimodal distribution is negative evidence that only one population is present. A trimodal distribution such as we have here has no statistical meaning at all. In particular, it is not evidence for the existence of a third population in the material. An analysis starting with only two criterion groups is not designed to detect that possibility; and if a third population were present there is no reason why its members should all obtain similar scores on the function. Why then has this trimodal distribution emerged? It is not due to a single point of rarity appearing in slightly different positions in the four series, since both clefts appear in three of them. By far the most likely explanation is that this non-normal distribution is a statistical artifact which has arisen in one of two ways: either because the data from which the weightings of the scale were originally derived did not satisfy the requirement, basic to discriminant function analysis, that the two criterion populations should have multivariate normal distributions and equal variance/co-variance matrices; or because the item distributions in our data were markedly skewed. Both these possibilities are inherently probable, and the likelihood that one or other is responsible for the uneven score distributions we obtained is strengthened by the fact that both discontinuities in the distribution involve only one or two scores, and disappear if the number of units in the distribution is reduced from 17 to 9 by combining adjacent scores. This is quite unlike Carney's original distribution, where the dip in the centre of the distribution extended all the way from +5 to +8.

We believe, therefore, that our data do not support the claim that there are two distinct forms of depressive illness, and that a dimensional model is still the most appropriate means of representing the variations between different patients.

In addition, we wish to repudiate Dr. Garside's suggestion that we have naïvely been trying to 'prove' a unimodal hypothesis. As he is really well aware, we have emphasized that this is impossible (Kendell and Gourlay, 1970). We also both started our researches in this area in the hope that we would be able to demonstrate the existence of two or more distinct

forms of depressive illness, but we have consistently failed to do so.

R. E. KENDELL.
FELIX POST.

The Maudsley Hospital,
Denmark Hill,
Camberwell,
London SE5 8AZ.

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CREATINE PHOSPHOKINASE ACTIVITY IN PSYCHIATRIC PATIENTS

DEAR SIR,

Gosling *et al.*, in your *Journal* (1), found that half of their newly-admitted psychotic patients had raised creatine phosphokinase (CPK) activity. This was specifically associated with the psychosis: none of their neurotic control patients showed an elevated CPK.

We have investigated newly-admitted female patients to the psychiatric ward of the Johannesburg General Hospital during a period of 30 days. This ward admits unselected cases across the whole psychiatric spectrum. Patients were only excluded from this investigation if they had received intramuscular chlorpromazine, as this can raise CPK values (2). The CPK assay was carried out by means of the *accu-zyme enzyme reagent* kit. Normal range for females is 0 to 30 I.U. Clinical assessment of the patient's condition was carried out independently by two psychiatrists, who were asked to allocate the patient to a psychotic or non-psychotic group. Where there was disagreement on the allocation (3 cases), the cases were omitted (the CPK levels for these three cases were within normal limits).

Details are set out in the following table:

TABLE

	Age	Clinical diagnosis	CPK I.U.
		Psychotic group	
1.	56	Manic-depressive psychosis (depressed phase)	29·9
2.	51	Manic-depressive psychosis (manic phase)	28·7
3.	49	Schizo-affective disorder	21·1
4.	36	Schizophrenia	30·4
5.	57	Presenile dementia	23·4
6.	16	Schizophrenia	26·7
7.	23	Schizo-affective disorder	28·4
8.	32	Schizophrenia	65·0
		Non-psychotic group	
1.	60	Anxiety state and hypochondriasis	29·3
2.	65	Personality disorder and alcoholism	24·6
3.	22	Neurotic depression	56·5
4.	31	Anxiety state	58·4
5.	19	Personality disorder and epilepsy	31·8
6.	46	Personality disorder	53·6
7.	70	Toxic confusional state	31·0
8.	21	Neurotic depression	28·4
9.	28	Neurotic depression	26·6
10.	25	Dissociative hysterical illness	30·0

The mean CPK level of the psychotic group was 31·7 (S.D. 13·8) and of the non-psychotic group 37·0 (S.D. 13·4). This difference is not statistically significant.

We have therefore failed to confirm that raised CPK activity indicates the presence of psychotic illness. In those cases showing abnormal levels the elevations were small. We suggest that patients manifesting elevated CPK levels may share a common factor, but that this is not the presence *per se* of psychosis. This factor may be a subclinical myopathy (3), a possibility further supported by the finding (4) that the raised serum CPK is of the muscle isoenzyme type. Furthermore, it is interesting that Meltzer (4) noted a familial tendency, in that some parents of psychotic patients had continuous CPK elevations while the patients themselves only demonstrated these intermittently.

J. P. LOEBEL.
A. H. ROBINS.

Department of Psychiatry,
Johannesburg General Hospital and the
University of the Witwatersrand,
Johannesburg, South Africa.

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